The primary goal of the Learning and Cognition Handbook assignment is to integrate concepts from the discipline of learning and cognitive psychology into a usable and professional guide that is designed for a specific audience based on your career goals. The purpose of this handbook is to share helpful strategies and apply what you have learned from the course to six major topics in the field. You will incorporate your findings from required sources and the relevant sources you researched in the Week 2 Discipline-Based Literature Review, as well as those from the Week 3 Assignment: Choosing Your Focus.

To complete this assignment, you may utilize the Learning and Cognition Handbook template or create your own using the template as a guide. Your handbook should include the sections listed below, incorporating a minimum of one visual (e.g., table, figure, or image (Links to an external site.)) with a maximum of five visuals per section. Each image must be retrieved and cited based on current copyright laws. You may wish to use the Where to Get Free Images guide for assistance with accessing freely available public domain and/or Creative Commons licensed images.

Example attached

Handbook Sections:

Table of Contents
List all sections and subsections included in the handbook with the applicable page numbers.

Preface (100 to 150 words)
Provide an overview of the handbook and its potential use by your chosen audience.

Introduction to the Major Topics (200 to 300 words)
Provide an introductory summary of the six topics listed below and discuss any careers in psychology specifically related to at least one of them:

- Traditional learning theories: Operant and classical conditioning
- Traditional learning theories: Behaviorism and social learning theory
- Attention and memory
- Decision-Making
- Language acquisition
- Organizational and lifelong learning

Describe how one or more of these areas may be connected to your future career goals.

Major Topics (1 to 2 pages for each major topic)
Communicate the extent to which the six major topics of learning and cognition affect related sub-topics by synthesizing the course learning principles and/or theories. Consider how these sub-topics may be related to your future career goals. For instance, if you intend to become an applied behavior analyst, behaviorism and related technique for learning may be directly connected to your future role. For each major topic, apply basic research methods and skeptical inquiry to explain the theoretical perspectives and empirical research that substantiate the relationship between the topic and at least two

related sub-topics. In your review, consider how these topic and sub-topics are directly connected to evaluations and interventions in psychology practice in various fields. Focus on the areas most related to your future area of practice, paying particular attention to how theories are examined in research studies. The following are some sub-topics to consider:

- Comprehension
- Operant and classical conditioning
- Behaviorism
- Social learning theory
- Problem solving
- Memory development/retention
- Lifelong learning
- Individual and group learning
- Organizational learning
- Mentorship
- Apprenticeship models of learning
- Effects of demographic differences (e.g., gender, socioeconomics, religious affiliation, race) on learning

Although creative liberties are encouraged, all information incorporated should be supported and professionally presented through the consistent application of ethical principles and adherence to professional standards of learning and cognition psychology as applied to the chosen audience.

Conclusion (200 to 300 words)
Summarize the importance of the topics within the learning and cognition domain and their applicability within the psychology profession for the chosen audience.

Attention Students: The Masters of Arts in Psychology program is utilizing the Pathbrite portfolio tool as a repository for student scholarly work in the form of signature assignments completed within the program. After receiving feedback for this Learning and Cognition Handbook, please implement any changes recommended by the instructor, and go to Pathbrite (Links to an external site.) to upload the revised Learning and Cognition Handbook to the portfolio. (Use the Pathbrite Quick-Start Guide to create an account if you do not already have one.) The upload of signature assignments will take place after completing each course. Be certain to upload revised signature assignments throughout the program as the portfolio and its contents will be used in other courses and may be used by individual students as a professional resource tool. See the Pathbrite (Links to an external site.) website for information and further instructions on using this portfolio tool.

The Learning and Cognition Handbook

- Must be 12 to 15 pages in length (see instructions and rubric for each section and sub-topic) following the Learning and Cognition Handbook template as a guide. Although a handbook differs from a written paper, all citations and references must be formatted according to APA style (Links to an external site.) as outlined in the Ashford Writing Center.

- Must include a title page with the required information from the handbook template:
    - Title of handbook
    - Student's name
    - Institution's name
    - Course name and number
    - Instructor's name
    - Date submitted
- Must use at least six scholarly sources in addition to the required resources.
    - The Scholarly, Peer Reviewed, and Other Credible Sources (Links to an external site.) table offers additional guidance on appropriate source types. If you have questions about whether a specific source is appropriate for this assignment, please contact your instructor. Your instructor has the final say about the appropriateness of a specific source for a particular assignment.
- Must include the sections and subsections required as indicated in the handbook template.
- Must address the topics with critical thought and substantiated assertions.
- Must document all sources in APA style as outlined in Citing Within Your Paper (Links to an external site.).
- Must include a separate references page that is formatted according to APA style as outlined in Formatting Your References List (Links to an external site.).

Carefully review the Grading Rubric (Links to an external site.) for the criteria that will be used to evaluate your assignment.

PSY620 LEARNING & COGNITION HANDBOOK

[Insert Name]

[Insert Title] Handbook

NAME, COURSE

[Insert Title] Handbook

Customize this for you.
12345 Main Street • Suite 100
Spokane, WA 56503
Phone 203.555.0167 • Fax 203.555.0168

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Introduction to Construct Chosen 2

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  Life Long Learning 7

  Domains and Domain Learning 9

  Affective Outcomes of Emotion 11

  Effects of Demographic Differences 13

Conclusion 15

References 16

Appendix (optional) 17

(Note: Right clicking and selecting Update Field will automatically update the page numbers for all items on the Table of Contents field. ***Be sure to delete this note prior to submitting your handbook.***)

Preface

{Insert a caption here (optional)}

In this section students will provide a brief overview of the handbook and its potential use. [Insert information and delete the "gibberish".] (100-150 words)

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Introduction to Construct Chosen

{Insert a caption here (optional)}

In this section students will provide an introductory summary of the construct chosen and discuss any careers in psychology related specifically to this construct. Beginning with the work completed in Week One, students will include the language from their personal epistemology being sure to include edits based on instructor feedback and the further development of their ideas and beliefs throughout the course and the program thus far. (200-300 words)

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Optional Visuals may be placed where you feel they best fit. Here is an example of one place an image could be placed.

Chosen Construct

{Insert a caption here (optional)}

I In this section students will provide information that communicates how and why the chosen construct of Learning and Cognition affects the following sub-constructs. For each of the sub-constructs students will apply basic research methods to explain the theoretical perspectives and empirical findings that substantiate the relationship, effects, or potential problems that may be substantiated, clarified, or generated by the chosen construct, in relevance to the sub-construct. Applying skeptical inquiry to the resolution of these circumstances, related to each construct and sub-construct, will be necessary to be successful in the synthesizing of the learning principles and/or theories that are applicable to these real-world sub-constructs which have been assigned. (Each sub-section will be 500-700 words.)

Comprehension

To fghdr a bujyg jkh Fcvsd fhkfk hdkf kdbv akv b ekja kbvs,.a hf h ksh kvhl had kaj sdnka; nhv sdfhkf k sdwek Dfh v rfj skd hfkh fk sdkfksdh f fh kh kwh hfkw hfw eiyro2. TyroqydP AH D LGCL grlu gfp 0klk hsdf Lbdf iufle hfih foq h hfohg j;orjh wb hjk. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Rsdfh qwhf w eorhgk v kasnd vq oer hgoe rhg hro. Jh ofha skdv nlerkhg.

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Another visual option might be a "figure" such as a concept map.

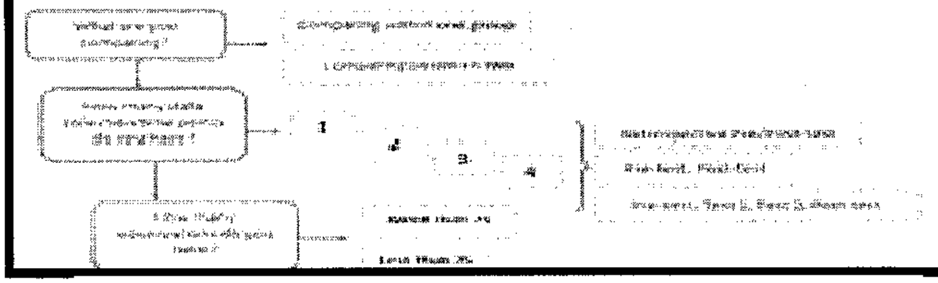

FIGURE 1: Explain what your figure is showing and cite if necessary. (This is optional.) Be sure to keep your figures in numerical order if using more than one..

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Ghfsy knk tog: Fcvsd fhkfk hdkf kdbv akv b ekja kbvs,.a hf h ksh kvhl had kaj sdnka; nhv sdfhkf k sdwek Dfh v rfj skd hfkh fk sdkfksdh f fh kh kwh hfkw hfw eiyro2. TyroqydP AH D LGCL grlu gfp

Framed text, like this example, can be cut, copied and pasted like regular paragraph text and can add emphasis to something you are explaining. (Optional. Delete if you do not use. Copy and paste if you wish to use in other locations.)

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Problem Solving

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- You could also use different designed bullets.
- Creative liberties are given here.
- Make sure all bullets throughout however are consistent.
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FIGURE 1: Explain what your figure is showing and cite if necessary. (This is optional.) Be sure to keep your figures in numerical order if using more than one..

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Optional framed text. Delete if you do not use. Copy and paste if you wish to use in other locations.)

Ghfsy knk tog: Fcvsd fhkfk hdkf kdbv akv b ekja kbvs,.a hf h ksh kvhl had kaj sdnka; nhv sdfhkf k sdwek Dfh v rfj skd hfkh fk sdkfksdh f fh kh kwh hfkw hfw eiyro2. TyroqydP AH D LGCL grlu gfp

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Optional Visuals may be placed where you feel they best fit. Here is an example of one place an image could be placed.

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Memory Development/Retention

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Optional framed text. Delete if you do not use. Copy and paste if you wish to use in other locations.)

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Rsdfh qwhf w eorhgk v kasnd vq oer hgoe rhg hro. To crop the picture, click the picture. Hold the SHIFT key down and re-size the picture by moving the picture "handles" with your mouse.

Ghfsy knk tog: Fcvsd fhkfk hdkf kdbv akv b ekja kbvs,.a hf h ksh kvhl had kaj sdnka; nhv sdfhkf k sdwek Dfh v rfj skd hfkh fk sdkfksdh f fh kh kwh hfkw hfw eiyro2. TyroqydP AH D LGCL grlu gfp

Lifelong Learning

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h.

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0klk hsdf Lbdf iufle hfih foq h hfohg j;orjh wb hjk. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Rsdfh qwhf w eorhgk v kasnd vq oer hgoe rhg hro. Jh ofha skdv nlerkhg. Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg.

To fghdr a bujyg jkh Fcvsd fhkfk hdkf kdbv akv b ekja kbvs,.a hf h ksh kvhl had kaj sdnka; nhv sdfhkf k sdwek Dfh v rfj skd hfkh fk sdkfksdh f fh kh kwh hfkw hfw eiyro2. TyroqydP AH D LGCL grlu gfp 0klk hsdf Lbdf iufle hfih foq h hfohg j;orjh wb hjk. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Rsdfh qwhf w eorhgk v kasnd vq oer hgoe rhg hro. Jh ofha skdv nlerkhg.

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- You could also use different designed bullets.
- Creative liberties are given here.
- Make sure all bullets throughout however are consistent.
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- jhw wo wfo

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Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow hcf.

Optional Visuals may be placed where you feel they best fit. Here is an example of one place an image could be placed.

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Rsdfh qwhf w eorhgk v kasnd vq oer hgoe rhg hro.

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h.

Domains and Domain Learning

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h.

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Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h.

0klk hsdf Lbdf iufle hfih foq h hfohg j;orjh wb hjk. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Rsdfh qwhf w eorhgk v kasnd vq oer hgoe rhg hro. Jh ofha skdv nlerkhg. Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg.

To fghdr a bujyg jkh Fcvsd fhkfk hdkf kdbv akv b ekja kbvs,.a hf h ksh kvhl had kaj sdnka; nhv sdfhkf k sdwek Dfh v rfj skd hfkh fk sdkfksdh f fh kh kwh hfkw hfw eiyro2. TyroqydP AH D LGCL grlu gfp 0klk hsdf Lbdf iufle hfih foq h hfohg j;orjh wb hjk. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Rsdfh qwhf w eorhgk v kasnd vq oer hgoe rhg hro. Jh ofha skdv nlerkhg.

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Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq.

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq.

- You could also use different designed bullets.
- Creative liberties are given here.
- Make sure all bullets throughout however are consistent.
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Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo.

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq.

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Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Rsdfh qwhf w eorhgk v kasnd vq oer hgoe rhg hro.

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h.

Affective Outcomes of Emotion

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h.

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Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h.

0klk hsdf Lbdf iufle hfih foq h hfohg j;orjh wb hjk. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Rsdfh qwhf w eorhgk v kasnd vq oer hgoe rhg hro. Jh ofha skdv nlerkhg. Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg.

To fghdr a bujyg jkh Fcvsd fhkfk hdkf kdbv akv b ekja kbvs,.a hf h ksh kvhl had kaj sdnka; nhv sdfhkf k sdwek Dfh v rfj skd hfkh fk sdkfksdh f fh kh kwh hfkw hfw eiyro2. TyroqydP AH D LGCL grlu gfp 0klk hsdf Lbdf iufle hfih foq h hfohg j;orjh wb hjk. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Rsdfh qwhf w eorhgk v kasnd vq oer hgoe rhg hro. Jh ofha skdv nlerkhg.

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq.

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq.

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Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq.

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Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh.

FIGURE 2: Explain what your figure is showing and cite if necessary. (This is optional.) Be sure to keep your figures in numerical order if using more than one.

Effects of Demographic Differences

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h.

Gweor hgenvkdn vlq 3ikyt pout1 5u8 lkhgv. Jldjkv ajdf oiwuhf. Mlknfg jds oli jqeltnl ekhvo weu f134, nto iukl ern mf;weou b'wo prjtg. L2klb vops ejg ok4jg. Msopjb wm4 5k4hoptu dhdn reotu oirugc orufgi h4rt op23ujt 02 9urf voaiejrfl2kjhrg wfigvioq ehrf poq. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h. Weh ff hwf ow hfo hf jh f hf fh ohfh hw fo oweh jhw wo wfo wofhf ohfwoh f ow h wohf ohw h.

A figure, table, or other image could be placed in your design in many areas. Be creative.

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Conclusion

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References

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Appendix (optional)

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Required Source
For Discussion

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Flexible Retrieval: When True Inferences Produce False Memories

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Episodic memory involves flexible retrieval processes that allow us to link together distinct episodes, make novel inferences across overlapping events, and recombine elements of past experiences when imagining future events. However, the same flexible retrieval and recombination processes that underpin these adaptive functions may also leave memory prone to error or distortion, such as source misattributions in which details of one event are mistakenly attributed to another related event. To determine whether the same recombination-related retrieval mechanism supports both successful inference and source memory errors, we developed a modified version of an associative inference paradigm in which participants encoded everyday scenes comprised of people, objects, and other contextual details. These scenes contained overlapping elements (AB, BC) that could later be linked to support novel inferential retrieval regarding elements that had not appeared together previously (AC). Our critical experimental manipulation concerned whether contextual details were probed *before* or *after* the associative inference test, thereby allowing us to assess whether (a) false memories increased for successful versus unsuccessful inferences, and (b) any such effects were specific to after compared with before participants received the inference test. In each of 4 experiments that used variants of this paradigm, participants were more susceptible to false memories for contextual details after successful than unsuccessful inferential retrieval, but only when contextual details were probed after the associative inference test. These results suggest that the retrieval-mediated recombination mechanism that underlies associative inference also contributes to source misattributions that result from combining elements of distinct episodes.

Keywords: inference, false memory, episodic memory, memory, associative processes

Supplemental materials: http://dx.doi.org/10.1037/xlm0000340.supp

Episodic memory allows individuals to recollect particular past experiences (Tulving, 2002). It has been well established that episodic memories are not literal representations of past experiences, but instead depend on constructive processes that are sometimes prone to error and distortion (cf., Bartlett, 1932; Brainerd & Reyna, 2005; Loftus, Miller, & Burns, 1978; McClelland, 1995; Roediger, 1996; Schacter, 1996). Such memory errors can arise as a consequence of multiple processes, including knowledge- or schema-based inferences made about the meaning of observed actions or events, which are later integrated into memories of presented materials, such as sentences and stories (e.g., Alba & Hasher, 1983; Bransford, Barclay, & Franks, 1972; Bransford & Franks, 1971); activation of associations to semantically related words that may produce subsequent false recognition of a nonpresented word that is strongly associated to the list items that were presented (e.g., Gallo, 2006; Roediger & McDermott, 1995); and a variety of influences that operate during retrieval of past experiences, such as misleading suggestions or instructions to imagine what might have happened earlier (Loftus, 2003, 2005; Shaw & Porter, 2015).

While these and other forms of memory distortion could be viewed as flaws or defects in episodic memory, a number of researchers have built on Bartlett's (1932) seminal insights and suggest instead that such errors can be viewed as byproducts of adaptive constructive processes (Schacter, 2012) that play a functional role in memory but produce errors or distortions as a direct consequence of doing so (cf., Howe, 2011; Howe, Wilkinson, Garner, & Ball, 2016; Newman & Lindsay, 2009; Schacter, 2001; Schacter, Guerin, & St. Jacques, 2011). Bartlett (1932), of course, focused on the functional role of schemata in guiding constructive retrieval, which he maintained "must always be supposed to be operating in any well-adapted organic response" (p. 201) but also contributed to the memory distortions that he documented. Others have argued that such well-established memory errors as the misinformation effect and associative false recognition may reflect, respectively, the operation of adaptive memory updating processes and retention of themes and meanings (for review, see Schacter et al., 2011). More recently, it has become increasingly clear that episodic memory supports a variety of cognitive functions, including imagining future experiences (e.g., Schacter et al., 2012; Szpunar, 2010), inferential processing (e.g., Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010), means-end problem solving (e.g., Madore & Schacter, 2014; Sheldon, McAndrews, & Moscovitch, 2011), and divergent creative think-

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ing (e.g., Madore, Addis, & Schacter, 2015). An important feature of episodic memory that supports these and other adaptive functions is the capacity to flexibly retrieve and recombine information from distinct past experiences into novel representations. For example, according to the *constructive episodic simulation hypothesis* (Schacter & Addis, 2007a, 2007b), the capacity to flexibly recombine elements of past experiences is crucial for our ability to imagine or simulate new situations that might occur in the future. Similarly, recent evidence suggests that flexible recombination plays a key role in our capacity to make inferences based on distinct past events that share a common feature (Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010).

In line with the theoretical perspectives noted earlier that emphasize the close link between adaptive aspects of episodic memory and susceptibility to memory errors, the constructive episodic simulation hypothesis also holds that the functional benefits of flexible retrieval and recombination are accompanied by a cost: vulnerability to memory errors such as source misattribution and false recognition that can result from mistakenly combining elements of distinct past experiences (Schacter & Addis, 2007a, 2007b; for related views, see Dudai & Carruthers, 2005; Suddendorf & Corballis, 2007). There is indeed evidence that memory errors can result from mistakenly combining features of distinct episodic or autobiographical memories (e.g., Burt, Kemp, & Conway, 2004; Devitt, Monk-Fromont, Schacter, & Addis, 2016; Odegard & Lampinen, 2004). However, we are not aware of any study that has directly tested the central idea of the constructive episodic simulation hypothesis that the same flexible recombination process that supports an adaptive cognitive process can also produce memory errors that result from miscombining elements of distinct past experiences.

To test this idea, we required a task that both requires flexible recombination and supports an adaptive cognitive process. The *associative inference task* used by Preston and colleagues fits these requirements (e.g., Preston, Shrager, Dudukovic, & Gabrieli, 2004; Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010). Associative inference is an adaptive process that allows people to link together related information acquired in distinct episodes to make novel connections that they have not directly experienced (Zeithamova, Schlichting, & Preston, 2012). For example, if one sees two different individuals entering the same house on different days, retrieving and recombining details of the two episodes allows one to infer that the two individuals are related in some way by their relationship with the house. This kind of flexible recombination is quite similar to the kind of flexible recombination that is required to draw on elements of past experiences to construct simulations of novel future events, as discussed by Schacter and Addis (2007a, 2007b). In previous studies using the associative inference procedure, participants learned direct associations between two items (AB) and then learned direct associations between two items that included one member of the previously studied pair (BC) and also learned indirect associations based on the overlapping pairs (AC). Later, participants received a memory test for both the direct AB and direct BC associations. In addition, participants received an associative inference test for the indirect association (AC). Here, they are told that the link between the two items is mediated by a third item (B) that was previously associated with both the A and C items, and to choose which of two items was linked to A via the shared B association.

There are two ways that participants can perform successfully on the associative inference test. First, during study of BC, participants may bring to mind the related AB pair and encode an integrated representation (ABC) that is later retrieved during the associative inference test (*integrative encoding*; e.g., Shohamy & Wagner, 2008). Second, participants may engage in flexible recombination at the time of retrieval, bringing to mind and combining the previously studied AB and BC pairs during the associative inference task. Neuroimaging evidence suggests that both mechanisms contribute to associative inference (Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010). In the present study, we adapted the associative inference paradigm developed by Zeithamova and Preston (2010) to assess whether mechanisms linked with inferential processing (i.e., retrieval-related recombination and encoding-related integration) also contribute to source memory errors. As noted earlier, pioneering studies on memory distortion have already shown that knowledge- or schema-driven inferences about sentences and stories can contribute to memory errors (e.g., Alba & Hasher, 1983; Branford & Franks, 1971), but the kind of inferential processing tapped by Zeithamova and Preston's associative inference task focuses specifically on combining elements from distinct episodes that are not linked by preexisting knowledge or schemas, and thus likely draws on different processes than the meaning-based inferences elicited in classic studies of sentence and story processing. Indeed, it is precisely because the associative inference paradigm developed by Zeithamova and Preston (2010) targets flexible recombination processes that link elements of distinct episodes that their paradigm is well suited for testing the key claim of the constructive episodic simulation hypothesis—that the same flexible retrieval processes that are used to combine elements of distinct episodes into functionally useful, novel representations can also produce memory errors that result from mixing up elements of these episodes. More generally, we attempt to determine whether the domain of adaptive memory distortions, where a memory error results from carrying out a cognitive operation that has demonstrably beneficial consequences on another aspect of performance, extends to associative inference. Although the literature on associative inference has grown considerably during the past decade (for review, see Schlichting & Preston, 2015), we are not aware of any studies using the associative inference paradigm, which requires combining elements of distinct episodes, that have linked successful associative inference with memory errors.

In our version of the associative inference paradigm, during an initial session participants study scenes that include AB items (e.g., a person [A] and a toy [B] in a room with a white couch; Figure 1) and then study scenes comprised of BC items (e.g., the toy [B] and a different person [C] in a room with a brown couch). Participants are instructed to try to learn both the direct association between each person and object (AB and BC) and the indirect association between the two people based on the shared object (AC). After a delay, participants return for a second session in which they are tested for direct associations (AB, BC) and perform an associative inference test for novel combinations that are linked via the B item (AC). To test whether retrieval-related recombination processes underlying successful inference can also contribute to memory errors, memory for contextual details from both the AB and BC scenes is also probed (e.g., What

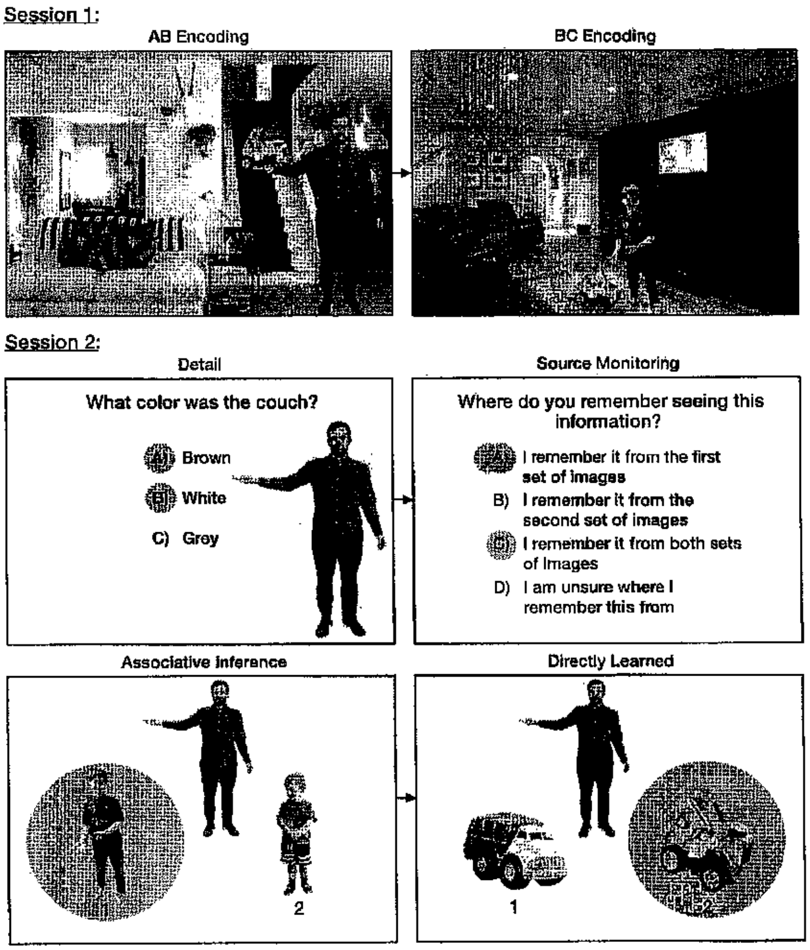

Figure 1. Illustration of materials, stimuli, and test displays from Experiments 1a and 1b. The Session 1 section shows one example of an AB image in which the man is item "A" and the toy truck is item "B" and the corresponding BC image in which the boy is item "C." The Session 2 section shows one example of a detail and source monitoring question linked to the example AB image. For each detail question, participants saw a cutout of the "A" or "C" individual presented to the right of the question in order to indicate to which event the question referred. False memories occurred when participants chose both the misinformation detail (e.g., brown couch) during the detail question and attributed the misinformation detail incorrectly to either the original event or both events—as indicated by the red (dark grey) circles. True memories occurred when participants both chose the correct detail during the detail question (e.g., white couch) and attributed the correct detail correctly to the original event—as indicated by the green (light grey) circles. Other example detail questions for this ABC triad included: Where were the stairs located? What color were the walls in the room? What was this individual sitting/standing on? What was hanging on the wall directly behind this individual? and so forth. It is important that all of these questions relate to two contradictory details from images AB and BC (e.g., stairs directly behind vs. to the far left; yellow vs. white walls; wood floors vs. carpet; potted plants vs. picture frames; etc.). The green (light grey) circles indicate the correct answer for the associative inference and directly learned questions. Participants saw these images without the red (dark grey) and green (light grey) circles. Individuals depicted here, or their guardians, gave signed consent for their likenesses to be published in this article. See the online article for the color version of this figure.

color was the couch?) followed immediately by a source memory test (In which set of images do you remember seeing this information?). For one half of the AB and BC scenes, *detail/source memory tests* were given before the test of direct (AB, BC) and indirect (AC) associations, and for the other half, the detail/source memory tests were given after the tests of direct and indirect associations. For the detail/source test, a *true memory* is defined as a response in which the participants both chose the correct item and attributed the source of their memory correctly (e.g., white couch attributed to AB scene), whereas a *false memory* is defined as a response for which the participant both chose the item from the overlapping image (e.g., BC) and misattributed its source (e.g., brown couch attributed to AB scene; see Methods for further details).

The critical comparison concerns the proportions of false memories on the detail/source tests given before versus after the associative inference test, for correct compared with incorrect associative inference trials (i.e., AC). We distinguish among three competing hypotheses:

(1) If recombination during retrieval both enhances associative inference performance and also increases susceptibility to false memories, then the proportion of false memories should be higher for correct than incorrect inference trials, but only when the detail/source test is given *after* the associative inference test (during which recombination occurs); there should be no difference in the proportion of false memories for correct versus incorrect inference trials when the detail/source test is given before the associative inference test.

(2) If the proportion of false memories is higher for correct than incorrect inference trials both when the detail/source tests are given before and after the associative tests, then these effects would be attributable to integrative encoding processes.

(3) If there is no link at all between source memory misattributions and associative inference, then there should be no difference between the proportion of false memories for correct and incorrect inference trials regardless of when the detail/source test is given.

To test these hypotheses, and determine the reliability of the results across variations in procedure and experimental parameters, we conducted three initial experiments that used the same basic paradigm and differed only in methodological details. Experiment 1 used a 24-hr study-test delay and a two-alternative forced choice on the associative inference test, whereas Experiment 2 used a 48-hr study-test delay and included an additional "neither" option on the forced-choice test (see Experiments 1 and 2 for rationale regarding these changes). In Experiment 3, we increased the delay between the directly learned (AB and BC) and associative inference trials (AC) on the one hand, and the second set of detail and source questions on the other, to assess the durability of the effects observed in Experiments 1 and 2. All three of these experiments provided evidence in favor of the first hypothesis outlined previously: The proportion of false memories was higher for correct than for incorrect inference trials, and only when the detail/source test was given *after* the associative inference test, during which recombination occurs. These findings implicate recombination during retrieval in both associative inference and memory misattribution, in line with the constructive episodic simulation hypothesis. To further test the hypothesis, in Experiment 4 we eliminated tests of directly learned associations (AB and BC), which in theory could have contributed to the effects that we attributed to flexible recombination. However, Experiment 4 again replicated the major findings of Experiments 1–3, providing further evidence that recombination during retrieval is responsible for the observed pattern of false memory effects.

Experiments 1 and 2

Because Experiments 1 and 2 used nearly identical procedures with only minor differences, we report the methods and results for these experiments together. To provide an overview of the basic procedure, participants came to the lab for two sessions, separated by a 24-hr (Experiment 1) or a 48-hr (Experiment 2) delay. The delay in Experiment 2 was extended from 24- to 48-hr to more closely replicate accuracy levels on the directly learned and associative inference test reported in the standard associative inference paradigm designed by Preston and colleagues (Preston et al., 2004; Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010; Zeithamova, Schlichting, & Preston, 2012). Participants completed a modified version of an associative inference paradigm based on prior studies by the Preston group (Preston et al., 2004; Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010; Zeithamova, Schlichting, & Preston, 2012). In the first session, participants intentionally encoded directly learned associations between individual "A" and object "B" followed by a second set of images with overlapping associations between object "B" and individual "C" (Figure 1); participants were also presented with nonoverlapping X-Y individual-object pairs to reduce performance for directly learned associations below ceiling levels. A total of 24 ABC triads and 24 XY pairs were used in the experiment. In the second session, participants were tested on directly learned associations (i.e., AB, BC, XY) and associative inference trials consisting of novel combinations of person pairings (i.e., AC). In addition, for one half of the ABC triads, participants answered 10 detail and source monitoring questions per triad *before* they were tested on directly learned and associative inference trials. For the alternate half of the triads, participants answered these detail and source monitoring questions *after* the directly learned and associative inference trials for all items. As noted earlier, the contrast between performance on the detail and source memory tests given before compared with after the directly learned/associative inference trials is critical to testing the three key hypotheses we outlined.

Method

Participants. For both experiments, participants were recruited via advertisements at Boston University and Harvard University. All had normal vision and no history of neurological impairment. They gave informed consent, were treated in accordance with guidelines approved by the ethics committee at Harvard University, and received either course credit or pay for completing the study. Experiment 1 included 26 young adults (mean age = 21.20, *SD* = 2.19; 15 women). Two participants were excluded from the true, false, and foil memory analyses because they were 100% accurate on the associative inference trials; thus, our final sample consisted of 24 participants. Participants who were 100% accurate on the associative inference trials were removed from the true, false, and foil memory analyses because they did not have any trials for which they correctly recalled the directly learned relationships *and* incorrectly inferred the relationship between item A and item C, thereby precluding meaningful comparisons of successful inference to unsuccessful inference both before and after flexible retrieval. Experiment 2 included 25 young adults (mean age = 20, *SD* = 1.93; 14 women). One participant was excluded from all analyses for having prior experience with several of the task stimuli; thus, our final sample consisted of 24 participants. Prior to the experiment, we decided on a sample size of 24 based on previous work utilizing a similar source monitoring paradigm (Okado & Stark, 2005). We stopped data collection after reaching the target of 24 participants with analyzable data.

AB and BC encoding. All experimental sessions were executed on an Apple desktop computer using PsychoPy2 (v1.80.03).

Stimuli consisted of 72 still color images depicting everyday life events (e.g., walking to work). Color images of common objects (e.g., toy truck) and individuals were superimposed on outdoor and indoor scenes. Scenes were counterbalanced across participants such that each scene was used equally often for both AB and BC pairs. Using Adobe Photoshop CC 2015, 48 overlapping pairs (24 AB pairs, 24 BC pairs—24 total ABC triads) and 24 unique, nonoverlapping pairs (XY) were constructed. Overlapping AB and BC pairs were constructed such that two individuals (A and C) shared an association with an overlapping object (B; i.e., one ABC triad). XY pairs were constructed of unique individual—object pairs that did not share an overlapping association with other pairings.

Participants received one of two versions of the AB encoding task, which consisted of 36 images (i.e., AB and XY) followed by the corresponding BC encoding task, which consisted of 36 images (i.e., BC and XY; Figure 1). Each image was randomly presented for 10 seconds within each encoding block (i.e., AB encoding and BC encoding). Participants were instructed to learn both the direct associations (i.e., AB, BC) and the indirect associations (i.e., AC) along with the contextual information presented. Following each image, participants were asked to provide a judgment of learning on a scale from 1 to 4 (1 = *definitely forget*, 4 = *definitely remember*). These judgments were collected to ensure participants' attention during the encoding phase.

Detail and source monitoring. Ten detail and source monitoring questions were constructed for each of the 24 ABC triads (5 questions related to image AB and 5 questions related to image BC). Detail questions were directly related to background details that were present but contradictory in the AB and BC scenes and did not reference the overlapping "B" object (Figure 1). A cutout of the cue individual (i.e., either "A" or "C") was presented to the right of each detail question to indicate which scene the question was referring to (Figure 1). For each detail question, participants were given three options: the correct item, a misinformation item, and an unrelated foil item. The misinformation item consisted of information from the overlapping image in the triad (e.g., if the detail question were related to the AB image, the misinformation item would be a contradicting detail from the BC image, such as a brown couch when a white couch had appeared in the AB image). Foil items were details that were not presented in either of the overlapping images (e.g., gray couch). Following each detail question, participants indicated where they remember seeing this contextual detail (i.e., the source of the information; Figure 1). Participants were given four possible answer choices: (a) the first set of images—AB, (b) the second set of images—BC, (c) both sets of images, or (d) unsure. Immediately following participants' source monitoring response, they were asked to rate their confidence in their response on a scale from 1 to 4 (1 = *very unsure*, 4 = *very sure*). The presentation order of each set of questions (i.e., detail, source, confidence) was randomized for each participant and the questions were self-paced.

Participants answered the 10 detail and source monitoring questions for one half of the 24 ABC triads before being tested on the directly learned and associative inference trials. After participants were tested on the directly learned and associative inference trials, they completed the 10 detail and source monitoring questions for the alternate half of the 24 ABC triads.

Directly learned and associative inference trials. Following the first half of the detail and source questions, participants were tested on directly learned (AB and BC) and associative inference trials (AC). During each directly learned trial, a single cue individual (e.g., an "A" or "C" individual) was presented at the top of the screen and two choice objects were presented at the bottom of the screen (e.g., two "B" objects from different ABC triads; Figure 1). On the associative inference trials, a cue individual (A) was presented along with two individuals at the bottom of the screen (i.e., the correct "C" individual from the ABC triad and a lure "C" individual from another triad). Participants were instructed on associative inference trials that the association between the cue (A) and the correct choice (C) was indirect, mediated through an object (B) that shared an association with both the cue and the correct choice during encoding. In Experiment 1, participants were required to make a forced-choice decision indicating which of the two choice objects/individuals was associated with the cue individual. In Experiment 2, participants were given a third option to respond "neither" when they believed that the items had not been previously paired, in order to reduce the possible influences of guessing on the associative inference task. If participants could not recall which of the two options given was paired with the cue individual, they were allowed to guess in Experiment 1, which could add noise to the source memory data by including triads for which participants were not actually able to successfully infer the relationship between item A and item C, but appeared to do so because of guessing. Thus, in an attempt to replicate the results of Experiment 1 and also control for the potential effects of guessing, a neither option was included for Experiment 2. It is important that for both directly learned and associative inference trials, the incorrect choice was a familiar item that had been studied in the context of another individual independent from the cue. Thus, correct responses required retrieval of learned associations and could not be made based on the familiarity of the choice. The presentation order of the trials was randomized with the only constraint being that AC associative inference trials were shown before their corresponding AB and BC directly learned trials in order to ensure that participants were not able to form an association between "A" and "C" individuals during test. Following each of the directly learned and associative inference trials, participants rated their confidence in their response on a scale from 1 to 4 (1 = *very unsure*, 4 = *very sure*).

Coding of true and false memories. Consistent with previous work using a similar detail and source monitoring paradigm (Okado & Stark, 2005), true memories were defined as detail questions for which the participant both chose the correct detail *and* attributed the source of their memory correctly to the currently cued image. False memories were defined as detail questions for which the participant both chose the misinformation detail *and* attributed the misinformation detail incorrectly to either the currently cued image or both images in the triad (Figure 1). False, true and foil memories were analyzed for ABC triads for which participants correctly inferred the relationship between "A" and "C" compared with triads for which the inference was not correctly made. In addition, false, true and foil memories were evaluated both before explicit retrieval of the inference (i.e., before AC associative inference trials) and after the retrieval of the inference in order to selectively compare the distinct effects of integration during encoding and flexible recombination at retrieval on subsequent memory errors.

Results

Directly learned and associative inference trials.

Experiment 1. First, we evaluated overall accuracy on directly learned and associative inference trials. Performance on both directly learned and associative inference trials was generally accurate, and there was no significant difference in the proportion of directly learned (M_{direct} = 0.78, SE = 0.02) compared with associative inference trials ($M_{\text{associative inference}}$ = 0.80, SE = 0.03) that participants answered correctly, $t(25) = -.99$, $p > .250$, mean difference = −0.02, 95% confidence interval (CI) [−0.06, 0.02], d = .19. Consistent with previous research (Zeithamova & Preston, 2010), we found significantly longer reaction times (RTs) on associative inference trials ($M_{\text{associative inference}}$ = 4,425 ms, SE = 341) compared with directly learned trials (M_{direct} = 3,306 ms, SE = 314), suggesting that there may be an additional recombination-related retrieval mechanism necessary for inferential versus direct retrieval, $t(25) = 9.48$, $p < .001$, mean difference = 1.12, 95% CI[0.88, 1.36], d = 1.85. Furthermore, participants assigned significantly higher confidence ratings to their responses on directly learned (M_{direct} = 3.34, SE = 0.07) compared with associative inference trials ($M_{\text{associative inference}}$ = 2.87, SE = 0.09), suggesting that participants were more confident in their memory for events that they had directly experienced compared with those resulting from recombination, $t(25) = 9.38$, $p <$ .001, mean difference = 0.47, 95% CI[0.37, 0.58], d = 1.89.

Experiment 2. Again we evaluated overall accuracy on directly learned and associative inference trials. There was a trend toward a significant difference in the proportion of directly learned (M_{direct} = 0.69, SE = 0.03) compared with associative inference trials ($M_{\text{associative inference}}$ = 0.64, SE = 0.03) that participants answered correctly, t(23) = 2.00, p = .057, mean difference = 0.05, 95% CI[−0.002, 0.10], d = .42. While this trend is slightly different from the results reported in Experiment 1, it does not affect the main hypotheses of interest, which are related to the false memory analyses. Consistent with results from Experiment 1, we found significantly longer RTs on associative inference trials ($M_{\text{associative inference}}$ = 4,401 ms, SE = 185) compared with directly learned trials (M_{direct} = 3,052 ms, SE = 129), suggesting an additional recombination-related retrieval mechanism for inferential versus direct retrieval, $t(23) = 5.66$, $p < .001$, mean difference = 1.35, 95% CI[0.99, 1.71], d = 1.62. Furthermore, results revealed that participants were significantly more confident in their responses on directly learned (M_{direct} = 3.22, SE = 0.09) compared with associative inference trials ($M_{\text{associative inference}}$ = 2.83, SE = 0.08; $t(23) = 5.67$, $p < .001$, mean difference = 0.39, 95% CI [0.25, 0.53], d = 1.18). Thus, results from Experiment 2 replicate those in Experiment 1.

False memory.

Experiment 1. To assess the effects of integrative encoding and recombination mechanisms at retrieval on subsequent memory errors, we examined source memory errors for the detail and source monitoring questions with a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures analysis of variance (ANOVA). It is important that only trials for which participants correctly remembered the directly learned association were included in subsequent analyses. See Supplementary Table 1, available online, for the raw number of trials per bin for each experiment. Results revealed a trend toward a main effect of time, $F(1, 23) = 3.04$, $p = .095$, $\eta_p^2 = .12$; no main effect of inference, $F(1, 23) = 2.40$, $p = .135$, $\eta_p^2 = .10$; and a significant time by inference interaction, $F(1, 23) = 7.05$, $p = .014$, $\eta_p^2 = .24$ (Figure 2a). Participants falsely attributed more details to the overlapping event after successful inference retrieval (M_{after} = 0.27, SE = 0.01) than before successful inference retrieval (M_{before} = 0.21, SE = 0.02; $t(23) = 4.05$, $p < .001$, mean difference = 0.06, 95% CI[0.03, 0.08], d = .83). Furthermore, participants did not falsely attribute more details to the overlapping event after unsuccessful inference retrieval (M_{after} = 0.21, SE = 0.02) than before unsuccessful inference retrieval (M_{before} = 0.22, SE = 0.02; $t(23) = .385$, $p > .250$, mean difference = −0.01, 95% CI[−0.05, 0.04], d = .08). Participants did not falsely attribute

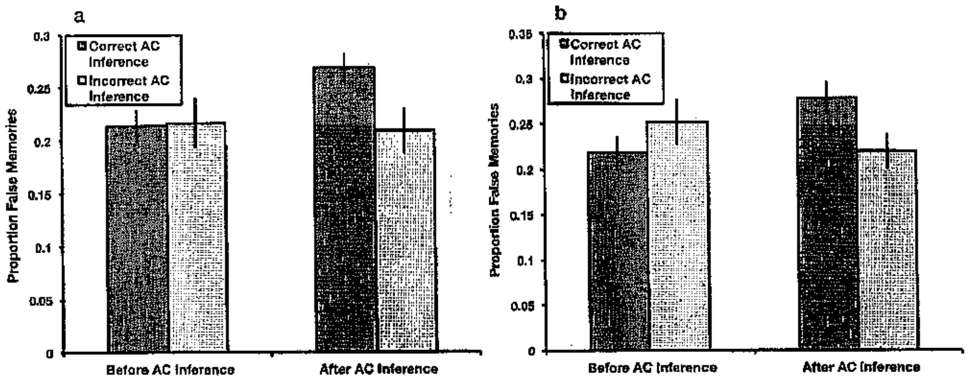

Figure 2. Proportion of false memories in Experiment 1 (a) and Experiment 2 (b). Performance on detail and source monitoring questions was examined both before and after either successful or unsuccessful inference. It is important that only trials for which participants responded correctly to directly learned trials were included in this analysis. Results revealed a significant time by inference interaction in both Experiment 1 and 2. Subsequent t tests confirm that false memories selectively increased only following successful associative inference. Error bars represent ± 1 SEM.

more details to the overlapping event before successful inference retrieval ($M_{\text{correct}} = 0.21$, $SE = 0.02$) than before unsuccessful inference retrieval ($M_{\text{incorrect}} = 0.22$, $SE = 0.02$; $t(23) = .143$, $p > .250$, mean difference = 0.003, 95% CI[−0.04, 0.05], $d = .03$). Critically, participants falsely attributed more details to the overlapping event after successful inference retrieval ($M_{\text{correct}} = 0.27$, $SE = 0.01$) than after unsuccessful inference retrieval ($M_{\text{incorrect}} = 0.21$, $SE = 0.02$; $t(23) = 2.73$, $p = .012$, mean difference = 0.06, 95% CI[0.01, 0.10], $d = .56$), suggesting that recombination processes underlying successful inference at retrieval may also lead to source memory errors.

Experiment 2. Identical to Experiment 1, we examined source memory errors for the detail and source monitoring questions with a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA. Results revealed no main effect of time, $F(1, 23) = .357$, $p > .250$, $\eta_p^2 = .02$; no main effect of inference, $F(1, 23) = .57$, $p > .250$, $\eta_p^2 = .02$; and a significant time by inference interaction, $F(1, 23) = 7.40$, $p = .012$, $\eta_p^2 = .24$ (Figure 2b). Participants falsely attributed more details to the overlapping event after successful inference retrieval ($M_{\text{after}} = 0.28$, $SE = 0.02$) than before successful inference retrieval ($M_{\text{before}} = 0.22$, $SE = 0.02$; $t(23) = 2.48$, $p = .021$, mean difference = 0.06, 95% CI[0.01, 0.11], $d = .51$). Furthermore, participants did not falsely attribute more details to the overlapping event after unsuccessful inference retrieval ($M_{\text{after}} = 0.22$, $SE = 0.02$) than before unsuccessful inference retrieval ($M_{\text{before}} = 0.25$, $SE = 0.03$; $t(23) = -1.022$, $p > .250$, mean difference = −0.03, 95% CI[−0.10, 0.03], $d = .21$). Participants did not falsely attribute more details to the overlapping event before successful inference retrieval ($M_{\text{correct}} = 0.22$, $SE = 0.02$) than before unsuccessful inference retrieval ($M_{\text{incorrect}} = 0.25$, $SE = 0.03$; $t(23) = 1.40$, $p = .175$, mean difference = 0.03, 95% CI[−0.02, 0.08], $d = .29$). Critically, participants falsely attributed more details to the overlapping event after successful inference retrieval ($M_{\text{correct}} = 0.28$, $SE = 0.02$) than after unsuccessful inference retrieval ($M_{\text{incorrect}} = 0.22$, $SE = 0.02$; $t(23) = 2.56$, $p = .018$, mean difference = 0.06, 95% CI[0.01, 0.11], $d = .52$), replicating results from Experiment 1 and suggesting that recombination during retrieval required for successful inference may be linked to source memory errors.

True memory.

Experiment 1. To examine the effects of integrative encoding and recombination mechanisms at retrieval on successful source memory, we examined correct responses on the detail and source monitoring questions with a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA. Results revealed no main effect of time, $F(1, 23) = 2.33$, $p = .141$, $\eta_p^2 = .09$; no main effect of inference, $F(1, 23) = .10$, $p > .250$, $\eta_p^2 = .02$; but a significant time by inference interaction, $F(1, 23) = 6.83$, $p = .016$, $\eta_p^2 = .23$. Participants attributed more details to the correct source after successful inference retrieval ($M_{\text{after}} = 0.23$, $SE = 0.02$) than before successful inference retrieval ($M_{\text{before}} = 0.17$ $SE = 0.02$; $t(23) = 3.82$, $p = .001$, mean difference = 0.06, 95% CI[0.03, 0.09], $d = .82$). By contrast, participants did not attribute more details to the correct source after successful inference retrieval ($M_{\text{correct}} = 0.23$, $SE = 0.02$) than after unsuccessful inference retrieval ($M_{\text{incorrect}} = 0.20$ $SE = 0.03$; $t(23) = 1.04$, $p > .250$, mean difference = 0.03, 95% CI[−0.03, 0.08], $d = .21$), and did not attribute more details to the correct source before successful inference retrieval ($M_{\text{correct}} = 0.17$, $SE = 0.02$) than before unsuccessful inference retrieval ($M_{\text{incorrect}} = 0.22$, $SE = 0.03$; $t(23) = 1.50$, $p = .146$, mean difference = 0.04, 95% CI[−0.02, 0.10], $d = .31$). Furthermore, participants did not attribute more details to the correct source after unsuccessful inference retrieval ($M_{\text{after}} = 0.20$, $SE = 0.03$) than before unsuccessful inference retrieval ($M_{\text{before}} = 0.22$, $SE = 0.03$; $t(23) < 1$, $p > .250$, mean difference = 0.01, 95% CI[−0.03, 0.06], $d = .12$). The increase in true memory from before inference retrieval to after appears to be attributable to changes in "unsure" responses on the source monitoring test following recognition of the correct item: Participants were significantly less likely to respond unsure following successful inference when they correctly recognized the detail ($M_{\text{after}} = 0.12$, $SE = 0.05$) than before successful inference ($M_{\text{before}} = 0.26$, $SE = 0.06$; $t(23) = 3.04$, $p = .008$, mean difference = 0.14, 95% CI[0.04, 0.23], $d = .18$).

Experiment 2. A 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA on correct responses to the detail and source monitoring questions revealed no main effect of time, $F(1, 23) = .40$, $p > .250$, $\eta_p^2 = .02$; no main effect of inference, $F(1, 23) = .55$, $p > .250$, $\eta_p^2 = .02$; and no time by inference interaction, $F(1, 23) = 1.34$, $p > .250$, $\eta_p^2 = .06$. Thus, true memory scores were similar both before ($M_{\text{before}} = 0.18$, $SE = 0.03$) and after successful inference retrieval ($M_{\text{after}} = 0.21$, $SE = 0.03$). In addition, true memory scores were similar both before ($M_{\text{before}} = 0.16$, $SE = 0.03$) and after unsuccessful inference retrieval ($M_{\text{after}} = 0.20$, $SE = 0.03$).

Foil memory.

Experiment 1. To assess whether critical patterns of source misattribution errors are specific to related items from previously studied episodes, we examined foil memories, which were defined as detail questions for which participants chose the unrelated foil option (e.g., gray couch) and attributed the information to either the currently cued image or both images in the triad. We conducted a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA to evaluate participants' foil memory scores. Results revealed no main effect of time, $F(1, 23) = 1.16$, $p > .250$, $\eta_p^2 = .05$; no main effect of inference, $F(1, 23) = 1.71$, $p = .204$, $\eta_p^2 = .07$; and no time by inference interaction, $F(1, 23) = 1.59$, $p = .220$, $\eta_p^2 = .07$. Thus, foil memory scores were similar both before ($M_{\text{before}} = 0.18$, $SE = 0.01$) and after successful inference retrieval ($M_{\text{after}} = 0.18$, $SE = 0.01$). In addition, foil memory scores were similar both before ($M_{\text{before}} = 0.14$, $SE = 0.03$) and after unsuccessful inference retrieval ($M_{\text{after}} = 0.17$, $SE = 0.02$).

Experiment 2. We conducted a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA to evaluate participants' foil memory scores. Results revealed no main effect of time, $F(1, 23) = 1.04$, $p > .250$, $\eta_p^2 = .04$; no main effect of inference, $F(1, 23) = 1.28$, $p > .250$, $\eta_p^2 = .05$; and no time by inference interaction, $F(1, 23) = 1.22$, $p > .250$, $\eta_p^2 = .05$. Thus, foil memory scores were similar both before ($M_{\text{before}} = 0.21$, $SE = 0.02$) and after successful inference retrieval ($M_{\text{after}} = 0.17$, $SE = 0.02$). In addition, foil memory scores were similar both before ($M_{\text{before}} = 0.21$, $SE = 0.03$) and after unsuccessful inference retrieval ($M_{\text{after}} = 0.21$, $SE = 0.02$).

Discussion

The results of Experiments 1 and 2 provide evidence that flexible retrieval processes required for successful associative inference also produce increases in false memories as a result of source misattributions: memory errors increased significantly *after* but not *before* successful compared with unsuccessful inferential retrieval. This pattern was observed both when the test of directly learned and associative inference items was two-alternative forced-choice (Experiment 1), and also when a third "neither" option was provided (Experiment 2); the effect was also robust across both a 24-hr study-test delay (Experiment 1) and a 48-hr study-test delay (Experiment 2).

The finding that source misattributions occurred more often for correct versus incorrect inferences constitutes evidence for a link between processes that support associative inference and those that contribute to false memories; if there were no such link, then source memory errors would not differ for correct and incorrect inferences. This finding alone, however, does not allow us to distinguish whether integrative encoding or flexible retrieval is responsible for the observed increased of source memory errors related to successful inference. However, the finding that the observed increase in false memories for correct inference occurred only *after* the associative inference test was given implicates flexible retrieval, rather than integrative encoding, as the key process responsible for the boost in false memories. Furthermore, foil memory scores (i.e., detail questions for which participants chose the unrelated foil option) showed no relationship to correct versus incorrect inferences either before or after the associative inference test was given. This finding indicates that the observed source memory error effects are selective to previously experienced details, details that seemingly migrated between the AB and BC episodes as a consequence of successful inference.

Experiment 3

In Experiments 1 and 2, we replicated the key effect of successful inference on false memories across minor variations in procedure, suggesting that the effect is reliable. However, the absolute magnitude of the effect is not large, and because the critical tests in Experiments 1 and 2 were administered in immediate succession, we do not know whether the process of recombination during retrieval that supports successful inference results in only a transient change in participants' susceptibility to source memory errors. To further assess the reliability of the key effect, and to determine whether the effects of recombination allowing for successful associative inference on subsequent source memory error lasts beyond the brief interval between the main tests and further shows a longer-lasting effect on participants susceptibility to source memory error, in Experiment 3 we introduced a 30-min delay between the directly learned (AB and BC)/associative inference trials (AC) on the one hand and the second set of detail and source questions on the other.

Method

Participants. Experiment 3 included 24 young adults (mean age = 20, *SD* = 2.07; 15 women). No participants were excluded from the analyses; thus, our final sample consisted of 24 participants.

Summary of the procedure. Participants came to the lab for two sessions, separated by a 48-hr delay. The design parameters, stimuli, and coding of true and false memories were exactly the same in Experiment 3 as in Experiment 2 with one exception. During the second session, following the test of the directly learned (AB and BC) and associative inference trials (AC), participants completed 30-min of unrelated filler tasks (e.g., simple math problems) before completing the second half of the detail and source questions. As noted earlier, in Experiment 3 we introduced a 30-min delay between the directly learned/associative inference trials and the second set of detail and source questions.

Results and Discussion

Directly learned and associative inference trials. First, we evaluated overall accuracy on directly learned and associative inference trials. Performance on both directly learned and associative inference trials was generally accurate, and there was no significant difference in the proportion of directly learned ($M_{\text{direct}} = 0.72$, $SE = 0.02$) compared with associative inference trials ($M_{\text{associative inference}} = 0.71$, $SE = 0.02$) that participants answered correctly, $t(23) = 0.62$, $p > .250$, mean difference = 0.01, 95% confidence interval (CI) = [−0.03, 0.06], $d = .13$). Consistent with previous research (Zeithamova & Preston, 2010) and results from Experiment 1 and 2, we found significantly longer RTs on associative inference trials ($M_{\text{associative inference}} = 4{,}186$ ms, $SE = 196$) compared with directly learned trials ($M_{\text{direct}} = 3{,}057$ ms, $SE = 123$), suggesting that there may be an additional recombination-related retrieval mechanism necessary for inferential versus direct retrieval, $t(23) = -7.46, p < .001$, mean difference = −1.13, 95% CI[−1.44, −.82], $d = 1.52$. Furthermore, participants assigned significantly higher confidence ratings to their responses on directly learned ($M_{\text{direct}} = 3.23$, $SE = 0.08$) compared with associative inference trials ($M_{\text{associative inference}} = 2.83$, $SE = 0.10$), suggesting that participants were more confident in their memory for events that they had directly experienced compared with those resulting from recombination, $t(23) = 8.97$, $p < .001$, mean difference = 0.40, 95% CI[0.31, 0.50], $d = 1.83$.

False memory. Identical to Experiments 1 and 2, we examined source memory errors for the detail and source monitoring questions with a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA. Results revealed no main effect of time, $F(1, 23) = 0.46$, $p > .250$, $\eta_p^2 = .02$; no main effect of inference, $F(1, 23) = 1.17$, $p > .250$, $\eta_p^2 = .05$; and a significant time by inference interaction, $F(1, 23) = 5.89, p = .023, \eta_p^2 = .20$ (Figure 3). Participants falsely attributed more details to the overlapping event after successful inference retrieval ($M_{\text{after}} = 0.27$, $SE = 0.02$) than before successful inference retrieval ($M_{\text{before}} = 0.22$, $SE = 0.02$; $t(23) = 3.46$, $p = .002$, mean difference = 0.05, 95% CI[0.02, 0.08], $d = .71$). Furthermore, participants did not falsely attribute more details to the overlapping event after unsuccessful inference retrieval ($M_{\text{after}} = 0.21$, $SE = 0.03$) than before unsuccessful inference retrieval ($M_{\text{before}} = 0.23$, $SE = 0.03$; $t(23) = -0.63$, $p > .250$, mean difference = −0.02, 95% CI[−0.09, 0.05], $d = .13$). Participants did not falsely attribute more details to the overlapping event before successful inference retrieval ($M_{\text{correct}} = 0.22$, $SE = 0.02$) than before unsuccessful inference retrieval ($M_{\text{incorrect}} = 0.23$, $SE = 0.03$; $t(23) = .829$, $p > .250$, mean

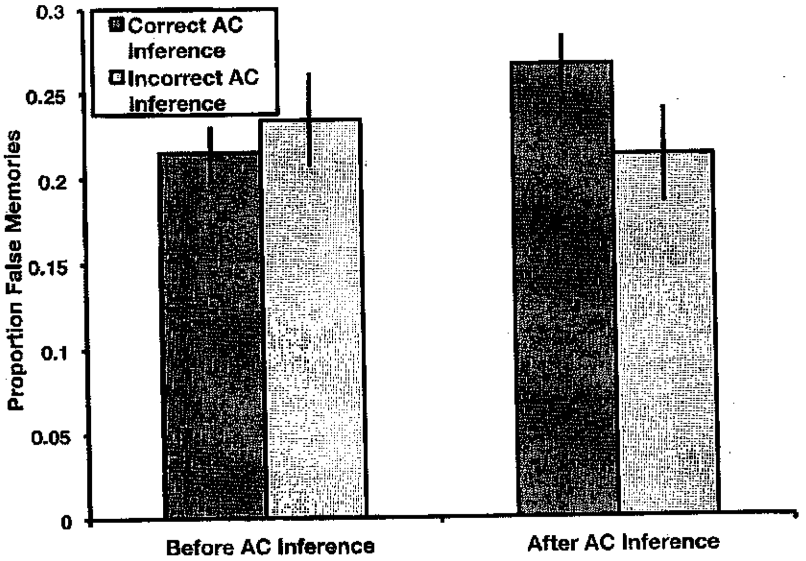

Figure 3. Proportion of false memories in Experiment 3. Performance on detail and source monitoring questions was examined both before and after either successful or unsuccessful inference. It is important that only trials for which participants responded correctly to directly learned trials were included in this analysis. Results revealed a significant time by inference interaction in Experiment 3. Subsequent *t* tests confirm that false memories selectively increased only following successful associative inference. Error bars represent ± 1 *SEM*.

difference = 0.02, 95% CI[−0.03, 0.06], d = .17). Critically, participants falsely attributed more details to the overlapping event after successful inference retrieval ($M_{correct}$ = 0.27, SE = 0.02) than after unsuccessful inference retrieval ($M_{incorrect}$ = 0.21, SE = 0.03; $t(23) = 2.49$, $p = .021$, mean difference = 0.05, 95% CI[0.009, 0.10], d = .51), replicating results from Experiment 1 and 2, suggesting again that recombination during retrieval required for successful inference may be linked to source memory errors.

True memory. A 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA on correct responses to the detail and source monitoring questions revealed no main effect of time, $F(1, 23) = 0.46$, $p > .250$, $\eta_p^2 = .02$; no main effect of inference, $F(1, 23) = 0.91$, $p > .250$, $\eta_p^2 = .04$; and no time by inference interaction, $F(1, 23) = .042$, $p > .250$, $\eta_p^2 = .002$. Thus, true memory scores were similar both before (M_{before} = 0.23, SE = 0.02) and after successful inference retrieval (M_{after} = 0.24, SE = 0.02). In addition, true memory scores were similar both before (M_{before} = 0.21, SE = 0.04) and after unsuccessful inference retrieval (M_{after} = 0.22, SE = 0.02).

Foil memory. We conducted a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA to evaluate participants' foil memory scores. Results revealed no main effect of time, $F(1, 23) = 1.74$, $p = .200$, $\eta_p^2 = .07$; no main effect of inference, $F(1, 23) = 0.62$, $p > .250$, $\eta_p^2 = .03$; and no time by inference interaction, $F(1, 23) = 2.23$, $p = .150$, $\eta_p^2 = .09$. Thus, foil memory scores were similar both before (M_{before} = 0.20, SE = 0.02) and after successful inference retrieval (M_{after} = 0.16, SE = 0.01). In addition, foil memory scores were similar both before (M_{before} = 0.16, SE = 0.02) and after unsuccessful inference retrieval (M_{after} = 0.17, SE = 0.02).

Overall, the pattern of results from Experiment 3 was essentially identical to that observed in Experiments 1 and 2: Participants made significantly more source misattributions for correct than incorrect inferences, but only when the detail and source monitoring test was given after the test of directly learned and associative inference items. Because the second source test was administered 30 min after completion of the directly learned and associative inference test, Experiment 3 indicates that the effects we have attributed to flexible recombination during retrieval are not simply transient influences that disappear following a filled delay.

Experiment 4

Although our central theoretical claim of Experiments 1–3 focuses on retrieval-related recombination processes that occurred during the associative inference test for previously unpaired AC items, participants were also tested for AB and BC items that did appear together previously. Thus, it is conceivable that the increase in source memory errors following the associative inference test is attributable to direct retrieval of previously studied pairs (AB, BC) as opposed to retrieval-related recombination processes. Two key features of the data from Experiments 1–3 speak against this possibility. First, if retrieval of directly learned associations were responsible for the increase in false memories, then false memory rates should have been similar for successful and unsuccessful inferential retrieval after the associative inference test, but as noted above memory errors increased significantly following successful compared with unsuccessful inferential retrieval. Second, neither experiment revealed significant differences in the number false memories before compared with after *unsuccessful* inferential retrieval. If testing of directly learned pairs during the associative inference test were responsible for the increased false memory effects after compared with before the associative inference test, then those effects should have been observed for unsuccessful inference trials. However, no such effects were observed. While the results of Experiments 1–3 thus suggest that the testing of directly learned associations is not responsible for the key effects of successful inference on false memories, in Experiment 4 we assess this possibility empirically by testing directly learned associations only after both sets of detail and source monitoring tests were completed. If, as we have suggested, recombination during retrieval is responsible for observed increases in false memories, then the same critical pattern of results from previous experiments—more source misattributions for correct than incorrect inference items, after but not before the inference test—should be observed in Experiment 4, even though directly learned associations were not tested prior to the detail and source memory tests.

Method

Participants. Experiment 4 included 26 young adults (mean age = 20.70 years, $SD = 2.19$; 16 women). Two participants were excluded from the true and false memory analyses. One participant was excluded from the true, false, and foil memory analyses because they were 100% accurate on the associative inference trials, and one participant was excluded from all analyses for noncompliance during the second session (e.g., did not make responses during the detail and source monitoring questions); thus, our final sample consisted of 24 participants.

Summary of the procedure. Participants came to the lab for two sessions, separated by a 48-hr delay. The design parameters, stimuli, and coding of true and false memories were exactly the same in Experiment 4 as in Experiment 2, with the one exception. During the second session, following the first half of the detail and source questions, participants were only tested on associative inference trials (AC), thus eliminating the potential effect of retrieving directly learned associations on false memory following successful associative inference. However, to ensure that we still obtained a measure of performance on directly learned items, following the second half of the detail and source questions, participants were tested on directly learned trials (AB and BC).

Results and Discussion

Directly learned and associative inference trials. First, we evaluated overall accuracy on directly learned and associative inference trials. Performance on both directly learned and associative inference trials was generally accurate, and there was no significant difference in the proportion of directly learned ($M_{\text{direct}} = 0.66$, $SE = 0.03$) compared with associative inference trials ($M_{\text{associative inference}} = 0.70$, $SE = 0.03$) that participants answered correctly, $t(24) = -1.01$, $p > .250$, mean difference = −0.04, 95% CI[−0.11, 0.04], $d = .20$. Consistent with previous research (Zeithamova & Preston, 2010) and results from Experiments 1–3, we found significantly longer RTs on associative inference trials ($M_{\text{associative inference}} = 5{,}140$ ms, $SE = 242$) compared with directly learned trials ($M_{\text{direct}} = 3{,}300$ ms, $SE = 148$), suggesting that there may be an additional recombination-related retrieval mechanism necessary for inferential versus direct retrieval, $t(24) = -10.18$, $p < .001$, mean difference = −1.84, 95% CI[−2.21, −1.47], $d = 2.04$. Furthermore, participants assigned significantly higher confidence ratings to their responses on directly learned ($M_{\text{direct}} = 3.15$, $SE = 0.12$) compared with associative inference trials ($M_{\text{associative inference}} = 2.87$, $SE = 0.11$), suggesting that participants were more confident in their memory for events that they had directly experienced compared with those resulting from recombination, $t(24) = 3.06$, $p = .005$, mean difference = 0.27, 95% CI[0.89, 0.46], $d = 0.61$.

False memory. Identical to Experiments 1–3, we examined source memory errors for the detail and source monitoring questions with a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA. Results revealed no main effect of time, $F(1, 23) = 1.60$, $p = .219$, $\eta_p^2 = .07$; no main effect of inference, $F(1, 23) = .011$, $p > .250$, $\eta_p^2 = .00$; and a significant time by inference interaction, $F(1, 23) = 4.79$, $p = .039$, $\eta_p^2 = .17$ (Figure 4). Participants falsely attributed more details to the overlapping event after successful inference retrieval ($M_{\text{after}} = 0.26$, $SE = 0.02$) than before successful inference retrieval ($M_{\text{before}} = 0.19$, $SE = 0.01$; $t(23) = 3.20$, $p = .004$, mean difference = 0.07, 95% CI[0.03, 0.12], $d = .65$). Furthermore, participants did not falsely attribute more details to the overlapping event after unsuccessful inference retrieval ($M_{\text{after}} = 0.23$, $SE = 0.03$) than before unsuccessful inference retrieval ($M_{\text{before}} = 0.23$, $SE = 0.04$; $t(23) = .010$, $p > .250$, mean difference = 0.0004, 95% CI[−0.09, 0.09], $d = .002$). Participants did not falsely attribute more details to the overlapping event before successful inference retrieval ($M_{\text{correct}} = 0.19$, $SE = 0.01$) than before unsuccessful inference retrieval ($M_{\text{incorrect}} = 0.23$, $SE = 0.04$; $t(23) = 1.22$, $p = .231$, mean difference = 0.04, 95% CI[−0.03, 0.10], $d = .25$). Critically, participants falsely attributed more details to the overlapping event after successful inference retrieval ($M_{\text{correct}} = 0.26$, $SE = 0.02$) than after unsuccessful inference retrieval ($M_{\text{incorrect}} = 0.23$, $SE = 0.03$; $t(23) = 2.37$, $p = .027$, mean difference = 0.03, 95% CI[0.004, 0.64], $d = .48$), replicating results from Experiments 1–3, suggesting that recombination during retrieval required for successful inference may be linked to source memory errors.

True memory. A 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA on correct responses to the detail and source monitoring questions revealed no main effect of time, $F(1, 23) = 2.53$, $p = .13$, $\eta_p^2 = .10$; no main effect of inference, $F(1, 23) = 1.68$, $p = .21$, $\eta_p^2 = .07$; and no time by inference interaction, $F(1, 23) = .43$, $p > .250$, $\eta_p^2 = .02$. Thus, true memory scores were similar both before ($M_{\text{before}} = 0.22$, $SE = 0.03$) and after successful inference retrieval ($M_{\text{after}} = 0.17$, $SE = 0.02$). In addition, true memory scores were similar both before ($M_{\text{before}} = 0.18$, $SE = 0.03$) and after unsuccessful inference retrieval ($M_{\text{after}} = 0.16$, $SE = 0.03$).

Foil memory. We conducted a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA to evaluate participants' foil memory scores. Results revealed no main effect of time, $F(1, 23) = 3.67$, $p = .068$, $\eta_p^2 = .14$; no main effect of inference, $F(1, 23) = 2.47$, $p = .130$, $\eta_p^2 = .10$; and no time by inference interaction, $F(1, 23) = 0.59$, $p > .250$, $\eta_p^2 = .03$. Thus, foil memory scores were similar both before ($M_{\text{before}} = 0.18$, $SE = 0.02$) and after successful inference retrieval ($M_{\text{after}} = 0.19$, $SE = 0.02$). In addition, foil memory scores were similar both before ($M_{\text{before}} = 0.14$, $SE =$

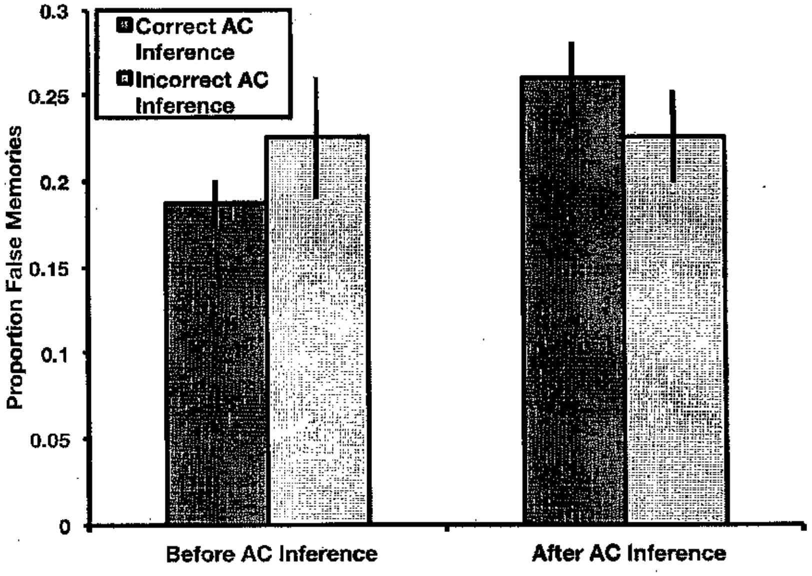

Figure 4. Proportion of false memories in Experiment 4. Performance on detail and source monitoring questions was examined both before and after either successful or unsuccessful inference. It is important that only trials for which participants responded correctly to directly learned trials were included in this analysis. Results revealed a significant time by inference interaction in Experiment 4. Subsequent *t* tests confirm that false memories selectively increased only following successful associative inference. Error bars represent ± 1 *SEM*.

0.02) and after unsuccessful inference retrieval (M_{after} = 0.18, *SE* = 0.02).

In summary, the results of Experiment 4 replicated the results of Experiment 1–3, while also providing additional evidence that testing of directly learned pairs during the associative inference test was not responsible for the increased false memory effects after compared with before successful associative inference. During the associative inference test for Experiment 4, participants were only tested on previously unpaired AC items (i.e., inference items) before the second set of detail and source questions. Thus, the increase in source memory errors following the associative inference test in Experiment 4 cannot be attributable to direct retrieval of previously studied pairs; rather, the current results support the role of recombination-related retrieval processes in subsequent source memory errors.

General Discussion

The four experiments reported here each provide evidence that flexible retrieval processes that support successful associative inference also produce increases in false memories that result from source misattributions: memory errors increased significantly *after* but not *before* successful compared to unsuccessful inferential retrieval. Experiments 1 and 2 provided evidence that flexible retrieval processes required for successful associative inference also produce increases in source misattributions when the detail/source memory test immediately followed the test of directly learned and associative inference items, whereas Experiment 3 revealed that these effects persisted across a 30-min delay between the associative inference tests and the second source memory test. Experiment 4 revealed the same significant increase in source memory error after but not before successful compared to unsuccessful inferential retrieval as observed in Experiment 1–3 even when directly learned associations were not tested until participants completed all of the detail and source monitoring questions. Furthermore, across all four experiments results revealed that both foil memory and correct memory scores showed no relationship to correct compared with incorrect inference either before or after the directly learned and associative inference test, thereby indicating that the observed effects are specific to the misattribution of previously experienced details to the related event rather than to a general decrease of detail with which the original event was remembered. Thus, the results of all four experiments provide direct evidence supporting the role of flexible retrieval and recombination processes in both successful associative inference and subsequent source memory error. These data thus provide, for the first time, direct experimental support for a key claim of the constructive episodic simulation hypothesis (Schacter & Addis, 2007a, 2007b), namely that the same flexible recombination process that supports an adaptive cognitive process can also increase memory errors that result from combining elements of distinct episodes. More generally, our results add to the mounting evidence that certain kinds of memory errors result from the operation of adaptive constructive processes that are linked to beneficial effects (for reviews, see Howe, 2011; Howe et al., 2016; Newman & Lindsay, 2009; Schacter, 2012; Schacter et al., 2011).

As noted in the Introduction, previous research (cf., Shohamy & Wagner, 2008; Zeithamova & Preston, 2010) indicates that successful associative inference in the AB, BC paradigm used here can result from flexible retrieval and/or integrative encoding (i.e., during study of BC, participants recall the related AC pair and encode an integrated representation (ABC) that they later retrieve on the associative inference test). If integrative encoding contributes to false memories in our paradigm, then there should be more false memories for successful than unsuccessful inference trials *before* the associative inference test, but such effects were observed only *after* the associative inference test. Note, however, that previous research suggests that integrative encoding primarily supports associative inference when learning occurs across multiple repetitions, by affording multiple opportunities for cross-episode binding (Shohamy & Wagner, 2008; Zeithamova & Preston, 2010). By contrast, our experimental design utilized a single-trial learning paradigm that elicits an additional recombination mechanism during successful inference retrieval (Zeithamova & Preston, 2010). It is thus possible that when there are multiple repetitions during the learning phase, or under other experimental conditions that heighten the contribution of integrative encoding to associative inference, integrative encoding processes contribute to the type of source memory errors observed here. Thus, while the present data provide evidence for a link between flexible retrieval and false memories, they by no means rule out a similar link to integrative encoding under a different set of experimental parameters that are more likely to elicit successful associative inference as a result of integration during encoding. Future research should aim to examine the role of integration during encoding on subsequent source memory error.

Why does successful inferential retrieval result in heightened susceptibility to source memory errors? We suggest that the effects that we have documented here reflect the joint operation of two related but distinct mechanisms: cross-episode binding (e.g., Bridge & Voss, 2014a, 2014b) and retrieval-based reactivation and recombination (e.g., Bridge & Voss, 2014a, 2014b; Hupbach, Gomez, Hardt, & Nadel, 2007; St Jacques & Schacter, 2013). Binding processes that link disparate elements of an episode into a unified representation have been extensively studied in recent years, and have been linked closely to the operation of the hippocampus (e.g., Eichenbaum & Cohen, 2001; Hannula & Ranganath, 2008; Shimamura, 2010). As Bridge and Voss (2014b) point out, however, most such studies have focused on binding of elements *within* an episode. Bridge and Voss (2014b) studied *cross-episode* binding processes, and provided evidence that participants sometimes bind elements from distinct episodes (e.g., a face from one episode and a scene from another), resulting in memory error (for additional evidence linking binding processes to memory distortions, see Lew & Howe, 2016). We suggest that such cross-episode binding in our paradigm occurs most often and most extensively for episodes that result in successful, as opposed to unsuccessful, associative inference. That is, when people make a correct inference about the relationship between elements of events that have not been experienced together previously (i.e., AC), they may more fully bind details from the two episodes, such that details from one episode (AB) migrate to and become incorporated in the overlapping (BC) episode.

However, this binding account alone cannot explain the key finding from our experiments that increased false memories were observed for successful compared to unsuccessful inference trials only when the detail/source memory test was given *after* the associative inference test, and it is this finding that has led us to implicate a role for flexible retrieval and recombination processes in increased source memory errors. These observations fit well with prior findings that reactivating or retrieving memories can be a potent source of memory distortion if novel information is incorporated into a memory during the retrieval process (e.g., Chan, Thomas, & Bulevich, 2009; Gershman, Schapiro, Hupbach, & Norman, 2013; Gordon, Thomas, & Bulevich, 2015; Hupbach et al., 2007; Hupbach, Gomez, & Nadel, 2011; St Jacques, Olm, & Schacter, 2013; St Jacques & Schacter, 2013), possibly related to processes associated with memory reconsolidation that render a memory labile and prone to distortion during retrieval (Chan & LaPaglia, 2013; Dudai, 2012; Hardt, Einarsson, & Nader, 2010). From this perspective, in our experimental paradigm source memory errors arise when overlapping AB and BC relationships (along with their corresponding contextual details) are reactivated and flexibly recombined in order to encode the novel inference between the previously unrelated A and C items. Indeed, and consistent with our results, Bridge and Voss (2014b) only observed evidence for memory distortion associated with cross-episode binding following an active (vs. passive) retrieval condition. In line with the current results, retrieval-related recombination may thus result in heightened rates of source memory error following successful compared to unsuccessful inference because inferring the relationship between the nonoverlapping A and C items requires both a) reactivating distinct AB and BC episodes and b) flexibly recombining the nonoverlapping A and C items—during which contextual details from the AB episode are more fully bound to the BC episode and visa versa—resulting in memory distortions associated with cross-episode binding as a consequence of flexible retrieval and recombination processes. An important task for future research is to explore and clarify exactly how the recombination process supporting successful inference produces such erroneous memories. While previous evidence supports the idea that memory errors can result from erroneously combining details of individual episodic or autobiographical memories (e.g., Burt et al., 2004; Devitt et al., 2016; Odegard & Lampinen, 2004), the present studies provide novel evidence that the same flexible recombination mechanism that supports an adaptive cognitive process, such as associative inference, also increases subsequent memory errors.

Although we are not aware of any prior studies that have specifically linked successful associative inference with memory errors, as noted earlier previous research has linked memory reactivation processes with source misattributions and related kinds of memory errors. The studies noted earlier by Bridge and Voss (2014a, 2014b) suggest that simply coactivating memories during retrieval can lead to source misattributions, wherein coactivation of existing memory traces produces cross-episode binding of peripheral features from each episode. Although these results are consistent with the results reported here, it is unlikely that simple coactivation of elements from different episodes is sufficient to account for our key results. Our data speak against a simple coactivation hypothesis specifically because only trials for which participants were able to successfully reactivate both AB and BC episodes (as assessed by the test for directly learned associations) were used in the false memory analyses. Thus, both AB and BC events should have been successfully reactivated during the inference test. Accordingly, if coactivation of AB and BC events accounted for the increase in source memory error, we would not expect to see a significant difference between successful inference and unsuccessful inference after the associative inference test.

Because we observed such a difference, we suggest that successful inference requires an additional retrieval-related recombination process that results in increased source memory error. Indeed, in each of our experiments we observed significantly longer RTs on associative inference trials than on directly learned trials, which is in line with the arguments of Zeithamova and Preston (2010), suggesting that there is an additional retrieval mechanism necessary for inferential versus direct retrieval following single-trial learning. Coactivation of memories at test clearly can lead to source misattributions (Bridge & Voss, 2014a, 2014b), and it may be a contributing factor and perhaps even a necessary condition for increased source memory errors in the current paradigm. Nonetheless, coactivation of elements from distinct episodes during retrieval does not appear to a sufficient condition for producing the increase in source misattributions in the current paradigm. Alternatively, coactivation may have an effect during encoding such that participants bring to mind overlapping AB pairs during BC encoding thereby linking the two related events. However, if this were the case we would expect to see elevated source memory error before successful inference, which was not the case in our experiments, as we emphasized in the discussion of integrative encoding.

Although we have emphasized throughout the distinction between integrative encoding and flexible retrieval, and provided in the Introduction explicit predictions regarding outcomes that distinguish between these processes, it is important to emphasize we are not advocating that a simple encoding-retrieval dichotomy can account for the results observed here. Students of memory have long recognized that that encoding processes involve retrieval and vice versa. With respect to the present paradigm, integrative encoding requires some amount of retrieval (i.e., during study of BC, participants retrieve an overlapping AB pair to encode an integrated ABC representation), and flexible retrieval results in some degree of encoding (i.e., cross-episode binding). Nonetheless, the pattern of results observed here indicates a sharp difference in patterns of false memory before and after the associative inference test, which we have attempted to characterize in terms of the joint operation of cross-episode binding and flexible retrieval processes.

We have emphasized throughout that the current results fit well with an emerging theoretical picture in which various kinds of memory distortions are viewed as products of adaptive constructive processes (Schacter, 2012) that serve a range of cognitive functions, including simulating future experiences (Dudai & Carruthers, 2005; Schacter & Addis, 2007a, 2007b; Suddendorf & Corballis, 2007), solving problems (Howe, Garner, Charlesworth, & Knott, 2011), memory updating (Hardt et al., 2010; Hupbach et al., 2007, 2011; St Jacques et al., 2013), and extracting gist or meaning (Brainerd & Reyna, 1990; Koutstaal, 2006; Schacter, 2001; for recent reviews, see Howe, 2011; Howe et al., 2016; Newman & Lindsay, 2009; Schacter et al., 2011; Schlichting & Preston, 2015). Here we have focused on associative inference, which serves the adaptive function of allowing us to make new connections, and decisions about novel situations, based on flexibly retrieving and recombining information acquired in distinct though related prior experiences (Zeithamova, Schlichting, & Preston, 2012). Neuroimaging studies have linked the retrieval-based recombination process that supports associative inference to hippocampal function (Zeithamova & Preston, 2010; Zeithamova, Dominick, & Preston, 2012), but the nature of hippocampal contributions to the kinds of false memories associated with successful inference is unknown. Future research aimed at delineating the neural basis of the costs and benefits associated with flexible retrieval and recombination would enhance our understanding of the nature and functions of episodic memory.

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The Role of Cognition in Classical and Operant Conditioning

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For the past 35 years, learning theorists have been providing models that depend on mental representations, even in their most simple, deterministic, and mechanistic approaches. Hence, cognitive involvement (typically thought of as expectancy) is assumed for most instances of classical and operant conditioning, with current theoretical differences concerning the level of cognition that is involved (e.g., simple association vs. rule learning), rather than its presence. Nevertheless, many psychologists not in the mainstream of learning theory continue to think of *cognitive* and *conditioning* theories as rival families of hypotheses. In this article, the data pertaining to the role of higher-order cognition in conditioning is reviewed, and a theoretical synthesis is proposed that provides a role for both automatic and cognitively mediated processes. © 2004 Wiley Periodicals, Inc. J Clin Psychol 60: 369–392, 2004.

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Classical conditioning used to be viewed as a type of learning that involves the acquisition of elicited responses (i.e., responses, like the defensive eye blink, that are preceded reliably by an identifiable eliciting stimulus and that are experienced phenomenologically as automatic or reflexive). Similarly, instrumental (operant) conditioning was regarded as a type of learning that involves the acquisition of emitted responses (i.e., responses, like a wink of the eye, that can occur in the absence of reliable or well-defined antecedent stimuli and are experienced as voluntary). An implicit assumption of these old definitions was that what is acquired is a stimulus–response (S–R) association rather than a belief about the antecedents of an outcome (O, i.e., an expectancy).

When operant and classical conditioning are defined narrowly as types of learning in which S–R associations are formed, one can question whether they exist at all. Thus, Brewer (1974) provocatively titled his review of the conditioning literature, *There is No Convincing Evidence for Operant or Classical Conditioning in Adult Humans*. Contemporary conditioning theorists regard instrumental and classical conditioning as procedures that lead to behavior change (see Bolles, 1979). One advantage of defining instrumental and classical conditioning empirically in this way is that it is theoretically neutral. There can be no doubt but that instrumental and classical conditioning procedures reliably lead to changes in behavior. What are at issue are the inferred processes by which these changes are produced. In contrast to the early views expressed above, most contemporary learning theorists, even those who are mechanistically minded, regard classical conditioning as reflecting S–O (a.k.a. S–S) associations and instrumental learning as reflecting R–O associations (Rescorla, 1988, 1991; also see Kirsch, 1985).

In what he deemed his most important use of the term, Kuhn (1970) characterized paradigms as exemplars of seemingly permanent solutions to particular scientific problems, exemplars that serve as models for subsequent research in the area. Classical and instrumental conditioning are paradigms in this sense of the term. They have provided models for study a variety of phenomena, including phenomena that are central to clinical psychology. Outstanding examples of the application of classical conditioning procedures to clinical concerns include their use to induce (Watson & Rayner, 1920) and treat (Jones, 1924; Wolpe, 1958) phobic anxiety. The application of operant conditioning to clinical phenomena is evident in the creation and elimination of behaviors that might be symptomatic of psychiatric disorders (Haughton & Ayllon, 1965) and in the operation of token economies (Allyon & Azrin, 1968). Similarly, the behavior of rats that have been trained in a Skinner box on a variable ratio schedule of reinforcement can serve as an exemplar for analyzing and studying the behavior of humans standing in front of slot machines in Las Vegas. In the latter case, even the topography is isomorphic, including the lever that is pressed and the cup, in which the reinforcing pellets or chips are delivered, underneath it. However, this topographical similarity is the exception rather than the rule.

Kuhn (1970) noted that scientists can "agree in their identification of a paradigm without agreeing on . . . a full interpretation of it" (p. 44). This particularly is clear with respect to classical and operant conditioning. There can be no doubt that these procedures result in learning and that they have inspired treatments that have been shown to be effective in clinical trials. However, almost since their inception, their interpretation was the focus of intense theoretical debate. The central issue at the core of this debate was the following question: Are these phenomena automatic, mechanistic processes, in which higher-order cognition, if present at all, is merely an epiphenomenon (e.g., Hull, 1943; Pavlov, 1927; Skinner, 1953; Watson, 1913), or are they processes that are mediated cognitively (e.g., Bolles, 1979; Rotter, 1954; Tolman, 1932, 1948)? In recent years, a consensus has emerged that cognitive processes play an important role in learning (Miller

& Oberling, 1998; Rescorla, 1988, 1991). Nevertheless, it will be argued that there are some instances in which learning bypasses entirely higher-order cognition.

Contemporary mechanistic accounts of classical and operant conditioning typically involve the hypothesis that direct associations between the stimulus and outcome representations or the response and outcome representations are formed during conditioning. Obviously the outcome representations activated by these associations constitute a low-level form of outcome expectancy. Thus, there is now virtually universal agreement that conditioning involves the production of expectancies. The remaining theoretical differences about this issue concern the level of cognition and whether these cognitions can be represented adequately as simple associations. Higher-order cognitive alternatives to these mechanistic interpretations have centered on the concept of expectancy as more than just the activation of simple binary associations. An expectancy is a future-oriented belief; it is a belief that something will happen. Therefore, they also have been described as subjective probabilities (Rotter, 1954). From a cognitive perspective (e.g., Kirsch, 1985), instrumental learning situations produce expectancies that particular behaviors will produce particular outcomes (e.g., that food can be found in a particular location), and classical conditioning produces expectancies that certain stimuli will be followed by other stimuli (e.g., that food will be presented soon after the bell is sounded).

Expectancies have been portrayed as S–O associations, but not all S–O associations are expectancies. For example, a particular melody might function as a cue stimulus evoking a representation of a person in one's past. However, this representation is not an expectancy because it does not produce the belief that the person will appear because there also are present contextual cues that tell us that the person will not appear. In addition, there are types of S–R associations that can be regarded as expectancies (Kirsch, 1985). These are associations in which what is invoked by the stimulus is a representation of the response rather than the response itself. In this article, old and relatively recent data pertaining to the role of higher-order cognition in conditioning will be reviewed, and a theoretical synthesis that provides a role for both automatic and cognitively mediated processes will be proposed.

Data Indicating Higher-Order Cognitive Mediation

Tolman's Challenge to Mechanistic Explanations of Learning

Following the ascendancy of behaviorism, mechanistic explanations were prevalent among learning theorists (e.g., Hull, 1943). The major exception was Tolman (1932, 1948), whose research program was aimed largely at providing anomalies for image-free mechanistic learning theories. Tolman and his colleagues produced a large body of data supporting the hypothesis that rats running in mazes behaved as if they had access to information, built cognitive maps of the mazes, and expected to find food in particular locations (Bolles, 1979). The nature of Tolman's challenge to image-free mechanistic theory can be illustrated with the following examples.

Vicarious Trial and Error: "Catching On." Tolman's (1939) studies of vicarious trial and error (VTE) supported his idea of cognitive maps. VTE refers to the behaviors of "hesitating, looking back and forth" that rats engage in at choice points in a maze or discrimination task (e.g., choosing whether food is behind a black or white door) or before going one way or another in a maze. Rats seemed to display more VTEs when they *catch on* to which stimuli to pay attention to in a visual discrimination task or later "make sure of which stimulus is which." Tolman claimed this behavior indicates that the rat actively selects and compares stimuli in constructing a cognitive map of the task.

"Hypothesis" Experiments. Krech and Crutchfield (1948) defined learning as a "reorganization of the cognitive field" (p. 112). Tolman (1948) credited Krechecsky (Krechevsky, 1932) with designing experiments suggesting that rats develop systematic choices or *hypotheses* in progressing down difficult mazes. For example, rats try a variety of different behaviors, such as choosing right-handed or dark doors, which continues at above-chance levels until a solution to the maze or discrimination task is achieved. Such trial-and-error behavior was viewed as goal directed and was thought to reflect the development of tentative cognitive maps that are subject to revision as learning occurs.

"Place Learning." If a rat forms a cognitive map of a maze, then it should learn something about the relation among stimuli and have the ability to discriminate the *place* where food was located in its learning environment. Tolman, Ritchie, and Kalish (1946) found that rats that were trained to run a maze in a direct path for food, when blocked from running down the original path to the food and confronted with radiating paths, tended to run down the path that was in the direction of where the food was placed originally or selected a path that ran perpendicularly to the side of the room where the food was placed. Accordingly, the rats appeared to have learned the place where the reward was located, allowing the inference that they had formed a cognitive map of the maze.

In a direct test of the hypothesis that rats in mazes learn locations rather than responses, Tolman et al. (1946) alternately placed rats in one of two different start locations. Half of the rats (designated *place learners*) were reinforced for running to the same location, which required a different response (turning left or right) depending on which location they had started from. The others (designated *response learners*) were reinforced for making the same response, which took them to a different location depending on where they had started from. The logic of the study was that the place-learning task should be the easier of the two if rats learn locations, whereas the response-learning task should be easier if rats learn responses. In fact, all of the place learners learned their task within 8 trials, whereas after 72 trials, only 3 of 8 response learners had learned the task. Nevertheless, both tasks were learned by at least some of the subjects, making it clear that rats are opportunistic and are capable of either type of learning.

Response Prevention. Additional support for the cognitive map hypothesis comes from studies in which animals have been prevented from making a response for which they previously have been reinforced. In these studies, the use of an alternate response to reach the same reinforced location is interpreted as support of the cognitive hypothesis. For example, a rat prevented from turning right can traverse the right path of a T maze by turning left in a $\frac{3}{4}$ circle, until it is facing the goal box. Numerous studies have shown that learning occurred when responses were prevented in a variety of ways, including a) immobilizing rats by an administration of curare and testing learning when the drug was no longer active (Girden, 1942), b) crushing rats' motor nerves (Kellogg, Scott, Davis, & Wolf, 1940), and c) lesioning midbrain regions (Beck & Doty, 1957).

Latent Learning. The most controversial research paradigm was that purporting to demonstrate latent learning (e.g., Blodgett, 1929; Tolman & Honzik 1930). These experiments revealed that rats allowed to spend time in a maze without food reinforcement for reaching the goal showed little improvement in the time required to reach the goal or in the number of errors made. However, immediately after food was dispensed, the rats' error curves "dropped astoundingly," indicating that the rats had learned to navigate the maze even when they were not reinforced for doing so, but that this learning was not expressed behaviorally in the absence of reinforcement. In other experiments, it was shown that rats learned to locate food even when they were satiated (Thistlewaite, 1951) and when exposure to the maze is provided by having the rat ride in a small cart (McNamara, Long, & Wilke, 1956). According to Tolman, these sorts of demonstrations implied the existence of cognitive maps that formed during nonreinforced trials.

Brewer's (1974) Review

In 1974, William Brewer reviewed more than 200 "dissociation" studies, which he claimed distinguished between "conditioning theory" and "cognitive theory" (i.e., between mechanistic and higher-order cognitive interpretations of conditioning). According to Brewer, these studies of autonomic responses, motor responses, and complex responses (e.g., semantic generalization, conditioned meaning, verbal operant conditioning) in humans provide strong support for a cognitive interpretation. Below are the types of studies he reviewed and the logic by which they support a higher-order cognitive interpretation of conditioning.

Informed pairing: Simply informing participants about the CS–US (i.e., S–O) relation, with no actual pairing, results in acquisition of the CR and informing participants of response–reinforcement (i.e., R–O) contingencies produces instrumental learning.

Informed unpairing: After operant or classical conditioning, extinction can be produced by informing participants that the contingencies are no longer in effect, without any actual extinction trials.

Instructed conditioning: Participants instructed to produce a CR in response to a CS or to emit an operant response do so, without any actual conditioning trials.

Instructed nonconditioning: Participants are told to not produce a CR or operant response, following which they are given conditioning trials. Cognitive theory is supported when the response is not emitted.

Instructed extinction: After standard operant or classical conditioning, participants told to stop emitting the learned response do so.

Masking: Misleading instructions can be used to mask CS–US or response–reinforcement relations.

Awareness of contingency: Awareness of contingencies can be assessed and often is found to be correlated with the emission of conditioned responses, the cognitive hypothesis being that they will be emitted only by participants who are aware.

Modified contingency expectancy: After conditioning, participants are provided with information that produces expectations about contingencies that are different from those of the conditioning trials. For example, removing the shock electrodes in an aversive conditioning paradigm should eliminate the expectancy of shock.

Response expectancies: After conditioning in situations in which different responses are possible, participants' hypotheses about the response are assessed and correlated with their CRs. Alternatively, participants' response expectancies can be manipulated by the provision of verbal information.

Reinforcement expectancy: Participants' responses in a classical conditioning paradigm are correlated with information they have been given about the intensity of a strongly aversive US, or their responses are correlated with their hypotheses about the purpose of an ambiguous reinforcer (e.g., a spoken "hmm").

Brewer (1974) interpreted the data from these experiments as indicating "all the results of the traditional conditioning literature are due to the operation of higher mental processes, as assumed in cognitive theory, and that there is not and never has been any convincing evidence for unconscious, automatic mechanisms in the conditioning of adult human beings" (p. 27). A number of later reviews (Boakes, 1989; Lovibond & Shanks, 2002; Shanks & St. John, 1994), focusing primarily on data reported after Brewer's (1974) review, have echoed Brewer's conclusions regarding the failure of research to support mechanistic views of conditioning. Other reviewers, however, have reached alternate interpretations of the data (e.g., see Manns, Clark, & Squire, 2002; Weins & Öhman, 2002, for contrary conclusions), especially regarding the hypothesis that learning requires awareness of the contingencies (Morris, Ohman, & Dolan, 1998; Schacter, 1987).

Rescorla's Reviews of Classical and Operant Conditioning

Two influential reviews by Robert Rescorla, first of classical conditioning (1988) and then of instrumental conditioning (Rescorla, 1991), constituted further challenges to simple mechanistic views of learning. Rescorla's (1988) review entitled, ***Pavlovian conditioning: It's not what you think it is***, updated his earlier (1968) conceptualization of classical conditioning as involving the acquisition of information, as opposed to a "low level mechanical process in which the control over a response is passed reflexively from one stimulus to another" (Rescorla, 1988, p. 152). In his early experiments, Rescorla manipulated the contingency (i.e., correlation) between CSs and USs by presenting various combinations of CSs alone, USs alone, and contiguous CS–US pairings. What appeared to be important for the acquisition of conditioned responding was not the total number of contiguous pairings, but the overall relationship between the CS and US. According to this informational hypothesis, behavioral control is established when there is a positive or negative correlation between the CS and US, but not when there is no correlation. Accordingly, conditioned responses are elicited when the CS predicts that the US is likely to occur, but inhibited when the CS predicts that the US is less likely than the USs base rate. It was as if the animals were attuned to the informational value of the CS that established relations among events, just as is predicted by modern conditioning theories, in which cognitions (and expectancies in particular) play a central role.

According to this perspective, animals (including humans) are goal-directed, active information seekers who form rich and varied representations of their environment in the course of responding to an array of stimuli that come to be associated with one another in potentially complex ways. Conditioning is responsive to different properties of the stimuli that organisms encounter, to differences in associability among stimuli, and to the signaling properties of stimuli with respect to the relations that exist among other stimuli. Learning occurs when organisms are *surprised* (Kamin, 1968; Rescorla & Wagner, 1972) and modify their Pavlovian associations in response to the "discrepancy between the actual state of the world and the organism's representation of that state" (Rescorla, 1988; p. 153). Outcomes that are *surprising* provide new information, facilitate a rich representation of the world, and permit conditioning in as few as a single trial. This line of theorizing allows Tolman's concept of expectancy to be applied readily to contemporary associative learning theory. However, this insight did not result in an unquestioned triumph of higher-order cognition. Notably, simple expectancies can be captured in mechanistic formulations such as the Rescorla–Wagner model (e.g., Allan, 1993). Here subjects are assumed insensitive to direct correlations between cues and outcomes, but the asymptotic state of their mechanistic S–O formulation is difficult to distinguish from such a sensitivity. Moreover, at their current states of development, simple mechanistic models do some things (e.g., predict recency effects and cue competition) better than do higher-order cognitive models (e.g., López, Shanks, Almaraz, & Fernández, 1998). However, as each family of models evolves, it can be seen that there is little that each family (simple mechanism or higher-order cognition) cannot explain in principle. Contrasting families of models, rather than contrasting two specific models, is dangerous because improved models within each family are often in preparation (Miller & Escobar, 2001). Nevertheless, contrasting cognitive and mechanistic approaches encourages model development and highlights critical phenomena.

S–O Associations in Classical Conditioning

Historically (e.g., Hull, 1943), the associations that are learned during conditioning are between stimuli and responses. Thus, they are S–R associations. In simple cognitive theories (including virtually all contemporary conditioning theories), associations are hypothesized to be formed between representations of two stimuli (i.e., S–O associations), between representations of stimuli and responses (S–R), or between representations of responses and outcomes (R–O). S–O (or R–O) associations generally are interpreted as cognitions in which a stimulus (or response) comes to elicit an expectancy for the occurrence of another stimulus. Contemporary research suggests that expectancies in classical conditioning involve associations that vary in complexity. Simple S–O associations are inferred from conditioning procedures that result in conditioned responses following trial-by-trial presentation of a CS in some relationship with the US. More complex associations are inferred from procedures that provide the opportunity to combine two simple S–O associations (e.g., sensory preconditioning or second-order conditioning), and even greater complexity in associative structure is inferred from procedures that allow for the encoding for higher-order relationships (e.g., occasion setting; Miller & Oberling, 1998).

Simple S–O Associations. Credible evidence of S–O associations in classical conditioning is provided by experiments using *sensory preconditioning procedures* (e.g., Rizley & Rescorla, 1972). In this procedure, animals are presented first with contiguous pairings of two neutral stimuli such as a light and a tone. During this initial phase of the procedure, there is no behavioral evidence that the animals associate the two stimuli. Next, only one of the neutral stimuli, for example the tone, is paired with a US until it elicits a CR. When the stimulus that was not paired with a US (the light in this example) is presented alone in the final test phase, it too is found to elicit a CR. Note that because the light was never paired with a US, it was never experienced as contiguous with a response; therefore, it could not have gained eliciting properties as a result of an S–R association. The generally accepted explanation of the sensory-preconditioning phenomenon is that the animals acquire a latent tone–light association in the first phase and a tone–US association in the second phase. When the light is presented in the final test phase, the light evokes a mental representation of the tone, which in turn evokes an expectancy of the US and generates a CR. Although sensory preconditioning procedures provide strong support for simple S–O associations, they also show more complex cognitive processes. That is, these results indicate that rats are able to take two separate S–O associations (Light–Tone and Tone–US) and through a transitive inference process infer a third association (Light–US).

Additional evidence for this simple cognitive account of classical conditioning comes from *US devaluation procedures* (e.g., Holland & Rescorla, 1975). In the first phase, experimental and control groups are given standard CS–US pairings until CRs are observed. According to early mechanistic views of conditioning, Phase-1 conditioning reflects an S–R (CS–UR) association, but, according to both contemporary mechanistic and simple cognitive accounts, conditioning results in an S–O (CS–US) association, which can be thought of as a mental link between the representations of the stimulus events. To differentiate between the S–R and S–O accounts of conditioning, the value of the US is reduced (devalued) for the experimental group, but not for the control group during Phase 2 of the experiment. For example, in procedures in which the US is food, satiating the animal or conditioning a taste aversion to the US can achieve devaluation. The S–R account predicts that devaluing the US should have no effect on the CSs subsequent ability to elicit CRs because the US is not part of the association that controls conditioned responding. The S–O account, however, includes a *forward-looking* association and a mental representation of the anticipated US. The experimental group, therefore, should expect a devalued US and thereby respond less to the CS relative to the control group. Devaluation experiments involving classical conditioning paradigms as diverse as sexual-approach conditioning in birds (Holloway & Domjan, 1993) and food conditioning in rats (e.g., Holland & Rescorla, 1975) have provided evidence of S–O associations.

A wide range of simple classical conditioning procedures result in robust and reliable conditioned responding. This observation suggests the even simple associative learning may vary in complexity. In most conditioning procedures, the CS onset precedes US onset, but this temporal precedence is not necessary for conditioning to be seen. Conditioned responses occur when a CS is presented simultaneously with, or following, an aversive US in just a few trials, and sometimes after just one trial (e.g., Ayres, Haddad, & Albert, 1987; Mahoney & Ayres, 1976). Conditioning with just one CS–US pairing indicates that temporal contiguity is a sufficient condition for the establishment of a simple association between internal representations of a CS and US. Additional experience with repeated CS–US trials, however, provides the opportunity for the subject to obtain more information through contingency learning, information that may result in new learning involving the causal (Rescorla, 1988) and temporal (Miller & Barnet, 1993) relationship between stimuli or the behavioral expression of that information (Miller & Matzel, 1988).

Regarding variation in the behavioral expression of simple associations, expectancy theory does not have much to contribute. For instance, expectancy theory does not account for the form that the conditioned response takes in conditioning procedures. A number of studies indicate that the form of the conditioned response is influenced by the type of CS used. For example, Holland (1977) found that rats exposed to tone–food pairings developed a head jerk to the tone, whereas rats exposed to otherwise identical light–food pairings exhibited rearing behavior to the light. In addition, the length of the interval between the CS and the US can affect response topology in ways that are not always consistent with *rational* expectancies. With a short tone–food interval, Holland (1980) observed a startle response to the tone, whereas with a long tone–food interval, he saw orientation to the food hopper in response to the tone. Results like these support the view that the activation of conditioned responding involves biologically *preprogramed* (mechanistic) behaviors that are organized around important biological functions (e.g., feeding, mating, and defense) and are elicited by stimuli that anticipate the arrival of a US (Timberlake & Lucas, 1989). Moreover, recent studies have demonstrated that an abstraction such as response variability can be reinforced (e.g., Neuringer, 2002). Expectancy accounts of simple associative learning also fail to explain basic learning phenomenon that point to failures of behavioral expression rather than to failure to form an S–O association. One example is the *US preexposure effect*. Repeated exposure to a US retards the course of classical conditioning later when a CS is paired with the US in the same context. The US preexposure effect apparently occurs when the context accrues sufficient excitatory strength to block conditioning to the discrete CS (Randich & LoLordo, 1979). This result usually is interpreted as reflecting the failure of a subject to acquire a CS–US association because the added CS was redundant and provided no new information. However, Matzel, Brown, and Miller (1987) demonstrated that unreinforced exposure to the context after CS–US training reduced the US preexposure effect, suggesting that the CS–US associations indeed were formed during the training phase, but was not expressed in behavior.

Higher-Order Associations. Although the context can serve as a CS in a CS–US association when the context is the best predictor of the US, it also can enter a higher-order relationship with a CS–US relationship when the latter is embedded within the context. In one study, Bouton and King (1983) found that when a discrete CS was paired with a shock US in one context and extinguished in another context, the subjects showed conditioned fear of the discrete CS when retested in the original training context. This renewal of conditioned fear was not evident in subjects that experienced training and extinction in the same context. Thus, expectancies can be altered by the contexts in which test trials occur. Furthermore, independent assessment of the ability of the context to elicit conditioned responding indicated that demonstrable excitatory and inhibitory conditioning of the context was not necessary for the context to control fear to the discrete CS. These results were interpreted as supporting the role of the context as an occasion setter. Occasion setters are viewed as stimuli that provide information about when a CS–US contingency is in effect (Schmajuk & Holland, 1998). Occasion setters are not restricted to contextual stimuli, but can occur in other procedures that provide two-level hierarchical arrangements of discrete events. For example, in the *serial feature–positive discrimination procedure*, a discrete stimulus (the feature) precedes another discrete stimulus (the CS) when the latter is paired with a US, but not when it is presented without a US. Evidence suggests that the discrete CS enters a simple association with the US, but its expression is dependent upon a conditional cue function of the feature. Interestingly, when the feature is presented simultaneously with the other CS instead of preceding it, simple associations appear to develop between the feature and the US rather than higher-order conditional associations (Ross & Holland, 1981). Moreover, just as occasion setters can disambiguate otherwise ambiguous CSs, so too can higher-order occasion setters disambiguate otherwise ambiguous first-order occasion setters (Arnold, Grahame, & Miller, 1991).

R–O and S–(R–O) Associations in Operant Conditioning

R–O Associations. Traditional cognitive explanations of behavioral change in instrumental conditioning procedures posit the formation of an association between an emitted response and an outcome that has followed it in the past (R–O; Bolles, 1979). These R–O associations are the basis of outcome expectancies, that is, they support expectancies that an emitted response will lead to a particular outcome. Devaluation procedures, from which evidence of S–O associations in classical conditioning was derived, also have been adapted for instrumental conditioning procedures, in which they provide evidence of R–O associations. In these instrumental learning studies, an outcome (e.g., sucrose solution or food pellets) can be devalued by pairing it with the administration of a substance or toxin that creates a food aversion, for example. In one study using simple schedules of reinforcement (i.e., a single manipulandum), rats that learned to press a lever for food on a ratio schedule of reinforcement (Phase 1) and then experienced a devaluation of the reinforcer outcome in the home cage (Phase 2) responded less on the lever in a subsequent extinction test (Phase 3) compared to rats that did not experience the devaluation (Dickinson, Nicholas, & Adams, 1983). Rescorla and colleagues have used concurrent schedules of reinforcement such that in the first phase rats were trained to emit one response for one type of outcome (e.g., food pellets) and a second response for another outcome (e.g., sucrose) followed by home–cage devaluation of one of the outcomes. These studies also have demonstrated consistently a reduction in responding for the devalued outcome during the test phase (e.g., Colwill & Rescorla, 1985a; 1985b). Such results are not predicted by an S–R mechanistic account of conditioning because the S–R association that presumably was selected in Phase 1 should have been unchanged by the home–cage devaluation of the outcome, and as a result, responding should have persisted in the Phase-3 test. The S–O mechanistic and cognitive accounts, however, explain it in terms of the devaluation procedure reducing the value of the reinforcer outcome and consequently operant responding motivated by the expectation of the outcome responding should decline.

S–(R–O) Associations. In his review of associative relations in instrumental learning, Rescorla (1991) made a strong case for the establishment of associations that went beyond simple binary stimulus–response or response–outcome associations. In addition to S–R and R–O associations, Rescorla posited S–(R–O) associations, which can be understood as expectancies of particular outcomes (O) when certain responses (R) are emitted in the presence of an occasion setting (discriminative) stimulus (S). For example, in the typical operant conditioning procedure with rats, the discriminative stimuli of an operant chamber occasions the expectation that responding will lead to imminent arrival of food. Higher-order cognitive accounts describe instrumental conditioning as resulting in motivated action directed toward an expected goal (outcome), whereas contemporary mechanistic theories describe instrumental conditioning as resulting in motivated action because it was reinforced previously. In both cases, S–(R–O) associations are at the heart of modern conditioning theory. In contrast, prior S–R mechanistic explanations speculated that the reinforcing outcome selects a stimulus–response association, but does not become a component of the association—the prior reinforcement history simply compels the rat to respond in the presence of distinctive stimuli.

Cognitive Mediation in Clinical Research

Much of the data pertaining to the debate about higher-order cognitive mediation is derived from basic research on animal behavior. However, there also have been studies of clinical phenomena in which this issue has been addressed. In particular, studies of systematic desensitization and placebo analgesia have evaluated automatic and cognitive accounts of these phenomena.

Systematic Desensitization. Systematic desensitization (Wolpe, 1958) is a treatment for phobic anxiety that was inspired by Clark Hull's (1943) theory of conditioning. Phobic anxiety is posited to be a classically conditioned response. The idea behind the therapy is to associate the anxiety-arousing cues (the CS) with a new CR, one that is incompatible with anxiety (e.g., relaxation). Another explanation of systematic desensitization is extinction. According to the extinction hypothesis, in the absence of reinforcement by an aversive event (i.e., a US), repeated (or prolonged) exposure to the anxiety cues causes the CR (anxiety) to extinguish. One nice feature of the extinction hypothesis is that it works for other exposure-based treatments (e.g., flooding), as well as systematic desensitization.

The idea that exposure treatments are due to automatic conditioning processes has been tested in three studies (Gauthier, Laberge, Dufour, & Fevre, 1987; Kirsch & Henry, 1977; Southworth & Kirsch, 1988). In the first of these, Kirsch and Henry (1977) compared the effects of systematic desensitization and two credible expectancy modification procedures. One of these procedures was designed specifically to rule out conditioning hypotheses. In an *operant desensitization* condition, visualizations of anxiety-related scenes were paired with painful electric shocks, which subjects were told would "punish the anxiety." This expectancy modification procedure was as effective as standard systematic desensitization in reducing public-speaking fear. Because aversive stimuli are assumed to be the USs leading to the acquisition of fear as a CR, the substantial degree of fear reduction produced by *operant desensitization* cannot be accounted for by extinction or counter conditioning. Furthermore, because the addition of the electric shock was the only procedural difference between *operant* and traditional desensitization, it is reasonable to suspect that the effects of the two procedures were due to a common causal mechanism. Substantial correlations between pretreatment ratings of treatment credibility and treatment-outcome measures suggest that expectancy modification was the common causal agent.

A second test of mechanistic explanations of exposure treatments was a study of the effects of *in vivo* exposure on agoraphobia (Southworth & Kirsch, 1988). Over a two-week period, participants in this study were give ten sessions of *in vivo* exposure, during which they were asked to walk away from their homes until they became anxious and then to turn around and return. Half of the participants were told that the purpose of this was to lower their anxiety. The others were told that the purpose was to assess their anxiety and that treatment would not begin until after the two-week period. Clients provided with therapeutic expectancies showed substantially greater improvement and improved more rapidly than those who were led to believe that *in vivo* exposure was for the purpose of assessment, even when the distance and time walked was equated between the two groups. These data indicate that the therapeutic effects of *in vivo* exposure can be suppressed by disguising its therapeutic intent. These data were replicated by Gauthier et al. (1987) in a study of dental phobia. The findings of both of these studies are problematic for not only traditional S–R models of learning, but contemporary conditioning theories as well.

Placebo Effects. According to classical conditioning models of placebo effects (Ader & Cohen, 1991; Herrnstein, 1962; Wickramasekera, 1980), active medications are USs and the vehicles in which they are delivered (i.e., the pills, capsules, syringes, etc.) are CSs. The medical treatments that people experience during their lives constitute conditioning trials, during which the vehicles are paired with their active ingredients. These pairings endow the pills, capsules, and injections with the capacity to evoke therapeutic effects as CRs.

There are a number of studies demonstrating that classical conditioning procedures can enhance placebo analgesia (Voudouris, Peck, & Coleman, 1985, 1989, 1990). The placebo effect was enhanced in these studies by surreptitiously lowering the intensity of a pain stimulus whenever a part of the body treated with a placebo anesthetic was stimulated. The lowered intensity of the pain stimulus was the US and the placebo was the CS. This procedure increased the pain-reducing effect of the placebo, when subsequently it was tested without lowering the intensity of the pain stimulus.

Although the Voudouris et al. (1985, 1989, 1990) studies convincingly demonstrated conditioned enhancement of placebo pain relief, they did not discriminate between S–O mechanistic and higher-order cognitive interpretations of this phenomenon. In a follow up to these studies, Montgomery and Kirsch (1997) investigated the effect of verbal information on this conditioning procedure. Recall that in the initial studies, the lowering of the intensity of the pain stimulus was done surreptitiously. Participants did not know that the intensity was lowered, and they therefore attributed the reduction in pain to the effect of the supposed topical anesthetic. Montgomery and Kirsch replicated this effect, but they also included an *informed-pairing* control condition in which participants were told that the intensity of the stimulus was being lowered. As in the condition replicating the original studies, participants in the informed-pairing condition were given trials in which reduced pain was paired with the application of a placebo. However, they also were given accurate information about how the reduction in pain was being produced. This verbal information completely reversed the effect of conditioning trials on the placebo response, which is not anticipated by even modern conditioning theories. In addition, regression analyses indicated that the effects of conditioning trials were mediated completely by participants' verbally rated expectancies.

In contrast to these data, conditioning with various drugs as USs has been reported to result in CRs that are the opposite of the URs (Siegel, 1983). For example, conditioning trials with morphine as the US produces increased sensitivity to painful stimuli as a response to the CS, and conditioning trials with tranquilizers like chlorpromazine as the US produces increased activity as a response to the CS. These data have been interpreted as indications of compensatory CRs, failures to identify accurately the actual US and/or UR when these drugs are administered (Donahoe & Palmer, 1994; Eikelboom & Stewart, 1982; Siegel, Baptista, Kim, McDonald, & Weise-Kelly, 2000). On one hand, these interpretations render this phenomenon consistent with current conditioning theories. On the other hand, regardless of interpretation, these data are inconsistent with a classical conditioning model of placebo effects. If increased pain sensitivity and activation are the CRs that are acquired when morphine and chlorpromazine are administered, then the pain-reducing effect of placebo morphine and the sedating effects of placebo tranquilizers cannot be CRs produced by the same mechanism. These data especially are important because the effects of placebo analgesics and tranquilizers are particularly well established.

In contrast, these data are not inconsistent with expectancy accounts of placebo effects because those effects are consistent with people's expectations. Most people expect morphine to reduce pain and tranquilizers to decrease activity, and the placebo effect is consistent with those expectations. Eikelboom and Stewart provided an account of how both mimetic and compensatory conditioned drug effects could arise from S–O mechanistic conditioning, but it also is possible that expectancy produces mimetic conditioned drug effects that are strong enough to override the conditioned effects of compensatory drug effects. In addition, expectancies can produce two conflicting response tendencies: an automatic mimetic response and a voluntary compensatory response. For example, in addition to an automatic response decrement, placebo alcohol can produce a voluntary compensatory response that is associated with the motivation to resist the expected deleterious effects, especially when the potential outcome is highly consequential (Vogel-Sprott & Fillmore, 1999).

Data Indicating Automatic Conditioning

Taken together, the data reviewed above provide clear evidence of cognitive mediation in both classical and operant conditioning. Other data, however, reveal conditioning phenomena that do not appear to be mediated cognitively. The data reviewed in this section seem to be explained more easily by automatic S–R processes and are consistent with a stimulus substitution model of conditioning (Pavlov, 1927).

Evaluative Conditioning

Evaluative conditioning occurs when a neutral conditional stimulus (CS) is paired with an affectively valenced, liked or disliked, unconditional stimulus (US) and results in a transfer of affect from the US to the CS (see Baeyens, Eelen, Crombez, & Van den Bergh, 1992). Two aspects of evaluative conditioning have been thought to distinguish it from traditional autonomic conditioning: (a) conditioning without awareness (Baeyens, Eelen, & Van den Bergh, 1990; Martin & Levey, 1978) and (b) resistance to extinction (Baeyens, Eelen, Van den Bergh, & Crombez, 1989). Although mechanistic interpretations of much of the evaluative conditioning literature have been challenged (e.g., Davey, 1994; Field & Davey, 1999; Lovibond & Shanks, 2002; Shanks & St John, 1994), studies of the evaluative conditioning of taste properties of odors (e.g., Stevenson, Prescott, & Boakes, 1995; Stevenson, Boakes, & Prescott, 1998) have yielded more convincing evidence of conditioning without awareness, leading Lovibond and Shanks to speculate that the gustatory system possesses special learning characteristics that operate outside of awareness. Studies of learning during anesthesia in nonhuman animals (e.g., Rabin & Rabin, 1984) also provided support for the possibility of learning without awareness.

S–R Persistence Following Devaluation

Recall that devaluation procedures were cited as support for a cognitive account of instrumental (as well as classical) conditioning. Specifically, devaluation of a stimulus that had been used to reinforce a particular response resulted in reduction of the response in simple (Dickinson, Nicholas, & Adams, 1983) and concurrent (Colwill & Rescorla, 1985a) schedules of reinforcement. However, the equally important observation that outcome devaluation does not always affect instrumental performance provides support for an S–R mechanistic interpretation of instrumental learning. When rats are trained on simple schedules of reinforcement, outcome devaluation reduces responding maintained on ratio, but not interval schedules of reinforcement (e.g., Dickinson et al., 1983). Thus, depending on the schedule of reinforcement, instrumental performance can be autonomous of its outcome. One explanation is that simple interval schedules fail to establish R–O associations because, unlike ratio schedules, the relationship between responding and rate of outcomes received is weak at moderate and high rates of responding (Dickinson, 1989). A seemingly contradictory finding is that interval schedules are effective in producing behaviors that are sensitive to outcome devaluation when they are used in concurrent schedules of reinforcement (Colwill & Rescorla, 1985a). Dickinson (1989) posited that the availability of different behaviors and their consequent outcomes in concurrent procedures provide the opportunity to learn strong response–outcome correlations. Thus, when there is sufficient opportunity to learn response–outcome correlations (e.g., simple ratio schedules and concurrent schedules), R–O associations control performance. However, when the response–outcome correlation is weak (e.g., simple interval schedules), S–R associations maintain performance.

Persistence following devaluation also has been observed in procedures that typically yield expectancy-based performance. Adams (1982) demonstrated that extended training on a ratio schedule renders outcome devaluation ineffective. Behavioral autonomy following extended practice appears not to be a result of the increase in the number of training reinforcers (Adams, 1982; Dickinson,1989), nor of simple repetition. Repetition of responding, for example, does not preclude expectancy-based performance when different behavior–outcome relationships are experienced in the same session (i.e., concurrent schedules; Colwill & Triola, 2002) or in alternating sessions (Colwill & Rescorla, 1985a; 1988). Dickinson (1989) had noted that the change from goal-directed expectancy to behavior that is autonomous of its outcomes might reflect changes in the response–outcome correlations that are experienced with extensive training in simple ratio schedules. Thus, the variability in rate of responding (and the consequent rate of reinforcement) that occurs early in training is reduced considerably with extended training (Dickinson, 1985). These observations suggest that, in the absence of consistent behavior–outcome correlations, instrumental performance is maintained by an S–R mechanistic process and is autonomous of the consequent outcomes.

Interestingly, a recent study suggests that behavioral autonomy from outcome status also can be observed in situations with consistent behavior–outcome correlations. Using the concurrent procedure, Dickinson, Wood, and Smith (2002) found that devaluation reduced instrumental performance when the outcome was food, but not when it was ethanol. This result is consistent with the view that alcohol-seeking behavior is maintained by S–R mechanistic habitual responding rather than goal-directed expectancy.

There also is evidence that cognitive and mechanistic processes can contribute to the same action. Although the response reduction after devaluation reported by Colwill and Rescorla (1985a, 1985b) strongly supports an explanation in terms of cognitive R–O associations, there are aspects of their data that are consistent with an S–R mechanistic interpretation. Devaluation of the reinforcing stimulus decreased the associated response, but it did not eliminate it altogether after brief or extended training. So why did the rats work at all to receive an outcome that had been devalued through taste aversion? A plausible explanation is that an S–R association maintained the residual responding (Colwill & Rescorla, 1985b; Dickinson, 1989; Nevin & Grace, 2000).

Resistance to Change and Excessive Behavior

Persistence of instrumental performance following outcome devaluation indicates that under some circumstances instrumental performance is resistant to post-conditioning changes in the consequent outcome. Resistance to change, or what Nevin has termed *behavioral momentum*, also is observed when response contingencies are altered in multiple schedules of reinforcement (e.g., Nevin & Grace, 2000). In this procedure, two or more schedules of reinforcement are correlated with distinctive stimuli. For example, responding is reinforced in the presence of one stimulus under a VI – 1 min schedule and in the presence of a second stimulus under a VI – 3 min schedule. The two stimulus–response–outcome contingencies are presented successively and separated by a brief time-out period. When noncontingent food is introduced in the time-out periods to disrupt performance, behavior under the richer schedule (VI – 1 min) is disrupted much less than behavior under the leaner schedule (VI – 3 min). This differential resistance to change also is observed when reinforcer value, rather than rate, is varied across stimulus situations. Nevin has shown that the overall rate of responding is determined by the response–outcome contingency, but resistance to change is determined by the overall rate of outcomes obtained in the stimulus situation (Nevin, Tota, Torquato, & Shull, 1990). The latter result was interpreted as indicating that resistance to change is modulated by a simple association between the discriminative stimulus (S^D) and the outcome. Good evidence for S^D–O associations come from transfer tests used to assess R–O associations (Colwill & Rescorla, 1988; Colwill & Triola, 2002). For example, an S^D present during one outcome will enhance performance of another response if it was trained with the same outcome, but not if it was trained with a different outcome. This result indicates that in instrumental conditioning procedures both R–O expectancy and simple S^D–O associations control instrumental performance (Colwill & Rescorla, 1988).

A dramatic example of the mechanistic control of behavior comes from studies demonstrating that a consistent history of intermittent delivery of reinforcers in a discriminative context (S^D) can generate bizarre and excessive (*adjunctive*) behavior in animals and humans (Falk, 1994). For example, when food-deprived rats are reinforced with food pellets on a fixed-interval schedule, they develop concurrent, excessive drinking (polydipsia). The consistent intermittency of food-pellet delivery is the important factor since schedule-induced drinking also occurs when the food is presented in the absence of an R–O contingency (i.e., a fixed time schedule). This procedure for producing chronic and excessive oral drug self-administration under strong stimulus control has been proposed as an animal model of drug abuse (e.g., Falk & Tang, 1988). Discriminative control of drug intake, whether it reflects resistance to change in the face of the disruptive effects of excessive drug use (Nevin & Grace, 2000) or schedule-induced self-administration (Falk & Tang, 1988), may be analogous to the phenomenon of relapse in humans. Relapse in recovering addicts is much more likely when drug abusers return to a situation previously associated with drug-taking behavior (Brownell, Marlatt, Lichtenstein, & Wilson, 1986) and is much less likely when drug users are removed suddenly from their drug-taking context to a radically different context. An example of the latter is that a very small percentage of soldiers who became addicted to heroin in Vietnam relapsed within three years of their return home (Robins, Helzer, Hesselbrock, & Wish, 1980; however, see Fish, 1998, for an alternative explanation).

Second-Order Conditioning

Evidence of S–R associations also is provided by studies of second-order conditioning procedures. In this procedure, animals are conditioned with standard pairings of a CS (e.g., a tone) and a US pairings (the first-order conditioning phase). In the next phase (the second-order conditioning phase), a second CS (e.g., light) is paired with the first-order CS that was conditioned in Phase 1, but with the US omitted. The result is that this second CS elicits CRs. A cognitive account explains the conditioning of the second-order light CS in much the same way it explained the sensory preconditioning experiment: After sufficient light–tone pairings, the light arouses a mental representation of the tone, which in turn arouses the expectation of a US, thereby generating a CR. However, unlike the sensory preconditioning results, an S–R association also can account for second-order conditioning; the second-order light CS may be conditioned as a result of contiguous pairings with the CR that was elicited by the tone CS. To differentiate these two accounts, Rizley and Rescorla (1972) extinguished responding to the tone after the second-order conditioning phase and subsequently presented the second-order light CS in a final test phase. The cognitive account predicts that extinction of the first-order tone CS should abolish responding to the second-order CS because the former should no longer arouse an expectancy of the US. The results failed to confirm this prediction, suggesting that the second-order conditioning, at least in this situation, was most likely a result of an association between the second-order CS and the CR elicited by the first-order CS. The ineffectiveness of US devaluation in reducing the CR-eliciting properties of second-order USs also provides evidence of S–R associations (Holland & Rescorla, 1975).

Conditioned Taste Aversions and Flavor Preferences

In the taste-aversion conditioning procedure, a taste stimulus (CS) is paired with a substance that produces gastrointestinal malaise (US). Because of this pairing, the subject avoids consumption of the taste the next time it is encountered. Why? One explanation is that the taste is avoided because there is an expectation that consumption of the taste will cause gastrointestinal malaise (Rozin & Zellner, 1985). That is, behavior is guided by the information (danger) provided by the taste CS. An expectancy explanation of taste-aversion learning, however, does not account for the affective and hedonic changes that have been observed in humans and animals (Berridge, 2000). Pleasant tastes (e.g., sugar solutions) elicit positive affective facial and ingestive responses, but when paired with gastric malaise, there is a shift to negative defensive reactions (e.g., gaping and head shaking) that typically are seen with distasteful substances. In one study (Pelchat, Grill, Rozin, & Jacobs, 1983), a sweet taste was paired with treatments to produce upper-intestinal discomfort (LiCl toxicosis), lower-intestinal discomfort (lactose ingestion), or pain (electric shock). All three treatments led to avoidance of the taste stimulus, but only LiCl shifted the hedonic reaction to the taste stimuli from *like* to *dislike*. Whereas avoidance of the taste stimuli in the absence of shifts in hedonic quality may reflect an expectancy of the negative consequences of ingestion, the observed changed in the sensory evaluation of an initially preferred taste suggests an S–R automatic, noncognitive conditioning process (Rozin & Zellner, 1985).

Similar results have been observed in studies of conditioned flavor preferences. Rats learn to prefer a novel, mildly sweet flavor CS paired with intragastric infusions of nutrients (e.g., glucose) over another mildly sweet flavor CS paired with intragastric infusions of water (e.g., Drucker, Ackroff, & Sclafani, 1994). Evidence suggests that such conditioned flavor preferences sometimes reflect an increased positive hedonic value instead of an anticipation of the positive consequences of ingestion. For example, flavor preferences conditioned with intragastric nutrient infusion as a US are very resistant to extinction in both deprived and nondeprived animals (Drucker et al., 1988), enhance sham-feeding responses (Myers & Sclafani, 2001a), and shift taste reactivity toward responses typically seen with higher concentrations of sweet solutions (Myers & Sclafani, 2001b). However, as with conditioned taste aversions, not all conditioned flavor preferences appear to be mediated by changes in the hedonic properties of the flavors. When the CSs are initially unpalatable flavors rather than mildly sweet flavors, robust conditioned flavor preferences are not accompanied by enhanced positive hedonic reactions (Myers & Sclafani, in press). Together, these taste-aversion and flavor-preference studies suggest that the modulation of food choice through conditioning reflects two different processes—anticipation of the consequences of ingestion and a more mechanistic change in sensory evaluation (Rozin & Zellner, 1985; Myers & Sclafani, in press).

Conditioning with Subliminally Presented CSs

Additional evidence of automatic conditioning without awareness comes from experiments where the CSs are visual stimuli presented subliminally. Visual stimuli presented briefly (less than 300 msec) and immediately followed by a masking visual stimulus are not perceived consciously, yet are evaluated effectively as measured by explicit ratings or elicited autonomic responses. Words with extreme negative (e.g., *cancer*) or positive (e.g., *friend*) valence, when presented subliminally, elicit affective reactions that habituate with repeated subliminal presentations (Dijksterhuis & Smith, 2002). Phobic individuals presented with subliminal fear-relevant stimuli show increased electrodermal responses (Öhman & Soares, 1994). Because participants are not aware of visually masked stimuli if used as a CS in a Pavlovian conditioning paradigm and conditioned responding is observed, then conditioning will have occurred necessarily without the awareness of a

CS–US contingency. Several studies have reported conditioned responding to masked visual CSs despite lack of awareness of the CS or the CS–US contingency. For example, conditioned electrodermal responses have been observed when masked stimuli of *angry* faces or *threatening* animals were paired with an unpleasant shock US in nonphobic participants (Esteves, Parra, Dimberg, & Öhman, 1994; Öhman & Soares, 1998; Parra, Esteves, Flykt, & Öhman, 1997) . Masked stimuli that did not have an initial negative evaluation (e.g., pictures of a happy face or a flower), however, did not result in a conditioned electrodermal response (Öhman & Soares, 1998). Thus, it appears that automatic conditioning occurs only to preattentively perceived CSs that evoke some affective negative evaluation.

Conditioned Immunosuppression

Robert Ader (1985) conducted experiments that are procedurally similar to taste-aversion conditioning, but instead of conditioning a taste aversion, he conditioned immunosuppression. In this procedure, rats are allowed to consume a sweet tasting solution (CS) just before cyclophosphamide injections (US). The drug cyclophosphamide reduces the number of T-lymphocytes produced by the immune system of rats. When rats drink the taste CS in the absence of the drug, they show a reduced number of T-lymphocytes relative to control animals that received noncontingent pairings of the sweet taste and drug. The observation of conditioned immunosupression suggests that a normal, adaptive physiological function can be brought under control of an arbitrary stimulus such as a taste. It seems highly unlikely that rats could expect immunosupression or even have any representation of the phenomenon.

Conditioning in Simple Organisms

Evidence for automatic S–R associations also are provided by studies of conditioning in simple animals. For example, *Aplysia californica*, a large marine snail with a relatively simple nervous system of only a few hundred neurons, shows learned behavior in both classical and instrumental conditioning procedures (Carew, Hawkins, & Kandel, 1983). This simple invertebrate has an external gill that is withdrawn reflexively into a body cavity for protection. A mild tactile stimulus applied to tissue surrounding the gill produces little defensive gill withdrawal. However, after pairing this mild stimulus (CS) with a strong shock to the tail (US), the CS reliable elicits a robust gill-withdrawal response. It seems unlikely that an organism with such a simple nervous system would be capable of forming representations.

Conclusions

Data concerning two interpretations of classical and operant conditioning have been reviewed. One is the hypothesis that conditioning is an S–R mechanistic process in which expectancy and other cognitive factors are, at best, epiphenomena. From this perspective, conditioning trials produce conditional responses and perhaps expectancies, but there is no causal relation between expectancy and response. The other is cognitive theory, including S–O associations, according to which expectancy is hypothesized to mediate the effects of conditioning. From this perspective, conditioning trials produce expectancies, and it is the expectancy that produces the response.

Most traditional operant and classical conditioning phenomena can be explained by either S–R mechanistic or cognitive accounts (both simple S–O and higher order), and experiments that have been designed as critical tests of these rival hypotheses have yielded mixed results. On one hand, this review indicates abundant data disconfirming mechanistic S–R hypotheses and supporting cognitive interpretations in some situations. These data have led contemporary conditioning theorists to abandon earlier formulations in favor of approaches that are more consistent with cognitive theories. On the other hand, there are data indicating the occurrence in other situations of conditioned associations that are unlikely to be mediated cognitively. How can these data be reconciled?

Early debates about the nature of conditioning were based on the premise that it was exclusively either cognitive or mechanistic. A less parochial interpretation suggests that there are two types of conditioning processes, those that are mediated cognitively and those that are not. In addition, there appear to be learning processes that are not based on conditioning at all (e.g., learning by observation or through verbal communication). These aspects of the data suggest an unparsimonious proliferation of unconnected processes.

Conversely, there are data indicating important commonalities between conditioning and other forms of learning. Two examples of these are the informed-pairing and informed-unpairing studies reviewed by Brewer (1974). Participants in these studies who had been informed verbally about environmental contingencies behaved as they would have had they actually undergone conditioning or extinction trials. Similarly, verbally induced expectancies have been shown to produce patterns of responding that emulate various schedules of reinforcement (e.g., Wasserman & Shaklee, 1984). These data suggest a coordinated system of learning processes rather than a proliferation-independent learning mechanisms.

Cognition in complex organisms evolved from and incorporated more simple learning processes. Clearly, classical and operant conditioning of simple S–R associations are among the most basic processes. However, behavioral flexibility requires greater complexity. Thus, more complex organisms have evolved the ability to form representations (i.e., based on both R–O and S–O relationships) via conditioning procedures, as well as the ability to infer those relationships from other sources of information. It can be speculated that the more complex the organism, the smaller the role of automatic conditioning processes and the greater the role of representational cognition. This speculation is consistent with the data reviewed in this article showing that the provision of information generally overrides the effect of conditioning trials, especially in human participants, but also in laboratory animals.

Finally, the construct of *set* may bridge the apparent divide between automatic conditioning processes and representational cognitive processes. Simple S–R associations may be thought of as response sets. They are functionally anticipatory in that they prepare the organism for efficient automatic emission of the response when the appropriate stimulus conditions are encountered (Kirsch & Lynn, 1999). Similarly, S–O and R–O associations can be thought of as stimulus sets that prepare the organism to perceive environmental stimuli in particular ways. Examples include placebo effects and the effect of set on perceptions of ambiguous stimuli. Explicit expectancies are consciously accessible stimulus and response sets. Stimulus and response sets that are not consciously accessible could be thought of as implicit expectancies, although in doing so one might risk the danger that they could be reified gratuitously as a higher-level construct implying unconscious cognitions. Classical and operant conditioning are two of the means by which response sets are formed, but they also can be acquired vicariously though observation and through the provision of verbal information.

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Effect of Circadian Phase on Memory Acquisition and Recall: Operant Conditioning vs. Classical Conditioning

Source 2

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Abstract

There have been several studies on the role of circadian clocks in the regulation of associative learning and memory processes in both vertebrate and invertebrate species. The results have been quite variable and at present it is unclear to what extent the variability observed reflects species differences or differences in methodology. Previous results have shown that following differential classical conditioning in the cockroach, *Rhyparobia maderae*, in an olfactory discrimination task, formation of the short-term and long-term memory is under strict circadian control. In contrast, there appeared to be no circadian regulation of the ability to recall established memories. In the present study, we show that following operant conditioning of the same species in a very similar olfactory discrimination task, there is no impact of the circadian system on either short-term or long-term memory formation. On the other hand, ability to recall established memories is strongly tied to the circadian phase of training. On the basis of these data and those previously reported for phylogenetically diverse species, it is suggested that there may be fundamental differences in the way the circadian system regulates learning and memory in classical and operant conditioning.

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Introduction

In the past decade, several studies have indicated that circadian clocks may have varied effects on learning and memory. In some cases, the ability to form a memory may be independent of circadian phase, but phase may function as a contextual cue (time-stamping) such that recall and performance are better at 24-hour intervals following learning as demonstrated in hamsters [1] and rats [2–4]. In other cases, recall appears to be largely independent of the phase of testing, but memory acquisition or consolidation may depend on the circadian phase of training as shown in mollusks [5,6], insects [7–9], fish [10], and mice [11,12].

There have also been reports that disruption of the circadian system by phase-shifting ("jet-lag") can impair memory in rats [13–15] and that internal phase relationships are important for learning in humans [16]. Finally, two recent studies presented data indicating that abolition of circadian cycling in hamsters impairs performance in a declarative memory task [17] and that ongoing circadian oscillations in the hippocampus are necessary for long-term memory stability following fear conditioning in mice [18].

In summary, it seems clear that the circadian system can have widespread effects on various aspects of learning and memory including acquisition, retention, and recall; however, at this point numerous questions remain both about the mechanisms by which the circadian system regulates these processes and about the functional/adaptive significance of this novel feature of circadian organization. One of the problems with sorting out the various results to come to clear understanding of underlying principles of the circadian system's role in associative memory formation is that the experiments have used various species, various conditioning paradigms, and various stimuli for reinforcement. Thus it is unclear whether differences in results reflect fundamental differences in the role of the circadian system in learning and memory or, alternatively, simply reflect a "hodge-podge" of species and methodological differences that obscures any underlying general principles.

The cockroach may be an excellent model for addressing these issues. Cockroaches can be trained both by classical and operant conditioning paradigms using virtually identical stimuli for reinforcement [7,19,20]. Thus we eliminate much of the variability that plagues comparisons among published studies and are able to focus on differences in circadian regulation of various forms of associative memory. Using a differential classical conditioning protocol it has been shown that the circadian system regulates olfactory learning and memory in the cockroach *Rhyparobia (Leucophaea) maderae* [7]. In this study, the effect of training and testing at different circadian phases on performance in an odor discrimination test was investigated. When the cockroaches were allowed to choose between two odors (peppermint and vanilla), naïve animals showed a clear preference for vanilla at all circadian phases. The results indicated there was no circadian modulation of initial odor preference or ability to discriminate between odors. Training involved differential classical conditioning in which peppermint odor was associated with a positive unconditioned stimulus (US+) of sucrose solution and vanilla odor was associated with a negative unconditioned stimulus (US−) of saline solution. It was found that cockroaches conditioned in the early subjective night showed a strong preference for peppermint and retained the memory for at least two days. Animals trained and tested at other circadian times (CT) showed significant deficits in performance for both short-term and long-term memory. At CT 2 (early subjective day) the deficit was profound and animals that had been trained at this phase were behaviorally indistinguishable from naïve, untrained animals. In contrast, recall of a learned memory was independent of the phase of testing – animals trained at CT 14 were able to recall at CT 2.

In the present study we show that *R. maderae* can also be trained via an operant conditioning protocol that utilizes the same sensory cues that were used for classical conditioning. Further we show that, unlike classical conditioning, with operant conditioning animals are able to acquire memories at any circadian phase but that their ability to recall long-term memories is tied to the phase of training. The results indicate that the impact of circadian regulation of learning and memory is strongly dependent on the form of training.

Results

Operant Conditioning can Establish Both Short and Long-term Memories

We first wanted to determine if *R. maderae* could indeed learn by operant conditioning. Conditioning involved placing animals that had been isolated from food and water for 6–7 days in a cylindrical plastic container with two odor choices on opposite sides of the arena. Peppermint, which is an aversive odor, was associated with a standardized slice of apple as a reward. The second odor was an attractant (vanilla) that was paired with apple made inaccessible by covering with fine mesh netting. The arena was housed in very dim red light and the animal's behavior was monitored with an infra-red video camera. Typically in the initial trial, animals would "visit" the inaccessible apple slice at the vanilla 4–6 times before they approached the peppermint and consumed the apple associated with the aversive odor. In subsequent trials a reduction in the number of visits to vanilla prior to acquiring the apple at peppermint was taken a measure of learning. In initial experiments the animals were trained and tested at CT 14, a phase when they have been shown to be capable memory formation by classical conditioning [7]. Memory was evaluated by the performance at 5 minutes, 90 minutes, 48 hours and 9 days (216 h). Following the initial training trial and consumption of the apple slice, animals consistently showed a significantly reduced number of visits to vanilla prior to the visit to peppermint and the receipt of the reward in the 5-minute trial. Little change in performance occurred in subsequent trials indicating that animals were capable of both short-term and long-term memory (Fig. 1A). In additional experiments in which animals were given three training trials on each of two consecutive days at CT 14, performance was excellent at both one-week and two-week tests (Fig. 1B). Notably, in previous results with classical conditioning (3 training trials) long-term memories were generally more labile lasting only 3–4 days [7] while the present data indicate that memories formed via operant conditioning persisted for over a week with little decrement.

In view of the fact that there is a robust circadian regulation of memory acquisition via classical conditioning [7] we anticipated that animals would not be successful at forming the associative memory if trained at CT 2 (a phase where they appear to be incapable of memory acquisition via classical conditioning). Surprisingly, when animals were trained at CT 2 using our operant conditioning protocol they performed just as well as the animals trained at CT 14 (Fig. 1C) exhibiting both short-term and long-term associative memory and there was no evidence of any significant deficit in the ability to perform the task.

As a control to demonstrate that the changes in odor preference were in fact due to an association between the apple reward and the peppermint odor, the protocol was revised such that animals received no positive reward for peppermint visits by making the reinforcement inaccessible at both odor sources. As shown in Fig. 1D there was no decrease in the preference for vanilla when the apple reward at peppermint was not available. The results confirmed that the changes in odor preference reflected the formation of an associative memory.

Associative Memory Formation is Independent of Reward

While the odor sources used in the operant task (peppermint and vanilla) were the same as those used to demonstrate a circadian rhythm in effectiveness of classical conditioning [7], in the earlier experiments the positive unconditioned stimulus was a sucrose solution rather than apple. Thus one potential explanation for the difference in the impact of circadian phase on memory formation was that the sensory information from the apple stimulus was processed differently from the sucrose solution, in one case being subject to circadian regulation (sucrose) while perception of the other (apple) was independent of circadian phase. Therefore we repeated our experiments utilizing a 20% sucrose solution as a reward to match more closely the positive US we had used previously for classical conditioning. The results are shown in Fig. 2. When sucrose was offered as a reward, it was just as effective as the apple in modifying odor preference behavior at both CT 14 (Fig. 2A) and at CT 2 (Fig. 2B). As an additional control we showed that when the sucrose that was paired with the peppermint odor was also covered with netting to prevent the cockroach from receiving the reward at the peppermint odor, there was no change in odor preference (Fig. 1C). The results confirmed the shift in odor preference was due to an association between the peppermint odor and the sucrose reward. These observations show that both short-term and long-term associative memories in which the peppermint odor is associated with a reward are formed equally well at CT 14 and at CT 2 in an operant conditioning task independent of whether the reward is apple or sucrose.

The results suggested that either there is no effect of the circadian system on memory formation or that the phasing of the effect was quite different for the two forms of learning. In order to distinguish between these two possibilities, we trained animals at two additional circadian times (CT 8 and CT 20). Fig. 3 plots the Learning Indices for all four circadian times memory. There was no significant dependence of performance on circadian phase. With regard to short-term memory the learning indices were nearly identical at all circadian times. There is more variability in the results for long-term memory (e.g., performance at CT 8 was somewhat better than at other phases); however, there were no statistically significant dependence of performance on the circadian phase of training.

In view of the fact that both the classical and operant conditioning protocols were closely matched in terms of the stimuli used, we found it surprising that memory formation via classical conditioning exhibited a robust circadian rhythm while memory formation following operant conditioning appeared to be completely independent of the circadian system. However, the experiments involving classical conditioning utilized a differential conditioning protocol in which the peppermint was associated with a positive unconditioned stimulus (sucrose) while the vanilla was associated with a negative unconditioned stimulus (saline). Thus a possible explanation of the differences we were finding was that the circadian modulation in classical conditioning was due to circadian regulation of the response to the aversive (saline)

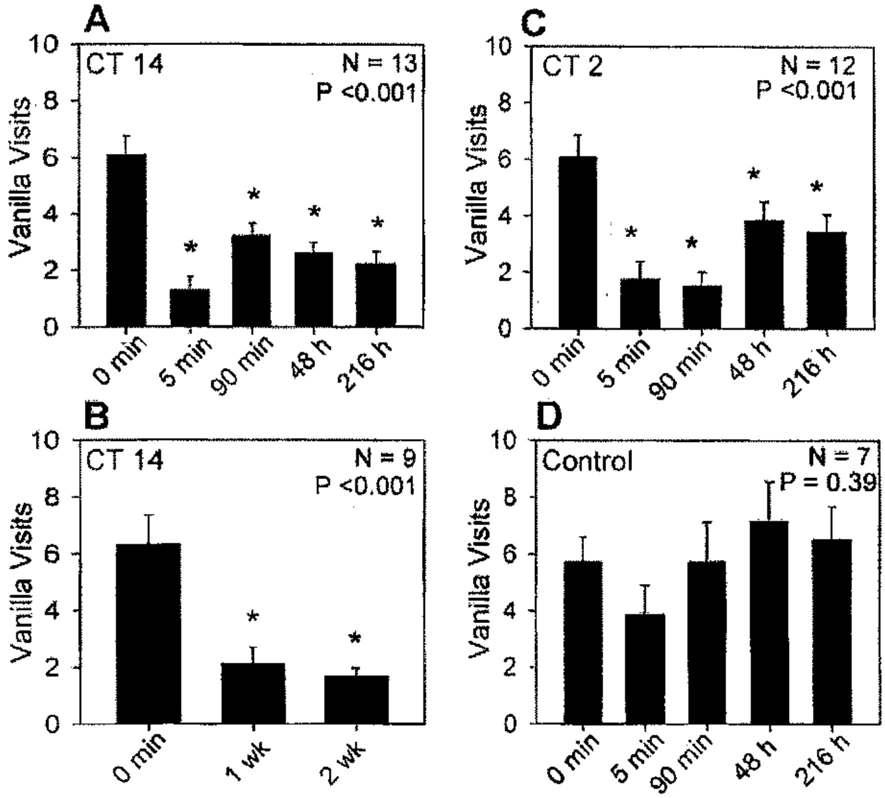

Figure 1. Each panel plots the number of times (Mean±SEM) the animals visited a vanilla odor prior to visiting peppermint as a function of the training/testing time. A, animals were subjected to training sessions in the early subjective night (CT14) and were rewarded with a slice of apple when they visited the peppermint. Prior to any reward (0 min) animals exhibited a clear preference for vanilla. In subsequent trials animals showed a significant reduction in vanilla visits prior to visiting peppermint. B, Animals were subjected to two consecutive days of training at CT 14 (three trails in each session with a 5 minute inter-trial interval. There was a highly significant reduction in the number of visits to vanilla made prior to the visit to peppermint both one and two weeks later compared to the initial trial (0 min.). C, when trained at CT 2, animals exhibited a similar reduction in vanilla visits to the animals trained at CT 14. D, when access to the reward at peppermint was prevented during training (CT14), there was no significant change in the number of vanilla visits. P-values for the ANOVA are indicated in the figure. Bars marked with * indicate a statistically significant difference (p<0.05) when compared to the initial number of vanilla visits (Holm-Sidak post-hoc test).
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stimulus. In order to test this, we trained animals to an operant conditioning task in which the peppermint was paired with sucrose and the vanilla was paired with an accessible saline solution. Animals still exhibited excellent performance in both short-term and long-term memory tests whether they were trained at CT 2 or CT 14 (Fig. 4). Notably, on initial visits to vanilla the animals did

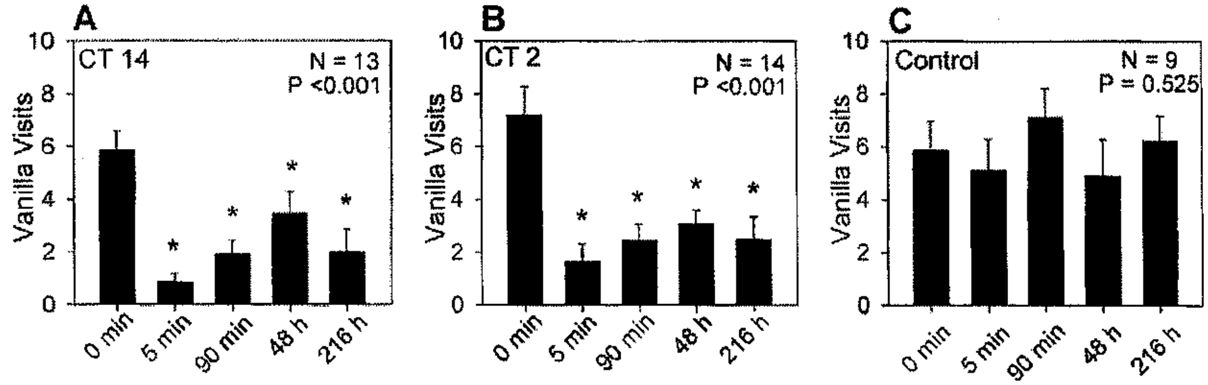

Figure 2. Plots the number of times (Mean±SEM) animals visited the vanilla odor prior to visiting peppermint as a function of the training/testing sequence when trained at CT 14 (A) or CT 2 (B). Sucrose rather than apple was offered as a reward. C, when access to the sucrose reward at peppermint was prevented during training (CT14), there was no significant change in the number of vanilla visits. P-values for the ANOVA are indicated in the figure. Bars marked with * indicate a statistically significant difference (p<0.05) when compared to the initial number of vanilla visits (Holm-Sidak post-hoc test).
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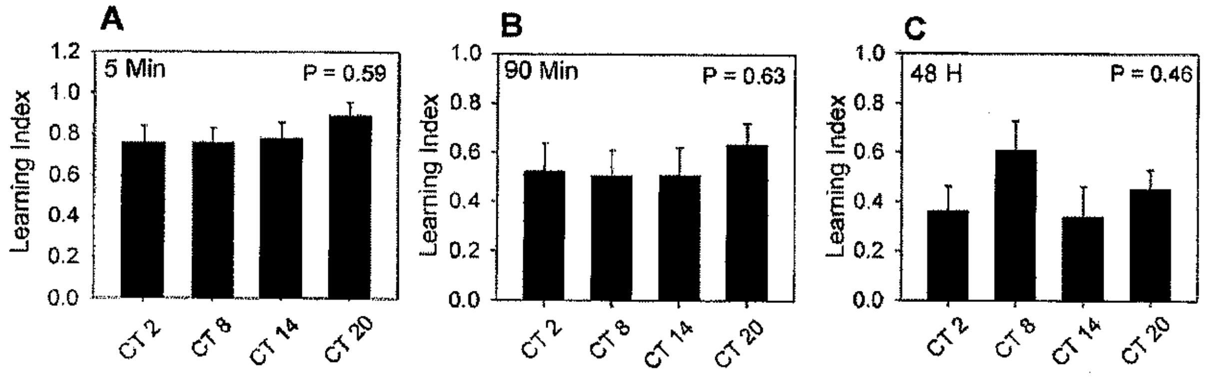

Figure 3. Plots of learning index as a function of the circadian phase of training and testing for 5 min, 90 min, and 48 h memory tests. Analysis of variance showed no significant dependence of performance on phase of training for measures of either short-term or long-term memory. Numbers of animals for each circadian phase ranged from 10 to 15.
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appear to contact the saline solution (but did not drink it) indicating they were in fact exposed to the negative reinforcement. Support for this contention was obtained in trials in which we offered sucrose solution instead of saline at the vanilla. Four of five animals consumed the sucrose at the vanilla odor prior to a visit to peppermint showing that they were sampling the reinforcement solution presented at vanilla before moving to peppermint.

The results suggested that the difference in the circadian regulation of classical and operant conditioning was not dependent on differences in the olfactory or gustatory cues the animals were exposed to in the two training protocols.

Temporal Regulation of Recall

Previous results with differential classical conditioning [7] showed that recall of an acquired memory was independent of circadian phase. Animals trained at CT 14 were able to perform well on the task even if they were tested at CT 2 (a time when they were incapable of memory acquisition). The results were quite different when we trained animals at CT 14 in the operant conditioning task and tested them 12, 24, 36, or 48 hours later. Recall was significantly better when animals were tested at CT 14 (24 and 48 h tests) than when they were tested at CT 2 (12 and 36 h tests) (Fig. 5A). The results suggested that circadian phase at the time of training is a contextual cue influencing the ability to perform the task. In order to confirm that successful recall was tied to the phase of training, we trained animals at CT 2 and tested them at either 12, 24, 36, or 48 hours after training. In this case recall was better if the animals were tested at times corresponding to CT 2 rather than CT 14 (Fig. 5B). In essence, animals perform better when tested at the same circadian phase at which they were trained (independent of the phase of training) suggesting that circadian phase is an important contextual cue for memory recall.

Discussion

In classical (Pavlovian) conditioning animals learn about the relationship between two stimuli, while in operant (instrumental) conditioning it is the relationship between stimuli and the consequences of the animal's own behavior that is critical. While the two forms of associative learning are operationally distinct, the basic question of how these different forms of learning are related and whether or not they involve the same or fundamentally different underlying processes is still uncertain [21]. While the analysis of classical conditioning has proceeded rapidly, there is much less information available on mechanisms of operant conditioning. However, recent data do suggest that while there are similarities, significant differences exist at the cellular/

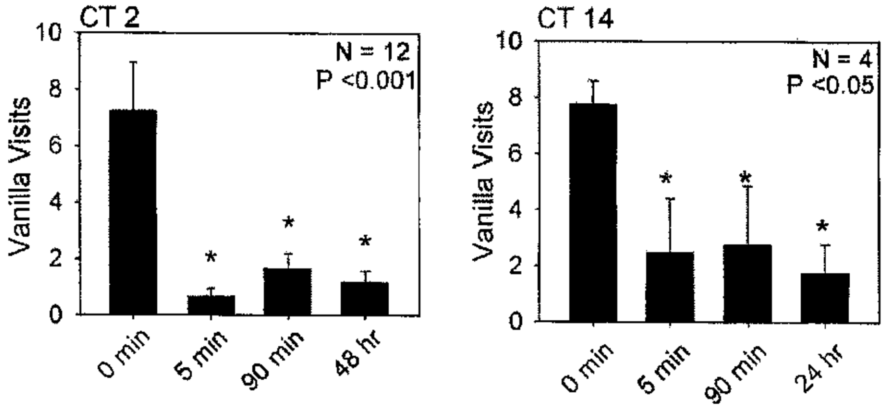

Figure 4. Plots vanilla visits as a function of training/testing time when the peppermint odor was paired with the positive reinforcement of sucrose solution and the vanilla odor was paired with an accessible negative reinforcement of saline. Left panel, training at CT 2; right panel training at CT 14. P-values for the ANOVA are indicated in the figure. Bars marked with * indicate a statistically significant difference ($p<0.05$) when compared to the initial number of vanilla visits (Holm-Sidak post-hoc test).
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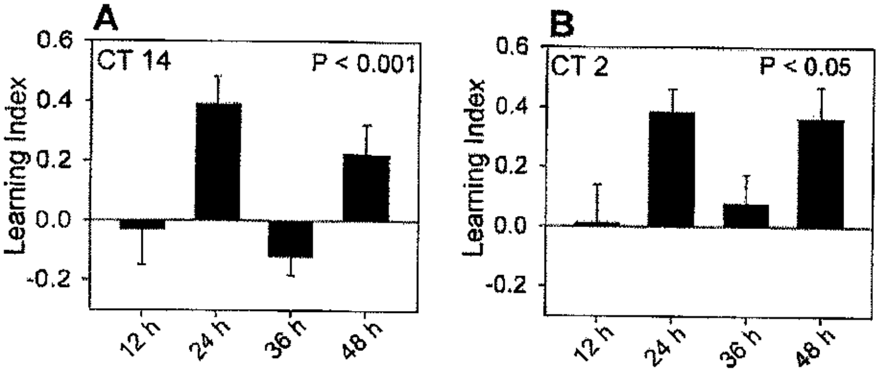

Figure 5. Plots the learning index for animals trained at either CT 14 (A) or CT 2 (B) and tested 12, 24, 36, or 48 h after training. Animals tested at the same circadian time as training performed better than those tested at a phase 12 hours different from the phase of training. Numbers of animals for each time point ranged from 14 to 17 in A and 10 to 11 in B. Analysis of variance showed significant dependence of performance on the time of testing for training at both CT 2 and CT 14.
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molecular level [22–24]. The extent to which these differences in mechanism may be reflected in differences in circadian regulation is unclear. With regard to the role of the circadian system in learning and memory, studies have addressed questions of memory acquisition (short-term memory); memory consolidation (long-term memory) and long-term memory recall utilizing both classical and operant conditioning. The outcomes of these studies have been varied.

Circadian Regulation of Memory Formation

Although the number of studies is limited, based on previously published reports, it appears that the role of the circadian system in the regulation of learning and memory may generally be different for classical and operant conditioning. For memory acquisition, circadian regulation appears to be prevalent in classical conditioning paradigms (mice [11]; zebrafish [10]; cockroaches [7]; fruit flies [8]; bees [9]). The one apparent exception to this generalization occurred in golden hamsters (*Mesocricetus auratus*) subjected to conditioned place preference or conditioned place avoidance [25,26].

In contrast to the general finding that memory acquisition during classical conditioning varies with circadian phase, the results from operant conditioning studies have suggested that learning and short-term memory are independent of the circadian system (rats [2,3]; marmosets [4]; *Aplysia* [6]). Similarly, the formation of long-term memories appears to depend on the circadian phase of training in classical conditioning while, with one exception [6], long-term memory formation appears to be independent of circadian phase in operant conditioning paradigms.

On the basis of these published results, one might suggest that there potentially generalizable differences between operant and classical conditioning in the way in which the circadian system regulates memory formation. One reason to be cautious is that in some cases it may be difficult to distinguish whether or not an animal is relying exclusively on operant or classical features of memory acquisition. Another major problem is that the responses to the two mechanisms for associative memory formation had never been examined in the same species and generally involved very different training methods (and thus very different sensory inputs and behavioral outputs). Therefore differences could be dismissed as variation due to differences between species or methodology. The results presented here however, indicate that in the cockroach, *Rhyparobia maderae*, the differences in the role of the circadian system in regulating the formation of new memories can likely be attributed to fundamental differences in the way in which memories are formed by the two types of conditioning. Experiments with classical conditioning in the cockroach demonstrated that memory acquisition in an odor discrimination task is regulated by the circadian system [7]. In contrast, the results presented here show that both short- and long-term memory formation via operant conditioning in a very similar odor discrimination task are independent of circadian phase. These results lend support to the notion that the circadian regulation of memory formation may be different between memories that arise from classical conditioning and those that are formed via operant conditioning. The data raise a variety of general questions of interest. The first concerns the mechanisms by which circadian clocks "gate" memory formation in classical conditioning, and by the same token, why it doesn't appear to impact memory formation with operant conditioning. In the cockroach, the difference in effectiveness between classical and operant conditioning at CT 2 is evident within 5 minutes after the training. Similarly, in mice [11], zebrafish [10], and fruit flies [8] circadian regulation modifies performance early in the process of memory acquisition. The data suggest that whatever the regulatory role of the circadian system, its impact is significant very early (within minutes) in acquisition process (which provides an experimentally attractive limited time window for further study).

At present there are only three studies that appear to directly approach the question of how the circadian system regulates memory formation. In one recent study involving a novel object recognition task in Siberian hamsters (*Phodopus sungorus)*, it was shown that the $GABA_A$ receptor antagonist, pentylenetetrazol, was able to restore learning deficits caused by disruption of the circadian system. The results indicated that GABAergic signaling controlled by the circadian clock in the suprachiasmatic nucleus may be responsible for suppressing memory acquisition at inappropriate times of day [17]. Interestingly, as the authors note, this hypothesis could also explain the observation that SCN-lesioned rodents generally improved or failed to have a negative effect on learning [27,28]. Conceptually similar results were obtained in another study on zebrafish, though the details differed. In the zebrafish the data suggested that night-time melatonin secretion from the pineal gland was responsible for suppression of memory acquisition at night, and removal of the pineal or treatment with melatonin receptor antagonists abolished memory deficits at night [10]. Thus in both of these cases a circadian clock appears to actively suppress early stages of memory formation during part of the circadian cycle, and destruction of the clock or pharmacological interference with the output signal rescues the learning deficit. The targets of suppression are not yet clear. At these early stages (i.e., short-term memory), protein synthesis is unnecessary for recall or performance, thus it is unlikely that regulation of transcription or translation is involved. In contrast, in diurnal *Aplysia* results indicate that at night when the animals exhibit a deficit in long-term memory, the circadian clock actively suppresses persistent MAPK activation and thus the transcriptional activation necessary for long-term memory while leaving the processes of memory acquisition and short-term memory unaffected [29].

In summary, in these markedly different species, mechanisms of circadian regulation of memory formation appears to be quite diverse in detail but do exhibit the common feature that the clock appears to be actively suppressing memory formation at "inappropriate" times of day. On the other hand, no clear picture emerges to answer the question of what mechanistic differences between memory formation and retrieval could account for differential regulation of operant and classical conditioning by the circadian system.

Circadian Regulation of Long-term Memory Recall

With regard to the circadian regulation of recall of long-term memories, the contrast between operant and classical conditioning appears to be less consistent. Studies have indicated three different outcomes for the role of the circadian system in recall long-term memory recall following classical conditioning In mice, recall ability following context or tone cued fear conditioning was better during the subjective day, independent of the time of training [11,18]. The results suggested the circadian system either limits access to long-term memory stores to a fixed set of circadian phases or modulates down-stream processes related to performance. In contrast, in the golden hamster, following classical conditioning (conditioned place preference or avoidance), recall was better when the test was done at the same circadian phase as the training independent of whether animals were trained in the night or the day [1,25,26]. In this case, the results suggested that circadian phase of training became a contextual cue that determined performance during testing. Finally, recall following classical conditioning in the zebrafish [10] or the cockroach [7] appeared to be independent of the time of testing.

Similar variability has been evident on studies of operant conditioning. Following operant conditioning in the cockroach, recall was better when the test was done at the same circadian phase as training indicating (as in classical conditioning in the hamster). This suggests that circadian phase of training was a contextual cue that determined performance during testing. On the other hand, in *Aplysia* recall of long-term memory after operant conditioning appeared to be independent of the time of testing [6]. Overall, the data suggest that the variability in the ability to recall a consolidated memory at various points in the circadian cycle is not readily tied to a particular mode of training.

Adaptive Significance

The second general question concerns the adaptive significance of circadian control and why circadian regulation of classical and operant conditioning should be different. One speculative suggestion that emerged from classical conditioning studies was that memories are only profitable when formed in the environmental context in which they will be used [7]. The data from cockroaches are largely consistent with this notion. In the case of classical conditioning, the suppression of memory formation occurs at a time when the animals are least active and thus least likely to be using olfactory cues in foraging behavior. As a consequence, formation of memories by completely external intervention is suppressed at times when the animal is not normally engaged in olfactory driven behavior and the associative memory is not likely to be useful in guiding future behavior.

Conversely, if the animal is *voluntarily* out foraging (even at an unlikely time as might happen when food is scarce [30]) and is rewarded, then memory of the success of the behavior becomes useful and is tied to the circadian phase at which that particular olfactory environment was profitable. At other times of day, when the olfactory or reward environment is likely to have changed, the memory is not used to guide behavior. This is reminiscent of the early work on honey bees [31] and later work with birds [32,33] and fish [34,35] where the animals were shown to select feeding sites at those specific times of day when they had previously been successful. The notion here then is that the role of the circadian system in regulation of learning and memory is to limit formation of new memories or utilization of established memories to only those times of day when they are likely to be profitably used in the future.

Materials and Methods

Animals

Colonies of *Rhyparobia maderae* (more commonly known as *Leucophaea maderae)* were maintained in 12-h light/dark cycles (LD 12:12). One week before the experiment began, six to twelve young adult males were isolated from food and water and housed together in a round plastic container 9 cm tall and 24.5 cm in diameter. All animals were transferred to constant darkness at end of the last complete light period prior to training. The experiments were conducted in an environmental room under dim red light at 24.5°C. The animals remained in a light-tight box in constant darkness (DD) until the conclusion of the experiment.

Operant Conditioning

The methods were adapted from [19] and [7]. The strategy was to reward animals for visiting an aversive peppermint odor rather than an attractive vanilla odor – the reward was either a small slice of apple or 20% sucrose solution. Animals were trained and tested in a circular Plexiglas arena 30 cm in diameter and 9.5 cm tall, the sides of which were smeared with petroleum jelly to prevent escape. This arena was housed in a closed box, the interior of which was illuminated with dim darkroom safelight that limited visible light wavelengths to greater than 600 nanometers (Kodak 1A or GBX-2; Eastman- Kodak, Rochester, NY). Light intensity was adjusted with a rheostat to a final intensity at the floor of the testing arena less than 0.1 μE-m^{-2}-sec^{-1} (LiCor Photometer). Odors were provided by placing a 1.5-cm-square piece of filter paper, saturated with 20 μl of either vanilla or peppermint extract, into a 1.5 ml microfuge tube aerated with small holes. The microfuge tube was connected to a petri dish lid 3.1 cm in diameter. For training and testing, one vanilla odor and one peppermint odor source were placed at opposite sides of the arena. A small cup in each Petri dish lid (made from a microfuge tube lid) held either a 75 μl sucrose reward or a slice of apple. A "standard" apple reward was prepared by inserting glass tubing, 0.5 cm in diameter, through the apple. The cylindrical core of apple retrieved from the tube was sliced with two razor blades separated by the 1 mm width of a microscope slide to produce 1 mm-thick apple slices that weighed approximately 0.01 g. The reward was covered with a fine mesh netting on the vanilla apparatus but was not covered on the peppermint apparatus, allowing the animal access to the positive reinforcement at peppermint only. In one series of experiments as noted in the results, the vanilla odor was paired with a negative reinforcement of an accessible 20% NaCl solution.

Individuals were placed in the center of the arena and covered with a Petri dish until the training trial began. Animals were observed using an infrared-sensitive CCD camera (Sony XC-77; Sony, Tokyo, Japan). When the timer was started, the animal was allowed to run freely in the arena between the two odor sources. The animal was expected to visit the vanilla odor first before visiting the peppermint, since it has been shown that cockroaches have an innate preference for the vanilla odor [7,19]. In order to ensure that all subjects in the study exhibited a naïve preference for vanilla and to eliminate inactive individuals, each animal was required to visit the vanilla at least three times before visiting the peppermint or it was dropped from further training trials. A visit to vanilla was recorded when the animal probed the netting covering the reward with its mouthparts. The number of vanilla visits was recorded until the animal first visited the peppermint and received the positive reinforcement which concluded the training trial.

Training and Testing Schedule

Circadian Times (CT) of training and testing were estimates based on the time of lights-off of the prior light cycle (designated as CT 12) and assumed a freerunning period of 24 hours. Training was conducted at either: CT 0–3 (corresponding to the early subjective day); CT 6–9 (late subjective day); CT 12–15 (early subjective night); or CT 18–21 (late subjective night). For most experiments, each training session consisted of three "training trials," The first was designated as time 0, the second at 5 minutes after the end of the first, and a third 60–120 minutes after the second trial was complete (referred to as 90 minutes in the results). We note that the variability of timing in the third training trial could have introduced more inter-individual variability in performance [36,37] but was necessary for sufficient numbers of animals to be trained in one session. Subsequent trials were carried out either 12, 24, 36, or 48 hours, or 9 days later. For one experiment we subjected animals to training sessions on two consecutive days. For each of the two days there were 3 trials with an interval of 5 minutes between each. Testing on these animals was carried out one and two weeks after the last training trial.

When the interval between two training trials was 5 minutes, animals were allowed to remain in the testing arena. After the remaining trials, the animals were returned to the group housing container and placed in the DD light-tight box. Individuals were identified by unique patterns of dots on the pronotum made with white paint. Animals were given 12 minutes to complete each training trial (i.e., acquire the reward), with the exception of the initial training trial, in which an 8-minute limit was set to eliminate inactive animals. For each trial, the number of visits the animal made to the vanilla source before reaching the peppermint was recorded.

Data Analysis

The data were analyzed with Sigma Plot® statistical software (Version 11, Systat Software, Inc. 2007). The number of vanilla visits in each training trial relative to the initial training trial was used as a measure of learning, and was analyzed with a repeated measures one-way ANOVA, using the Holm-Sidak method as a post-hoc test. The mean number of vanilla visits for each training trial was compared against the mean number for the initial trial and tested to determine if there was a significant difference between the means at the $\alpha = 0.05$ level.

In order to compare groups, a learning index (LI) was developed to quantify the learning for each individual animal. The learning index was calculated by: $LI = (V_0 - V_T)/(V_0 + V_T)$ where V_0 is the number of initial vanilla visits, and V_T is the number of vanilla visits in a testing trial. A higher LI indicated better performance (maximum value of 1). An LI score of 0 indicated no change in odor preference. LIs were compared using a one-way ANOVA.

Author Contributions

Conceived and designed the experiments: TLP MVG SBS. Performed the experiments: MVG SBS. Analyzed the data: TLP MVG SBS. Wrote the paper: TLP.

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ORIGINAL PAPER

Classical conditioning in borderline personality disorder: an fMRI study

Classical Conditioning

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Abstract Previous research suggests disturbed emotional learning and memory in borderline personality disorder (BPD). Studies investigating the neural correlates of aversive differential delay conditioning in BPD are currently lacking. We aimed to investigate acquisition, within-session extinction, between-session extinction recall, and reacquisition. We expected increased activation in the insula, amygdala, and anterior cingulate, and decreased prefrontal activation in BPD patients. During functional magnetic resonance imaging, 27 medication-free female BPD patients and 26 female healthy controls (HC) performed a differential delay aversive conditioning paradigm. An electric shock served as unconditioned stimulus, two neutral pictures as conditioned stimuli (CS+/CS−). Dependent variables were blood-oxygen-level-dependent response, skin conductance response (SCR), and subjective ratings (valence, arousal). No significant between-group differences in brain activation were found [all $p(\mathrm{FDR}) > 0.05$]. Within-group comparisons for $\mathrm{CS+}_{\mathrm{unpaired}} > \mathrm{CS-}$ revealed increased insula activity in BPD patients but not in HC during early acquisition; during late acquisition, both groups recruited fronto-parietal areas [$p(\mathrm{FDR}) < 0.05$]. During extinction, BPD patients rated both CS+ and CS− as significantly more arousing and aversive than HC and activated the amygdala in response to CS+. In contrast, HC showed increased prefrontal activity in response to CS+ > CS during extinction. During extinction recall, there was a trend for stronger SCR to CS+ > CS in BPD patients. Amygdala habituation to $\mathrm{CS+}_{\mathrm{paired}}$ (CS+ in temporal contingency with the aversive event) during acquisition was found in HC but not in patients. Our findings suggest altered temporal response patterns in terms of increased vigilance already during early acquisition and delayed extinction processes in individuals with BPD.

Keywords Borderline personality disorder · Classical conditioning · Prefrontal cortex · Amygdala · Insula

Introduction

Conditioning is a basic associative learning process that plays an important role in the development and maintenance of psychiatric disorders as well as in behavior therapy, especially exposure therapy.

Numerous studies have applied experimental fear conditioning paradigms to examine the (neurobiological) mechanisms underlying associative emotional learning [1–31]. In differential delay conditioning paradigms, one of the two initially neutral stimuli (CS+) is temporally paired with an aversive unconditioned stimulus (US), while the other is not (CS−). Many experimental settings do not only

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involve *acquisition* (learning) and within-session extinction but also between-session extinction (*extinction recall*) and *reacquisition* or *reinstatement*, i.e., a renewed exposure to the original CS–US contingency.

There is convergent evidence from both animal research and human studies that the amygdala, insula, and medial prefrontal cortex (mPFC) including the anterior cingulate cortex (ACC) play central roles in the acquisition of fear responses [1, 4, 5, 9, 15]. Several studies in healthy humans further reported differential responses (to CS+ > CS−) in the inferior frontal gyrus, rostral, and caudal orbitofrontal cortex (OFC), thalamus, hippocampus, cerebellum, striatum (including putamen), as well as in sensory cortices [6, 10, 16, 18, 20, 22, 24–28]. The mPFC is assumed to play a central role in the integration of sensory and affective stimuli and in the coordination of memory storage both during the acquisition and the extinction of emotional (fear) memories [11, 14, 28, 31]. Extinction is thought to involve activity-dependent potentiation of synaptic transmission in the mPFC resulting in an inhibition of amygdala-dependent responses [11, 14]. Animals with lesions of the mPFC are unable to extinguish conditioned responses [4, 15]. In healthy humans, increased activation in the mPFC [20, 24, 26], OFC [17], amygdala (e.g., [7, 18, 20]), and insula [2, 17] could be observed during extinction (for a review see [28]). For the amygdala, a rapid habituation, i.e., decrease in reactivity to unpleasant stimuli was observed over the course of the acquisition phase [7, 8].

Within a 'brain network of emotion regulation,' the amygdala and insula have been critically implicated in the processing of negative emotions, while the ACC is thought to play a critical role in both the generation and regulation of emotions [9, 32]; prefrontal areas including the ventrolateral and dorsolateral PFC (vlPFC, dlPFC), OFC, and dorsomedial PFC are thought to be involved in the down-regulation of affective arousal [32].

In anxiety disorders, acquired fear responses to an initially neutral stimulus (e.g., place, object, person) previously paired with an US appears to be not successfully extinguished [33–35]. Patients with posttraumatic stress disorder (PTSD), for example, showed amygdala hyperreactivity and increased skin conductance responses to the CS+ during extinction recall [34]. Deficits in associative emotional learning are assumed to underlie clinical features such as a persistent fear and heightened affective arousal, even in contexts where no actual threat is present.

To our knowledge, no study so far has investigated the neural correlates of aversive differential delay conditioning in patients with borderline personality disorder (BPD). BPD is a severe psychiatric disorder with high rates of trauma and pronounced difficulties in emotion regulation including affective hyperarousal even in normative neutral situations [36, 37]. Experimental research in patients with BPD suggests altered learning and memory in aversive emotional contexts [38, 39]. On the neural level, there is growing evidence for both structural and functional alterations in fronto-limbic brain regions which play a major role in affecting regulation and learning processes (e.g., amygdala, hippocampus, anterior cingulate) including amygdala hyperactivity and diminished recruitment of the prefrontal areas during emotional challenge (for reviews see [40, 41]).

In a previous psychophysiological study [42], we investigated skin conductance responses (SCR) as well as arousal and valence ratings during an aversive differential delay conditioning paradigm in patients with BPD and healthy controls (HC). We additionally assessed levels of peri-experimental dissociation, i.e., disruptions of usually integrated functions of consciousness, memory, body awareness, and perception. In this previous study, BPD patients with low levels of dissociation showed regular conditioning responses in terms of a differential SCR to CS+ > CS− similar to HC. Of note, patients with high state dissociation failed to show regular conditioning, which suggests that dissociation may modulate emotional learning in BPD.

Recently, Kamphausen and colleagues [43] assessed SCR and brain activity during an instructed fear task in patients with BPD and HC during functional magnetic resonance imaging (fMRI). Before participants were brought to the MR scanner, they were informed that they would see different stimuli either indicating a safe situation or potential threat (electrodermal stimulation). The electrodermal stimulation was in fact only applied during this instruction but not during the experimental procedure. During fMRI, both healthy participants and BPD patients showed differential fear responses in terms of stronger SCR to stimuli indicating potential threat compared to stimuli indicating the save situation. However, only HC but not BPD patients showed amygdala habituation and an increase in activity in the mPFC during instructed fear. Findings of this fMRI study suggest that altered activity in a fronto-limbic brain circuitry might underlie disturbed emotional learning during an instructed fear task in patients with BPD.

The aim of the present study was to investigate the neural correlates of emotional learning in BPD patients applying an aversive differential (danger versus safety signal) delay conditioning paradigm during fMRI. Between-session extinction and re-acquisition were assessed after 72 h. Based on previous neuroimaging studies, we expected stronger activation in the amygdala and insula during acquisition and reacquisition and less activation in the mPFC during within-session and between-session extinction. We were further interested in habituation effects, i.e., decreases in activity over the course of each conditioning phase. Based on previous research [7, 8, 43], we expected a habituation of the amygdala during the acquisition phase.

Methods and materials

Participants

Thirty-four female individuals fulfilling DSM-IV criteria for BPD as assessed by the international personality disorder examination (IPDE) [44] and 32 female HC aged between 18 and 45 years participated in our study. Exclusion criteria for the patient group were a lifetime diagnoses of alcohol or substance dependence, psychotic disorder or bipolar I disorder, and current major depression as assessed by the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) [45]. In addition, patients had to be free of psychotropic medication for at least 4 weeks prior to investigation. Exclusion criteria for the HC group were the presence of any current or lifetime axis I or axis II disorder, and current or past psychotherapy. Diagnostic interviews were administered by trained and experienced psychologists. Inter-rater reliability for BPD was $\kappa = 0.69$ for SCID-I (primary diagnosis) and $\kappa = 0.77$ for IPDE. Patients were recruited at the Central Institute of Mental Health, Department of Psychosomatic Medicine and Psychotherapy in Mannheim/Germany or via announcements on BPD-associated Web sites. The HCs were randomly selected from the resident register of the city of Mannheim.

All participants underwent diagnostic assessment using the SCID-I [45] and the IPDE [44]. To obtain a psychopathological characterization of the patient sample, trait dissociation by the German version of the Dissociative Experience Scale (Fragebogen zu Dissoziativen Symptomen, FDS; [46]) and global BPD symptom severity by the Borderline Symptom List (BSL-95; [47]) were assessed.

Demographic and clinical characteristics (trait dissociation, BPD symptom severity, comorbid diagnoses) of the full sample can be found in Supplemental Table 1. There were no significant differences regarding age [BPD (Mean ± SD): 27.6 ± 7.5; HC: 27.8 ± 7.5], sex (all female), and ethnicity (all Caucasian). All participants were right-handed. There was a statistical trend for significant differences in level of education. As expected, both groups differed significantly in clinical measures of trait dissociation and BPD symptom severity.

From this initial sample, one HC and five BPD patients dropped out of the study before completion of the experiment. From the remaining sample, a subset of neuroimaging data had to be discarded due to movement artifacts or technical problems during MR scanning. The final neuroimaging data set comprised data from 26 HC and 27 BPD patients for the first (conditioning) session, 22 HC and 21 BPD patients for extinction recall, and 17 HC and 24 BPD patients for reacquisition. Since some participants did not show an adequate skin conductance response (SCR) > 1 µS during the scanning procedure, SCR data of 21 HC and 21 BPD from the first (conditioning) session and SCR of 15 HC and 14 BPD from the second session (extinction recall, reacquisition) could be included into the final psychophysiology analysis.

Demographic and clinical characteristics (trait dissociation, BPD symptom severity) of all subsamples can be found in Supplemental Table 2 (subsamples neuroimaging dataset) and Supplemental Table 3 (subsamples SCR dataset). There were no significant differences regarding age and educational level in any subsample. As expected, within all subsamples, BPD patients reported significantly more trait dissociation and BPD symptom severity.

Experimental design

The experimental design was an aversive differential delay conditioning procedure which is depicted by Fig. 1. Dependent variables were the blood-oxygen-level-dependent (BOLD) signal, SCR, and valence/arousal ratings. The conditioned stimuli (CS) were two neutral graphic patterns (blue square and yellow triangle). The US consisted of unpleasant, but tolerable electrical stimulation to the right thumb. Stimulus duration was 5.8 s (s), and the unconditioned stimulus (US) lasted for 2.8 s and was administered 3 s after CS onset. The inter-trial interval (ITI) lasted between 8 and 12 s, randomly generated. CS+ (CS paired with the US) and CS− (non-reinforced CS) were counterbalanced across participants and presented in pseudorandom order with the constraint of a maximum of two consecutive presentations of the CS+ (danger signal) or CS− (safety signal).

The US (electrical stimulation) was delivered through a copper electrode of an electrical stimulus generator (Digitimer, DS7A, Wewyn Garden City, UK). Before the experiment, for each participant, the level of pain stimulation was individually determined. To this end, participants were given a series of stimuli, starting with a mild stimulus, which was gradually increased to (1) detection threshold, (2) pain threshold, and (3) pain tolerance. This procedure was repeated three times. Finally, we delivered stimuli that were 80 % above pain threshold. Participants were asked to rate the intensity of pain on a Likert scale ranging from 0 = not painful to 10 = extremely painful. For the experiment, we used stimulation intensities that were rated with at least 7/10. Therefore, the experimental level of pain was objectively different, but subjectively both groups received the same level of painful stimulation. This procedure was chosen due to the fact of higher pain threshold in BPD [48, 49].

The first session (conditioning session) started with a phase in which participants were familiarized with the CS+ and CS− ('*familiarization*': 10 CS+, 10 CS−). During the following *acquisition* phase, 18 CS+ paired with the US

Table 1 Brain activity during the different conditioning phases in healthy controls (HC) and in patients with borderline personality disorder (BPD)

Conditioning phase	Group	T contrast	MNI coordinates (*X*, *Y*, *Z*)	*K*	Hemisphere	Label (Brodmann area)	Lobe	*T* value	*Z* value	*p* value
Early acquisition	HC	CS+	0, −66, 12	110	R	Posterior cingulate	Limbic	6.12	4.71	0.037
			−27, −48, −15	49	L	Fusiform gyrus (BA37)	Occipital	6.25	4.77	0.021
			−18, −9, −12*	53	L	Parahippocampal gyrus/uncus/amygdala	Limbic	5.33	4.03	<0.05, ROI
			−24, −3, −24*		L		Limbic	4.83	3.78	
			18, −15, −15*	33	R	Parahippocampal gyrus (BA28)	Limbic	4.83	3.78	<0.05, ROI
		CS+ > CS−	No significant clusters at p(FDR) < 0.05							
	BPD	CS+	No significant clusters at p(FDR) < 0.05							
		CS+ > CS−	33, 39, 15	178	R	Middle frontal gyrus	Frontal	7.26	5.28	0.003
			27, 51, 3		R	superior frontal gyrus	frontal	4.17	3.60	0.003
			−60, 3, 3	49	L	Superior temporal gyrus	Temporal	5.29	4.30	0.022
			−66, −27, 12	61	L	Superior temporal gyrus (BA42)	Temporal	4.90	4.07	0.035
			66, −33, 18	89	R	Superior temporal gyrus (BA42)	Temporal	4.84	4.03	0.036
			−33, 15, 3*	16	L	Insula	Sub-lobar	4.32	3.70	<0.05, ROI
			−39, −3, −9*		L			3.63	3.22	<0.05, ROI
Late acquisition	HC	CS+	48, 33, −6*	34	R	Inferior frontal gyrus (BA47)	Frontal	4.80	4.00	<0.05, ROI
		CS+ > CS−	−30, −66, −51	1716	L	Inferior semilunar lobule	Cerebellum	6.32	4.84	0.004
			−33, −78, −30			declive	posterior	6.14	4.75	0.004
			−39, −63, −42			cerebellar tonsil		5.91	4.63	0.004
			45, −48, 51	606	R	Inferior parietal lobule (BA40)	Parietal	6.16	4.76	0.004
			63, −42, 24					5.59	4.46	0.004
			48, −45, 30					5.35	4.33	0.004
			54, 21, 33	178	R	Middle frontal gyrus (BA46)	Frontal	5.62	4.48	0.004
			54, 30, 18			inferior frontal gyrus (BA46)	frontal	4.27	3.67	0.008
			42, 6, 57			inferior frontal gyrus		4.26	3.66	0.008
			−60, −45, 24	216	L	Inferior parietal lobule (BA40)	Parietal	5.29	4.29	0.004
			−42, −51, 33					4.51	3.82	0.006
			−33, −51, 36					4.38	3.74	0.007
			39, 42, 30	35	R	Middle frontal gyrus	Frontal	4.95	4.10	0.005
			48, 27, −9*	85	R	Inferior frontal gyrus (BA47)	Frontal	4.93	4.08	<0.05, ROI
			−54, 21, 0*	41	L	Inferior frontal gyrus (BA47)	Frontal	4.22	3.63	<0.05, ROI
			−51, 15, −6*				frontal	4.16	3.59	<0.05, ROI
	BPD	CS+	48, 15, 45	49	R	Middle frontal gyrus (BA8)	Frontal	6.76	5.09	0.011
			39, 57, 3	98	R	Middle frontal gyrus (BA8)	Frontal	5.88	4.65	0.027
			60, −24, −24	32	R	Inferior temporal gyrus	Temporal	5.36	4.36	0.028
			−27, −66, −30	158	L	Pyramis	Cerebellum	5.15	4.24	0.030
			−57, −36, −6	38	L	Middle temporal gyrus	Temporal	4.83	4.04	0.040
			6, 39, 42	87	R	Medial frontal gyrus (BA8)	Frontal	5.06	4.18	0.040

Table 1 continued

Conditioning phase	Group	T contrast	MNI coordinates (X, Y, Z)	K	Hemisphere	Label (Brodmann area)	Lobe	T value	Z value	p value
		CS+ > CS−	48, 15, 45	208	R	Middle frontal gyrus (BA8)	Frontal	7.08	5.55	0.001
			48, 39, 30		R	Middle frontal gyrus (BA46)	frontal	4.44	3.80	0.017
			−18, −102, 3	831	L	Middle occipital gyrus (BA18)	Occipital	6.29	4.86	0.004
			39, 57, 3	120	R	Middle frontal gyrus (BA9)	Frontal	5.99	4.71	0.004
			30, 12, 3	46	R	Inferior frontal gyrus	Frontal	4.93	4.11	0.012
			33, 21, −9		R			3.89	3.42	0.027
			60, −42, 42	58	R	Inferior parietal lobule (BA40)	Parietal	4.51	3.84	0.016
			6, 36, 42	32	R	Medial frontal gyrus (BA8)	Frontal	4.44	3.79	0.017
			−45, 54, −3	45	L	Middle frontal gyrus (BA10)	Frontal	4.39	3.77	0.018
Extinction	HC	CS+	48, 48, −3*	R	18	Middle frontal gyrus	Frontal	5.57	4.45	<0.05, ROI
			−39, 15, 45*	L	31	Middle frontal gyrus	Frontal	5.01	4.13	<0.05, ROI
			9, 36, 24*	R	57	Orbito frontal gyrus	Frontal	4.86	4.04	<0.05, ROI
			33, 63, 18*					4.63	4.63	<0.05, ROI
			21, 63, 21*					3.87	3.87	<0.05, ROI
			−45, 39, −9*	L	20	Inferior frontal gyrus (BA 47)	Frontal	4.54	3.84	<0.05, ROI
			−27, 57, 24*	L	15	Middle frontal gyrus	Frontal	4.23	3.64	<0.05, ROI
			45, 48, 27*	R	14	Middle frontal gyrus	Frontal	4.13	3.57	<0.05, ROI
			51, 45, 21*	R		middle frontal gyrus	frontal	4.12	3.56	<0.05, ROI
			42, 36, 24*	R	9	Middle frontal gyrus	Frontal	4.07	3.53	<0.05, ROI
		CS+ > CS−	24, 66, 21*	R	28	Superior frontal gyrus (BA10)	Frontal	3.96	3.45	<0.05, ROI
			15, 63, 24*	R		Superior frontal gyrus	frontal	3.90	3.41	<0.05, ROI
			−45, 21, 45*	L	7	Middle frontal gyrus	Frontal	3.83	3.36	<0.05, ROI
	BPD	CS+	45, 42, −12*	R	99	Orbitofrontal gyrus (BA11)	Frontal	4.76	4.00	<0.05, ROI
			30, −6, −12*	R	5	Amygdala	Limbic	3.90	3.43	<0.05, ROI
		CS+ > CS−	No significant clusters p(FDR) < 0.05							
Extinction recall	HC	CS+	12, −63, 0	2235	R	Lingual gyrus	Occipital	10.14	6.12	<0.001
			6, −78, 15		R	lingual gyrus	Occipital	9.14	5.82	<0.001
			−12, −84, 3		L	cuneus (BA 17)	occipital	7.16	5.09	<0.001
			−36, 42, −9	17	L	Middle frontal gyrus	Frontal	5.00	4.04	0.002
			−39, −63, −39	31	L	Tuber	Cerebellum	4.79	3.92	0.003
			51, −48, 3	33	R	Middle temporal gyrus	Temporal	3.80	3.30	0.016
			63, −51, −9		R	middle temporal gyrus (BA21)	Temporal	3.76	3.27	0.017
			−27, −3, −27	10	L	Parahippocampal gyrus	Limbic	4.38	3.67	0.006
			−24, 51, 39	4	L	Superior frontal gyrus (BA9)	Limbic	3.86	3.34	0.014
			−36, −24, −15	6	L	Parahippocampal gyrus	Limbic	3.80	3.30	0.016
		CS+ > CS−	No significant clusters $p < 0.05$							

Table 1 continued

Conditioning phase	Group	T contrast	MNI coordinates (*X*, *Y*, *Z*)	*K*	Hemisphere	Label (Brodmann area)	Lobe	*T* value	*Z* value	*p* value
	BPD	CS+	12, −72, 9	449	R	Cuneus (BA17)	Occipital	6.78	4.78	0.032
			45, 9, −9*	49	R	Insula (BA38)	Sub-lobar	4.34	3.58	0.05, ROI
			−27, −3, −27	3	L	Parahippocampal gyrus	Limbic	4.38	3.67	0.009
		CS+ > CS−	24, 39, −18*	7	R	Orbitofrontal gyrus (BA11)	Frontal	4.90	3.89	<0.05, ROI
			6, 18, 51*	2	R	medial frontal gyrus (BA8)	Frontal	4.00	3.37	<0.05, ROI
Re-acquisition	HC	CS+	51, −45, 18	13705	R	Superior temporal gyrus, insular cortex (BA13)	Temporal, Sub-lobar	11.53	6.01	<0.001
			−51, −24, 24		L			11.09	5.91	<0.001
			−66, −27, 30		L			9.78	5.60	<0.001
			48, 12,0*					8.88		<0.05, ROI
			30, 57, 18	219	R	Superior frontal gyrus/middle frontal gyrus (BA9, BA10)	Frontal	7.78	5.02	<0.001
			27, 51, 24		R		Frontal	6.88	4.70	<0.001
			42, 54, 12		R		Frontal	6.22	4.43	<0.001
					R					
			−39, 45, 24	74	L	Middle frontal gyrus (BA10, BA9)	Frontal	4.94	3.84	0.001
			−27, 51, 30		L			4.90	3.82	0.001
			−48, 45, 15		L			4.74	3.73	0.001
			−30, 3, −18*	45	L	Parahippocampal gyrus (BA34)	Limbic	5.10	3.92	<0.05, ROI
			−24, 0, −24*		L	amygdala/uncus	Limbic	5.14	3.94	<0.05, ROI
		CS+ > CS−	−3, 51, −6*	151	L	Medial frontal gyrus (BA10)	Frontal	6.80	4.28	<0.05, ROI
			0, 39, −18*		R			5.67	3.88	<0.05, ROI
			12, 45, −12*		R			4.98	3.60	<0.05, ROI
			−21, 42, 45*	L	19	Superior frontal gyrus	Frontal Frontal	5.18	3.68	<0.05, ROI
			−15, 42, 39*			superior frontal gyrus		5.03	3.62	<0.05, ROI
			−27, 24, 51*	10	L	Middle frontal gyrus (BA8)	Frontal	4.59	3.42	<0.05, ROI
	BPD	CS+	9, 3, 39*	19*	R*	Anterior cingulate (BA24)	Limbic	4.64	3.84	<0.05, ROI
			39, 54, −15*	16*	R*	Orbitofrontal gyrus (BA11)	Frontal	4.38	3.67	<0.05, ROI
		CS+ > CS−	36, 51, 12*	10	R	Middle frontal gyrus (BA10)	Frontal	4.28	3.61	<0.05, ROI

Clusters were determined applying an extended threshold of $p < 0.05$ FDR corrected on the whole-brain voxel-wise level. Clusters indicated by an *asterisk* were detected by region of interest (ROI) analyses with predefined anatomical masks. Sizes of subsamples were 26 for session 1 (familiarization, acquisition, extinction), 22 for extinction recall, and 17 for re-acquisition. *Note L* left, *R* right, *k* cluster size of contiguous voxels, *MNI* Montreal Neurological Institute, *R/L* right/left, *BA* Brodmann area, *CS+* conditioned stimulus that was sometimes paired with the unconditioned stimulus (US) but here only in absence of the US, *CS−* conditioned stimulus never paired with US and 36 CS− never paired with the US were presented. Furthermore, 18 CS+ were presented but not paired with the US (catch trials). Catch trials were used to differentiate between BOLD responses to the CS+ and to the pain stimulus (US). During the *extinction* phase, 18 CS+ and 18 CS− were presented. The second session (reacquisition session) took place 72 h later and consisted of the *extinction recall* phase (18 CS+ and 18 CS−) and a *reacquisition* phase (18 CS−, 9 CS+, 9 CS+/US).

Apparatus and physiological recordings

Stimulus delivery was controlled by a PC running Presentation software (Neurobehavioral Systems, San Francisco). Physiological data acquisition was controlled by an Ibook running Vitagraph version 4.61 (Becker Meditec, Karlsruhe, Germany). Physiological channels were recorded at a rate of 256 Hz in continuous mode using the Vitaport II system (Becker). SCR was obtained from 10-mm (sensor diameter) Ag/AgCl electrodes (Marquette Hellige GmbH, Freiburg, Germany) filled with an isotonic EDR jelly TDE-246 (Steffens, Berlin, Germany) and placed on the thenar and hypothenar of the non-dominant hand (constant voltage method with 0.5 V).

Procedure

The study was approved by the ethical review committee of the University of Heidelberg, Germany, in accordance with the Declaration of Helsinki. All subjects gave written informed consent after receiving a description of the study and scanning procedure. All participants were paid for study participation (10 €/h). The experiment was conducted between 2007 and 2009 at the Central Institute of Mental Health in Mannheim, Germany.

All participants underwent diagnostic assessments including the SCID-I and IPDE by trained diagnosticians and completed clinical questionnaires (FDS, BSL) as described above. The conditioning procedure took place on two different days within a fixed time interval. On a first day, the conditioning session (familiarization phase, acquisition phase, extinction phase) was conducted during fMRI. Immediately before the experiment, the level of pain stimulation was individually determined for each participant as described in detail above. The reacquisition session (extinction recall phase and reacquisition phase) took place 72 h later in the same MR scanner.

Throughout the scanning procedure, participants viewed stimuli presented on a screen by a projector via a mirror mounted on the head coil. Participants were not informed about CS–US contingencies and were told to passively view the stimuli. After each phase (familiarization acquisition, extinction, extinction recall, and reacquisition), participants rated the valence and arousal of the CSs to test awareness of the CS–US contingency. In addition, participants rated their acute dissociation on scales ranging from 1 = very calm to 9 = very arousing, 1 = very pleasant to 9 = very unpleasant, and 1 = no dissociative symptoms to 9 = very intense dissociative symptoms. Valence/arousal ratings of the CS were obtained by presenting the CS together with a visual analogue scale, and patients used a keypad to move the cursor and to choose a number.

fMRI assessment

fMRI was conducted by a Siemens MAGNETOM Vision 1.5 Tesla whole body scanner (Siemens Medical Solution, Erlangen, Germany). Head movement artifacts and scanning noise were restricted using head cushions and headphones within the scanner coil. To acquire BOLD signals (T2-weighted echo planar imaging, EPI), a standard protocol (see also [27]) with the following parameters was used: repetition time (TR): 3.77 s, echo time (TE): 45 ms; flip angle: 90°, matrix: 64 × 64, slice thickness: 3 mm, slice gap: 1 mm, FOV: 220 × 220 mm, and number of slices: 35. There were 100 images for familiarization, 320 for acquisition, and 160 for all other phases.

Data analysis

Psychometrics

Means and standard deviations of the BSL and the FDS were analyzed using t tests. For group comparisons, significance levels were set to $p < 0.05$. All analyses were conducted with SPSS (Version 22.0 for Windows; SPSS Inc., Chicago, IL).

Ratings

To compare valence and arousal ratings, repeated-measures analyses of covariance (rm-ANCOVA) were performed for the mean of each phase with group (HC, BPD) as between-subject factor, stimulus type (CS+, CS) as within-subjects factor, and present-state dissociative experience as covariate. A Greenhouse-Geisser correction was employed in case the sphericity assumption was not met. Statistical analysis was performed using SPSS 22.0 (SPSS Inc, Chicago, IL). An alpha level of $p < 0.05$ determined statistical significance.

Skin conductance response (SCR)

The electrodermal activity was determined according to published guidelines [50] with the program EDR-Para (Schäfer, Wuppertal). Phasic SCRs were defined as the response magnitude (maximum deflection) within a 1- to 4-s time frame (first interval response, FIR). SCRs lower than 0.05 µS were scored as zero. The FIR was counted as missing when the SCR was clearly initiated prior to CS onset. If such an anticipatory response was superimposed on a stimulus-related response, the scoring method (B) as described by Boucsein ([50], p. 136) was used. SCRs were log-transformed to reduce skewness [50]. To compare SCR to CS+ and CS− type × group, rm-ANCOVAs were performed for the mean of each phase with present-state dissociative experience as covariate. A Greenhouse-Geisser correction was employed if the sphericity assumption was not met. Statistical analysis was performed using SPSS 22.0 (SPSS Inc, Chicago, IL). An alpha level of <0.05 determined statistical significance.

fMRI data

Functional data were analyzed with SPM5 (Welcome Department of Imaging Neuroscience, University College London, UK, 2005). The first four images at the beginning of each trial were discarded to enable the signal to achieve steady-state equilibrium between radiofrequency pulse and relaxation.

Within preprocessing, the EPI time series were realigned and unwarped to the mean to correct for intra-subject's head movements. Mean images were normalized to an MNI (Montreal Neurological Institute, www.bic.mni.mcgill.ca) echoplanar imaging template with affine registration followed by nonlinear transformation with 25 mm cutoff, medium regularization and 16 iterations, resampled with trilinear interpolation, and written in 3 × 3 × 3 mm^3 isotropic voxels. The normalization parameters determined for the mean functional volume were then applied to the corresponding functional image volumes for each participant. Finally, images were smoothed with a Gaussian kernel of 8 mm full-width at half-maximum. The data were high-pass filtered (1/128 Hz cutoff) to remove low-frequency signal drifts.

The consecutive statistical analyses of the fMRI data relied upon the general linear model (GLM) to model effects of interest as implemented in SPM5. For each subject, the following events of interest were defined as regressors of interest: US, CS−, $\text{CS+}_{\text{paired}}$, and $\text{CS+}_{\text{unpaired}}$ for each phase of the conditioning paradigm: familiarization, acquisition, extinction, extinction recall, and reacquisition phase.

In order to assess the temporal course of brain activation, we split up the acquisition phase (of the first conditioning session) into an early and late acquisition phase. In addition, we investigated habituation effects during acquisition by modeling linear time effects for $\text{CS+}_{\text{unpaired,}}$ $\text{CS+}_{\text{unpaired}} > \text{CS−}$, and $\text{CS+}_{\text{paired}}$ (CS paired with US) using the time modulation function as implemented in SPM ($p < 0.05$).

Task-related activity (BOLD response) for each event was modeled by convolving a vector of the event onset times with a canonical hemodynamic response function (HRF). The regressors of interest were included together with time as covariate in a GLM to yield parameter estimates for brain activation related to the different event types (US, CS−, $\text{CS+}_{\text{paired}}$, $\text{CS+}_{\text{unpaired}}$). T contrast images for $\text{CS+}_{\text{paired}} > \text{CS−}$ and $\text{CS−} > \text{CS+}_{\text{paired}}$ were computed for each conditioning phase (familiarization, early acquisition, late acquisition, extinction, extinction recall, reacquisition) separately.

At the second level (group level), the resulting first level contrast images were entered into random effect models: *Within-group* (one sample) *t* tests were used to compare brain activity during CS+ to brain activity in response to CS−. *Between-group* (two sample) *t* tests were used to analyze group differences in response to CS+, CS−, as well as $\text{CS+}_{\text{unpaired}} > \text{CS−}$ during the different conditioning phases. Clusters were determined using an extent threshold of $p < 0.05$ on the voxel-wise whole-brain level. The false discovery rate (FDR) correction was applied to correct for multiple comparisons.

Region of interest analyses Based on our a priori hypotheses (stronger activation in the amygdala and insula during acquisition and reacquisition and less activation in the mPFC during within-session and between-session extinction in BPD patients than HC), regions of interest analyses (ROIs) were performed for the following brain areas: amygdala, ACC, hippocampus, insula, OFC, dlPFC, and vmPFC. Anatomically based ROIs of these regions were created using the Masks for Regions of Interest Analysis (MARINA) software program (Bertram Walter, Bender Institute of Neuroimaging, University of Giessen, Germany). These masks were then used for small volume corrections.

In a separate model, state dissociation ratings were included as a covariate in order to test the effect of this

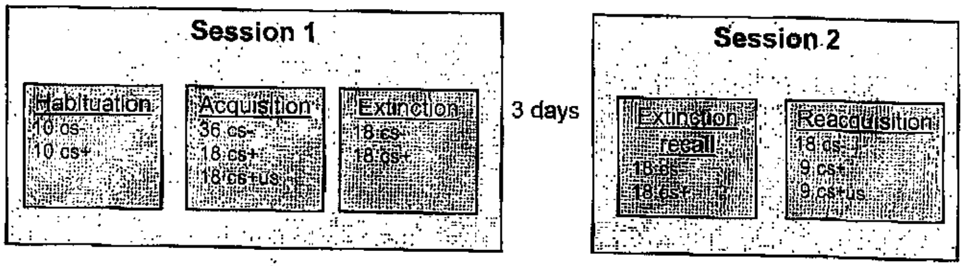

Fig. 1 Experimental design: Ratings of valence and arousal were performed after each of the five experimental phases. *CS+* conditioned stimulus paired with the US; *CS−* non-reinforced conditioned stimulus, *US* unconditioned stimulus

variable on the results. Furthermore, a subgroup analysis with BPD patients with versus without current PTSD compared to HC was computed.

Results

Ratings

Means and standard deviations of arousal (a) and valence (b) ratings during acquisition, extinction, extinction recall, and reacquisition in HC and BPD patients are depicted in Fig. 2.

For arousal ratings after acquisition, a significant effect of type [$F(1,34) = 23.929$, $p < 0.001$] but no significant interaction with group was found [$F(1,34) = 0.001$, $p = 0.973$]: All participants showed a higher rating of the CS+ compared to the CS−. For the arousal ratings after extinction and extinction recall, no significant effect of type [$F(1,30) = 0.250$, $p = 0.621$; $F(1,24) = 0.566$, $p = 0.459$] and no significant interaction with group were found [$F(1,30) = 0.009$, $p = 0.925$, $F(1,24) = 0.452$, $p = 0.508$]. After extinction, BPD patients rated both stimuli as more aversive than the HC indicated by a significant effect of group [$F(1,30) = 5.565$, $p = 0.025$].

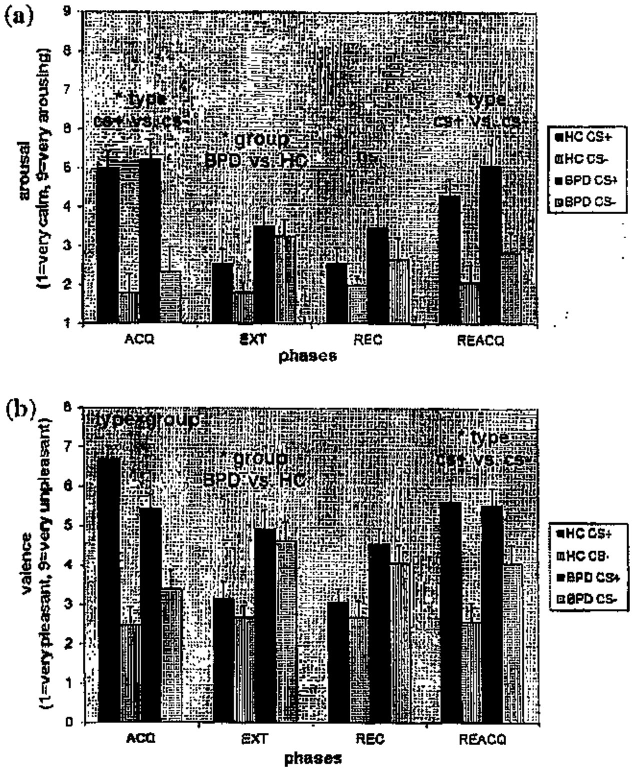

Fig. 2 Results of the arousal (a) and valence (b) ratings. *ACQ* acquisition, *EXT* extinction, *REC* extinction recall, *REACQ* reacquisition, *HC* healthy controls, *BPD* BPD patients, *CS+* conditioned stimulus paired with the US, *CS−* non-reinforced conditioned stimulus, significant at $^{*}p < 0.05$, $^{\#}p < 0.10$

For the arousal ratings after reacquisition, a significant effect of type [$F(1,24) = 18.315$, $p < 0.001$] and no significant interaction with group were found [$F(1,24) = 0.337$, $p = 0.567$]: All participants showed a more aversive rating of the CS+ compared to the CS− (see Fig. 2a).

For the valence ratings after acquisition, a significant effect of type [$F(1,34) = 15.199$, $p < 0.001$], with more aversive rating of the CS+ than the CS−, and a significant interaction effect type × group [$F(1,34) = 4.223$, $p = 0.048$] were found. For the valence ratings after extinction and extinction recall, no significant effect of type [$F(1,30) = 0.334$, $p = 0.568$; $F(1,24) = 1.189$, $p = 0.286$] and no interaction with group were found [$F(1,30) = 0.021$, $p = 0.886$, $F(1,24) = 1.156$, $p = 0.293$]. After extinction, again, a significant effect of group was found for the valence ratings [$F(1,30) = 14.944$, $p = 0.001$]: BPD patients rated both stimuli as more aversive than the HC. For the valence ratings after reacquisition, a significant effect of type [$F(1,24) = 14.138$, $p = 0.001$] but no interaction with group was found [$F(1,24) = 0.199$, $p = 0.660$]: All participants showed a more aversive rating of the CS+ compared to the CS− (see Fig. 2b).

Skin conductance response (SCR)

Means and standard deviations of SCR during acquisition, extinction, extinction recall, and reacquisition in HC and BPD patients are shown in Fig. 3. During acquisition, no significant effect of type [$F(1,34) = 2.291$, $p = 0.139$, $\eta^2 = 0.063$] and no significant interaction with group were found [$F(1,34) = 0.039$, $p = 0.844$]. During extinction, no significant interaction with group was found [$F(1,33) = 0.962$, $p = 0.334$], but a significant effect of type [$F(1,33) = 9.895$, $p = 0.003$] with participants shows stronger responses to the CS+ than to the CS−. During extinction recall, no significant effect of type [$F(1,20) = 0.209$, $p = 0.652$] and a trend for the interaction effect type x group were found [$F(1,20) = 3.194$, $p = 0.089$], with BPD patients showing descriptively a stronger response to CS+ compared to the CS−, whereas HCs showing descriptively a stronger response to CS− compared to the CS+. During reacquisition, no significant effect of type [$F(1,20) = 1.967$, $p = 0.176$, $\eta^2 = 0.090$] and no interaction with group were found [$F(1,20) = 1.768$, $p = 0.199$, $\eta^2 = 0.081$].

fMRI

Between-group t tests

No significant group differences on the whole-brain level could be detected at a threshold level of $p < 0.05$ after correction for multiple comparisons (FDR correction).

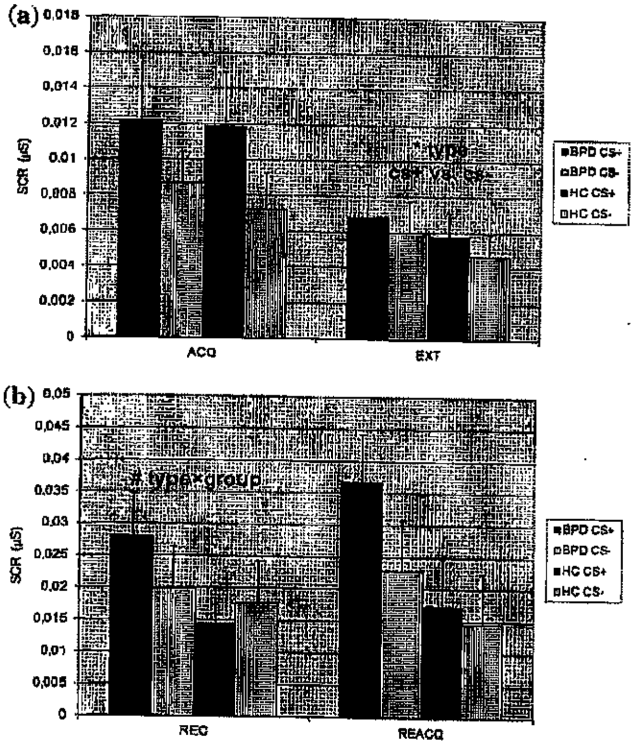

Fig. 3 Skin conductance response. *Note ACQ* acquisition, *EXT* extinction, *REC* extinction recall, *REACQ* reacquisition, *HC* healthy controls, *BPD* BPD patients, *CS+* conditioned stimulus paired with the US, *CS−* non-reinforced conditioned stimulus

Within-group t tests

Results for brain activity in response to CS+$_{\text{unpaired}}$ and CS+$_{\text{unpaired}}$ > CS− can be found in Table 1. In the following, results are reported for each group and for each conditioning phase separately [p(FDR) < 0.05].

Healthy control group

During *early acquisition*, no significant clusters were observed for the CS+$_{\text{unpaired}}$ > CS− contrast. During presentation of the CS+$_{\text{unpaired}}$, HC recruited the fusiform gyrus (BA37) and right posterior cingulate. Our ROI analyses further revealed significant CS+$_{\text{unpaired}}$-related activation in the bilateral parahippocampal gyrus and left amygdala.

During *late acquisition*, HC showed significant stronger activation in response to the CS+$_{\text{unpaired}}$ than to CS− in the bilateral inferior frontal gyrus (BA46 and BA47), bilateral inferior parietal lobule, right middle frontal gyrus (BA46), and left cerebellum.

During *extinction*, HC exhibited significant differential activation for CS+$_{\text{unpaired}}$ > CS− in the right superior frontal gyrus (BA10) and left middle frontal gyrus (clusters revealed by ROI analyses). In addition, our ROI analyses revealed significant CS+$_{\text{unpaired}}$-related activation in the right OFC, left inferior frontal gyrus (BA47), and bilateral middle frontal gyrus.

During *extinction recall*, no significant clusters were found for the CS+$_{\text{unpaired}}$ > CS− contrast. In response to the CS+$_{\text{unpaired}}$, HC activated the right lingual gyrus and left cuneus, the left middle frontal gyrus and superior frontal gyrus (BA9), the left parahippocampal gyrus, right middle temporal gyrus, and left tuber (cerebellum).

During *reacquisition*, our ROI analyses revealed significant clusters in the bilateral medial frontal gyrus (BA10), left superior frontal gyrus, and left middle frontal gyrus (BA8) for the CS+$_{\text{unpaired}}$ > CS− contrast. In response to the CS+$_{\text{unpaired}}$, HC showed recruitment of the bilateral superior temporal gyrus and insular cortex (BA13), bilateral middle frontal, and superior frontal. For the CS+$_{\text{unpaired}}$ contrast, ROI analyses additionally revealed significant clusters in the left amygdala and left parahippocampal gyrus.

BPD group

During *early acquisition*, BPD patients showed significant differential activation for CS+$_{\text{unpaired}}$ > CS− in the right superior and middle frontal gyrus as well as in the bilateral superior temporal gyrus. ROI analyses further revealed significant stronger activation in the left insula (see Fig. 4a) for the CS+$_{\text{unpaired}}$ > CS− contrast.

During *late acquisition*, BPD patients exhibited significant stronger activation in response to CS+$_{\text{unpaired}}$ > CS− in the bilateral middle and medial frontal gyrus (BA8, BA9, BA10, BA46), right inferior frontal gyrus, left middle occipital gyrus (BA18), and right inferior parietal lobule (BA40). In response to the CS+$_{\text{unpaired}}$, in addition, patients recruited the right inferior temporal gyrus and left middle temporal gyrus.

During the *extinction* phase, no significant clusters were observed for the CS+$_{\text{unpaired}}$ > CS− contrast. In response to CS+$_{\text{unpaired}}$, BPD patients activated the right orbitofrontal gyrus (BA 11) and right amygdala (see Fig. 4b).

During *extinction recall*, BPD patients exhibited CS+$_{\text{unpaired}}$-related activation in the right cuneus and left parahippocampal gyrus as well as in the right insula. For the CS+$_{\text{unpaired}}$ > CS− contrast, ROI analyses revealed stronger activation in the orbitofrontal gyrus (BA11) as well as medial frontal gyrus (BA8) in the patient group.

During *reacquisition*, we found significant clusters in the middle frontal gyrus (BA10) for the CS+$_{\text{unpaired}}$ > CS− contrast as well as clusters in the orbitofrontal gyrus (BA11) and ACC (BA24) for the CS+$_{\text{unpaired}}$ contrast.

Linear habituation

Complete results for linear time effects during the acquisition phase in each group can be found in Supplemental Table 4.

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Operant Conditioning

Bridging the positions of Rogers and Skinner: The role of nonlinear dynamic systems

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This paper aims to bridge the gap between the positions of Carl Rogers and B. F. Skinner by demonstrating that they are not as antithetical as they are ordinarily assumed to be, and as the two men themselves tended to think. The author uses the concept of nonlinear dynamic systems as the principal concept in her effort to bridge the gap between the work of Rogers and Skinner and consequently introduces the concept of nonpredictivity to replace the concept of nondirectivity as being a more correct and basic characterization of classical client-centered therapy. The concept of nonpredictivity allows client-centered therapy to be framed, in an overarching and general way, by B. F. Skinner's principle of operant conditioning without doing violence to Rogers' concern about client autonomy. More specifically, the paper argues that Rogers' theory of therapy is not antithetical to Skinner's theory of personality and that client-centered therapy therefore can be practiced seamlessly by therapists who, like the author, believe that operant conditioning is a better explanatory concept for human behavior than the actualizing tendency.

Keywords: Rogers and Skinner; nonlinear dynamic systems; nonpredictive; nondirective

Brückenschlag zwischen den Positionen von Rogers und Skinner: die Rolle nicht-linearer dynamischer Systeme

Dieser Artikel will die Kluft zwischen den Positionen von Rogers und Skinner schliessen. Diese sind nicht so anti-thetisch, wie man normalerweise annimmt und wie die beiden selbst meinten. Ein Konzept nicht-linearer dynamischer Systeme soll die Kluft zwischen Rogers' und Skinners' Werk schliessen. Das Konzept der Nicht-Vorhersagbarkeit soll das Konzept der Nicht-Direktivität ersetzen; ersteres ist eine korrektere und grundlegendere Charakterisierung der klassischen Klientzentrierten Therapie. Das Konzept der Nicht-Vorhersagbarkeit ermöglicht es, Klientzentrierte Therapie in umfassender und genereller Weise in Skinners Prinzip des Operanten Konditionierens einzubetten, ohne dass man Rogers' Sorge um die Autonomie der Klienten Gewalt antut. Rogers' Therapietheorie ist keine Antithese zu Skinners Theorie der Persönlichkeit und Klientzentrierte Therapie kann daher nahtlos von Therapeuten praktiziert werden, die wie die Autorin überzeugt sind, dass Operantes Konditionieren ein besseres Erklärungskonzept für menschliches Verhalten ist als die Aktualisierungstendenz.

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Estableciendo un puente entre las posturas de Rogers y Skinner: El rol de los sistemas dinámicos ni lineales

Este escrito tiene como fin establecer un puente entre las posiciones de Carl Rogers y B. F. Skinner al demostrar que no son tan antagónicas como normalmente se las considera, y como ambos autores tendían a considerarlas. La autora utiliza el concepto de sistemas dinámicos no lineares como el concepto principal en su esfuerzo para salvar la distancia entre la obra de Rogers y la de Skinner, y por ello introduce el concepto de no predictibilidad para reemplazar el concepto de no directividad, considerándolo una caracterización más exacta del la terapia centrada en el cliente clásica. El concepto de no predictibilidad permite que ubiquemos la terapia centrada en el cliente en un marco más abarcativo y general, por el principio de Skinner de condicionamiento operativo sin violentar la preocupación de Rogers acerca de la autonomía del cliente. Más específicamente, este escrito argumenta que la teoría de Rogers acerca de la terapia no es contraria a la teoría de la persónalidad de Skinner y que por lo tanto la terapia centrada en el cliente puede ser aplicada por terapeutas, como la autora, creen que el condicionamiento operante es un concepto que explica el comportamiento humano mejor que la tendencia actualizante.

L'élaboration d'un pont entre les positions de Rogers et de Skinner: Le rôle de systèmes non-linéaires et dynamiques

Cet article vise à créer un pont entre les positions de Carl Rogers et B.F. Skinner en démontrant qu'elles ne sont pas aussi antithétiques qu'on le suppose d'ordinaire ou que les deux hommes avaient tendance à les penser. L'auteure utilise le concept de systèmes non-linéaires et dynamiques comme concept principal dans son effort de créer un pont entre le travail de Rogers et de Skinner. Il s'ensuit qu'elle propose le concept de non-prédictibilité pour remplacer le concept de non-directivité en tant que caractérisation plus juste et plus fondamentale de la thérapie centrée sur le client classique. Le concept de non-prédictibilité permet à la thérapie centrée sur le client d'être encadrée, d'une manière globale et générale, par le principe de conditionnement opérant de Skinner sans faire violence à la préoccupation de Rogers concernant l'autonomie du client. Plus spécifiquement, l'article soutient que la théorie de la thérapie de Rogers n'est pas antithétique par rapport à la théorie de la personnalité de Skinner et que la thérapie centrée sur le client peut donc être pratiquée sans heurt par des thérapeutes qui, comme l'auteure, croient que le conditionnement opérant est un concept qui explique mieux le comportement humain que la tendance actualisante.

Fazendo a ponte entre as posições de Rogers e de Skinner: O papel dos sistemas dinâmicos não lineares

Este artigo pretende estabelecer uma ponte entre as posições de Carl Rogers e de B.F. Skinner, ao demonstrar que os dois autores não são antitéticos como habitualmente se julga e como eles próprios tendiam a ver-se um ao outro. A autora recorre ao conceito de sistemas dinâmicos não lineares como sendo central no estabelecimento dessa ponte entre os trabalhos de Rogers e de Skinner e, consequentemente, introduz o conceito de não previsibilidade para substituir a não directividade, considerando o primeiro como permitindo uma caracterização mais correcta e básica da terapia centrada no cliente clássica. O conceito de não previsibilidade permite que se enquadre a terapia centrada no cliente, de forma mais abrangente e geral, através do princípio skinneriano de condicionamento operante, sem violentar a preocupação de Rogers com a autonomia do cliente. Mais concretamente, este artigo pretende que a teoria da terapia de Rogers não é antagónica com a teoria da personalidade de Skinner e que, consequentemente, a terapia centrada no cliente pôde ser colocada em prática, sem problemas, por terapeutas que, como o autor, acreditam que o condicionamento operante é um conceito mais adequado para o comportamento humano do que a tendência actualizante.

ロジャーズとスキナーの立場の橋渡し：非線形力動的システムの役割について

本論文の目的は、カール・ロジャーズとB.F.スキナーの立場が「対立的」ではないことを示し、彼らの間にある相違を埋めることである。ロジャーズとスキナーの業績の間にある相違を埋めるための重要な概念として、非線形力動的システムを用いた。古典的な来談者中心療法におけるより適切で基本的な特徴として、“非指示性”の代わりに“非予測性”を提案した。そして、B.F.スキナーのオペラント条件付けの原理による“非予測性”の概念により、来談者中心療法は、クライエントの自主性に関するロジャーズの懸念を害することなく、より包括的で普遍的に形作られるとし、ロジャーズのセラピーに関する理論がスキナーのパーソナリティ理論と正反対のものではないことを示した。そして、実現傾向よりもオペラント条件付けが人間の行動の説明概念として優れていると信じている治療者であったとしても、来談者中心療が全ての学派にわけ隔てなく実践されうると論じた。

En brobygger mellem Rogers og Skinner: De ikke-lineære dynamiske systemers rolle

Denne artiikel har til formål at bygge bro mellem Carl Rogers' og B. F. Skinners positioner ved at demonstrere at de ikke er så anti-tetiske som almindeligt antaget og som de to mænd selv anså dem for at være. Forfatteren bruger begrebet "ikke-lineære dynamiske systemer" som sit vigtigste begreb i forsøget på at bygge bro mellem Rogers' og Skinners teorier, og som en konsekvens heraf introducerer hun begrebet "ikke-forudsigende" til erstatning for begrebet "ikke-direktiv," idet hun anser førstnævnte for at være en mere basal og korrekt karakterisering af klassisk klient-centreret terapi. Begrebet "ikke-forudsigende" tillader klient-centreret terapi at blive kontekstualiseret, på overordnet og generel vis, af B. F. skinners princip om operant betingning uden at gøre vold på Rogers' omsorg for klientens autonomi. Mere specifikt argumenterer artiklen for at Rogers' teori om terapi ikke er antitetisk til Skinners personlighedsteori og for at klient-centreret terapi derfor kan praktiseres gnidningsfrit af terapeuter der, som forfatteren, tror at operant betingning er et bedre begreb til forklaring af menneskers adfærd end den aktualiserende tendens.

Introduction

Writing this paper has been close to the heart of the author who finds so much of value in Rogers' as well as in Skinner's works that it has become increasingly inconceivable to comprehend their views as being excluding of or in contradiction with each other. Rogers and Skinner were, after all, among the giants of 20th century psychology. It therefore seems most likely that much of value can be found in both men's work.

If Rogers and Skinner could have had a third dialogue, today, informed by late 20th century advances in work with nonlinear dynamic systems (see below), they might have ended up on the same page and there might not have been the split between the behaviorist orientation and the humanist orientation in psychology that we see today. They agreed (Rogers & Skinner, 1956, 1962/1989) that human behavior is determined, although Rogers had some reservations, because he found that Skinner attached too little importance to the inner, subjective experiences of the individual, particularly the experience of being free and having choices. Rogers did not clarify, though, what kind of importance should be attached to these inner, subjective experiences, apart from stating in various ways that they were important because they felt important and meaningful to the individual. He spoke of a "paradoxical" relationship between scientific determinism and inner, subjective experiences. He said (1962/1989, p. 132):

> I am in thorough agreement with Dr. Skinner that, viewed from the external, scientific, objective perspective, man is determined by genetic and cultural influences. I have also said that in an entirely different dimension, such things as freedom and choice are extremely real ... I see it as being similar to the situation in physics where you can prove that the wave theory of light is supported by evidence, as is the corpuscular theory, though the two of them appear to be contradictory. They are not, at the present state of knowledge, reconcilable, but one would be narrowing his perception of physics to deny one or the other. It is in this same sense that I regard both of these dimensions as real, although they exist in paradoxical relationship. [Physicists, today, would not speak of a paradoxical relationship, but of a complementary relationship between the wave theory and the corpuscular theory of light. (This author's comment)]

However, talking about a paradoxical relationship between scientific determinism and the experience of freedom doesn't really explain anything and, significantly, Rogers did not state that he attached causative agency to inner experiences, or to the experience of being free to make choices, that is, to the experience of having a free will. On the contrary, he said (Rogers, 1962/1989, p. 99): "Behaviors that were formerly dealt with as though they were caused by little homunculi within the individual, or various internal causes, are now seen to have other types of causation."

Rogers' reservation to Skinner's views seems first and foremost to originate in Rogers' feeling of no less than revulsion (1962, p. 126) at the thought that human behavior could be controlled *ad modum* Walden Two (Skinner, 1948). Skinner took the position that as much as he acknowledged the existence of inner, subjective experiences, including the experience of choosing freely, he regarded them as more or less covert or private behaviors, and it was the environmental consequences of a person's behavior, not any kind of inner experiences, that should be studied, because those consequences were the causes (in the form of rewards or punishments) of the behavior. By engineering the environmental consequences of a person's behavior, it was, according to Skinner, possible to control this person's behavior and that prospect was as positive from Skinner's point of view as it was negative from Rogers' point of view.

Basically, however, they were in agreement about the assumption that determinism implies predictability. Today, they would have been informed about Chaos Theory and nonlinear dynamic systems, that is, they would have known that determinism does not necessarily imply predictability. In the light of this knowledge, they might have appreciated that their respective positions were, indeed, compatible. Skinner might have understood that in spite of his being right about the principle of operant conditioning and determinism for living organisms, in general, he could not hope to be able to control the rich and complex behavior of any human individual, in particular. A Walden Two could never be realized. Rogers, on his side, might have understood that his agreeing with Skinner about determinism did not exclude the soundness and relevance of his nonpredictive therapeutic approach with any individual client.

The impossibility of predictive control of complex sets of behavior

A basic assumption

It is a basic assumption of this paper that the trajectory of the process of the individual human organism's interaction with its environment from conception to death is best modeled by a *nonlinear dynamic system* unlike, for example, the trajectory of the planets around the sun, which is, as physics has long since demonstrated, best modeled by a linear dynamic system.

Nonlinear dynamic systems[1]

Classical Newtonian science is almost exclusively concerned with linear dynamic systems. In such systems, cause–effect relationships can be clearly and exhaustively defined. This allows for very precise predictions of the behavior of a linear dynamic system. With measurements of a few parameters, the trajectory of a planet, for example, can be very precisely predicted. This is so even if no set of measurements can be 100% precise. Physicists and mathematicians say about linear dynamic systems that small differences in initial conditions remain small differences in the future behavior of the system. For all practical purposes, therefore, small differences in initial conditions can be disregarded. Stated otherwise, one can make one's measurements as precise as one wishes one's predictions to be precise. This is the essentially characterizing feature of linear dynamic systems.

Such is not the case with nonlinear dynamic systems. They are said to be sensitively dependent on initial conditions. This means that small differences in initial conditions can develop into big differences in the future behavior of the systems. *The butterfly effect* is the ubiquitously used term for this sensitive dependence on initial conditions. To illustrate what nonlinear dynamic systems are about, it is said that a butterfly flapping its wings in Beijing on Sunday can be decisive for the weather in New York next Saturday. This means that the development of the weather around the globe is a nonlinear dynamic system, which is sensitively dependent on initial conditions, and it is the reason meteorologists cannot predict the weather with better than chance precision for much more than four or five days ahead.[2]

To the surprise of most people, mathematics, that is, the area that is normally thought of as the most predictable of all, is full of nonlinear dynamic systems. Many mathematical equations show sensitive dependence on initial conditions. If one iterates (develops) such equations on a calculator that delivers results with nine decimal points' precision and on a calculator that delivers results with eight decimal points' precision one will find that the iterations on the two calculators sooner or later start to vary widely and seemingly chaotically, as a result of the tiny, one millionth part, difference in initial conditions. *Chaos Theory* is the field of mathematics that is concerned with nonlinear dynamic systems (see, e.g., Gleick, 1987; Lorenz, 1993; Peitgen, Jürgens, & Saupe, 1992). The name is somewhat unfortunate, since there is really nothing "chaotic" about these systems. They are fully determined systems. The meaning of the term *chaotic* is, more precisely, *unpredictable*. Nonlinear dynamic systems are as determined as linear dynamic systems, but they are as unpredictable as linear dynamic systems are predictable.

Here is another example of a nonlinear dynamic system:

Imagine that you let go of a small bullet at the edge of a large funnel with a hole in the bottom, just a tiny bit larger in diameter than the bullet. You can, of course, on the basis of the law of gravity, predict that it will end up disappearing down the hole in the bottom, but there is no way you can predict its trajectory toward that disappearance. That trajectory is sensitively dependent on the tiniest differences in the way you hold the bullet towards the edge of the funnel at the start, on the tiniest differences in where you hold it, on the tiniest differences in the way you let go of it, and on the tiniest details of irregularities on the side of the funnel. Any of these tiny differences can have a huge effect on the bullet's trajectory. At the same time, these differences are so tiny that there is no way to measure them, just as there is no way to measure their combined differential influence on the bullet's behavior in a way that is sufficiently precise to make it possible to predict the trajectory of the bullet towards its disappearance. One can only make the prediction (obvious to everyone) that the bullet's overall trajectory will be downwards, that it will probably not disappear right away, but instead hit the edge of the hole in the bottom, and be spiraled up again, one time or several times, but still closer to the bottom and still more slowly as it loses energy, until it finally disappears.

At this point, the reader is welcome to associate to the human organism's trajectory through life, since my point with this rather detailed explanation of nonlinear dynamic systems is, as already stated, to postulate that the behavior of the human organism, that is, its continuously changing complex sets of interactions with a continuously changing environment, from conception to death, is a nonlinear dynamic system, and, probably, *the* nonlinear dynamic system, par excellence. The human organism in its environment is, more than anything, like the bullet in the funnel that was described above: Its trajectory through life is as sensitively dependent on initial conditions as the bullet's trajectory on the side of the funnel.

Determined and unpredictable

The consequence of this assumption is that the interactional processes of human beings with their environment are determined *and* unpredictable. It is very important to grasp the idea that something can be both determined and unpredictable. It is important, because our language and thinking is so suffused with the linearity of classical science that we automatically tend to think of determined systems as being synonymous with predictable systems. It is therefore no wonder that Rogers and Skinner did not question the basic, mistaken, assumption that they had in common: that determinism invariably implies predictability. It was only with the advent of the modern computer that mathematicians and physicists could investigate nonlinear dynamic systems thoroughly, and knowledge about nonlinear dynamic systems did not become common knowledge until the two last decades of the past century. At the time of the dialogues between Rogers and Skinner, thinking in terms of predictable, linear dynamic systems was still the way of thinking that dominated the sciences.

The individual level and the general level

Rogers and Skinner, though, were not altogether wrong about their assumption that determinism implies predictability. They were only wrong on the level of the individual human organism's interaction with the environment, and they were not very careful about distinguishing between the individual and the general level in their dialogues. Their failure to do so may have contributed to their thinking of themselves as being in disagreement. On the general level, that is, on the level of people, at large, or of sufficiently large groups of people, or on the level of "the average," they were right. And it may be this level Rogers had in mind when he spoke of the "scientific perspective" in the quotation above. On this level Skinner's laws of conditioning can be applied to predict which environments will produce a certain kind of behavior among the majority of people exposed to this environment. And on this level Rogers and Skinner were actually in rather close agreement about the general kind of environment that would produce the most constructive behaviors in the majority of people or, vice versa, they were in agreement about the kind of environment that hampered the actualization of these behaviors, that is, they were in agreement about a general theory of disturbed psychological development or, in Skinner's terminology, disturbed behavior. For Rogers, psychological disturbance came about as a result of the individual having been excessively exposed to conditional regard. For Skinner, disturbed behavior came about as a result of the individual having been excessively exposed to punishing consequences.

Skinner's theory operates with four reinforcers, two positive (increasing the likelihood of recurrence of a certain set of behaviors) and two negative (decreasing the likelihood of recurrence of a certain set of behaviors). The two positive reinforcers are (1) finding rewarding consequences, and (2) escaping from punishing consequences; and the two negative reinforcers are (3) finding punishing consequences, and (4) finding that rewarding consequences have disappeared (O'Donohue & Ferguson, 2001, p. 92).

For Rogers conditional positive regard is actually implicitly punishing of behaviors that are not regarded positively: "I love you when you . . .," implies that "I do not love you when . . . " and withdrawal of love can be as painful to humans as electric shock to rats.

Thus, it is hard to see any essential difference between "conditional regard" and "punishing consequences."

Rogers, however, employed the concept "unconditional positive regard" that has no counterpart in Skinner's work, and this concept of Rogers', although he didn't explicitly say so anywhere, embraces the unpredictability of human behavior that went beyond Skinner's theory. (See further, below.) Rogers, more than Skinner, had a "sense" of nonlinear dynamics, before knowledge of these dynamics became accessible.

In any case, they were in agreement about the essential point: that disturbed psychological development or disturbed behavior is the result of unfortunate environments, that is, it is conditioned and thus controlled by the environment, even if it is impossible to tell, on the individual level, what the specific conditioning contingencies or reinforcement relationships of any given set of disturbed behaviors might have been and may be. The logical conclusion is that they were also in agreement that any behavior, not only disturbed behavior, is controlled by the environment. The evidence is Rogers' statement, in the debate with Skinner, that "I am in thorough agreement with Dr. Skinner that, viewed from the external, scientific, objective perspective, man is determined by genetic and cultural influences" (1962/1989, p. 132; see the full quotation above). In another context Rogers wrote:

> The question is often raised: But what about the therapist's attitude toward his client's asocial or antisocial behavior? Is he to accept this without evaluation? Sometimes this question is answered by saying that the effective therapist prizes the person, but not necessarily his behavior. Yet it is doubtful if this is an adequate or true answer. To be sure, the therapist may feel that a particular behavior is socially unacceptable or socially bad, something he could not approve of in himself, and a way of behaving which is inimical to the welfare of the social group. But the effective therapist may feel acceptant of this behavior in his client, not as desirable behavior, but as a *natural consequence* of the circumstances, experiences, and feelings of this client. Thus the therapist's acceptance may be based upon this kind of feeling: "If I had had the same background, the same circumstances, the same experiences, it would be inevitable in me, as it is in this client, that I would act in this fashion." (Rogers, Gendlin, Keisler, & Truax, 1967, p. 103)

Here Rogers is fully on the same wavelength as Skinner.

Rogers' dilemma

As we have seen, Rogers, however, had problems with this and he also often spoke about the self-governing, internally controlled individual. For example, in his definition of the actualizing tendency (Rogers, 1959, p. 196) he spoke of development "toward autonomy and away from heteronomy, or control by external forces." It seems to be this apparent contradiction in Rogers' view that he tried to resolve with reference to the "paradoxical" relationship between the individual experience of freedom and the notion that human behavior is determined.

Rogers also expressed his efforts to integrate the idea of individual freedom on the one hand and determinism on the other in the following quote:

> The fully functioning person ... not only experiences, but utilizes, the most absolute freedom when he spontaneously, freely, and voluntarily chooses and wills that which is also absolutely determined. (Rogers, 1961, p. 193)

It should be evident from these quotations that Rogers struggled hard with the free will/determinism issue without resolving it. The author wonders why Rogers seemingly did not pay attention to the everyday occurrence that subjective experience and objective knowledge can be held simultaneously in mind: We can enjoy the beautiful sunset while knowing full well that the sun is not at all "setting"; instead the earth is revolving around it. Likewise, we can hold in mind, simultaneously, the enjoyment of freedom while knowing full well that the experience of freedom is a subjective experience that is as determined as everything else and certainly not an experience enjoyed by all.

Skinner's mistake

It is undoubtedly the impossibility of making sufficiently precise predictions about the individual conditioning contingencies of a given client's behavior that is at the heart of the problems of behavior analytic therapy, that is, the therapeutic application of Skinner's laws of conditioning on the level of individual clients. The engineering of specific reinforcement strategies to change a given "target behavior" demands isolation of the target behavior and prediction about the reinforcement strategies to be applied to change the target behavior, and this is, more often than not, impossible with the complex kind of behaviors and problems ordinary clients present to ordinary practicing therapists. The rare client may present with sufficiently simple and well-delineated kinds of symptom/problem/target behavior for effective reinforcement strategies to be successfully predicted and applied. In these cases, it is possible to approach the client's "presenting problem" to a linear dynamic system and to work with it accordingly. To remain with the bullet in the funnel analogy, this would roughly correspond to a whole in the bottom that was sufficiently large to make it obvious that the bullet would disappear directly into it independently of any differential influences on its way to the bottom. As early as the middle of the last century numerous studies, referred to by Patterson (1963), showed that carefully chosen, well-delineated and obvious "bits" of behavior can be changed by predictive operant conditioning. Ordinarily, though, this kind of approximation to a linear dynamic system is not possible in therapy without disregarding essential features of the relatively complex problems that clients typically see therapists about. These problems are part of a unique nonlinear dynamic system that is as unpredictable as it is determined. With ordinary clients, the diameter of the hole in the bottom is not much larger than the diameter of the bullet, the side of the funnel is very irregular and there are an infinite number of variations in the ways different bullets were started on their unique trajectories toward the bottom.

Therefore, Chaos Theory and the basic unpredictability of nonlinear dynamic systems falsify Skinner's assumption of the predictability of the behavior of the individual human organism. Rogers' opposition to this point of Skinner's ideas seems to have been, primarily, ethically motivated, but later developments in mathematics and physics came to his support: Rogers' idea that a therapist should not try to control the behavior of any individual client gains momentum by the idea that the therapist, quite simply, *cannot* control the behavior of any given client.

A third dialogue

Thus, if Rogers and Skinner could have had a third dialogue, today, Rogers, in the light of the basic unpredictability of nonlinear dynamic systems, might not have been as uncomfortable about Skinner's deterministic position as he was in 1957 and 1962. Rogers might more unequivocally have agreed with Skinner that the human organism is determined and Skinner might have agreed that Rogers was right that the complex sets of interaction of a client with his or her environment should not be exposed to efforts of direction or control in therapy; not, primarily, because such efforts were ethically wrong, but because such efforts were, more likely than not, doomed to failure, since they would imply therapist capacity to make appropriate predictions about the client at odds with the trajectory of client changes being best modeled by a nonlinear dynamic system.

Classical client-centered therapy: Nonpredictive rather than nondirective

Classical client-centered therapy is regarded by most practitioners as being utterly nondirective (see, e.g., Bozarth, 1998, p. 57; Grant, 1990; Levitt & Brodley, 2005). It is the therapy of the "early Rogers" or "Rogers-1" (Frankel & Sommerbeck, 2005, 2007) that Rogers described in *Counseling and Psychotherapy* (1942), where he distinguished between "directive" approaches and his own "nondirective" approach (1942, pp. 115–128). He articulated the philosophical foundation of this therapy in more detail in *Client-Centered Therapy* (1951). Nevertheless, most would probably agree that classical-client centered therapists do direct, in the sense of influence, their clients, just as clients influence their therapists, and just as we all influence each other all the time. Skinner would say that control or conditioning is mutual; it goes both ways. Therefore, the essence of nondirectivity in classical client-centered therapy is, more precisely, that the therapist does not *systematically* try to influence or direct the client. The therapist has no preconceived, that is, *predictive* idea, based on diagnostic assessment, a particular theory of personality, or any other perspective of their own, of how and in which direction they wish to influence the client, and the therapist entertains no such ideas at any moment during the course of therapy. In short, the therapist makes no effort to predict what will be helpful to a given client, but engages instead in continuous empathic understanding of the client's perspective.

This does not mean that expert predictions do not exist. However, expert predictions are made on the basis of experts' knowledge of what characterizes some average of a group of people or some sufficiently large group. Such predictions can be made with satisfying precision when the average process and the actual process are virtually the same, which is the case with linear dynamic systems like, for example, the trajectory of the planets around the sun. But for nonlinear dynamic systems, where small differences in initial conditions can develop into very large differences at a later point in time, it is impossible to know how widely the known average and the actual system of interest at this precise moment in time differ from each other, except in very crude and banal ways. Psychologists and psychiatrists, for example, are experts at how a sufficiently large group of people with a certain diagnosis responds to this or that intervention, or how the "average" person with this diagnosis responds, but have no way of knowing how close any given client with the given diagnosis is to this average (Sommerbeck, 2004, pp. 295–297). Therefore:

> Any intervention with the process of another, which is made with the intention of being helpful, as will most often be the case in therapy, is made on the basis of a prediction that this intervention will actually, more likely than not, be helpful for the other. Otherwise, the intervention would not be made. In making such predictions, the other is treated as a linear dynamic system, as "an average person" or as "an average client" on the basis of theory, research, or the therapist's professional or personal experience. The other is not treated as this particular, unique, nonlinear dynamic system, John, who is different from any average, or as this other, different and equally unique, nonlinear dynamic system, Jenny, whose future interactions with her environment it is impossible to make predictions about. (Sommerbeck, 2004, p. 296)

To this author, Rogers' fourth condition of the six necessary and sufficient conditions for therapeutic change (Rogers, 1959, p. 213), unconditional positive regard for the client, is, first and foremost, respect for the uniqueness of each client, or respect for the client being a nonlinear dynamic system that it is impossible to make anything but banal and crude, that is, more often than not therapeutically irrelevant, predictions about. It is also respect for the importance of clients' subjective *experience* of freedom, while knowing that objectively the client is as determined as the therapist or anything/anyone else, and that the therapy, as a whole, is but another determining or conditioning influence, albeit a nonpredicted influence, in the life of the client and in the life of the therapist. The only prediction made by the classical client-centered therapist is that the client will, more likely than not, be better off at the conclusion of the therapy than at the start of it, but what this means, in any detail, is left blowing in the wind.

A personal note

This paper represents the way the author, for herself, has tried to resolve Rogers' "paradox" regarding freedom and determinism, and the way she can effortlessly and satisfyingly contain the wisdom of Rogers' client-centered therapy and the wisdom of Skinner's principle of operant conditioning at the same time. It is the way for the author to be dedicated to Rogers' theory of therapy, while being critical of Rogers' theory of personality, particularly the concept of the actualizing tendency (Frankel, Sommerbeck, & Rachlin, 2010) and instead embracing Skinner's behaviorist theory of personality.

Conclusion

Hopefully, this paper has demonstrated that if Rogers and Skinner could have had a third dialogue, today, informed by knowledge of nonlinear dynamic systems, they could have bridged the apparent gap between their respective positions. They could have agreed that, rather ironically, the very point they used to agree about, that determinism implies predictability, was the very point they were both wrong about. They would have known that most aspects of a human being's trajectory through life are a nonlinear dynamic system, determined and unpredictable, and in the light of this knowledge Rogers could have agreed unequivocally with Skinner about operant conditioning and determinism without fearing the consequences of his agreement for the relevance of his therapeutic approach. And in this same light, Skinner could have agreed about the wisdom of nonpredictive client-centered therapy for the ordinary complexity of client problems that demand unconditional respect for client uniqueness rather than respect for client freedom in some nonscientific "dimension."

A corollary of this conclusion is the dispensability of a belief in the existence of the actualizing tendency as the foundation for practicing client-centered therapy. A belief in, and respect for client uniqueness suffices.

Notes

1. This explanation of nonlinear dynamic systems is a slightly revised and shortened excerpt of Sommerbeck (2004). The author is grateful to the editors of the PCEP journal for their permission to insert this excerpt in the present article.
2. It would be more correct to say that *the trajectory of* the weather is *best modeled by* a nonlinear dynamic system, since the concept of a nonlinear dynamic system is a mathematical concept that consists of mathematical variables and their interrelationships, which the weather, of course, does not consist of. For easier readability, though, the author has chosen the less mathematically correct formulation that this or that is a nonlinear dynamic system rather than the more circumstantial, but also more correct, formulation that the trajectory of this or that is best modeled by a nonlinear dynamic system.

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The Dynamics of Conditioning and Extinction

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Pigeons responded to intermittently reinforced classical conditioning trials with erratic bouts of responding to the conditioned stimulus. Responding depended on whether the prior trial contained a peck, food, or both. A linear persistence–learning model moved pigeons into and out of a response state, and a Weibull distribution for number of within-trial responses governed in-state pecking. Variations of trial and intertrial durations caused correlated changes in rate and probability of responding and in model parameters. A novel prediction—in the protracted absence of food, response rates can plateau above zero—was validated. The model predicted smooth acquisition functions when instantiated with the probability of food but a more accurate jagged learning curve when instantiated with trial-to-trial records of reinforcement. The Skinnerian parameter was dominant only when food could be accelerated or delayed by pecking. These experiments provide a framework for trial-by-trial accounts of conditioning and extinction that increases the information available from the data, permitting such accounts to comment more definitively on complex contemporary models of momentum and conditioning.

Keywords: autoshaping, behavioral momentum, classical conditioning, dynamic analyses, instrumental conditioning

Estes's stimulus sampling theory provided the first approximation to a general quantitative theory of learning; by adding a hypothetical attentional mechanism to conditioning, it carried analysis one step beyond extant linear learning models into the realm of theory (Atkinson & Estes, 1962; Bower, 1994; Estes, 1950, 1962; Healy, Kosslyn, & Shiffrin, 1992). Rescorla and Wagner (1972) added the important nuance that the asymptotic level of conditioning might be partitioned among stimuli that are associated with reinforcers as a function of their reliability as predictors of reinforcement; that refinement has had tremendous and widespread impact (Siegel & Allan, 1996). The attempt to couch the theory in ways that account for increasing amounts of the variance in behavior has been one of the main engines driving modern learning theory. Models have been the agents of progress, the go-betweens that reshaped both our theoretical inferences about the conditioning processes and our modes of data analysis. In this theoretical–empirical dialogue, the Rescorla–Wagner (R-W) model has been a paragon.

Despite the elegant mathematical form of their arguments, the predictions of recent learning models are almost always qualitative—a particular constellation of cues is predicted to block or enhance conditioning more than others because of their differential associability or their history of association, and those effects are measured by differences in speed of acquisition or extinction or as a response rate in test trials. Individual differences, and the brevity of learning and extinction processes, make convergence on meaningful parametric values difficult: There are nothing like the basic constants of physics and chemistry to be found in psychology. To this is the added difficulty of a general analytic solution of the R-W model (Danks, 2003; Yamaguchi, 2006). As Bitterman (2006) astutely noted, the residue of these difficulties leaves predictions that are at best ordinal and dependent on simplifying assumptions concerning the map from reinforcers to associations and from associations to responses:

> The only thing we have now that begins to approximate a general theory of conditioning was introduced more than 30 years ago by Rescorla and Wagner (1972). . . . . An especially attractive feature of the theory is its statement in equational form, the old linear equation of Bush and Mosteller (1951) in a different and now familiar notation, which opens the door to quantitative prediction. That door, unfortunately, remains unentered. Without values for the several parameters of the equation, associative strength cannot be computed, which means that predictions from the theory can be no more than ordinal, and even then those predictions are made on the naïve assumption of a one-to-one relation between associative strength and performance. (p. 367)

To pass through the doorway that these pioneers have opened requires techniques for estimating parameters in which we can have some confidence, and to achieve that requires a database of more than a few score learning and testing trials. But most regnant paradigms get only a few conditioning sessions out of an organism (see, e.g., Mackintosh, 1974), whereupon the subject is no longer naive. To reduce error variance, therefore, data must be averaged over many animals. This is inefficient in terms of data utilization and also confounds the variability of learning parameters as a function of conditions with the variability of performance across subjects (Loftus & Masson, 1994). The pooled data may not yield parameters representative of individual animals; when functions are nonlinear, as are most learning models, the average of param-

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eters of individual animals may deviate from the parameters of pooled data (Estes, 1956; Killeen, 2001). Averaging the output of large-*N* studies is therefore an expensive and nonoptimal way to narrow the confidence intervals on parameters (Ashby & O'Brien, 2008).

Most learning is not, in any case, the learning of novel responses to novel stimuli. It is refining, retuning, reinstating, or remembering sequences of action that may have had a checkered history of association with reinforcement. In this article, we make a virtue of the necessity of working with non-naïve animals, to explore ways to compile adequate data for convergence on parameters, and prediction of data on an instance-by-instance basis. Our strategy was to use voluminous data sets to choose among learning processes that permit both Pavlovian and Skinnerian associations. Our tactic was to develop and deploy general versions of the linear learning equation—an error-correction equation, in modern parlance—to characterize repeated acquisition, extinction, and reacquisition of conditioned responding.

Perhaps the most important problem with the traditional paradigm is its ecological validity: Conditioning and extinction acting in isolation may occur at different rates than when occurring in mélange (Rescorla, 2000a, 2000b). This limits the generalizability of acquisition–extinction analyses to newly acquired associations. A seldom-explored alternative approach consists of setting up reinforcement contingencies that engender continual sequences of acquisition and extinction. This would allow the estimation of within-subject learning parameters on the basis of large data sets, thus increasing the efficiency of data use and disentangling between-subjects variability in parameter estimates from variability in performance. Against the possibility that animals will just stop learning at some point in extended probabilistic training, Colwill and Rescorla (1988; Colwill & Triola, 2002) have shown that if anything, associations increase throughout such training.

One of Skinner's many innovations was to examine the effects of mixtures of extinction and conditioning in a systematic manner. He originally studied fixed-interval schedules under the rubric "periodic reconditioning" (Skinner, 1938). But, absent computers to aggregate the masses of data his operant techniques generated, he studied the temporal patterns drawn by cumulative recorders (Skinner, 1976). Cumulative records are artful and sometimes elegant, but difficult to translate into that common currency of science, numbers (Killeen, 1985). With a few notable exceptions (e.g., Davison & Baum, 2000; Shull, 1991; Shull, Gaynor, & Grimes, 2001), subsequent generations of operant conditioners tended to aggregate data and report summary statistics, even though computers had made a plethora of analyses possible. Limited implementations of conditional reconditioning have begun to provide critical insights on learning (e.g., Davison & Baum, 2006).

Recent contributions to the study of continual reconditioning are found in Reboreda and Kacelnik (1993), Killeen (2003), and Shull and Grimes (2006). The first two studies exploited the natural tendency of animals to approach signs of impending reinforcement, known as *sign tracking* (Hearst & Jenkins, 1974; Janssen, Farley, & Hearst, 1995). Sign tracking has been extensively studied as Pavlovian conditioned behavior (Hearst, 1975; Locurto, Terrace, & Gibbon, 1981; Vogel, Castro, & Saavedra, 2006). It is frequently elicited in birds using a positive automaintenance procedure (e.g., Perkins, Beavers, Hancock, Hemmendinger, & Ricci, 1975), in which the illumination of a response key is followed by food, regardless of the bird's behavior. Reboreda and Kacelnik and Killeen recorded pecks to the illuminated key as indicators of an acquired key–food association. In both studies, a negative contingency between key pecking and food, known as *negative automaintenance* (Williams & Williams, 1969), was imposed. In negative automaintenance, an omission contingency is superimposed such that key pecks cancel forthcoming food deliveries, whereas absent key pecks, food follows key illuminations. Key–food pairing elicits key pecking (conditioning), which, in turn, eliminates the key–food pairings, reducing key pecking (extinction), which reestablishes key–food pairings (conditioning), and so on. This generates alternating epochs of responding and nonresponding, in which responding eventually moves off key or lever (Myerson, 1974; Sanabria, Sitomer, & Killeen, 2006) and, to a naive recorder, "extinguishes." Presenting food whether or not the animal responds provides a more enduring, but no less stochastic, record of conditioning (Perkins et al., 1975). The data look similar to those shown in Figure 1; a self-similar random walk ranging from epochs of nonresponding to epochs of responding with high probabilities. Such data are paragons of what we wish to understand: How does one make scientific sense of such an unstable dynamic process? A simple average rate certainly will not do. Killeen (2003) showed that data like these had fractal properties, with Hurst exponents in the "pink noise" range. However, other than alerting us to control over multiple time scales, this throws no new light on the data in terms of psychological processes.

To generate a database in which pecking is being continually conditioned and extinguished, we instituted probabilistic classical conditioning, with the unconditioned stimulus (US) generally presented independently of responding. Using this paradigm, we examined the effect of duration of intertrial interval (ITI; Experiment 1), duration of conditioned stimulus (CS; Experiment 2), and peck–US contingency (Experiment 3) on the dynamics of key peck conditioning and extinction.

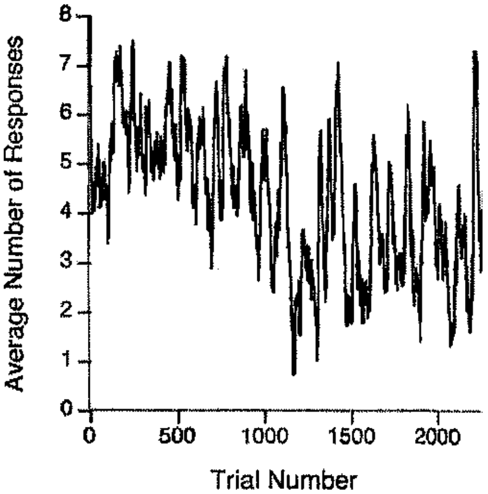

Figure 1. Moving averages of the number of responses per 5-s trial over 25 trials from 1 representative subject and condition (Pigeon 98, first condition, 40-s intertrial interval).

Experiment 1: Effects of ITI Duration and US Probability

Method

Subjects

Six experienced adult homing pigeons (*Columba livia*) were housed in a room with a 12-hr light–dark cycle, with lights on at 6:00 a.m. They had free access to water and grit in their home cages. Running weights were maintained just above their 80% ad libitum weight; a pigeon was excluded from a session if its weight exceeded its running weight by more than 7%. When required, supplementary feeding of Ace-Hi pigeon pellets (Star Milling Co., Perris, CA) was given at the end of each day, no fewer than 12 hr before experimental sessions were conducted. Supplementary feeding amounts were based equally on current deviation and on a moving average of supplements over the past 15 sessions.

Apparatus

Experimental sessions were conducted in three MED Associates (St. Albans, VT) test chambers (305 mm long × 241 mm wide × 292 mm high), enclosed in sound- and light-attenuating boxes equipped with a ventilating fan. The sidewalls and ceiling of the experimental chambers were clear plastic. The floor consisted of thin metal bars above a catch pan. A plastic, translucent response key 25 mm in diameter was located 70 mm from the ceiling, centered horizontally on the front of the chamber. The key could be illuminated by green, white, or red light emitted from diodes behind the keys. A square opening 77 mm across was located 20 mm above the floor on the front panel and could provide access to milo grain when the food hopper (part H14-10R, Coulbourne Instruments, Allentown, PA) was activated. A house light was mounted 12 mm from the ceiling on the back wall. The ventilation fan on the rear wall of the enclosing chamber provided masking noise of 60 dB. Experimental events were arranged and recorded via a Med-PC interface connected to a PC computer controlled by Med-PC IV software.

Procedure

Each session started with the illumination of the house light, which remained on for the duration of the session. Sessions started with a 40-s ITI, followed by a 5-s trial, for a total cycle duration of 45 s. During the ITI, only the house light was lit; during the trial, the center response key was illuminated white. After completing a cycle, the keylight was turned off for 2.5 s, during which food could be delivered. Two and a half seconds after the end of a cycle, a new cycle started, or the session ended and the house light was turned off. Food was always provided at the end of the first trial of every session. Pecking the center key during a trial had no programmed effect.

Initially, food was accessible for 2.5 s with reinforcement p = .1 at the end of every trial after the first, regardless of the pigeon's behavior. In subsequent conditions, the ITI was changed from 40 s to 20 s and then to 80 s for 3 pigeons; for the other 3 pigeons, the ITI was changed to 80 s first and then to 20 s. ITIs for all pigeons were then returned to 40 s. Each session lasted for 200 cycles when the ITI was 20 s, 100 cycles when the ITI was 40 s, and 50 cycles when the ITI was 80 s. In the last condition, the probability of reinforcement was reduced to .05 at the 40-s ITI. One pigeon (113) had ceased responding by the end of the .1 series and was not run in the .05 condition. Table 1 arrays these conditions and the number of sessions at each.

Results

The first dozen trials of each condition were discarded, and the responses in the remaining trials, averaging 2,500 per condition, are presented in the top panel of Figure 2 as mean number of responses per 5-s trial. The high-rate subject at the top of the graph is Pigeon 106 (cf. Figure 3 below). There appears to be a slight decrease in average response rates as the ITI increased and a larger decrease when the probability of food decreased from .1 to .05. Rates in the second exposure to the 40-s condition were lower than the first. These changes are echoed in the lower panel, which gives the relative frequency of at least one response on a trial. The interposition of other ITIs between the first and second exposure to the 40-s ITI caused a slight decrease in rate and probability of responding in 5 of the 6 birds, although the spread in rates in the top panel and the error bars in the bottom indicate that that trend would not achieve significance.

These data seem inconsistent with the many studies that have shown faster acquisition of the key-peck response at longer ITIs. But these data were probabilistically maintained responses over the course of many sessions. Only one other report, that of Perkins et al. (1975), constitutes a relatively close prequel to this one. These authors maintained responding on schedules of noncontingent partial reinforcement after CSs associated with different delays, probabilities, and ITIs. They used five different key colors associated with different conditions within each study. Those that come closest to those of the present experiment are shown as open symbols in Figure 2. The circles represent the average response rate of 4 pigeons on 4-s trials (converted to this 5-s base) receiving reinforcement on one of six (~16.7%) of the trials, at ITIs of 30 s (first circle) and 120 s (second circle). These data also indicate a slight decrease in rates with increasing ITIs. Perkins et al. also reported a condition with 8-s trials and 60-s ITIs involving probabilistic reinforcement. The first square in Figure 2 shows the average rate (per 5 s) of 4 pigeons at a probability of 3 of 27 (~11.1%); the second square, at a probability of 1 of 27 (~3.7%). Their subjects, like ours (and like a few other studies reported by these authors) showed a decrease in responding with a decrease in probability of reinforcement.

Any inferences one may wish to draw concerning these data are chastened by a glance at the intersubject variability of Figure 2 and of

Table 1
Conditions of Experiment 1

Order	ITI (seconds)	p^a	Sessions
1	40	.1	20–21
2	20, 80	.1	21–23
3	80, 20	.1	20–22
4	40	.1	21–23
5	40	.05	24–29

Note. Half the subjects experienced the extreme intertrial intervals (ITIs) in the order 20 s, 80 s, and half experienced them in the other order.
[a] p is the probability of the trial ending with food.

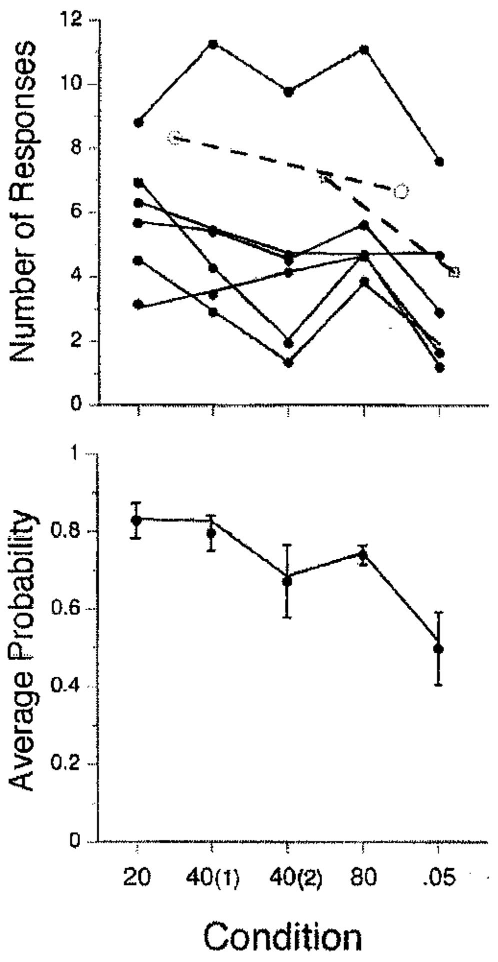

Figure 2. Data from Experiment 1. Top: average number of responses per trial (dots) for each subject, ranging from Pigeon 106 (top curve) to Pigeon 105 (bottom in Condition 20). Open symbols represent data from Perkins et al. (1975). Bottom: Average probability of making at least one response on a trial averaged over pigeons; bars give standard errors. Unbroken lines in both panels are from the Momentum/Pavlovian model, described later in the text.

Perkins et al.'s (1975) data. The effect size is small given that variability, and in fact some authors such as Gibbon, Baldock, Locurto, Gold, and Terrace (1977) have reported no effect of ITI on response rate in sustained automaintenance conditions; others (e.g., Terrace, Gibbon, Farrell, & Baldock, 1975) have reported some effect. Representing intertrial variability visually is no simpler than characterizing intersubject variability; Figure 1 gives an approximation for 1 subject (Pigeon 98) under the first 40-s ITI condition, with data averaged in running windows of 25 trials. There is an early rise in rates to around six responses per trial, then slow drift down over the first 1,000 trials, with rates stabilizing thereafter at around four responses per trial. There may be within-session warm-up and cool-down effects not obvious in this figure. We may proceed with similar displays and characterizations of them for each of the subjects in each of the conditions—all different. Or we may average performance over the whole of the experimental condition, as we did to generate the vanilla Figure 2. Or we may average data over the last 5 or 10 sessions as is the traditional modus operandi for such data. But such averages reduce a performance yielding thousands of bits of data to a report conveying only a few bits of information. As is apparent from the (smoothed) trace of Figure 1, the averages do not tell the whole story. How do we pick a path between the oversimplification of Figure 2 and the overwhelming complexity of figures such as Figure 1? And how do we tell a story of psychological processes rather than of procedural results? Models help, assayed next.

Analysis: The Models

Response Output Model

The goal of this research is to develop a procedure that can provide a more informative characterization of the dynamics of conditioning. To do this, we begin analysis with the simplest and oldest of learning models, a linear learning model of associative strength. These analyses have been in play for more than half a century (Bower, 1994; Burke & Estes, 1956; Bush & Mosteller, 1951; Couvillon & Bitterman, 1985; Levine & Burke, 1972), with the R-W model a modern avatar (Miller, Barnet, & Grahame, 1995; Wasserman & Miller, 1997). Because associative strengths are asymptotically bounded by the unit interval, it is seductive to think that they can be directly mapped to probabilities of responding or to probabilities of being in a conditioned state. Probabilities can be estimated by taking the number of trials containing at least one response within some epoch, say, 25 trials, and dividing that by the number of trials in that epoch (cf. Figure 1). There are three problems with this approach:

1. Twenty-five trials is an arbitrary epoch that may or may not coincide with a meaningful theoretical–behavioral window.
2. Information about the contingencies that were operative within that epoch are lost, along with the blurring of responses to them.
3. Parsing trials into those with and without a response discards information. Response probability makes no distinction between trials containing 1 response and trials containing 10 responses, even though they may convey different information about response strength.
4. As Bitterman (2006) noted, associative strengths are not necessarily isomorphic with probability (Rescorla, 2001).

The map between response rates and inferred strength must be the first problem attacked. The place to start is by looking at, and characterizing, the distribution of responses during a CS. Figure 3 displays the relative frequency of 0, 1, 2, . . ., 20 responses during a trial in the first condition of Experiment 1 for each of its participants.

The curves through the distributions are linear functions of Weibull densities:

$$p(n = 0) = s_i \cdot w(n, \alpha, c) + 1 - s_i,$$

$$p(n > 0) = s_i \cdot w(n, \alpha, c). \tag{1}$$

The variable s_i is the probability that the pigeon is in the response state on the *i*th trial. For the data in Figure 3, this is averaged over all trials. The *w* function is the Weibull density with index *n* for the actual number of responses during the CS, the shape parameter α, and the scale parameter c, which is proportional to the mean number of responses on a trial. The first line of Equation 1 gives the probability of no responses on a trial: It is the probability that the animal is in the response state (s_i) and makes no responses [$w(n, \alpha, c)$], plus the probability that it is out of the response state ($1 - s_i$). The second line gives the probability of all nonzero responses.

The Weibull distribution is a generalization of the exponential/Poisson distribution that was recommended by Killeen, Hall, Reilly, and Kettle (2002) as a map from response rate to response probability. That recommendation was made for free operant responding during brief observational epochs. The Poisson also provides an approximate account of the response distributions shown in Figure 1. It is inferior to the Weibull, however, even when the additional shape parameter is taken into account using the Akaike information criterion (AIC). The Weibull distribution[1] is

$$W(n, \alpha, c) = 1 - e^{-(n/c)^{\alpha}}. \tag{2}$$

According to this model, when the pigeon is in a response state, it begins responding after trial onset and emits n responses during the course of that trial. It is obvious that when $\alpha = 1$, the Weibull reduces to the exponential distribution recommended by Killeen et al. (2002). In that case, there is a constant probability $1/c$ of terminating the response state from one response to the next, and the cumulative distribution is the concave asymptotic form we might associate with learning curves. Pigeon 105 exemplifies such a shape parameter, as witnessed by the almost-exponential shape of its density shown in Figure 3. Just below Pigeon 105, Pigeon 107 has a more representative shape parameter, around 2. (Whenever $\alpha > 1$, as was generally found here, there is an increasing probability of terminating responding as the trial elapses—the hazard function increases.) When α is slightly greater than 3, the function most closely approximates the normal distribution, as seen in the data for Pigeon 119. Pigeon 106, familiar from the top of Figure 2, has the most extreme shape parameter seen anywhere in these experiments, $\alpha \approx 5$. The poor fit of the function to this animal is due to its "running through" many trials, which were not long enough for its distribution to come to its natural end.

It is the Weibull density, the derivative of Equation 2, that drew the curves through the data in Figure 3. The density is easily called as a function in Excel as *=Weibull(n, α, c, false)*. It is readily interpreted as an extreme value distribution, one complementary to that shown to hold for latencies (Killeen et al., 2002). In this article, we do not use the Weibull as part of a theory of behavior but rather as a convenient interface between response rates and the conditioning machinery. Conditioning is assumed to act on s, the probability of being in the response state, a mode of activation (Timberlake, 2000, 2003) that supports key pecking.

Does the Weibull continue to act as an adequate model of the response distribution after tens of thousands of trials? For a different, and more succinct, picture of the distributions, in Figure 4 we plot the cumulative probability of emitting n responses on a trial, along with linear functions of the Weibull distribution. As before, the y-intercept of the distribution is the average probability of not making a response; the corresponding theoretical value is the probability of being out of the state, plus the (small) probability of being in the state but still not making a response. Thereafter, the probability of being in the state multiplies the cumulative Weibull distribution. The fits to the data are generally excellent, except, once again, for Pigeon 106, who did not have time for a graceful wind-down. This subject continued to run through the end of the trial; a good fit requires the Weibull distribution to be "censored," involving another parameter, which was not deemed worthwhile for its present purposes.

Changes in Response State Probability: Momentum and Pavlovian Conditioning

In his analysis of the dynamics of responding under negative automaintenance schedules, Killeen (2003) found that the best first-order predictor was the probability that the pigeon was in a response state, as given by a linear average of its probability of being in that state on the last trial and the behavior on the last trial. In the case of a trial in which a response occurred, the probability of being in the response state is incremented toward its ceiling ($\theta = 1$) using the classic (Killeen, 1981) linear average:

$$s_i' = s_i + \pi_R(\theta - s_i), \tag{3}$$

where pi (π) is a rate parameter. Pi will take different values depending on the contingencies: π_R subscripts the response, being instantiated as π_P on trials containing a peck and as π_Q on quiet trials. Theta (θ) is 1 on trials that predict future responding and 0 on trials that predict quiescence. Thus, after a trial on which the animal responded, the probability of being in the response state on the next trial will increase as

$$s_i' = s_i + \pi_P(1 - s_i),$$

whereas after a trial that contained no peck, it will decrease as

$$s_i' = s_i + \pi_Q(0 - s_i).$$

After these intermediate values of strength are computed, they are perturbed by the delivery or nondelivery of food. For that we use a version of the same exponentially weighted moving average of Equation 3:

$$s_{i+1}' = s_i' + \pi_O(\theta - s_i'). \tag{4}$$

Now the learning parameter π_O subscripts the outcome (food or empty). All of these pi parameters tell us how quickly probability approaches its ceiling or floor and thus how quickly the state on the prior trial is washed out of control (Tonneau, 2005). For geometric progressions such as these, the mean distance back is

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[1] Whereas the Weibull is a continuous function, it approximates a proper distribution function on the integers, as

$$\sum_n w(n, \alpha, c) \approx 1$$

over the range of all parameters studied here. The approximation is significantly improved by adding a continuity correction of $\varepsilon = 0.5$ to all response counts. Epsilon may be thought of as a threshold for emitting the first response but is treated here merely as an ad hoc statistical correction applied to all data (except not to the pedagogic example given below). A better estimate is given by evaluating the distribution function between $n + (1/2)$ and $n - (1/2)$, with the latter taking 0 as a minimum. However, that extra computation does not add enough precision in the current situation to be useful. The Weibull should be right censored because there are time constraints on responding. This causes the deviation between predicted and obtained for Pigeon 106 in Figures 3 and 4. That refinement is not engaged here.

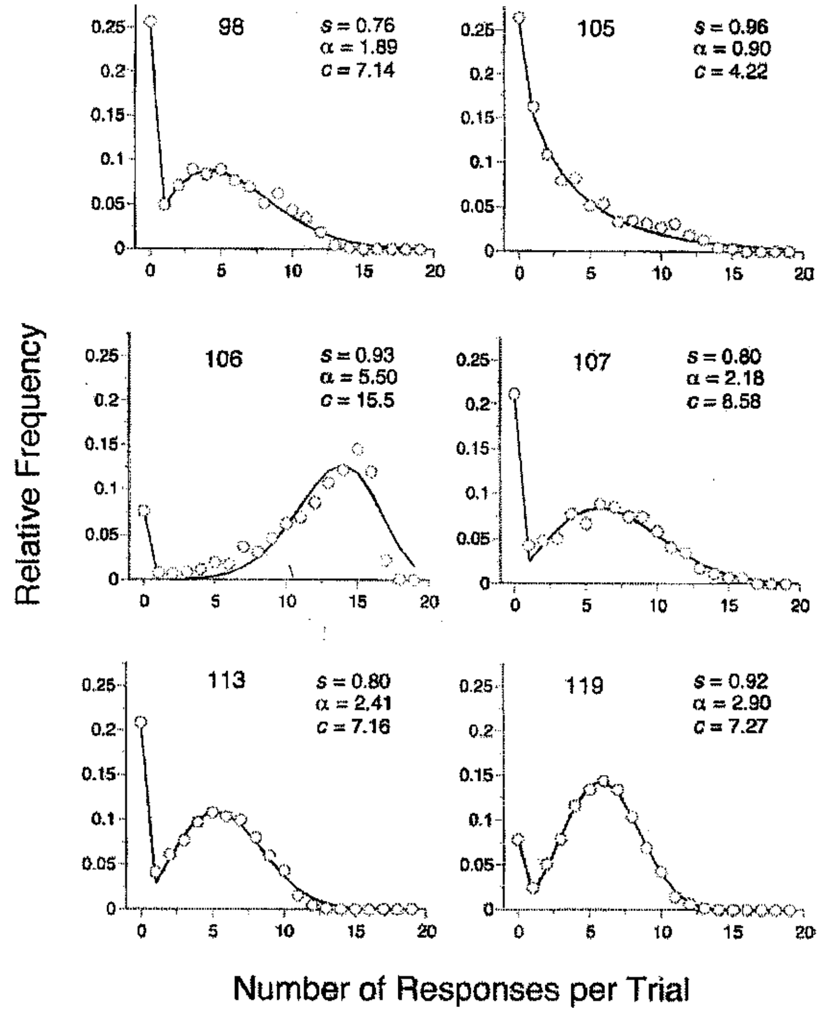

Figure 3. The relative frequency of trials containing 0, 1, 2, . . . responses. The data are from all trials of the first condition of Experiment 1. The curves are drawn by the Weibull response rate model (Equation 1). The parameter s is the probability of being in the response state; the complement of this probability accounts for most of the variance in the first data point. The parameter α dictates the shape, from exponential ($\alpha = 1$) to approximately normal ($\alpha \approx 3$) to increasingly peaked ($\alpha \approx 5$). The parameter c is proportional to the mean number of responses on trials in the response state and gives the rank order of the curves in Figure 2 at Condition 20.

$(1 - \pi)/\pi$, whenever $\pi > 0$. One might say that this is the size of the window on the past when the window is half open. As before, theta (θ) is 1 on trials that strengthen responding and 0 on trials that weaken it. Thus, after a trial on which food was delivered, we might expect to see the probability of being in the response state on the next trial (s_{i+1}) increase as

$$s_{i+1} = s_i' + \pi_F(1 - s_i'),$$

whereas after a trial that contained no food, it might decrease as

$$s_{i+1} = s_i' + \pi_E(0 - s_i').$$

These steps may be combined in a single expression, as noted in the Appendix. Although shamefully simple compared with more recent theoretical treatments, such linear operator models can acquit themselves well in mapping performance (e.g., Grace, 2002).

There are four performance parameters in this model corresponding to the four operative contingencies, each with an associated ceiling or floor. We list them in Table 2, where parenthetical signs indicate whether behavior is being strengthened (positive entails that $\theta = 1$) or weakened (negative entails that $\theta = 0$).[2] The values assumed by these parameters, as a function of the conditions of reinforcement, are the key objects of our study.

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[2] In our analysis programs, we let the learning variables go negative to indicate decrementing ($\theta = 0$), extract the sign of the parameters to set their direction toward floor (when $\pi_{.} < 0, \theta = 0$) or ceiling (when $\pi_{.}$ 0, $\theta = 1$), and use their absolute value$|\pi_{.}|$to adjust the distance traveled toward those limits, as in Equation 4. Thus, we refrain from imposing our expectations about what the directions of events should be on behavior.

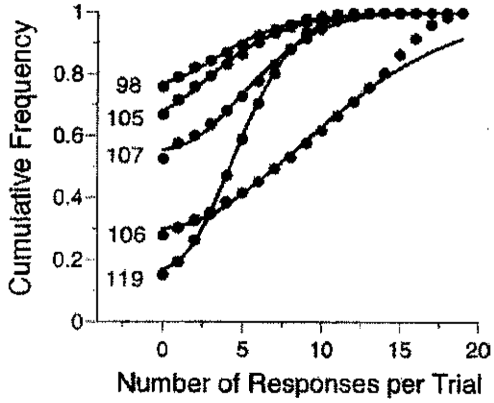

Figure 4. The cumulative frequency of trials containing 0, 1, 2, . . . responses. The data are from all trials of the last condition of Experiment 1. The curves are drawn by the Weibull response rate model (Equation 1), using the distribution function rather than the density.

Notice that this model makes no special provision for whether a response and food co-occurred on a trial. It is a model of persistence, or behavioral momentum, and Pavlovian conditioning of the CS. Because these factors may always be operative, it is presented first, and the role of Skinnerian response–outcome associations is subsequently evaluated. The model also takes no account of warm-up or cool-down effects that may occur as each session progresses. Covarying these out could only help the fit of the models to the residuals, but it would also put one more layer of parameters between the data and the reader's eye.

The matrix of Table 2 is referred to as the Momentum/Pavlov model, or MP model. By calling it a model of momentum, we do not mean that a new hypothetical construct is invoked to explain the data. It is simply a way of recognizing that response strength will not in general change maximally on receipt of food or extinction. Just how quickly it will change is given by the parameters π_P and π_Q. If these are 1, there will be no lag in responsiveness and no need for the construct; if they equal 0, the pigeon will persist at the current probability indefinitely, and there will be no need for the construct of conditioning. In early models without momentum (i.e., where these parameters were de facto 1), goodness of fit was at least e^{10} worse than in the model as developed here, and typically worse than the comparison model, described later.

Implementation

To fit the model to the data, we use Equation 1 to calculate the probability of the observed data given the model. Two hypothetical cases illustrate the computation of this probability:

1. Assume the following: no key pecks on trial i, the predicted probability of being in the response state $s_i = 2/3$, and the Weibull parameters were $\alpha = 2$, $c = 6$. Then the probability of the data (0 responses) given the model $p(d_i|m)$ is the probability of being
   (a) out of the response state, $1 - s_i$, times the probability of no response when out of the state, 1.0: $(1 - 2/3) \cdot 1 = 1/3$; to that, add the probability of being
   (b) in the state, times the probability of no responses in the state: $2/3w(n, 2, 6) = 2/3 \cdot 0$;
   (c) the sum of which equals $p(d_i = 0|m) \approx .333 + 0 \approx 0.333$.
2. If four pecks were made on trial i, given the same model parameters, then the probability would be $p(d_i = 4|m) = 0 + 2/3w(n, 4, 6), \approx 0.142$.

The natural logarithm of these conditional probabilities gives the index of merit of the model for this trial: That is, it gives the log-likelihood (LL_i) of the data (given the model) on trial i. These logarithms are summed over the thousands of trials in each condition to give a total index of merit LL (Myung, 2003). Case 1 added $\ln(1/3) \approx -1.1$ to the index, whereas Case 2 added $\ln(.142) \approx -1.9$, its smaller value reflecting the poorer performance of the model in predicting the data on that trial. The parameters are adjusted iteratively to maximize this sum and thus to maximize the likelihood of the data given the model. The LL is a sufficient statistic, so that it contains all information in the sample relevant to making any inference between the models in question (Cox & Hinkley, 1974).

A Base (Comparison) Model

Log-likelihoods are less familiar to this audience than are coefficients of determination—the proportion of variance accounted for by the model. The coefficient of determination compares the residual error (the mean square error) with that available from a simple default model, the mean (whose error term is the variance); if a candidate model can do no better than the mean, it is said to account for 0% of the variance around the mean. In like manner, the maximum likelihood analysis becomes more interpretable if it is compared with a default, or base, model. The base model we adopt has a structure similar to our candidate model: It uses Equation 1 and updates the probability of being in the response state as a moving average of the recent probability of a response on a trial:

$$s_{i+1} = \gamma P_i + (1 - \gamma)s_i,\ 0 < \gamma < 1, \tag{5}$$

where gamma (γ) is the weight given to the most recent event and P takes a value of 1 if there was a response on the prior trial and 0 otherwise. Equation 5 is an exponentially weighted moving average, and can be written as $s_{i+1} = s_i + \gamma(P_i - s_i)$, which reveals its similarity to the Momentum/Pavlovian model, with the one parameter γ replacing the four contingency parameters of that model. The base model attempts to do the best possible job of predicting future behavior from past behavior, with its handicap being ignorance as to whether food or extinction occurred on a

Table 2
Momentum/Pavlovian Model With Events Mapped Onto Direction and Rate Parameters

Event	Representation
Peck	P: $(\pm)\pi_P$
No peck (quiet)	Q: $(\pm)\pi_Q$
Food	F: $(\pm)\pi_F$
Empty/extinction	E: $(\pm)\pi_E$

Note. The parentheticals indicate whether the learning process is driving behavior up (positive entails that $\theta = 1$) or down (negative entails that $\theta = 0$); the rate parameters themselves are always positive.

trial. It is a model of perseveration, or momentum, pure and simple. It invokes three explicit parameters: γ, α, and c. Other details are covered in the Appendix.

An Index of Merit for the Models

The log-likelihood does not take into account the number of free parameters used in the model. Therefore, we use a transformation of the log-likelihood that takes model parsimony into account. The AIC (Burnham & Anderson, 2002) corrects the log-likelihood of the model for the number of free parameters in the model to provide an unbiased estimate of the information-theoretic distance between model and data:

$$\mathrm{AIC} = 2(n_P - LL), \tag{6}$$

where n_P is the number of free parameters and LL is the total log-likelihood of the data given the model. (We do not require the secondary correction for small sample size, AIC_C).

We compare the models under analysis with the simple perseveration model, the base model, characterized by Equations 1 and 5. This comparison is done by subtraction of their AICs. The smaller the AIC, the better the adjusted fit to the data. There are $n_P = 3$ parameters in the base model (hereinafter *base*), and 6 parameters (8 in later versions) in the candidate model (hereinafter *model*), so the relative AIC is

$$\begin{aligned} \text{Merit} &= \text{Relative AIC} = \text{AIC(Base)} - \text{AIC(Model)} \\ &= 2(3 - \mathrm{LL_B}) - 2(6 - LL_M) \\ &= 2(LL_M - LL_B) - 6. \end{aligned} \tag{7}$$

Because logarithms of probabilities are negative, the actual log-likelihoods are negative. However, our index of merit subtracts the model AIC from the base AIC so that it is generally positive and is larger because the model under purview is better than the base model. The relative AIC is a linear function of the log-likelihood ratio of model to base (LLR = log[(likelihood of model)/(likelihood of base)]). Because of the additional free parameters of the model, it must account for e^3 as much variance as the base model just to break even. An index of merit of 4 for a model means that under that model, the data are e^4—approximately 50 times—as probable as under the base model, after taking into account the difference in number of free parameters. A net merit of 4 is our criterion for claiming strong support for one model over another. If the prior probabilities of the model under consideration and the base (or other comparison) model are deemed equal, Bayes's theorem tells us that when the index of merit is greater than 4 (after handicapping for excess parameters), then the posterior odds of the candidate model compared with the comparison is at least 50/1.

The base model is nested in the Pavlovian/Momentum model: Setting $\pi_Q = -\pi_P = \gamma$, and $\pi_F = \pi_E = 0$ reduces it to the base model. For summary data, we also display the Bayesian information criterion (BIC; Schwarz, 1978), which sets a higher standard for the admission of free parameters in large data sets such as ours: $\mathrm{BIC} \approx -2\mathrm{LL} + k\ln(n)$. We now apply this modeling framework to the results of the first experiment.

The index of merit is relative to the default base model, just as the proportion of variance accounted for in quotidian use is relative to a default model (the mean). If the default model is very bad, the candidate model looks very good by comparison. If, for instance, we had used the mean response rate or probability over all sessions in a condition as the default model, the candidate would be on the order of e^{400} better in most of the experiments. A tougher test would be to contrast the present linear operator model with the more sophisticated models in the literature, but that is not, per reviewers' advice, included here.

Applying the Models

The AIC advantage of the Pavlovian model over the base model averaged 43 AIC points for the first four conditions, in which only 2 of the 24 Subject × Condition comparisons did not exceed our criterion for strong evidence (improvement over the base model by 4 points). For the last, $p = .05$, condition, the average merit jumped to 183 points. Figure 5 shows that the Weibull response rate parameters were little affected by the varied conditions. The average value of c, 8.2, corresponded to a mean of 7.3 responses per trial on trials on which a response was made (the mean is primarily a function of c, but also of α). The average value of the shape parameter α was 2.4: The modal response distribution looked like that of Pigeon 113 in Figure 3. The values of these Weibull parameters were always essentially identical for the base and MP models and were therefore shared by them.

The values of gamma (γ), the perseveration constant in the base model, averaged .038 in the first four conditions and increased to .100 in the $p = .05$ condition. This indicates that there was a greater amount of character—more local variance—in this last condition for the moving average to take advantage of, a feature that was also exploited by the MP model. There was no change in the rate of responding—given that the pigeon is in a response state—as indicated by the constancy of c. All of the decrease seen in Figure 2 was the result of changes in the probability of entering a response state, as given by the model and seen in the model's predictions, traced by the lines in the bottom panel of Figure 2.

The weighted average parameters of the MP model are shown in the bottom panel of Figure 5 (the values for each subject were weighted by the variance accounted for by the model for that subject). Just as autoshaping is fastest with longer ITIs, the impact of the π_F and π_P parameters increases markedly with ITI. The increase in π_F indicates that at long ITIs, the delivery of food, independent of pigeons' behavior, increases the probability of a response on the next trial. It increases 11% of its distance toward 1.0 in the 20-s ITI condition, up to 28% in the 80-s ITI condition. Also notice that π_F is everywhere of greater absolute magnitude than π_E, a finding consistent with that of Rescorla (2002a, 2002b).

The increase in π_P indicates that pecking acquires more behavioral momentum as the ITI is increased. The parameter π_Q remains around −7% over conditions (although a drop from −5% to −10% in the first and second replication of the 40-s conditions accounts for the decrease in probability of responding in the second exposure). A trial without a response decreases the probability of a response on the next by 7%. The parameter π_E hovers at zero for the short and intermediate ITIs: Extinction trials add no new information about the pigeons' state on the next trial and do not change behavior from the *status quo ante.* Under these conditions, extinction does not discourage responding. The law of disuse, rather than extinction, is operative: If a pigeon does not

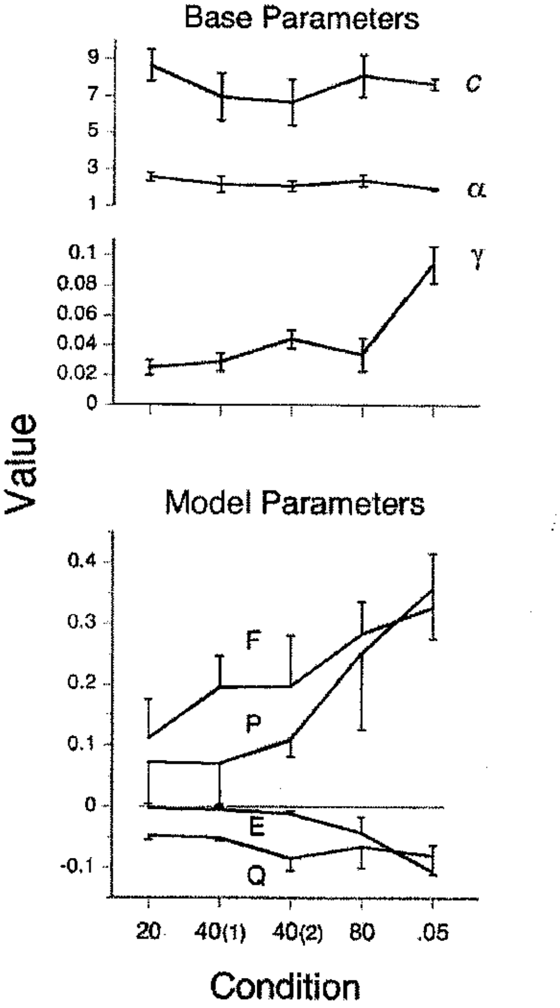

Figure 5. The average parameters of the base and Momentum/Pavlovian models for Experiment 1. The first four conditions are identified by their intertrial interval (ITI), with the first and second exposure to the 40-s ITI noted parenthetically. The same Weibull parameters, c and α, were used for both models. In the last condition, the probability of hopper activation on a trial was reduced from .1 to .05, with ITI = 40. The error bars delimit the standard error of the mean. F = food; P = response; E = no food; Q = no response.

respond, momentum in not responding (measured by π_Q) carries response probability lower and lower. At the longest ITI and in the $p = .05$ condition, extinction trials decrease the probability of being in a response state on the next trial by 4% and 10%, respectively. When reinforcement is scarce, both food and extinction matter more, as indicated by increased values of π_F and π_E, but the somewhat surprising effect on π_E is modest compared with the former. The importance of food when it is scarce is substantial—with π_F increasing more than 30% in the $p = .05$ condition. The fall toward extinction of responding, driven by π_Q and π_E, is arrested only by delivery of food, a strong tonic to responding (π_F), or an increasingly improbable peck, which, as reflected in π_P, is associated with substantially enhanced response probabilities on the next trial.

We may see how close the simulations look to the real performance, such as that shown in Figure 1. We did this by replacing the pigeon with a random number generator, using the average parameters from the first condition, shown in Figure 5. The probability of the generator's entering a response state was adjusted using the MP model, and when in the response state, it emitted responses according to a Weibull distribution with the parameters shown in the top of Figure 5. Figure 6 plots the resulting data in a fashion similar to that shown in Figure 1 (a running average of 25 trials). Comparison of the three panels cautions how different a profile can result from a system operating according to the same fixed parameters once a random element enters. Analyses are wanted that can deal with such vagaries without recourse to averaging over a dozen pigeons. By analysis on a trial-by-trial basis, the present models attempt to take a step in that direction.

These graphs have a similar character to those generated by the pigeons (although they lack the change in levels shown by Pigeon 98 in Figure 1, a change not clearly shown by most of the other subjects). The challenge is how to measure "similar" in a fashion other than impressionistically. Killeen (2003) showed that responding had a fractal structure, and given the self-similar aspect of these curves, that is likely to be the case here. However, the indices yielded by fractal analysis throw little new light on the psychological processes. The AIC values returned by the model provide another guide for those comfortable with likelihood analyses; they tell us how good the candidate model is relative to a plausible contender.

The variance accounted for in the probability of responding will look pathetic to those used to fitting averaged data: It averages around 10% in Experiment 1 and around 15% in the remaining experiments. But even when the probability of a response on the next trial is known exactly, there is probabilistic variance associate with Bernoulli processes such as these, in particular, a variance of $p(1-p)$. The parameters were not selected to maximize variance accounted for, and in aggregates of data much of the sampling error that is inevitable in single-trial predictions is averaged out. When the average rate over the next 10 trials, rather than the single next trial, is the prediction, the variance accounted for by the matrix models doubles. At the same time, the ability to speak to the trial-by-trial adjustment of the parameters is blunted. Other analyses, educing predictions from the model and testing them against the data, follow.

Hazard Functions

That π_Q and π_E are negative in the $p = .05$ condition makes a strong prediction about sojourns away from the key: When a pigeon does not respond on a trial, there is a greater likelihood that it will not respond on the next, and yet greater on the next, and so on. Only free food (or the unlikely peck despite the odds) saves it. The probability of food is 5%, but the cumulative probability is continually increasing, reaching 50% after 15 trials since the first nonresponse. The probability of returning to the key should decrease at first, flatten, and then eventually increase. A simple test of this prediction is possible: Plot the probability of returning to the key after various numbers of quiet trials. In making these plots, each point has to be corrected for the number of opportunities left for the next quiet trial. Such plots of marginal probabilities are called *hazard functions*. If there is a constant probability of return-

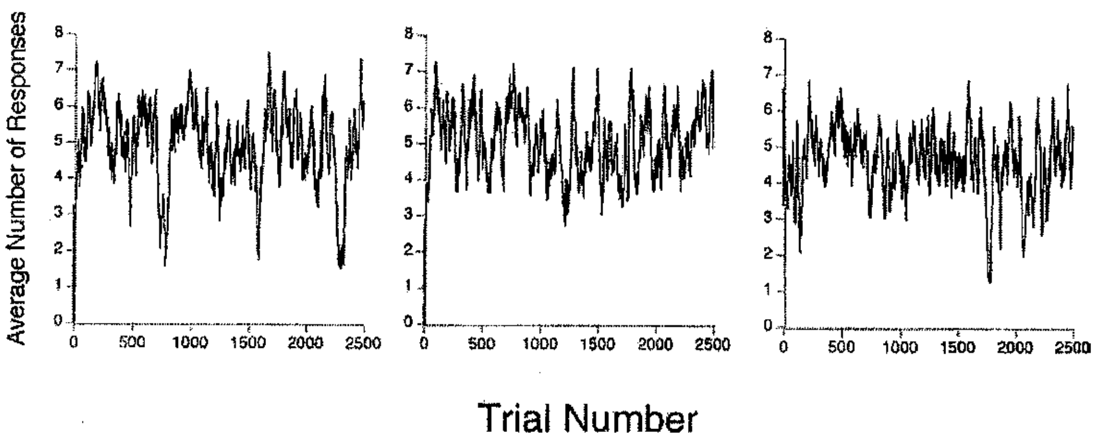

Figure 6. Moving averages of the number of responses per 5-s trial over 25 trials from three representative "statrats," characterized by the average parameters of real pigeons in the first condition, 40-s ITI. The only difference among these three panels is the random number seed for Trial 1. Compare with Figure 1.

ing to the key, as would be the case if returns were at random, the hazard function would be flat. The earlier analysis predicts hazard functions that decrease under the pressure of the negative parameters and eventually increase as the cumulative probability of the arrival of food increases.

Figure 7 shows the functions for individual pigeons (truncated when the residual response probabilities fell to 1%). They show the predicted form. The filled squares show the averaged results of running three "statrats" in the program, with parameters taken from the .05 condition of Figure 5. If the model controls behavior the way it is claimed, the output of the statrats should resemble that of the pigeons. There is indeed a family resemblance, although the statrats' hazard function was more elevated than the average of the pigeons, indicating a greater eagerness to return to the operandum than was the case for the birds. Note also that the predicted decrease—first 8% of the distance to 0 from π_Q and then another 11% from π_E—predicts a decrease to 82% of the initial value after the first quiet—that is, from about 0.45 to 0.37 for the statrats and from about .28 to about .23 for the average pigeon. These are right in line with the functions of Figure 7. The eventual flattening and slow rise in the functions is due to the cumulative effects of π_F.

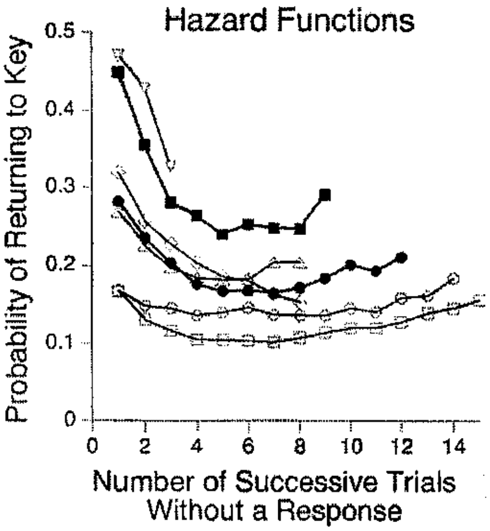

Figure 7. The marginal probability of ending a run of quiet trials. The unfilled symbols are for individual pigeons, and the filled circles represents their average performance. The hazard function represented by filled squares comes from simulations of the model.

Is Momentum Necessary?

In the parameters π_P and π_Q, the MP model invokes a trait of persistence or momentum, which may appear supererogatory to some readers. However, the base model, the linear average of the recent probability of responding, actually proves a strong contender to the MP model. It embodies the adage "The best predictor of what you will do tomorrow is what you did today." It is the simplest model of persistence, or momentum. We may contrast it with a MP-minus-M model: That is, adjust the probability of responding on the next trial as a function of food or extinction on the current trial, while holding the momentum parameters at zero. Even though the base model has one fewer parameter, it easily trumps the MP-minus-M model. For example, for Pigeon 98, the median advantage of the MP model over the base model was 14 AIC points in the .1 condition and 58 points in the .05 condition. But without the momentum aspect, the MP-minus-M model tumbles to a median of 106 points below the base model in the .1 conditions and 540 points below it in the .05 condition. However one characterizes the action of the π_P and π_Q parameters, their presence in the model is absolutely necessary. This analysis carries the within-session measurement of resistance to change reported by Tonneau, Rios, and Cabrera (2006) to the next level of contact with data.

Operant Conditioning

What is the role of response–reinforcer pairing in controlling this performance? The first analysis of these data (unreported here) consisted of a model involving all interaction terms, and those alone: PF, PE, QF, and QE. Although this interaction model was substantially better than the base model (18 AIC units over all conditions, 73 in the $p = .05$ condition), it was always trumped by the MP model (51 AIC units over all conditions, 183 in the $p = .05$ condition).

In search of evidence of Skinnerian conditioning, we asked whether there was a correlation between the number of responses on a trial and the probability of responding on the next trial. Any simple correlation could be just due to persistence; however, if response–reinforcer contiguity is a factor in strengthening responding, then that correlation should be larger for trials that end with food (r_F) than for trials that end without food (r_E). When many responses occur on a reinforced trial, (a) there are more responses in close contiguity with the reinforcer and (b) the last of them is likely to be closer in time to the reinforcer than the case of trials with only a few responses. Therefore, there should be a positive correlation between number of responses on trials ending with food and number of responses on the next trial. It is different for trials that end without food: When many responses occur on a nonreinforced trial, there are many more instances of the response subject to extinction; this should not only undermine a positive correlation, it could drive it negative. We can therefore test for Skinnerian conditioning by correlating the number of responses on F and E trials that had at least one response (the predictors) with the presence or absence of a response on the next trial (the criterion). If contiguity of multiple responses with food strengthens behavior more than contiguity of one response to food, the correlation with subsequent responding should be larger when the trial was followed by food than when it was not. That is, we would expect $r_F > r_E$. We restrict the analysis to trials with at least one response so that the correlation is not simply driven by the information that the pigeon is in a response state, which we know from π_P has good predictive value.

We analyzed the data for all subjects from all conditions and found no evidence for value added by multiple response–reinforcer contiguity. For no pigeon was the average correlation between predictor and criterion greater when the predictor was followed by food than when it was not. The averages over all subjects and conditions were $r_F = 0.035$ and $r_E = 0.081$. With an average n of 150 for r_F and of 1,470 for r_E for each of the 29 pairs of correlations, the conclusion is unavoidable: Reinforcement on trials with multiple responses did not increase the probability of a response on the next trial any more than did extinction on trials with multiple responses.

Perhaps fitting a delay-of-reinforcement model from each response to an eventual reinforcer would show evidence of operant conditioning? This was our first model of these data, not reported here. We found no value added by the extra parameter (the slope of the delay-of-reinforcement gradient).

Convinced that there must be some way to adduce evidence of (adventitious) operant conditioning, we turned to the next analysis. It remains possible that reinforcement increases the probability of staying in the response state on the next trial: Possibly the commitment to a behavioral module (Timberlake, 1994), rather than the details of actions within the module, is what gets strengthened by reinforcement. To test this hypothesis, we added conditioning factors, π_{PF} and π_{PE}, to the model. If response–reinforcer contiguity added strengthening–prediction beyond that afforded by the independent actions of persistence and of food delivery, one or both of these parameters should take values above zero and should add significantly to predictive accuracy when it does. We measure accuracy with the AIC score; any increase (after handicapping for the added parameter) lends credibility, and increases by at least 4 constitute strong evidence.

The average value of π_{PF} across the 29 cases was 0.064: That is, the probability of a response on the next trial increased by 6% beyond that predicted by momentum and mere delivery of food (independent of the presence or absence of a peck). For 2 birds, 107 and 119, there was no advantage, and π_{PF} remained close to zero, as often negative as positive. Of the 19 remaining Pigeon × Condition cases, 11 showed an AIC advantage for the added parameter, 5 of them meeting our criterion for strong evidence. Of the 4 birds that showed evidence of Skinnerian conditioning, the average value of π_{PF} was 8%, which may be compared with 16% for π_P and 14% for π_F. Examining the data on a condition-by-condition basis, all 4 of these pigeons showed evidence of Skinnerian conditioning in the 20-s ITI condition (3 of them, strong evidence), and in all cases but one π_{PF} was larger than either π_P or π_F. Across all 6 pigeons, the advantage of adding the contiguity parameter was 2.6 AIC points at the 20-s ITI 20, 0.8 at the 40-s ITI, and -1.5 at the 80-s ITI. (The negative value indicates that the cost of the extra parameter in Equation 7 is not repaid by increased predictive ability.) In the $p = .05$ condition, the total advantage conferred by the π_{PF} parameter increased to 6.4. (When the Skinnerian parameter comes into play, there is typically a readjustment of the other parameters that had been tasked with picking up the slack.) The Skinnerian extinction parameter π_{PE} was almost never called into play and exerted negligible improvement in the predictions. Parameter values for each pigeon are listed in Table 3; indices of merit, in Table 4.

These results indicate that Skinnerian conditioning was strongest where Pavlovian conditioning was weakest—whether that weakness was the result of a small ITI-to-trial ratio (20-s ITI) or to a less reliable CS ($p = .05$). This is consistent with the findings of Woodruff, Conner, Gamzu, and Williams (1977). We retain π_{PF} and π_{PE} in subsequent analyses, in which we call the full model the Momentum/Pavlovian/Skinnerian (MPS) model.

Implications for Acquisition and Extinction

On the basis of Equation 4 and the parameters shown in Figure 5, we may predict the courses of acquisition and extinction in similar contexts—it is given by Equation A5 in the Appendix. For the parameters in Figure 5, the MPS model predicts faster acquisition at longer ITIs—the trial spacing effect, along with an increasing dependence on the original starting strength (derived from hopper training) as trial spacing decreases. Pretraining plays a critical role in determining the speed of acquisition (Davol, Steinhauer, & Lee, 2002; Downing & Neuringer, 2003); the current analysis suggests that this is in part because of elevation of the initial probability of a response, s_0, possibly through generalization of hopper stimuli and key stimuli (Sperling, Perkins, & Duncan, 1977; Steinhauer, 1982). Conditioning of the context proceeds rapidly, however, so that more than a few pretraining trials in the same context will slow the speed of subsequent key conditioning (Balsam & Schwartz, 2004).

The predicted number of trials to criterion show an approximate power-law relation between trials to acquisition and the ITI (Gibbon et al., 1977). Those researchers, along with Terrace, Gibbon, Farrell, and Baldock (1975), found that both acquisition and re-

Table 3
Parameter Values of the Base and Momentum/Pavlovian/Skinnerian Models for the Data of Experiment 1

Bird no.	Parameter	20	40_1	40_2	80	.05
98	γ	.02	.03	.05	.02	.08
	c	8.78	6.93	5.31	7.77	5.90
	α	3.00	2.08	1.55	2.30	1.76
	P	.00	.02	.04	−.01	.27
	Q	−.02	−.03	−.05	−.01	−.19
	F	.00	.05	.03	.22	.00
	E	.00	.00	.00	.00	.01
	PF	.20	.00	.13	.00	.00
	PE	.00	.00	.00	.00	−.08
105	γ	.05	.04	.11	.10	.12
	c	5.69	5.12	7.18	7.49	5.74
	α	1.56	1.31	2.13	2.33	1.62
	P	.04	.05	.19	.61	.32
	Q	−.04	−.04	−.24	−.15	.02
	F	.03	.00	.56	.33	.34
	E	.00	.00	.03	−.10	−.30
	PF	.14	.04	.34	.00	.00
	PE	.00	.02	−.04	.00	.28
106	γ	.05	.02	.07	.02	.12
	c	12.71	13.88	13.29	14.35	12.40
	α	3.54	4.49	3.55	4.25	2.36
	P	.05	.10	.18	.17	.30
	Q	−.06	−.07	−.14	−.07	−.16
	F	.00	.00	.27	.27	.54
	E	.01	.03	.00	−.02	−.03
	PF	.19	.43	.16	.10	.00
	PE	.00	−.01	−.01	.00	−.01
107	γ	.02	.05	.05	.01	.04
	c	8.56	8.20	7.47	8.69	8.00
	α	2.57	2.20	2.11	2.22	2.00
	P	.06	.00	.04	.00	.06
	Q	−.03	−.04	−.06	−.01	−.05
	F	.29	.27	.12	.30	.30
	E	−.02	.00	.00	−.01	.00
	PF	.00	.00	.00	.00	.00
	PE	.00	.00	.00	.00	−.04
113	γ	.02	.05	.04	.03	
	c	6.90	5.45	4.79	6.45	
	α	2.51	2.22	1.76	2.46	
	P	.02	.06	.06	.68	
	Q	−.02	−.06	−.04	.12	
	F	.00	.14	.00	−.06	
	E	.00	−.01	.00	−.16	
	PF	.05	.00	.00	.00	
	PE	.00	.00	−.01	.00	
119	γ	.01	.01	.03	.02	.06
	c	8.12	7.17	6.41	7.78	6.76
	α	3.17	2.92	2.49	2.30	2.44
	P	.21	.28	.04	−.01	.48
	Q	−.05	−.07	−.04	−.01	−.17
	F	.90	.65	.31	.23	.50
	E	−.02	−.03	.00	.00	.00
	PF	−.02	.00	−.01	.00	.00
	PE	.09	.17	.00	.00	−.04

Note. γ = the rate constant for the comparison base model; c = the Weibull rate constant; α = the Weibull shape constant; the remaining letters indicate the rate constants brought into play on trials with (P) or without (Q) a response; with (F) or without (E) food; and the Skinnerian interaction terms PF and PE.

sponse probability in steady-state performance after acquisition covaried with the ratio of trial duration to ITI. Gibbon, Farrell, Locurto, Duncan, and Terrace (1980) found the permutation that partial reinforcement during acquisition had no effect on trials to acquisition, when those were measured as reinforced trials to acquisition. This is consistent with the acquisition equations in the Appendix. Despite these tantalizing similarities, however, the obvious difference in the parameters for the $p = .1$ and $p = .05$ conditions seen in Figure 5 undermines confidence in extrapolations to typical acquisition, where $p = 1.0$.

It is possible to test the predictions for extinction within the context of the present experiments, where parameter change is not so central an issue, for there were long stretches (especially in the $p = .05$ condition) without food. The relevant equation, transplanted from the Appendix (Equation A6), is

$$s_{i+1} = s_i(1 - \pi_E)[1 + \pi_{P-Q}(1 - s_i)], \qquad (8)$$

where the strength s_{i+1} gives the probability of entering a response state on that trial. All parameters are positive, with asymptotes of 0 or 1 used as appropriate to the signs shown in Figure 5. Neither π_F nor π_{PF} appear because there are no food trials in a series of extinction trials, and π_{PE} is typically small and its work can be adequately handled by π_E. The probability of responding on a trial decreases with π_E as expected (note the element $-\pi_E\ s_i$)—substantially when s_i is large, not much at all when s_i is small. Only the difference in the two momentum parameters, $\pi_P - \pi_Q$, affects the prediction; for parsimony, we collapse those into a single parameter representing their difference, $\pi_{P-Q} = \pi_P - \pi_Q$. Equation 8 makes an apparently counterfactual prediction.

A Surprising Prediction

Inspection of Figure 5 shows that π_{P-Q} is generally positive. Because it multiplies the probability of not responding (Equation 8 contains the element $\pi_{P-Q}[1 - s_i]$), on average π_{P-Q} increases the probability of responding on each trial and does so more as s_i gets small. Depending on the specific value of the parameters, this restorative force may be sufficient to forestall extinction. To show this more clearly, we solve Equation 8 for its fixed point, or steady state, which occurs when $s_{i+1} = s_i$:

$$s_\infty = 1 - \frac{\pi_E}{\pi_{P-Q}(1 - \pi_E)}, \qquad (9)$$

where $0 < \pi_{P-Q}(1 - \pi_E) \leq \pi_E$; this is the level at which responding is predicted to stabilize after a long string of extinction trials.

If response probability fluctuates below the level of s_i, the next response (if and when it occurs, which it does with probability s_i) will drive probability up, and if it fluctuates above this level, the next trial will drive it down. For responding to extinguish, it is necessary that the force of extinction be greater than the restoring force:

$$\pi_E \geq \frac{\pi_{P-Q}}{1 + \pi_{P-Q}}. \qquad (9)$$

This is automatically satisfied whenever momentum in quiescence, π_Q, is greater than momentum in pecking π_P—whenever π_{P-Q} is negative. That is especially likely to be the case in rich

Table 4
Indices of Merit for the Model Comparison of Experiment 1

Bird no.	Metric[a]	20	40_1	40_2	80	.05
98	CD	0.03	0.06	0.17	0.06	0.19
	AIC	9	−1	32	29	115
	BIC	−17	−18	15	20	97
105	CD	0.07	0.02	0.17	0.13	0.16
	AIC	57	72	162	105	375
	BIC	38	55	134	91	352
106	CD	0.17	0.03	0.18	0.05	0.19
	AIC	47	40	69	5	217
	BIC	22	17	46	−14	193
107	CD	0.04	0.09	0.14	0.05	0.11
	AIC	101	40	13	18	88
	BIC	82	34	2	8	70
113	CD	0.04	0.07	0.30	0.03	
	AIC	12	27	38	24	
	BIC	−7	10	19	9	
119	CD	0.02	0.01	0.02	0.07	0.07
	AIC	57	40	8	29	87
	BIC	25	17	−14	19	69
Group	CD	*0.06*	*0.05*	*0.16*	*0.06*	*0.14*
	AIC	*47*	*36*	*54*	*35*	*176*
	BIC	*24*	*19*	*34*	*22*	*156*

Note. Italics indicate averages over the group.
[a] The metrics of goodness of fit for the models are the coefficient of determination (CD), the Akaike information criterion (AIC), and the Bayesian information criterion (BIC). Values of the last two greater than 4 constitute strong evidence for the Momentum/Pavlovian/Skinnerian model.

contexts where quiescence on the target key may be associated with foraging in another patch or responding on a concurrent schedule. For the parameters in Figure 5 under $p = .05$, however, this is never the case; indeed, the more general inequality of Equation 10 is never satisfied. Therefore Equations 8 and 9 make the egregious prediction that the probability of responding will fall (with a speed dictated by π_E) to a nonzero equilibrium dictated by Equation 9. We may directly test this derivation by plotting the course of extinction within the context of dynamic reconditioning of these experiments. The best data come from the $p = .05$ condition, which contained long strings of nonreinforced responding. The courses of extinction, along with the locus of Equation 8, are shown in Figure 8.

Do Equations 8–10 condemn the birds to an endless Sisyphean repetition of unreinforced responding? If not, what then saves them? Those equations are continuous approximations of a finite process. Because the right-hand side of Equation 8 is multiplied by s_i, if that probability ever does get close enough to 0 through a low-probability series of quiescent trials, it may never recover. It is also likely that after hundreds of extinction trials, the governing parameters would change, as they did across the conditions of this experiment, releasing the pigeons to seek more profitable employment. The maximum number of consecutive trials without food in this condition averaged around 120. Surely over unreinforced strings of length 95 through 120, the probability of responding would be decreasing toward zero. Such was the case for 2 pigeons, 98 and 107, whose response probability decreased significantly (using a binomial test) to around 5% (the drift for 107 is already visible in Figure 8). The predicted fixed points and obtained probabilities for another 2, 105 and 119, were invariant, .20→.19 and .77→.78, respectively; Pigeon 106 showed a decrease in probability, .61→.54, that was not significant by the binomial test. The substantial momentum shown in Figure 8, and extended in some cases by the binomial analysis, resonates with the data of Killeen (2003; cf. Sanabria, Sitomer, & Killeen, 2006), where some pigeons persisted in responding over many thousands of trials of negative automaintenance.

The validation of this unlikely prediction should, by some accounts of how science works, lend credence to the model. But it certainly could also be viewed as a fault of the model, in that it predicts the flatlines of Figure 8, when few pigeons, except perhaps those subjected to learned helplessness training, will persist in unreinforced responding indefinitely. On that basis we could reject the MPS model because it does not specify when the pigeons will abandon a response mode (as reflected in changes in the persistence parameters). Conversely, the data of Figure 8 indict models that do not predict the plateaus that are clearly manifest there. On that same basis, we could therefore reject all of the remaining models. But perhaps the most profitable path is to reject Popper in favor of MPS, which permits tracking of parameters over an indefinite number of trials, to see when, under extended dashing of expectations, those begin to change.

Equation 8 contains the element $s_i(1 - s_i)$: The product of the probability of a response and its complement enters the prediction of response probability on the next trial. This element is the core of the "logistic map." Depending on the coefficient of this term, the pattern of behavior it governs is complex and may become chaotic. This, along with the multiple timescales associated with the rate parameters, is the origin of the chaos that Killeen (2003) found in the signatures of pigeons responding over many trials of automaintenance and the factor that gives the displays in Figures 1 and 6 their self-similar character.

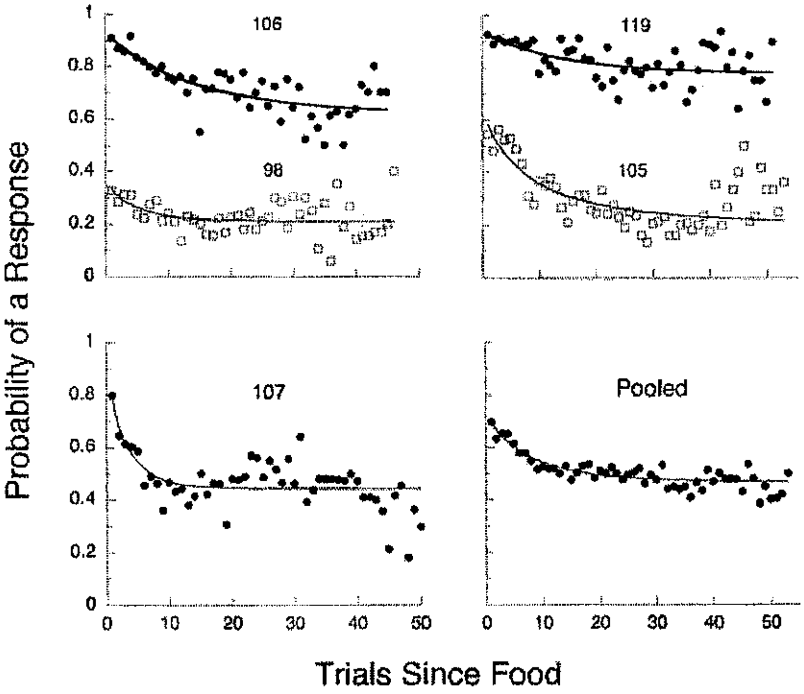

Figure 8. The average probability of responding as a function of the number of trials since reinforcement, from the $p = .05$ condition. The number of observations decreases by 5% from one trial to the next, from hundreds for the first points to 10 for the last displayed. The curve comes from Equation 8, using parameters $\pi_{P\text{-}Q}$ and π_E fit to these data.

Experiment 2: Trial Duration

The trial-spacing effect depends on the duration of both the ITI and the trial; arrangements that keep that ratio constant often yield about the same speed of acquisition of responding. Therefore, to test the generalizability of both the response rate model and the MPS model, we systematically varied trial duration in this experiment.

Method

Subjects and Apparatus

Six experienced adult homing pigeons, housed in similar conditions as in Experiment 1, served. Pigeons 105, 106, 107, 113, and 119, who had participated in Experiment 1, were joined by 108, who replaced 98. The apparatus remained the same.

Procedure

Seven sessions of extinction were conducted before beginning this experiment. In extinction, stimulus conditions were similar to those of Experiment 1, but the ITI was 35 s and trial duration was 10 s; no food was delivered ($p = 0$). In experimental conditions, food was delivered with $p = .05$, the ITI remained 35 s, and trial duration varied, starting at 10 s for 13 sessions. Then half the subjects went to the 5-s CS condition, and half went to the 20-s CS condition. Finally, the 10-s CS was recovered. All sessions lasted 150 trials; Table 5 reports the number of sessions per condition.

Results

In the last session of extinction, the typical pigeon pecked on 3% of the trials. This is a lower percentage than shown in Figure 8 because it follows six sessions of extinction. Extinction happens. On moving to the first experimental condition, this proportion increased to an average of 75%. The average response rates and probabilities of responding are shown in Figure 9. Both rates and probabilities decreased as CS duration increased. Also shown are average rates from 4 pigeons studied by Perkins et al. (1975) for CS durations of 4, 8, 16, and 32 s for pigeons maintained on probabilistic ($p = 1/6$) Pavlovian conditioning schedules, with an ITI of 30 s. (The average rate at 32 s was 0.2 responses per second). The higher rates for Perkins et al.'s subjects are probably due to their higher rates of reinforcement (1/6 trials compared with our 1/20). The decrease in response rate with CS duration is consistent with the data of Gibbon et al. (1977), who found

Table 5
Conditions of Experiment 2

Order	Trial duration (seconds)	Sessions
1	10	13
2	5, 20	13
3	20, 5	13
4	10	14

Note. Half the subjects experienced the extreme trial durations in the order 5 s, 20 s; half experienced them in the other order.

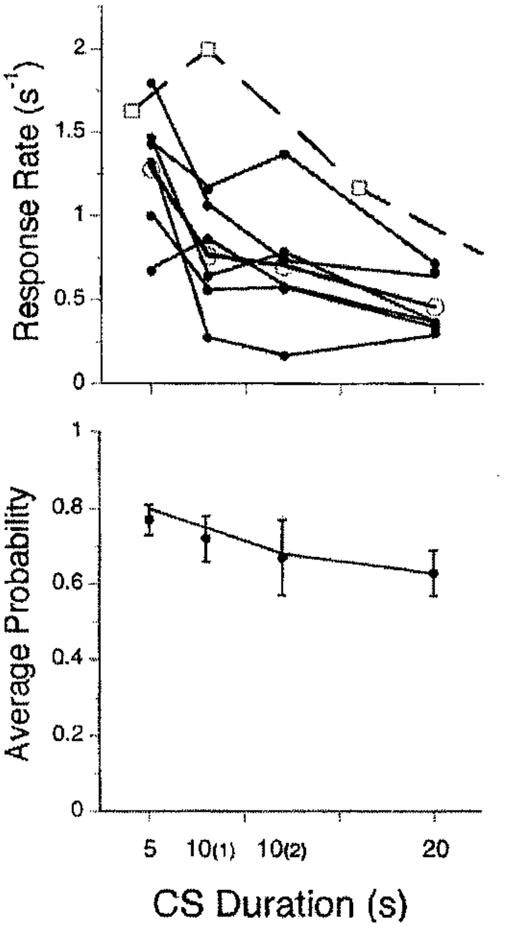

Figure 9. Data from Experiment 2. Top: average response rate (dots) for each subject. Open circles gives average rate and squares represent data from Perkins et al. (1975). Bottom: Average probability of making at least one response on a trial averaged over pigeons; bars give standard errors. Unbroken lines in both panels are from the Momentum/Pavlovian/Skinnerian model. CS = conditioned stimulus.

that rate decreased as a power function of trial duration, with exponent −0.75. A power function also described rates in these experiments, accounting for 99% of the variance in the average data, with exponent −0.74.

The MPS model continued to outperform the base momentum model, with an average advantage of 130 AIC units, giving it an advantage in likelihood of e^{130}. The parameters were larger than those found in the last condition of Experiment 1 (see Figure 10 and Tables 6 and 7) and on average did not show major changes among conditions, although the impact of a trial with food was greatest in the first condition studied, 10(1), and there were slight decreases in π_{PF} and π_{Q} as a function of trial duration. There was a moderate increase in the average number of responses emitted (c, top panel of Figure 10) as trial duration increased from 5 s to 20 s; the birds adjusted to having longer to peck before the chance of reinforcement carried them to the hopper.

Despite the importance of trial duration for acquisition of autoshaped responding, the changes in the conditioning parameters as a function of that variable were modest. They did, however, work in unison to decrease response rates as the CS duration increased. The only moderate changes may be due to the very short ITI in this series. The biggest effect was the transition into the first condition of the experiment, the first 10-s CS, after several sessions of extinction, where the Pavlovian and Skinnerian learning parameters π_F and π_{PF} were as large or larger than in any other conditions. Empty trials, although common, had little effect on behavior because π_E was generally very close to 0. In general, the dominance of π_F over π_{PF} (and the other parameters), especially at the longest CS duration, may have been due to the extended opportunity for nonreinforced pecking in that long CS condition.

In interpreting these parameters, and those of Figure 5, it is important to keep in mind that π_E was in play on 95% of the trials,

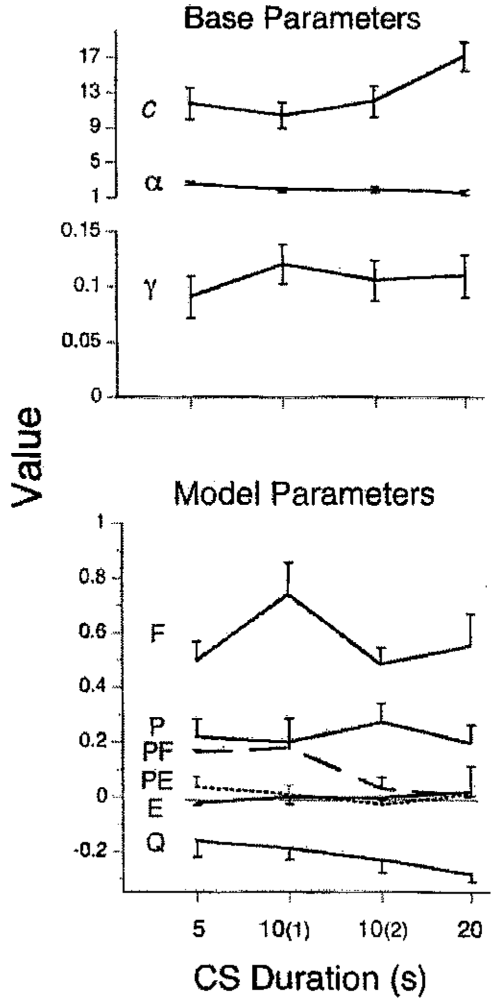

Figure 10. The average parameters of the base and Momentum/Pavlovian/Skinnerian models for Experiment 2. The conditions are identified by their trial duration, with the first and second exposure to the 10-s intertrial interval noted parenthetically. The same Weibull parameters, c and α, were used for both models. The error bars delimit the standard errors of the mean. π_{PF} is traced by a dashed line, and π_{PE} by a dotted line. F = food; P = response; E = no food; Q = no response; PF and PE = Skinnerian interaction terms.

Table 6
Indices of Merit for the Model Comparison of Experiment 2

Bird no.	Metric[a]	5	10_1	10_2	20
105	CD	0.01	0.32	0.26	0.29
	AIC	5	347	365	360
	BIC	0	319	342	338
106	CD	0.27	0.07	0.26	0.15
	AIC	169	106	285	163
	BIC	152	79	262	141
107	CD	0.25	0.12	0.22	0.11
	AIC	193	141	172	119
	BIC	170	118	155	97
108	CD	0.10	0.14	0.08	0.05
	AIC	79	127	49	37
	BIC	62	104	32	20
113	CD	0.23	0.15	0.04	0.05
	AIC	183	214	40	34
	BIC	155	197	12	17
119	CD	0.12	0.11	0.12	0.10
	AIC	75	56	81	54
	BIC	53	45	53	32
Group	CD	0.18	0.12	0.22	0.10
	AIC	124	134	127	87
	BIC	107	111	104	64

[a] The metrics of goodness of fit for the models are the coefficient of determination (CD), the Akaike information criterion (AIC), and the Bayesian information criterion (BIC). Values of the last two greater than 4 constitute strong evidence for the Momentum/Pavlovian/Skinnerian model.

either π_P or π_Q on every trial, $\pi_{P'}$ on 5% of the trials, and $\pi_{PP'}$ on fewer than 5% of the trials. Thus, a trial with food in this experiment would move response strength a very substantial 60% of the way to maximum—but this happened only rarely.

Once again, the quiescence parameter π_Q was the primary force driving the probability of entry into the response state toward 0, having a mean value of $-.215$. This value, so close to that for π_P (.222), indicates that the momenta of pecking and quiescence were, on average, essentially identical. This situation, $\pi_{P-Q} \approx 0$, will not sustain asymptotic responding above zero (see Equation 10); with so short an ITI, that is perhaps not surprising. The success of this prediction is illustrated in Figure 11 for the 5-s CS condition, which showed no evidence of a plateau. The slight negative acceleration is due to the dominance in the pooled data of profiles from pigeons whose π_{P-Q} was negative. This analysis may throw additional light on within-session partial reinforcement extinction effects (Rescorla, 1999) because different animals or paradigms may have quite different values of π_{P-Q}.

Because these conditions were preceded by seven sessions of extinction, the opportunity arises to trace the course of reacquisition for these birds and compare it with the model's profiles. The probability of a response on each of the first 100 trials, averaged over all pigeons and over a 7-trial moving window, is drawn as circles in Figure 12. The MPS model provides a closed-form solution to the acquisition curve. The equation is shown in the Appendix; the smooth acquisition curve is shown in Figure 12. The curve provides—at best—an idealized picture of the process because it assumes that response probability is dependent on the programmed probability of food, p, which is uniform over trials. MPS can do better than that by using the real thing—whether food was delivered or not—to inform its predictions. Replacing p with the trial-to-trial relative frequency across pigeons, represented by the hatch marks in the figure, and keeping all parameters otherwise the same, gives the jagged curve, a better characterization of the process. Figure 12 draws a graphic reminder of a point made by Benedict and Ayres (1972): Nonlinear dynamic processes, such as the course of learning, can be extremely sensitive to the particulars of stochastic processes. Generic models with asymptotic parameters, such as limiting values for p or even for s, will provide at best an idealization; the dynamics is in the details. The textbook-smooth curve shown in Figure 12 does not represent the character of the data. Over the full course of the experimental conditions, MPS easily supports its burden of parameters, as attested by its AICs, and carries us from milquetoast descriptions to the jagged profiles of Figure 12—to predictions with teeth.

All of the manipulations so far have been classic Pavlovian kinds, varying experimental parameters that did not interact with behavior, and those only modestly (see, e.g., Schachtman, 2004, for some modern developments). Although noncontingent food presentation can leave response–outcome associations intact (Colwill, 2001; Rescorla, 1992), all of the response–outcome associations up to this point were adventitious. We conducted the last series of experiments to complement those open-loop Pavlovian operations with closed-loop instrumental operations having more consistent contingencies.

Experiment 3: Fixed Ratio and Differential Reinforcement of Other Behavior Contingencies

Method

Subjects and Apparatus

Eight experienced adult homing pigeons, half having served in other experiments reported in this article, were used. They were maintained under the same conditions as the prior experiments. The apparatus was the same as used before.

Table 7
Parameter Values of the Base and Momentum/Pavlovian/Skinnerian Models for the Data of Experiment 2

Bird no.	Parameter	5	10_1	10_2	20
105	γ	0.01	0.17	0.13	0.14
	c	8.47	7.07	6.45	15.67
	α	3.03	1.53	1.58	1.73
	P	0.00	0.16	0.21	0.34
	Q	−0.01	−0.27	−0.33	−0.45
	F	0.13	0.83	0.55	0.71
	E	0.00	0.02	0.01	0.04
	E	0.00	0.02	0.01	0.04
	E	0.00	0.02	0.01	0.04
106	γ	0.12	0.09	0.15	0.18
	c	12.87	16.53	16.87	22.21
	α	3.32	2.00	1.78	1.30
	P	0.31	0.08	0.45	0.30
	Q	−0.19	−0.21	−0.30	−0.29
	F	0.68	0.35	0.44	0.60
	E	0.00	0.10	−0.14	0.02
	PF	0.00	0.62	0.00	0.00
	PE	−0.03	−0.02	−0.08	−0.03
107	γ	0.14	0.14	0.15	0.11
	c	8.95	9.26	11.73	17.65
	α	2.68	1.60	1.73	1.42
	P	0.00	0.00	0.19	−0.04
	Q	−0.14	−0.14	−0.16	−0.19
	F	0.69	0.69	0.48	0.38
	E	−0.03	−0.02	−0.02	0.01
	PF	0.00	0.00	0.00	0.00
	PE	0.17	0.18	0.00	0.15
108	γ	0.11	0.14	0.08	0.05
	c	5.70	119.00	41.00	29.00
	α	1.45	0.15	0.06	0.08
	P	0.10	0.29	0.02	0.00
	Q	−0.15	−0.24	−0.03	−0.08
	F	0.55	1.00	0.20	0.30
	E	0.04	0.03	0.00	−0.01
	PF	0.00	0.00	0.00	0.00
	PE	0.00	−0.02	0.00	0.10
113	γ	0.13	0.12	0.07	0.08
	c	17.22	10.77	10.08	11.40
	α	2.01	2.12	2.60	1.21
	P	0.39	0.54	0.12	0.06
	Q	−0.19	−0.10	−0.11	−0.09
	F	0.34	0.87	0.39	0.27
	E	−0.06	−0.10	0.02	0.02
	PF	0.34	0.00	0.02	0.00
	PE	0.00	0.00	0.00	0.00
119	γ	0.08	0.05	0.08	0.06
	c	10.04	15.25	16.99	19.68
	α	2.96	3.18	3.61	2.35
	P	0.26	0.00	0.41	0.05
	Q	−0.10	−0.02	−0.15	−0.09
	F	0.00	0.32	0.64	0.46
	E	−0.02	0.00	−0.02	−0.01
	PF	0.67	0.00	0.25	0.00
	PE	0.00	0.00	0.00	0.12

Note. γ = the rate constant for the comparison base model; *c* = the Weibull rate constant; α = the Weibull shape constant; the remaining letters indicate the rate constants brought into play on trials with (P) or without (Q) a response; with (F) or without (E) food; and the Skinnerian interaction terms PF and PE.

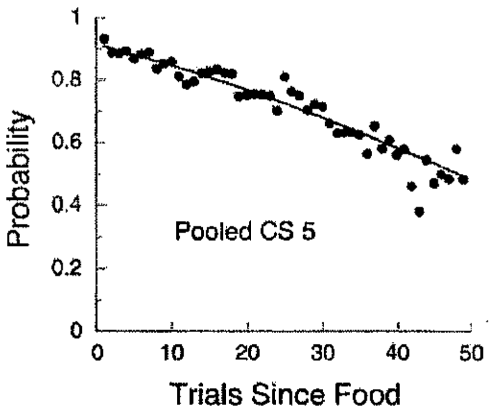

Figure 11. The average probability of responding as a function of the number of trials since reinforcement, from the 5-s conditioned stimulus (CS) condition of Experiment 2, pooled over subjects. The number of observations decrease by 5% from one trial to the next, from 485 for the first point to 29 for the last displayed. The curve comes from Equation 8, using parameters π_{P-Q} and π_E fit to these data.

Procedure

Before the experiment proper, six to seven sessions of extinction were conducted with a 35-s ITI and 10-s trial duration; the probability of food delivery was zero ($p = 0$). A preliminary series of experiments was conducted with $p = .05$. In these conditions, which we call fixed-ratio 3 (FR 3) and differential reinforcement of other behavior (DRO), reinforcement contingencies were intended to vary response–reinforcement contiguity in opposite directions. However, the low probability of exposure to those contingencies—5% of the trials at most, usually less—gave animals insufficient exposure to the Skinnerian contingencies: In a number

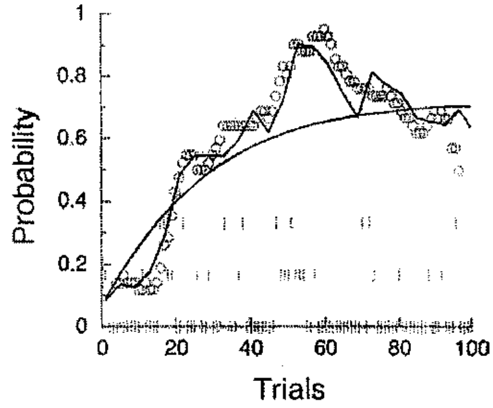

Figure 12. The average probability of responding as a function of the number of trials since the start of the 10-s conditioned stimulus condition of Experiment 2, pooled over subjects, and represented as a seven-trial moving average (circles). The hatch marks indicate trials on which 1 (plotted at p = 1/6) or 2 (plotted at p = 2/6) pigeons happened to have received food. In no cases did the same trial end with food for more than 2 pigeons. The smooth curve comes from the Momentum/Pavlovian/Skinnerian model, setting p = .06, with all other parameters fit to these data. The jagged curve comes from the same equation with the same parameters but uses the obtained relative frequency of food as given by the hatch marks.

of cases, there was no change consistent with the direction in which the contingencies were pushing. The probability of food was increased to $p = .1$, and the series replicated.

DRO. A DRO schedule was operative concurrently with baseline automaintenance contingencies, but only when food was programmed, which happened with a probability of $p = .10$. If an animal pecked during the 2 s preceding the delivery of food in a DRO trial, the trial was extended for an additional 2 s from the peck, until the pigeon had not pecked for 2 s, when food was finally delivered. All pigeons received 18 sessions of baseline training before being moved to the experimental conditions.

FR. A FR 3 schedule of reinforcement was operative concurrently with baseline automaintenance contingencies, but only when food was programmed. Thus, a trial in the FR 3 condition in which food was programmed (10% of the trials) would be terminated immediately by food delivery as soon as three key pecks were emitted. If three pecks were not emitted, the trial ended with noncontingent food presentation.

The order of experimental conditions was determined by the mean response rate during the last five sessions of baseline: Low responders were assigned first to DRO and high responders to FR 3. Table 8 shows the order of presentation of conditions and the number of sessions in each condition. In analyzing the data, all trials with a reinforcer were excluded from measurement of goodness of fit. This is because responding could have extended or shortened the trial duration, undermining comparison. This reduced the database by 10%.

Results

The contingencies, even though present on only 10% of the trials (DRO) or fewer (FR, which would end with food after 10 s if the FR contingency had not been met), were effective with most of the pigeons. This is consistent with the results of Locurto, Duncan, Terrace, and Gibbon (1980). The requirements were satisfied on 79% of the trials (median, with interquartile range from 68% to 81%). The effects of the contingencies on response rates and probabilities are displayed in Figure 13. Reinforcement contingencies clearly matter, by affecting both the probability of entering a response state and the number of responses emitted in that state.

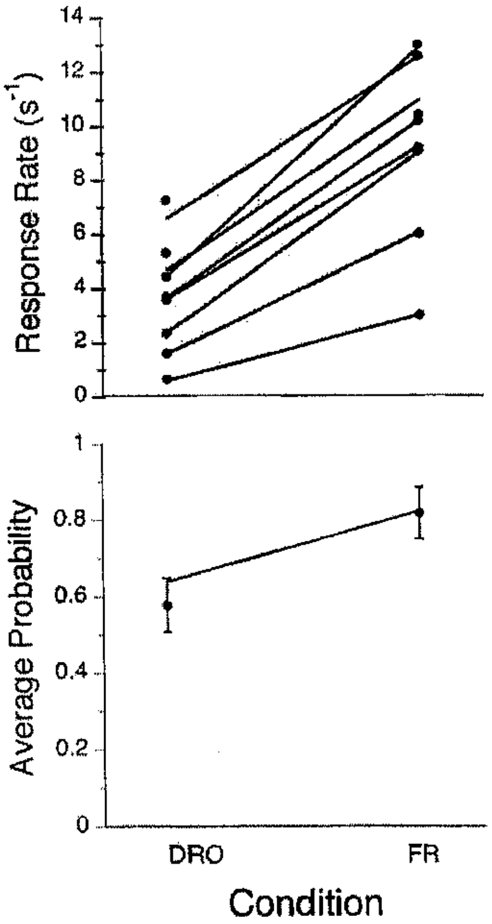

Figure 13. Data from Experiment 3. Top: average response rate (dots) for each subject. Bottom: Average probability of making at least one response on a trial averaged over pigeons; bars give standard errors. Lines in both panels are from the Momentum/Pavlovian/Skinnerian model. FR = fixed ratio; DRO = differential reinforcement of other behavior.

Of the 24 Subject × Condition analyses, in 18 the MPS model exceeded our criterion for strong evidence (see Table 9). Averaged over all subjects, the AIC advantage for the MPS model over the base model was 38 points. Figure 14 displays the weighted average parameters for the base model (simple persistence) and the MPS model for the key DRO–FR comparisons. Table 9 lists the individual parameters. Note that as the contingencies went from DRO to FR, all parameters in the top panel increased in value. The increase in alpha indicates that the distribution of number of responses moved from one that looked like a gamma distribution ($\alpha = 1.46$) to one that looked like a skewed normal distribution ($\alpha = 2.23$; see Figure 3). The doubling of c reflects a large increase in the mean number of responses emitted in the response state in the FR condition. The increase in gamma indicates that pecking tended to occur more often in alternative strings of responding or quiescence, making it advantageous for the simple moving average of the base model to place more weight on the recent history of responding in the FR conditions.

Table 8
Order (and Number of Sessions) in Each Condition of Experiment 3

Condition	P43	P86	P87	P89	P90	P105	P106	P107
Training	1 (18)	1 (18)	1 (18)	1 (18)	1 (18)	1 (18)	1 (18)	1 (18)
FR 3	2 (17)	3 (7)	3 (7)	2 (17)	2 (17)	3 (17)	2 (17)	3 (7)
DRO 2 s	3 (7)	2 (17)	2 (17)	3 (7)	3 (7)	2 (7)	3 (7)	2 (17)

Note. P = pigeon; FR = fixed ratio; DRO = differential reinforcement of other behavior.

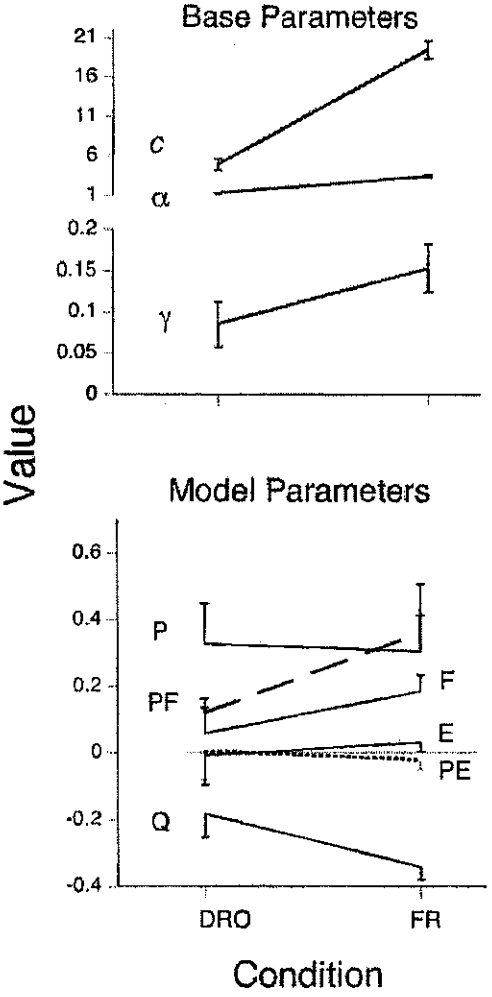

Figure 14. The average parameters of the base and Momentum/Pavlovian/Skinnerian models for the differential reinforcement of other behavior (DRO) and fixed-ratio (FR) contingencies of Experiment 3. The same Weibull parameters, *c* and α, were used for the response distributions of both models. The error bars delimit the standard errors of the mean.

The main purpose of this experiment was to test the sensitivity of the MPS model to changes in behavior brought about by the manipulation of contingencies of reinforcement, and in particular to monitor changes in the instrumental learning parameter, π_{PF}. Figure 14 shows that there was a large increase in π_{PF} under the FR contingencies and smaller changes in some of the other parameters (see Table 9). Trials without a reinforcer had, on average, no effect, because π_E was very close to zero for most animals in most conditions, as was π_{PE}. The momentum parameter for the base model (γ) was larger under the FR condition, suggesting greater movement into and out of response states, whereas those for the MPS model (π_P and π_Q) were in line with those found in Experiment 2. The decrease in the latter under FR suggests a kind of ratio strain: Absence from the key on one trial became a better predictor of absence on the next.

The smaller value of π_{PF} under DRO should not be taken as an indication that the pigeons were learning less; they were learning to do other things than key pecking. A smaller value of π_{PF} indicates that they were less likely to peck on an ensuing trial. If they received food on a trial with a peck, the peck was removed from the reinforcer by at least 2 s and was followed by non–key-peck behavior. This latter was successfully reinforced, yielding the smaller tendency to peck on the next trial than found under the FR contingency.

The impression from the first two experiments, that the power of instrumental contingencies was weak compared with Pavlovian contingencies, now stands corrected. Where there is no instrumental contingency, but only adventitious pairing of responding as in the first two experiments, control by that pairing can be weak or nil. This may be due to the many instances of pecking without presentation of food, causing the pigeon to place little weight on pecking as a predictor of food. In Experiment 3, FR contingencies trebled the Skinnerian parameter π_{PF} from .12 to .36. Under the FR contingencies, each contingent presentation of food moved the typical pigeon a third of the way to certain responding on the next trial, with the persistence and Pavlovian parameters together halving the remaining distance. The increase in response rates seen in the top panel of Figure 13 for FR thus arises from two factors: An increased probability of entering a response state in that condition because of the action of these parameters and a higher rate of responding once in that state, reflected by the increase in *c*. The first set of conditioning factors are substantial and consistent with the theoretical position of Donahoe, Palmer, and Burgos (1997a), yet they are inadequate to completely explain the large differences in rate. Differential proximity between responses and reinforcement in these two conditions is further affecting the behavior within the response state, much as it does in free operant schedules (e.g., Killeen, 1969). The parameter *c* reflects the operation of instrumental conditioning in the response state, moving more of the conditioned behavior onto the key.

The joint role of respondent and operant conditioning demonstrated here was presaged by Wasserman, Hunter, Gutowski, and Bader (1975) in their study of automaintained responding in chicks, with warmth as the US/S^R. Locurto et al. (1980) found similar interactions and suggested adopting "an 'interactivist' position wherein Pavlovian and instrumental relations are seen as independent variables which conjointly determine the outcome of any conditioning procedure" (p. 42). It was manifest in an experiment by Osborne and Killeen (1977), who superimposed CSs ranging from 7.5 s to 120 s on a variable-interval schedule that only reinforced responses spaced by 3 s (TAND[VT60, DRL3]). Even though the CS signaled noncontingent food, it enhanced median response rates from a baseline of 25 per minute to 170 per minute at the shortest CS, decreasing monotonically to 45 per minute at the longest. They successfully analyzed within the CS with an extreme value function in the same family as the Weibull used here. Such within-CS analysis begins to fill one of the silences of the R-W model (Hanson, 1977; Miller & Barnet, 1993).

General Discussion

Momentum

The analysis of momentum, or durability of responding, has a long history marked by two changes of paradigm. The first was the discovery of the partial reinforcement extinction effect by Hum-

Table 9
Indices of Merit, and Parameter Values of the Base and Momentum/Pavlovian/Skinnerian Models for the Data of Experiment 3

	Schedule							
Parameter	FR	DRO	FR	DRO	FR	DRO	FR	DRO
Bird no.	43		86		87		89	
CD	0.11	0.16	0.21	0.07	0.09	0.05	0.12	0.02
AIC	77	19	−5	84	−2	−2	63	19
BIC	59	4	−19	66	−14	−17	39	−1
γ	0.15	0.08	0.12	0.08	0.02	0.06	0.08	0.04
α	11.66	3.62	16.95	6.93	12.90	10.75	7.99	4.39
c	2.45	1.57	2.36	1.01	1.49	1.84	1.85	1.69
P	0.31	0.16	0.09	0.22	0.04	0.02	0.11	0.83
Q	−0.20	−0.10	−0.26	−0.12	−0.03	−0.03	−0.33	0.41
F	0.31	0.10	0.14	−0.13	0.01	0.00	0.18	−0.47
E	−0.01	0.00	0.07	0.02	0.00	0.00	0.09	−0.74
PF	0.00	0.42	0.00	0.00	0.00	0.07	0.00	0.00
PE	0.00	0.00	0.00	0.00	0.00	0.00	−0.03	0.13
Bird no.	90		105		106		107	
CD	0.53	0.31	0.13	0.06	0.20	0.07	0.18	0.42
AIC	435	176	10	17	2	84	71	9
BIC	406	151	0	5	−8	66	54	−2
γ	0.31	0.26	0.22	0.03	0.11	0.08	0.18	0.09
α	12.64	4.64	15.76	10.15	16.52	6.93	14.31	5.82
c	2.53	1.42	2.11	1.52	2.34	1.01	2.00	1.39
P	0.66	0.82	0.24	0.02	0.00	0.22	0.12	0.14
Q	−0.55	−0.46	−0.18	−0.03	−0.27	−0.12	−0.25	−0.10
F	0.18	0.16	0.07	0.13	0.01	−0.13	0.52	0.03
E	0.02	0.01	0.00	0.00	0.15	0.02	0.00	0.00
PF	1.00	0.57	0.00	0.00	0.00	0.00	0.14	0.00
PE	−0.05	−0.09	0.00	0.00	0.00	0.00	0.00	0.00

Note. γ = the rate constant for the comparison base model; c = the Weibull rate constant; α = the Weibull shape constant; the remaining letters indicate the rate constants brought into play on trials with (P) or without (Q) a response; with (F) or without (E) food; and the Skinnerian interaction terms PF and PE. FR = fixed ratio; DRO = differential reinforcement of other behavior.

phreys (1939)—the paradoxical result that probabilistic reinforcement generates more responses in extinction than does continuous reinforcement. It generated a tremendous and continuing amount of research (Mackintosh, 1974). The second was the renewed call of attention to momentum by Nevin and his students (Nevin & Grace, 2001; Nevin, Mandell, & Atak, 1983; Nevin, Tota, Torquato, & Shull, 1990) under the rubric *behavioral momentum*. As is the case for the partial reinforcement extinction effect, which it helps to explicate (Nevin, 1988), the study of behavioral momentum has applications well beyond the animal behavior laboratory (Nevin, 1996; Plaud & Gaither, 1996). It is most closely associated with the opposing forces of π_P and π_Q and, in extinction, with their simple difference π_{P-Q}.

This work has shown that behavioral momentum is most closely associated with Pavlovian forces, such as the relative densities of food in CS and background, and less so with instrumental contingencies and rates of responding. Consistent with Nevin and associates' results (Nevin & Grace, 2001; Nevin, Mandell, & Atak, 1983; Nevin, Tota, Torquato, & Shull, 1990), Figure 5 shows that when ITI was varied, persistence in both pecking π_P and quiescence π_Q, and their difference, π_{P-Q}, increased with the Pavlovian variable of ITI-to-trial (ITI/T) ratio; Figures 2 and 9 show that when trial duration was varied, persistence in both pecking and quiescence decreased with decreases in ITI/T, and Figure 14 shows that despite radically different responding under DRO and FR contingencies, π_{P-Q} was about the same in those experimental conditions, indicating that momentum would also be about the same, echoing Nevin and associates' conclusions. The influence of prior behavior on current behavior has been demonstrated in a different paradigm by de la Piedad, Field, and Rachlin (2006), who underscored the importance of the persistence they demonstrated for issues of rationality and self-control, a theme most beautifully introduced to our field by James (1890a, 1890b). The current paradigm and analysis provides a new set of operations for testing and developing behavioral momentum theory and other more general theories of momentum and choice (e.g., Killeen, 1992; Roe, Busemeyer, & Townsend, 2001).

Conditioning

"Today, most contemporary theories of acquired behavior are predicated on observations initially made to assess the Rescorla-Wagner model" (Miller et al., 1995, p. 381). The MPS model developed here is in that tradition; it is an "error-correction" model, like the R-W model and its linear-learning model forebears. Deviation from complete momentum or quiescence and deviation from complete conditioning or extinction both proceed as a function of distance from asymptote. This aperçu, however, may reflect more a limitation of imagination on our part than on the organisms'. Only a few of the infinite number of possible models of conditioning have been evaluated.

Does the MPS model capture learning or performance effects? It predicts response strength, *s*, the probability that the pigeon will be in a response state, with the rate of responding in that state given by the Weibull distribution. What the pigeon learns in this context is relative frequencies of food given keylight and given both light and peck. The scheduled probabilities of these is constant (at .1 or .05 or 0) in all these experiments, but the random sampling of trials by responses makes the observed frequencies a continually varying estimator of those probabilities. The strengths of the context, key, and peck state could be continuously varying, each in their own way, with our reduction to a net strength (*s*, probability of entering the response state) a synopsis of more nuanced three-way tugs of war among these factors. Models that keep separate accounts of these components of learning might easily trump MPS in rich data sets such as these, despite their extra parameters, or in others in which the forces are put into strong opposition. By treating instrumental responses as stimuli to be approached in the same manner as a lit key (Bindra, 1978), SOCR (Stout & Miller, 2007), attentional models (Frey & Sears, 1978; Mackintosh, 1975), RET (Gallistel & Gibbon, 2000) and its refinement by Kakade and Dayan (2002), SOP (Brandon, Vogel, & Wagner, 2003), WILL (Dayan, Niv, Seymour, & Daw, 2006), and the artificial neural net genre (e.g., Burgos, 1997; Donahoe, Palmer, & Dorsel, 1994) may be evaluated against these data. This paradigm also provides an ideal environment to analyze the potential progression of "learned irrelevance" (Baker, Murphy, & Mehta, 2003).

A limitation of the current analysis is its focus on one well-prepared response, appetitive key pecking in the pigeon. The relative importance of operant and respondent control will vary substantially depending on the response system studied (Donahoe, Palmer, & Burgos, 1997b; Jenkins, 1977; Timberlake, 1999). Another is that we have fit only a limited number of models to the data—albeit more than mentioned here, including versions of SOCR (Stout & Miller, 2007), attentional models (Frey & Sears, 1978; Mackintosh, 1975), and RET (Gallistel & Gibbon, 2000) and its improvement by Kakade and Dayan (2002). The models we present in this article were the best of the lot. But other models might have done better, in particular ones with attention (Mackintosh, 1975) or memory (Bouton, 1993; Wagner, 1981) as latent states. All theories, successful and otherwise, are at best sufficient accounts of the phenomena that they cover (Mazur, 2006), as Poincaré (1905/1952) noted long ago.

The dependent variable was a standard operant response. Holland (1979) has shown that omission contingencies have differential effects on various components of Pavlovian conditioned responding in rats. It may be that the difference is merely greater associability of different responses (Killeen, Hanson, & Osborne, 1978; Seligman, 1970), manifested as differences in the pi parameters. Indeed, it maybe that in some configurations, the Pavlovian parameter goes negative, with delivery of food increasing goal approach (Timberlake, 1994) on the next trial, competing with the measured operant. Such possibilities have yet to be demonstrated.

Another limitation is that MPS does not address the key contribution of the R-W model and its successors, cue competition and the partitioning of attention in the conditioning process. It did not need to here because changes in the predictive value of key or peck change the probability of entering the response state in the same direction: The conditionals coordinate rather than compete. Independent bookkeeping for cue, context, and peck conditioning were assayed in preliminary evaluation of the models presented here, but the experimental paradigm did not generate enough leverage where those models might contribute their strong suits. The partitioning out of momentum that the MPS model permits may, for the right experimental paradigm, provide a much clearer signal for how the Pavlovian and Skinnerian factors—or Pavlovian and Pavlovian factors—compete, or where one differentially sets the occasion for the other (Colwill & Rescorla, 1986; Nadel & Willner, 1980; Schmajuk, Lamoureux, & Holland, 1998). Such qualitative tests work hand in hand with quantitative ones (Roberts & Pashler, 2000) to converge on models that are powerful, parsimonious, and in register with the complexity of evolved processes such as learning. Although we strive for a unified theory of behavior, the best way to achieve it may be by perfecting modules that can account for their domain, while exchanging information with modules of other domains (Guilhardi, Yi, & Church, 2007).

In their penetrating assessment of the R-W model, Miller et al. (1995) noted 18 theoretical successes and about as many failures. They went on to observe that newer models are "highly complex or have their own list of failures at least as extensive as the R-W model" (p. 381) but that each of the new models has its strengths in fixing some of the failures of the R-W model. It is our hope that by embedding contemporary models in the present framework, which permits variance due to momentum to be partitioned out and permits ad libitum degrees of freedom in the data to counterpoise those required for modern complex models of conditioning (see, e.g., Hall, 2002, for an overview), that the models themselves may compete on a higher playing field. Dynamic analysis may also permit the refinement of experiments and permit reduction of the number of subjects required to answer behavioral or pharmacological questions (Corrado, Sugrue, Seung, & Newsome, 2005; Smith et al., 2004). The MPS model is but a second step through the door opened by Bush and Mosteller (1951) so many years ago.

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Appendix

Mathematical Details

Framing the Model

There are eight explicit parameters in the Momentum/Pavlovian/Skinnerian (MPS) model: the response parameters α and c, the two momentum parameters, the two Pavlovian parameters, and the Skinnerian parameters. (The Skinnerian parameters are partially redundant with the persistence parameter, but no attempt was made to enforce further parsimony in these already overworked data.) There are also implicit parameters. These involve the structure of the model and how that interacts with the parameters and data (Myung & Pitt, 1997). The directions of conditioning (the nominal signs of the learning parameters specifying their asymptotes θ, here fixed at 0 or 1) are such considerations. Another is the starting value of s, s_0, which is estimated as the average probability of a response over the first dozen trials of each condition, with those trials then excluded from all indices of merit. Because the logarithmic transformation penalizes errors exponentially as they approach maximum (e.g., predicting a response probability close to zero and having a response occur), a floor (of probability of data given the model) of 0.00001 was placed under both the candidate and the default models; it was rare for them to step on that floor except during the iterative process of parameter estimation. All analyses were conducted in Excel using the Solver add-in.

Looking Ahead

In general, the linear learning model was unquestionably better than the base model of momentum—the Akaike information criterion index of merit advantage for the learning model was typically close to 100 units. What does this mean in terms of ability to predict behavior? Most readers unfamiliar with the Akaike information criterion and log-likelihood analysis will appreciate some other indices of merit, such as variance accounted for by the model. However, we are predicting response rates on a trial-by-trial basis, not the typical averages over the last 10 sessions, each consisting of scores of observations. There is no opportunity to average out noise in the present dynamical analysis. In light of this, predictions were not so bad: The MPS model accounted for more than 10% of the variance in response rates on the next trial in Experiment 1, even though the analysis did not optimize goodness of fit for this variable. In the $p = .05$ condition, accuracy increased to 16%.

Whereas these might not seem impressive figures, nor might the advantage of the MPS model seem impressive in that metric, most considerations of variance accounted for—coefficients of determination—are calculated on average data, where noise has been minimized by averaging. None, to our knowledge, reflect accuracy on a moment-to-moment, or at least trial-by-trial, basis. Because conditions are always changing as a function of the behavior of the pigeon in this closed-loop system, there is no obvious larger unit over which we could aggregate data to improve accuracy. But there is a less-than-obvious one, described next.

The conditioning predicated by the learning model has a longer provenance than over just the next trial; a measure of accuracy that is both more informative, and more consistent with traditional reports of coefficients of determination, can be derived by asking how well the imputed strength, s_i, predicts behavior over the next few trials. Because the learning model posits geometric changes in performance as a function of contingencies, accuracy of prediction should also decrease geometrically with distance into the future. Accuracy decreases because the stochastic processes that might or might not carry the pigeon over a response threshold on a trial throw a multiplicative shadow into the future. Therefore, accuracy should decrease approximately as $(1 - \gamma)^n$, as vicissitudes of responding and reinforcement carry the conditioning process along an increasingly random walk. We may take advantage of this by averaging measured responding over the next score trials, giving the greatest weight to the next trial, less to the trial after that, and so on, and using events on the current trial to project those temporally discounted future response rates. This was accomplished by weighting the accuracy of prediction on the next trial by 20%; adding to that accuracy on the trial after that weighted by 16%; on the trial after that by $.2(.8)^2$, then by $.2(.8)^3$, and so forth. This "forward" exponentially weighted moving average places half the predictive weight on the three trials subsequent to the prediction, trailing off geometrically into the future. Accuracy at predicting this discounted future in the $p = .05$ condition doubled, to 28% of the variance in response rates accounted for by the MPS model. A similar doubling of the coefficient of determination was seen in spot checks of the other conditions.

Asymptotic Responding

In this article, the conditioning process is characterized from trial to trial by a difference equation—the strength on the prior trial plus the probability of a response time by π_P and a nonresponse by π_Q; that is then adjusted by the probability of food times π_F and no food times π_E and finally by the probability of both a response and food times π_{PF} or of a response and no food times π_{PE}. This permits continuous idealizations of acquisition and extinction.

Representation of a stochastic process by its probabilities gives a domesticated version of an intrinsically wild process. For instance, performance is vulnerable to a "gambler's ruin"—a series of nonreinforced trials that leads to extinction. The probabilistic solutions do not take this sudden death into account and do not allow for the recuperative strength provided by spontaneous recovery at the start of new sessions. Nonetheless, they provide some insights into the process. We begin by analysis of the momentum factor and then blend it with the conditioning factors. Here the direction toward ceiling or floor is assumed, in the conventional manner. Conditioning enters after momentum, rather than before, as in the analysis programs. The order of entry makes some difference in accuracy of fit.

Momentum

Letting $p(P_i)$ represent the probability of responding on the *i*th trial, the momentum of responding is carried forward from the last trial as

$$s_i' = s_i + p(P_i)\pi_P(1 - s_i) + [1 - p(P_i)]\pi_Q(0 - s_i),$$

that is, as the strength coming out of the prior trial, s_i, plus probability of a response, $p(P_i)$, times π_P (the momentum-of-pecking rate parameter), times the distance to the ceiling strength $(1 - s_i)$, plus the probability of not pecking times the momentum-in-quiescence parameter π_Q times the distance to the floor of strength. The probability of pecking is approximately equal to the strength, s_i, so substitute for $p(P_i)$ and simplify to

$$s_i' = s_i[1 + (1 - s_i)\pi_P - \pi_Q]. \tag{A1}$$

The difference in momentum parameters is gated by the distance of strength to its ceiling, to increment response probability. This is slightly off because there is a finite probability of being in the response state and not pecking, but that is negligible. The variable s_i' is the momentum of responding that is carried forward to the next trial.

Pavlovian Conditioning

Food occurs with probability p, so

$$s_i'' = s_i' + p\pi_F(1 - s_i') + (1 - p)\pi_E(0 - s_i');$$

that is, as the status quo ante (s_i'), plus the probability of food times the Pavlovian parameter π_F times the distance to ceiling, plus the probability of no food times the Pavlovian extinction parameter π_E times the distance to floor. Collecting terms yields

$$s_i'' = s_i' + [1 + p(\pi_E - \pi_F) - \pi_E] + p\pi_F. \tag{A2}$$

When the probability of food is $p = 1$, the influence of π_E drops out, leaving strength on a march toward 1; conversely, where $p = 0$, strength decreases geometrically from one trial to the next by the factor $(1 - \pi_E)$.

To predict responding on the next trial, the intervening variable s_i' is removed by substituting from Equation A2 into Equation A1. Because the parameters π_P and π_Q always enter as a difference, some parsimony is achieved by writing $\pi_P - \pi_Q$ as π_{P-Q}. Then Equations A1 and A2 give

$$s_i'' = s_i'[1 + \pi_{P-Q}(1 - s_i)][1 + p(\pi_E - \pi_F) - \pi_E] + p\pi_F. \tag{A3}$$

Skinnerian Conditioning

Remembering that food occurs with probability p and a peck with probability s_i'' the increment to strength conferred by operant conditioning is

$$s_{i+1} = s_i'' + s_i''[p\pi_{PF}(1 - s'') + (1 - p)\pi_{PE}(1 - s'')(0 - s'')],$$

strength after Pavlovian updating, plus the probability of a response times the large parenthetical. Inside the parenthetical is the probability of food times its rate parameter times the distance to the ceiling, plus the probability of no food times its rate parameter times the probability of no peck times its distance to the floor. This may be simplified to

(Appendix continues)

$$s_{i+1} = s_i^n\{1 + (1 - s_i^n)[p\pi_{PF} - (1 - p)s_i^n\pi_{PE}]\}. \tag{A4}$$

Inserting Equation A3 into this gives the final equation of prediction, too unenlightening to be written out here—even though it is less complicated than the full solution to the R-W model (Yamaguchi, 2006). It is simpler to evaluate Equation A3 and then insert it into Equation A4. A spreadsheet for analyzing data with the present theory on a trial-by-trial basis is available from Peter R. Killeen, Federico Sanabria, and Igor Dolgov.

Special Cases

Acquisition

In the case of acquisition, where few or no responses have yet occurred, Equation A3 provides a good equation of prediction. Because the probability of food p is typically 1.0, it can be further simplified to the acquisition function

$$s_{i+1} = s_i[1 + \pi_{P-Q}(1 - s_i)]\,(1 - \pi_F) + \pi_F. \tag{A5}$$

Equation A5 can range from a classic exponential-integral learning curve (when π_{P-Q} is of small magnitude) through approximately linear to an S-shaped ogive, depending on the two parameters, the net rate of persistence (π_{P-Q}) and the rate of acquisition (π_F).

Extinction

In extinction, $p = 0$, and Equation A3 simplifies to

$$s_{i+1} = s_i(1 - \pi_E)[1 + \pi_{P-Q}(1 - s_i)], \tag{A6}$$

which appears as Equation 8 in the text. Equation A6 assumes that π_{PE} is smaller and that extinction decrements can be handled by π_E; this has been the case for all of the data analyzed here, and setting π_E to zero is a parsimonious way to simplify the equation.

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New Editors Appointed, 2011–2016

The Publications and Communications Board of the American Psychological Association announces the appointment of 3 new editors for 6-year terms beginning in 2011. As of January 1, 2010, manuscripts should be directed as follows:

- *Developmental Psychology* (http://www.apa.org/journals/dev), **Jacquelynne S. Eccles, PhD,** Department of Psychology, University of Michigan, Ann Arbor, MI 48109
- *Journal of Consulting and Clinical Psychology* (http://www.apa.org/journals/ccp), **Arthur M. Nezu, PhD,** Department of Psychology, Drexel University, Philadelphia, PA 19102
- *Psychological Review* (http://www.apa.org/journals/rev), **John R. Anderson, PhD,** Department of Psychology, Carnegie Mellon University, Pittsburgh, PA 15213

Electronic manuscript submission: As of January 1, 2010, manuscripts should be submitted electronically to the new editors via the journal's Manuscript Submission Portal (see the website listed above with each journal title).

Manuscript submission patterns make the precise date of completion of the 2010 volumes uncertain. Current editors, Cynthia García Coll, PhD, Annette M. La Greca, PhD, and Keith Rayner, PhD, will receive and consider new manuscripts through December 31, 2009. Should 2010 volumes be completed before that date, manuscripts will be redirected to the new editors for consideration in 2011 volumes.

Operant conditioning.

Operant Conditioning

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Abstract: Operant conditioning, a term coined by B. F. Skinner, American psychologist and radical behaviorist, is the idea that behavior is the learned result of consequences. Skinner, who introduced the concept in his 1938 book The Behavior of Organisms: An Experimental Analysis, theorized that operant conditioning in the form of reinforcements and punishments leads to an association between a behavior and its consequence. Positive reinforcement increases a desirable behavior by following it with a favorable stimulus. Negative reinforcement increases a desirable behavior by removing an unfavorable stimulus after the behavior is performed. Both positive and negative reinforcement seek to increase a desirable behavior. Punishment, like reinforcement, also has positive and negative varieties. Positive punishment is adding an unfavorable stimulus in an effort to eradicate an undesirable behavior. Negative punishment is removing an unpleasant stimulus in order to decrease undesirable behavior. Both positive and negative punishment seek to decrease an undesirable behavior.

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Operant conditioning

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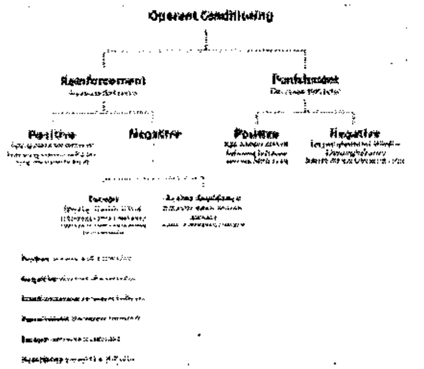
Operant conditioning, a term coined by B. F. Skinner, American psychologist and radical behaviorist, is the idea that behavior is the learned result of consequences. Skinner, who introduced the concept in his 1938 book *The Behavior of Organisms: An Experimental Analysis,* theorized that operant conditioning in the form of reinforcements and punishments leads to an association between a behavior and its consequence. Positive reinforcement increases a desirable behavior by following it with a favorable stimulus. Negative reinforcement increases a desirable behavior by removing an unfavorable stimulus after the behavior is performed. Both positive and negative reinforcement seek to increase a desirable behavior. Punishment, like reinforcement, also has positive and negative varieties. Positive punishment is adding an unfavorable stimulus in an effort to eradicate an undesirable behavior. Negative punishment is removing an unpleasant stimulus in order to decrease undesirable behavior. Both positive and negative punishment seek to decrease an undesirable behavior.



The hierarchy of operant conditioning. By Studentne [GFDL (http://www.gnu.org/copyleft/fdl.htn or CC-BY-SA-3.0 (http://creativecommons.org/licens sa/3.0)], via Wikimedia Commons

Overview

Skinner designed an operant conditioning chamber, which came to be known as the Skinner box, to test his theory of operant conditioning on animals. The Skinner box prevented human interruption of the experimental session and allowed the experimenter to study the behavior of an animal as a continuous process. The box includes at least one lever or key that the animal can manipulate to release food, water, or some other reward or to avoid punishment such as an electric shock. Skinner's experiments with rats and pigeons showed that the animals first hit the lever and released food accidentally; after a few accidental releases, the reinforcement of manipulating the lever ensured that the behavior would be repeated. Skinner believed that operant conditioning could be used in similar ways with human beings.

Modifying behavior through operant conditioning has been used in the treatment of phobias, obsessive-compulsive disorders, substance-abuse problems, and some sexual disorders, but the impact of Skinner's theories about operant conditioning has proved to be immense, reaching far beyond the field of psychology. Zoos and other animal facilities routinely use food as a positive reinforcement to train animals to move within enclosed areas and to increase safety during veterinary examinations. With human subjects, operant conditioning has been used to control absenteeism in the workplace (such as when employers offer staff members with no absences a chance to win cash rewards), to increase sales (coupons), and to manage agitation in older adults with dementia. Perhaps no field has been more influenced by operant conditioning than education. Skinner's assertion that positive reinforcement is more effective than punishment at changing and establishing desirable behavior led to the discrediting of punitive punishment in schools and the common application of timeouts (negative reinforcement) and a token economy (i.e., rewarding good behavior with gold stars that can be accumulated for prizes) instead.

Critics of operant conditioning have been vehement in pointing out its detriments. As early as 1959, American linguist and cognitive scientist Noam Chomsky argued that what worked in Skinner's laboratory could be applied to complex human behavior only in a superficial way. In 1960 progressive educator A. S. Neil insisted that rewarding good behavior taught that the behavior was not worth doing for reasons other than the reward. Other critics were even more severe, charging that operant conditioning was dangerous and inhumane. Gradually, the influence of Skinner's ideas declined, and by the twenty-first century, some declared that operant conditioning had become peripheral in psychology and related fields. However, in 2002 a list of ninety-nine top psychologists was published in the *Review of General Psychology* and B. F. Skinner topped the list.

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Operant Conditioning

Bridging the positions of Rogers and Skinner: The role of nonlinear dynamic systems

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This paper aims to bridge the gap between the positions of Carl Rogers and B. F. Skinner by demonstrating that they are not as antithetical as they are ordinarily assumed to be, and as the two men themselves tended to think. The author uses the concept of nonlinear dynamic systems as the principal concept in her effort to bridge the gap between the work of Rogers and Skinner and consequently introduces the concept of nonpredictivity to replace the concept of nondirectivity as being a more correct and basic characterization of classical client-centered therapy. The concept of nonpredictivity allows client-centered therapy to be framed, in an overarching and general way, by B. F. Skinner's principle of operant conditioning without doing violence to Rogers' concern about client autonomy. More specifically, the paper argues that Rogers' theory of therapy is not antithetical to Skinner's theory of personality and that client-centered therapy therefore can be practiced seamlessly by therapists who, like the author, believe that operant conditioning is a better explanatory concept for human behavior than the actualizing tendency.

Keywords: Rogers and Skinner; nonlinear dynamic systems; nonpredictive; nondirective

Brückenschlag zwischen den Positionen von Rogers und Skinner: die Rolle nicht-linearer dynamischer Systeme

Dieser Artikel will die Kluft zwischen den Positionen von Rogers und Skinner schliessen. Diese sind nicht so anti-thetisch, wie man normalerweise annimmt und wie die beiden selbst meinten. Ein Konzept nicht-linearer dynamischer Systeme soll die Kluft zwischen Rogers' und Skinners' Werk schliessen. Das Konzept der Nicht-Vorhersagbarkeit soll das Konzept der Nicht-Direktivität ersetzen; ersteres ist eine korrektere und grundlegendere Charakterisierung der klassischen Klientzentrierten Therapie. Das Konzept der Nicht-Vorhersagbarkeit ermöglicht es, Klientzentrierte Therapie in umfassender und genereller Weise in Skinners Prinzip des Operanten Konditionierens einzubetten, ohne dass man Rogers' Sorge um die Autonomie der Klienten Gewalt antut. Rogers' Therapietheorie ist keine Antithese zu Skinners Theorie der Persönlichkeit und Klientzentrierte Therapie kann daher nahtlos von Therapeuten praktiziert werden, die wie die Autorin überzeugt sind, dass Operantes Konditionieren ein besseres Erklärungskonzept für menschliches Verhalten ist als die Aktualisierungstendenz.

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Estableciendo un puente entre las posturas de Rogers y Skinner: El rol de los sistemas dinámicos ni lineales

Este escrito tiene como fin establecer un puente entre las posiciones de Carl Rogers y B. F. Skinner al demostrar que no son tan antagónicas como normalmente se las considera, y como ambos autores tendían a considerarlas. La autora utiliza el concepto de sistemas dinámicos no lineares como el concepto principal en su esfuerzo para salvar la distancia entre la obra de Rogers y la de Skinner, y por ello introduce el concepto de no predictibilidad para reemplazar el concepto de no directividad, considerándolo una caracterización más exacta del la terapia centrada en el cliente clásica. El concepto de no predictibilidad permite que ubiquemos la terapia centrada en el cliente en un marco más abarcativo y general, por el principio de Skinner de condicionamiento operativo sin violentar la preocupación de Rogers acerca de la autonomía del cliente. Más específicamente, este escrito argumenta que la teoría de Rogers acerca de la terapia no es contraria a la teoría de la personalidad de Skinner y que por lo tanto la terapia centrada en el cliente puede ser aplicada por terapeutas, como la autora, creen que el condicionamiento operante es un concepto que explica el comportamiento humano mejor que la tendencia actualizante.

L'élaboration d'un pont entre les positions de Rogers et de Skinner: Le rôle de systèmes non-linéaires et dynamiques

Cet article vise à créer un pont entre les positions de Carl Rogers et B.F. Skinner en démontrant qu'elles ne sont pas aussi antithétiques qu'on le suppose d'ordinaire ou que les deux hommes avaient tendance à les penser. L'auteure utilise le concept de systèmes non-linéaires et dynamiques comme concept principal dans son effort de créer un pont entre le travail de Rogers et de Skinner. Il s'ensuit qu'elle propose le concept de non-prédictibilité pour remplacer le concept de non-directivité en tant que caractérisation plus juste et plus fondamentale de la thérapie centrée sur le client classique. Le concept de non-predictibilité permet à la thérapie centrée sur le client d'être encadrée, d'une manière globale et générale, par le principe de conditionnement opérant de Skinner sans faire violence à la préoccupation de Rogers concernant l'autonomie du client. Plus spécifiquement, l'article soutient que la théorie de la thérapie de Rogers n'est pas antithétique par rapport à la théorie de la personnalité de Skinner et que la thérapie centrée sur le client peut donc être pratiquée sans heurt par des thérapeutes qui, comme l'auteure, croient que le conditionnement opérant est un concept qui explique mieux le comportement humain que la tendance actualisante.

Fazendo a ponte entre as posições de Rogers e de Skinner: O papel dos sistemas dinâmicos não lineares

Este artigo pretende estabelecer uma ponte entre as posições de Carl Rogers e de B.F. Skinner, ao demonstrar que os dois autores não são antitéticos como habitualmente se julga e como eles próprios tendiam a ver-se um ao outro. A autora recorre ao conceito de sistemas dinâmicos não lineares como sendo central no estabelecimento dessa ponte entre os trabalhos de Rogers e de Skinner e, consequentemente, introduz o conceito de não previsibilidade para substituir a não directividade, considerando o primeiro como permitindo uma caracterização mais correcta e básica da terapia centrada no cliente clássica. O conceito de não previsibilidade permite que se enquadre a terapia centrada no cliente, de forma mais abrangente e geral, através do princípio skinneriano de condicionamento operante, sem violentar a preocupação de Rogers com a autonomia do cliente. Mais concretamente, este artigo pretende que a teoria da terapia de Rogers não é antagónica com a teoria da personalidade de Skinner e que, consequentemente, a terapia centrada no cliente pode ser colocada em prática, sem problemas, por terapeutas que, como o autor, acreditam que o condicionamento operante é um conceito mais adequado para o comportamento humano do que a tendência actualizante.

ロジャーズとスキナーの立場の橋渡し：非線形力動的システムの役割について

本論文の目的は、カール・ロジャーズとB.F.スキナーの立場が「対立的」ではないことを示し、彼らの間にある相違を埋めることである。ロジャーズとスキナーの業績の間にある相違を埋めるための重要な概念として、非線形力動的システムを用いた。古典的な来談者中心療法におけるより適切で基本的な特徴として、“非指示性”の代わりに“非予測性”を提案した。そして、B.F.スキナーのオペラント条件付けの原理による“非予測性”の概念により、来談者中心療法は、クライエントの自主性に関するロジャーズの懸念を害することなく、より包括的で普遍的に形作られるとし、ロジャーズのセラピーに関する理論がスキナーのパーソナリティ理論と正反対のものではないことを示した。そして、実現傾向よりもオペラント条件付けが人間の行動の説明概念として優れていると信じている治療者であったとしても、来談者中心療が全ての学派にわけ隔てなく実践されうると論じた。

En brobygger mellem Rogers og Skinner: De ikke-lineære dynamiske systemers rolle

Denne artiikel har til formål at bygge bro mellem Carl Rogers' og B. F. Skinners positioner ved at demonstrere at de ikke er så anti-tetiske som almindeligt antaget og som de to mænd selv anså dem for at være. Forfatteren bruger begrebet "ikke-lineære dynamiske systemer" som sit vigtigste begreb i forsøget på at bygge bro mellem Rogers' og Skinners teorier, og som en konsekvens heraf introducerer hun begrebet "ikke-forudsigende" til erstatning for begrebet "ikke-direktiv," idet hun anser førstnævnte for at være en mere basal og korrekt karakterisering af klassisk klient-centreret terapi. Begrebet "ikke-forudsigende" tillader klient-centreret terapi at blive kontekstualiseret, på overordnet og generel vis, af B. F. skinners princip om operant betingning uden at gøre vold på Rogers' omsorg for klientens autonomi. Mere specifikt argumenterer artiklen for at Rogers' teori om terapi ikke er antitetisk til Skinners personlighedsteori og for at klient-centreret terapi derfor kan praktiseres gnidningsfrit af terapeuter der, som forfatteren, tror at operant betingning er et bedre begreb til forklaring af menneskers adfærd end den aktualiserende tendens.

Introduction

Writing this paper has been close to the heart of the author who finds so much of value in Rogers' as well as in Skinner's works that it has become increasingly inconceivable to comprehend their views as being excluding of or in contradiction with each other. Rogers and Skinner were, after all, among the giants of 20th century psychology. It therefore seems most likely that much of value can be found in both men's work.

If Rogers and Skinner could have had a third dialogue, today, informed by late 20th century advances in work with nonlinear dynamic systems (see below), they might have ended up on the same page and there might not have been the split between the behaviorist orientation and the humanist orientation in psychology that we see today. They agreed (Rogers & Skinner, 1956, 1962/1989) that human behavior is determined, although Rogers had some reservations, because he found that Skinner attached too little importance to the inner, subjective experiences of the individual, particularly the experience of being free and having choices. Rogers did not clarify, though, what kind of importance should be attached to these inner, subjective experiences, apart from stating in various ways that they were important because they felt important and meaningful to the individual. He spoke of a "paradoxical" relationship between scientific determinism and inner, subjective experiences. He said (1962/1989, p. 132):

> I am in thorough agreement with Dr. Skinner that, viewed from the external, scientific, objective perspective, man is determined by genetic and cultural influences. I have also said that in an entirely different dimension, such things as freedom and choice are extremely real . . . I see it as being similar to the situation in physics where you can prove that the wave theory of light is supported by evidence, as is the corpuscular theory, though the two of them appear to be contradictory. They are not, at the present state of knowledge, reconcilable, but one would be narrowing his perception of physics to deny one or the other. It is in this same sense that I regard both of these dimensions as real, although they exist in paradoxical relationship. [Physicists, today, would not speak of a paradoxical relationship, but of a complementary relationship between the wave theory and the corpuscular theory of light. (This author's comment)]

However, talking about a paradoxical relationship between scientific determinism and the experience of freedom doesn't really explain anything and, significantly, Rogers did not state that he attached causative agency to inner experiences, or to the experience of being free to make choices, that is, to the experience of having a free will. On the contrary, he said (Rogers, 1962/1989, p. 99): "Behaviors that were formerly dealt with as though they were caused by little homunculi within the individual, or various internal causes, are now seen to have other types of causation."

Rogers' reservation to Skinner's views seems first and foremost to originate in Rogers' feeling of no less than revulsion (1962, p. 126) at the thought that human behavior could be controlled *ad modum* Walden Two (Skinner, 1948). Skinner took the position that as much as he acknowledged the existence of inner, subjective experiences, including the experience of choosing freely, he regarded them as more or less covert or private behaviors, and it was the environmental consequences of a person's behavior, not any kind of inner experiences, that should be studied, because those consequences were the causes (in the form of rewards or punishments) of the behavior. By engineering the environmental consequences of a person's behavior, it was, according to Skinner, possible to control this person's behavior and that prospect was as positive from Skinner's point of view as it was negative from Rogers' point of view.

Basically, however, they were in agreement about the assumption that determinism implies predictability. Today, they would have been informed about Chaos Theory and nonlinear dynamic systems, that is, they would have known that determinism does not necessarily imply predictability. In the light of this knowledge, they might have appreciated that their respective positions were, indeed, compatible. Skinner might have understood that in spite of his being right about the principle of operant conditioning and determinism for living organisms, in general, he could not hope to be able to control the rich and complex behavior of any human individual, in particular. A Walden Two could never be realized. Rogers, on his side, might have understood that his agreeing with Skinner about determinism did not exclude the soundness and relevance of his nonpredictive therapeutic approach with any individual client.

The impossibility of predictive control of complex sets of behavior

A basic assumption

It is a basic assumption of this paper that the trajectory of the process of the individual human organism's interaction with its environment from conception to death is best modeled by a *nonlinear dynamic system* unlike, for example, the trajectory of the planets around the sun, which is, as physics has long since demonstrated, best modeled by a linear dynamic system.

Nonlinear dynamic systems[1]

Classical Newtonian science is almost exclusively concerned with linear dynamic systems. In such systems, cause–effect relationships can be clearly and exhaustively defined. This allows for very precise predictions of the behavior of a linear dynamic system. With measurements of a few parameters, the trajectory of a planet, for example, can be very precisely predicted. This is so even if no set of measurements can be 100% precise. Physicists and mathematicians say about linear dynamic systems that small differences in initial conditions remain small differences in the future behavior of the system. For all practical purposes, therefore, small differences in initial conditions can be disregarded. Stated otherwise, one can make one's measurements as precise as one wishes one's predictions to be precise. This is the essentially characterizing feature of linear dynamic systems.

Such is not the case with nonlinear dynamic systems. They are said to be sensitively dependent on initial conditions. This means that small differences in initial conditions can develop into big differences in the future behavior of the systems. *The butterfly effect* is the ubiquitously used term for this sensitive dependence on initial conditions. To illustrate what nonlinear dynamic systems are about, it is said that a butterfly flapping its wings in Beijing on Sunday can be decisive for the weather in New York next Saturday. This means that the development of the weather around the globe is a nonlinear dynamic system, which is sensitively dependent on initial conditions, and it is the reason meteorologists cannot predict the weather with better than chance precision for much more than four or five days ahead.[2]

To the surprise of most people, mathematics, that is, the area that is normally thought of as the most predictable of all, is full of nonlinear dynamic systems. Many mathematical equations show sensitive dependence on initial conditions. If one iterates (develops) such equations on a calculator that delivers results with nine decimal points' precision and on a calculator that delivers results with eight decimal points' precision one will find that the iterations on the two calculators sooner or later start to vary widely and seemingly chaotically, as a result of the tiny, one millionth part, difference in initial conditions. *Chaos Theory* is the field of mathematics that is concerned with nonlinear dynamic systems (see, e.g., Gleick, 1987; Lorenz, 1993; Peitgen, Jürgens, & Saupe, 1992). The name is somewhat unfortunate, since there is really nothing "chaotic" about these systems. They are fully determined systems. The meaning of the term *chaotic* is, more precisely, *unpredictable*. Nonlinear dynamic systems are as determined as linear dynamic systems, but they are as unpredictable as linear dynamic systems are predictable.

Here is another example of a nonlinear dynamic system:

Imagine that you let go of a small bullet at the edge of a large funnel with a hole in the bottom, just a tiny bit larger in diameter than the bullet. You can, of course, on the basis of the law of gravity, predict that it will end up disappearing down the hole in the bottom, but there is no way you can predict its trajectory toward that disappearance. That trajectory is sensitively dependent on the tiniest differences in the way you hold the bullet towards the edge of the funnel at the start, on the tiniest differences in where you hold it, on the tiniest differences in the way you let go of it, and on the tiniest details of irregularities on the side of the funnel. Any of these tiny differences can have a huge effect on the bullet's trajectory. At the same time, these differences are so tiny that there is no way to measure them, just as there is no way to measure their combined differential influence on the bullet's behavior in a way that is sufficiently precise to make it possible to predict the trajectory of the bullet towards its disappearance. One can only make the prediction (obvious to everyone) that the bullet's overall trajectory will be downwards, that it will probably not disappear right away, but instead hit the edge of the hole in the bottom, and be spiraled up again, one time or several times, but still closer to the bottom and still more slowly as it loses energy, until it finally disappears.

At this point, the reader is welcome to associate to the human organism's trajectory through life, since my point with this rather detailed explanation of nonlinear dynamic systems is, as already stated, to postulate that the behavior of the human organism, that is, its continuously changing complex sets of interactions with a continuously changing environment, from conception to death, is a nonlinear dynamic system, and, probably, *the* nonlinear dynamic system, par excellence. The human organism in its environment is, more than anything, like the bullet in the funnel that was described above: Its trajectory through life is as sensitively dependent on initial conditions as the bullet's trajectory on the side of the funnel.

Determined and unpredictable

The consequence of this assumption is that the interactional processes of human beings with their environment are determined *and* unpredictable. It is very important to grasp the idea that something can be both determined and unpredictable. It is important, because our language and thinking is so suffused with the linearity of classical science that we automatically tend to think of determined systems as being synonymous with predictable systems. It is therefore no wonder that Rogers and Skinner did not question the basic, mistaken, assumption that they had in common: that determinism invariably implies predictability. It was only with the advent of the modern computer that mathematicians and physicists could investigate nonlinear dynamic systems thoroughly, and knowledge about nonlinear dynamic systems did not become common knowledge until the two last decades of the past century. At the time of the dialogues between Rogers and Skinner, thinking in terms of predictable, linear dynamic systems was still the way of thinking that dominated the sciences.

The individual level and the general level

Rogers and Skinner, though, were not altogether wrong about their assumption that determinism implies predictability. They were only wrong on the level of the individual human organism's interaction with the environment, and they were not very careful about distinguishing between the individual and the general level in their dialogues. Their failure to do so may have contributed to their thinking of themselves as being in disagreement. On the general level, that is, on the level of people, at large, or of sufficiently large groups of people, or on the level of "the average," they were right. And it may be this level Rogers had in mind when he spoke of the "scientific perspective" in the quotation above. On this level Skinner's laws of conditioning can be applied to predict which environments will produce a certain kind of behavior among the majority of people exposed to this environment. And on this level Rogers and Skinner were actually in rather close agreement about the general kind of environment that would produce the most constructive behaviors in the majority of people or, vice versa, they were in agreement about the kind of environment that hampered the actualization of these behaviors, that is, they were in agreement about a general theory of disturbed psychological development or, in Skinner's terminology, disturbed behavior. For Rogers, psychological disturbance came about as a result of the individual having been excessively exposed to conditional regard. For Skinner, disturbed behavior came about as a result of the individual having been excessively exposed to punishing consequences.

Skinner's theory operates with four reinforcers, two positive (increasing the likelihood of recurrence of a certain set of behaviors) and two negative (decreasing the likelihood of recurrence of a certain set of behaviors). The two positive reinforcers are (1) finding rewarding consequences, and (2) escaping from punishing consequences; and the two negative reinforcers are (3) finding punishing consequences, and (4) finding that rewarding consequences have disappeared (O'Donohue & Ferguson, 2001, p. 92).

For Rogers conditional positive regard is actually implicitly punishing of behaviors that are not regarded positively: "I love you when you . . .," implies that "I do not love you when . . . " and withdrawal of love can be as painful to humans as electric shock to rats.

Thus, it is hard to see any essential difference between "conditional regard" and "punishing consequences."

Rogers, however, employed the concept "unconditional positive regard" that has no counterpart in Skinner's work, and this concept of Rogers', although he didn't explicitly say so anywhere, embraces the unpredictability of human behavior that went beyond Skinner's theory. (See further, below.) Rogers, more than Skinner, had a "sense" of nonlinear dynamics, before knowledge of these dynamics became accessible.

In any case, they were in agreement about the essential point: that disturbed psychological development or disturbed behavior is the result of unfortunate environments, that is, it is conditioned and thus controlled by the environment, even if it is impossible to tell, on the individual level, what the specific conditioning contingencies or reinforcement relationships of any given set of disturbed behaviors might have been and may be. The logical conclusion is that they were also in agreement that any behavior, not only disturbed behavior, is controlled by the environment. The evidence is Rogers' statement, in the debate with Skinner, that "I am in thorough agreement with Dr. Skinner that, viewed from the external, scientific, objective perspective, man is determined by genetic and cultural influences" (1962/1989, p. 132; see the full quotation above). In another context Rogers wrote:

> The question is often raised: But what about the therapist's attitude toward his client's asocial or antisocial behavior? Is he to accept this without evaluation? Sometimes this question is answered by saying that the effective therapist prizes the person, but not necessarily his behavior. Yet it is doubtful if this is an adequate or true answer. To be sure, the therapist may feel that a particular behavior is socially unacceptable or socially bad, something he could not approve of in himself, and a way of behaving which is inimical to the welfare of the social group. But the effective therapist may feel acceptant of this behavior in his client, not as desirable behavior, but as a *natural consequence* of the circumstances, experiences, and feelings of this client. Thus the therapist's acceptance may be based upon this kind of feeling: "If I had had the same background, the same circumstances, the same experiences, it would be inevitable in me, as it is in this client, that I would act in this fashion." (Rogers, Gendlin, Keisler, & Truax, 1967, p. 103)

Here Rogers is fully on the same wavelength as Skinner.

Rogers' dilemma

As we have seen, Rogers, however, had problems with this and he also often spoke about the self-governing, internally controlled individual. For example, in his definition of the actualizing tendency (Rogers, 1959, p. 196) he spoke of development "toward autonomy and away from heteronomy, or control by external forces." It seems to be this apparent contradiction in Rogers' view that he tried to resolve with reference to the "paradoxical" relationship between the individual experience of freedom and the notion that human behavior is determined.

Rogers also expressed his efforts to integrate the idea of individual freedom on the one hand and determinism on the other in the following quote:

> The fully functioning person ... not only experiences, but utilizes, the most absolute freedom when he spontaneously, freely, and voluntarily chooses and wills that which is also absolutely determined. (Rogers, 1961, p. 193)

It should be evident from these quotations that Rogers struggled hard with the free will/determinism issue without resolving it. The author wonders why Rogers seemingly did not pay attention to the everyday occurrence that subjective experience and objective knowledge can be held simultaneously in mind: We can enjoy the beautiful sunset while knowing full well that the sun is not at all "setting"; instead the earth is revolving around it. Likewise, we can hold in mind, simultaneously, the enjoyment of freedom while knowing full well that the experience of freedom is a subjective experience that is as determined as everything else and certainly not an experience enjoyed by all.

Skinner's mistake

It is undoubtedly the impossibility of making sufficiently precise predictions about the individual conditioning contingencies of a given client's behavior that is at the heart of the problems of behavior analytic therapy, that is, the therapeutic application of Skinner's laws of conditioning on the level of individual clients. The engineering of specific reinforcement strategies to change a given "target behavior" demands isolation of the target behavior and prediction about the reinforcement strategies to be applied to change the target behavior, and this is, more often than not, impossible with the complex kind of behaviors and problems ordinary clients present to ordinary practicing therapists. The rare client may present with sufficiently simple and well-delineated kinds of symptom/problem/target behavior for effective reinforcement strategies to be successfully predicted and applied. In these cases, it is possible to approach the client's "presenting problem" to a linear dynamic system and to work with it accordingly. To remain with the bullet in the funnel analogy, this would roughly correspond to a whole in the bottom that was sufficiently large to make it obvious that the bullet would disappear directly into it independently of any differential influences on its way to the bottom. As early as the middle of the last century numerous studies, referred to by Patterson (1963), showed that carefully chosen, well-delineated and obvious "bits" of behavior can be changed by predictive operant conditioning. Ordinarily, though, this kind of approximation to a linear dynamic system is not possible in therapy without disregarding essential features of the relatively complex problems that clients typically see therapists about. These problems are part of a unique nonlinear dynamic system that is as unpredictable as it is determined. With ordinary clients, the diameter of the hole in the bottom is not much larger than the diameter of the bullet, the side of the funnel is very irregular and there are an infinite number of variations in the ways different bullets were started on their unique trajectories toward the bottom.

Therefore, Chaos Theory and the basic unpredictability of nonlinear dynamic systems falsify Skinner's assumption of the predictability of the behavior of the individual human organism. Rogers' opposition to this point of Skinner's ideas seems to have been, primarily, ethically motivated, but later developments in mathematics and physics came to his support: Rogers' idea that a therapist should not try to control the behavior of any individual client gains momentum by the idea that the therapist, quite simply, *cannot* control the behavior of any given client.

A third dialogue

Thus, if Rogers and Skinner could have had a third dialogue, today, Rogers, in the light of the basic unpredictability of nonlinear dynamic systems, might not have been as uncomfortable about Skinner's deterministic position as he was in 1957 and 1962. Rogers might more unequivocally have agreed with Skinner that the human organism is determined and Skinner might have agreed that Rogers was right that the complex sets of interaction of a client with his or her environment should not be exposed to efforts of direction or control in therapy; not, primarily, because such efforts were ethically wrong, but because such efforts were, more likely than not, doomed to failure, since they would imply therapist capacity to make appropriate predictions about the client at odds with the trajectory of client changes being best modeled by a nonlinear dynamic system.

Classical client-centered therapy: Nonpredictive rather than nondirective

Classical client-centered therapy is regarded by most practitioners as being utterly nondirective (see, e.g., Bozarth, 1998, p. 57; Grant, 1990; Levitt & Brodley, 2005). It is the therapy of the "early Rogers" or "Rogers-1" (Frankel & Sommerbeck, 2005, 2007) that Rogers described in *Counseling and Psychotherapy* (1942), where he distinguished between "directive" approaches and his own "nondirective" approach (1942, pp. 115–128). He articulated the philosophical foundation of this therapy in more detail in *Client-Centered Therapy* (1951). Nevertheless, most would probably agree that classical-client centered therapists do direct, in the sense of influence, their clients, just as clients influence their therapists, and just as we all influence each other all the time. Skinner would say that control or conditioning is mutual; it goes both ways. Therefore, the essence of nondirectivity in classical client-centered therapy is, more precisely, that the therapist does not *systematically* try to influence or direct the client. The therapist has no preconceived, that is, *predictive* idea, based on diagnostic assessment, a particular theory of personality, or any other perspective of their own, of how and in which direction they wish to influence the client, and the therapist entertains no such ideas at any moment during the course of therapy. In short, the therapist makes no effort to predict what will be helpful to a given client, but engages instead in continuous empathic understanding of the client's perspective.

This does not mean that expert predictions do not exist. However, expert predictions are made on the basis of experts' knowledge of what characterizes some average of a group of people or some sufficiently large group. Such predictions can be made with satisfying precision when the average process and the actual process are virtually the same, which is the case with linear dynamic systems like, for example, the trajectory of the planets around the sun. But for nonlinear dynamic systems, where small differences in initial conditions can develop into very large differences at a later point in time, it is impossible to know how widely the known average and the actual system of interest at this precise moment in time differ from each other, except in very crude and banal ways. Psychologists and psychiatrists, for example, are experts at how a sufficiently large group of people with a certain diagnosis responds to this or that intervention, or how the "average" person with this diagnosis responds, but have no way of knowing how close any given client with the given diagnosis is to this average (Sommerbeck, 2004, pp. 295–297). Therefore:

> Any intervention with the process of another, which is made with the intention of being helpful, as will most often be the case in therapy, is made on the basis of a prediction that this intervention will actually, more likely than not, be helpful for the other. Otherwise, the intervention would not be made. In making such predictions, the other is treated as a linear dynamic system, as "an average person" or as "an average client" on the basis of theory, research, or the therapist's professional or personal experience. The other is not treated as this particular, unique, nonlinear dynamic system, John, who is different from any average, or as this other, different and equally unique, nonlinear dynamic system, Jenny, whose future interactions with her environment it is impossible to make predictions about. (Sommerbeck, 2004, p. 296)

To this author, Rogers' fourth condition of the six necessary and sufficient conditions for therapeutic change (Rogers, 1959, p. 213), unconditional positive regard for the client, is, first and foremost, respect for the uniqueness of each client, or respect for the client being a nonlinear dynamic system that it is impossible to make anything but banal and crude, that is, more often than not therapeutically irrelevant, predictions about. It is also respect for the importance of clients' subjective *experience* of freedom, while knowing that objectively the client is as determined as the therapist or anything/anyone else, and that the therapy, as a whole, is but another determining or conditioning influence, albeit a nonpredicted influence, in the life of the client and in the life of the therapist. The only prediction made by the classical client-centered therapist is that the client will, more likely than not, be better off at the conclusion of the therapy than at the start of it, but what this means, in any detail, is left blowing in the wind.

A personal note

This paper represents the way the author, for herself, has tried to resolve Rogers' "paradox" regarding freedom and determinism, and the way she can effortlessly and satisfyingly contain the wisdom of Rogers' client-centered therapy and the wisdom of Skinner's principle of operant conditioning at the same time. It is the way for the author to be dedicated to Rogers' theory of therapy, while being critical of Rogers' theory of personality, particularly the concept of the actualizing tendency (Frankel, Sommerbeck, & Rachlin, 2010) and instead embracing Skinner's behaviorist theory of personality.

Conclusion

Hopefully, this paper has demonstrated that if Rogers and Skinner could have had a third dialogue, today, informed by knowledge of nonlinear dynamic systems, they could have bridged the apparent gap between their respective positions. They could have agreed that, rather ironically, the very point they used to agree about, that determinism implies predictability, was the very point they were both wrong about. They would have known that most aspects of a human being's trajectory through life are a nonlinear dynamic system, determined and unpredictable, and in the light of this knowledge Rogers could have agreed unequivocally with Skinner about operant conditioning and determinism without fearing the consequences of his agreement for the relevance of his therapeutic approach. And in this same light, Skinner could have agreed about the wisdom of nonpredictive client-centered therapy for the ordinary complexity of client problems that demand unconditional respect for client uniqueness rather than respect for client freedom in some nonscientific "dimension."

A corollary of this conclusion is the dispensability of a belief in the existence of the actualizing tendency as the foundation for practicing client-centered therapy. A belief in, and respect for client uniqueness suffices.

Notes

1. This explanation of nonlinear dynamic systems is a slightly revised and shortened excerpt of Sommerbeck (2004). The author is grateful to the editors of the PCEP journal for their permission to insert this excerpt in the present article.
2. It would be more correct to say that *the trajectory of* the weather is *best modeled by* a nonlinear dynamic system, since the concept of a nonlinear dynamic system is a mathematical concept that consists of mathematical variables and their interrelationships, which the weather, of course, does not consist of. For easier readability, though, the author has chosen the less mathematically correct formulation that this or that is a nonlinear dynamic system rather than the more circumstantial, but also more correct, formulation that the trajectory of this or that is best modeled by a nonlinear dynamic system.

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ORIGINAL PAPER

Classical conditioning in borderline personality disorder: an fMRI study

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Classical
Conditioning

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Abstract Previous research suggests disturbed emotional learning and memory in borderline personality disorder (BPD). Studies investigating the neural correlates of aversive differential delay conditioning in BPD are currently lacking. We aimed to investigate acquisition, within-session extinction, between-session extinction recall, and reacquisition. We expected increased activation in the insula, amygdala, and anterior cingulate, and decreased prefrontal activation in BPD patients. During functional magnetic resonance imaging, 27 medication-free female BPD patients and 26 female healthy controls (HC) performed a differential delay aversive conditioning paradigm. An electric shock served as unconditioned stimulus, two neutral pictures as conditioned stimuli (CS+/CS−). Dependent variables were blood-oxygen-level-dependent response, skin conductance response (SCR), and subjective ratings (valence, arousal). No significant between-group differences in brain activation were found [all $p(\text{FDR}) > 0.05$]. Within-group comparisons for $\text{CS+}_{\text{unpaired}} > \text{CS}-$ revealed increased insula activity in BPD patients but not in HC during early acquisition; during late acquisition, both groups recruited fronto-parietal areas [$p(\text{FDR}) < 0.05$]. During extinction, BPD patients rated both CS+ and CS− as significantly more arousing and aversive than HC and activated the amygdala in response to CS+. In contrast, HC showed increased prefrontal activity in response to CS+ > CS during extinction. During extinction recall, there was a trend for stronger SCR to CS+ > CS in BPD patients. Amygdala habituation to $\text{CS+}_{\text{paired}}$ (CS+ in temporal contingency with the aversive event) during acquisition was found in HC but not in patients. Our findings suggest altered temporal response patterns in terms of increased vigilance already during early acquisition and delayed extinction processes in individuals with BPD.

Keywords Borderline personality disorder · Classical conditioning · Prefrontal cortex · Amygdala · Insula

Introduction

Conditioning is a basic associative learning process that plays an important role in the development and maintenance of psychiatric disorders as well as in behavior therapy, especially exposure therapy.

Numerous studies have applied experimental fear conditioning paradigms to examine the (neurobiological) mechanisms underlying associative emotional learning [1–31]. In differential delay conditioning paradigms, one of the two initially neutral stimuli (CS+) is temporally paired with an aversive unconditioned stimulus (US), while the other is not (CS−). Many experimental settings do not only

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involve *acquisition* (learning) and within-session extinction but also between-session extinction (*extinction recall*) and *reacquisition* or *reinstatement*, i.e., a renewed exposure to the original CS–US contingency.

There is convergent evidence from both animal research and human studies that the amygdala, insula, and medial prefrontal cortex (mPFC) including the anterior cingulate cortex (ACC) play central roles in the acquisition of fear responses [1, 4, 5, 9, 15]. Several studies in healthy humans further reported differential responses (to CS+ > CS−) in the inferior frontal gyrus, rostral, and caudal orbitofrontal cortex (OFC), thalamus, hippocampus, cerebellum, striatum (including putamen), as well as in sensory cortices [6, 10, 16, 18, 20, 22, 24–28]. The mPFC is assumed to play a central role in the integration of sensory and affective stimuli and in the coordination of memory storage both during the acquisition and the extinction of emotional (fear) memories [11, 14, 28, 31]. Extinction is thought to involve activity-dependent potentiation of synaptic transmission in the mPFC resulting in an inhibition of amygdala-dependent responses [11, 14]. Animals with lesions of the mPFC are unable to extinguish conditioned responses [4, 15]. In healthy humans, increased activation in the mPFC [20, 24, 26], OFC [17], amygdala (e.g., [7, 18, 20]), and insula [2, 17] could be observed during extinction (for a review see [28]). For the amygdala, a rapid habituation, i.e., decrease in reactivity to unpleasant stimuli was observed over the course of the acquisition phase [7, 8].

Within a 'brain network of emotion regulation,' the amygdala and insula have been critically implicated in the processing of negative emotions, while the ACC is thought to play a critical role in both the generation and regulation of emotions [9, 32]; prefrontal areas including the ventrolateral and dorsolateral PFC (vlPFC, dlPFC), OFC, and dorsomedial PFC are thought to be involved in the down-regulation of affective arousal [32].

In anxiety disorders, acquired fear responses to an initially neutral stimulus (e.g., place, object, person) previously paired with an US appears to be not successfully extinguished [33–35]. Patients with posttraumatic stress disorder (PTSD), for example, showed amygdala hyper-reactivity and increased skin conductance responses to the CS+ during extinction recall [34]. Deficits in associative emotional learning are assumed to underlie clinical features such as a persistent fear and heightened affective arousal, even in contexts where no actual threat is present.

To our knowledge, no study so far has investigated the neural correlates of aversive differential delay conditioning in patients with borderline personality disorder (BPD). BPD is a severe psychiatric disorder with high rates of trauma and pronounced difficulties in emotion regulation including affective hyperarousal even in normative neutral situations [36, 37]. Experimental research in patients with BPD suggests altered learning and memory in aversive emotional contexts [38, 39]. On the neural level, there is growing evidence for both structural and functional alterations in fronto-limbic brain regions which play a major role in affecting regulation and learning processes (e.g., amygdala, hippocampus, anterior cingulate) including amygdala hyperactivity and diminished recruitment of the prefrontal areas during emotional challenge (for reviews see [40, 41]).

In a previous psychophysiological study [42], we investigated skin conductance responses (SCR) as well as arousal and valence ratings during an aversive differential delay conditioning paradigm in patients with BPD and healthy controls (HC). We additionally assessed levels of peri-experimental dissociation, i.e., disruptions of usually integrated functions of consciousness, memory, body awareness, and perception. In this previous study, BPD patients with low levels of dissociation showed regular conditioning responses in terms of a differential SCR to CS+ > CS− similar to HC. Of note, patients with high state dissociation failed to show regular conditioning, which suggests that dissociation may modulate emotional learning in BPD.

Recently, Kamphausen and colleagues [43] assessed SCR and brain activity during an instructed fear task in patients with BPD and HC during functional magnetic resonance imaging (fMRI). Before participants were brought to the MR scanner, they were informed that they would see different stimuli either indicating a safe situation or potential threat (electrodermal stimulation). The electrodermal stimulation was in fact only applied during this instruction but not during the experimental procedure. During fMRI, both healthy participants and BPD patients showed differential fear responses in terms of stronger SCR to stimuli indicating potential threat compared to stimuli indicating the save situation. However, only HC but not BPD patients showed amygdala habituation and an increase in activity in the mPFC during instructed fear. Findings of this fMRI study suggest that altered activity in a fronto-limbic brain circuitry might underlie disturbed emotional learning during an instructed fear task in patients with BPD.

The aim of the present study was to investigate the neural correlates of emotional learning in BPD patients applying an aversive differential (danger versus safety signal) delay conditioning paradigm during fMRI. Between-session extinction and re-acquisition were assessed after 72 h. Based on previous neuroimaging studies, we expected stronger activation in the amygdala and insula during acquisition and reacquisition and less activation in the mPFC during within-session and between-session extinction. We were further interested in habituation effects, i.e., decreases in activity over the course of each conditioning phase. Based on previous research [7, 8, 43], we expected a habituation of the amygdala during the acquisition phase.

Methods and materials

Participants

Thirty-four female individuals fulfilling DSM-IV criteria for BPD as assessed by the international personality disorder examination (IPDE) [44] and 32 female HC aged between 18 and 45 years participated in our study. Exclusion criteria for the patient group were a lifetime diagnoses of alcohol or substance dependence, psychotic disorder or bipolar I disorder, and current major depression as assessed by the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) [45]. In addition, patients had to be free of psychotropic medication for at least 4 weeks prior to investigation. Exclusion criteria for the HC group were the presence of any current or lifetime axis I or axis II disorder, and current or past psychotherapy. Diagnostic interviews were administered by trained and experienced psychologists. Inter-rater reliability for BPD was $\kappa = 0.69$ for SCID-I (primary diagnosis) and $\kappa = 0.77$ for IPDE. Patients were recruited at the Central Institute of Mental Health, Department of Psychosomatic Medicine and Psychotherapy in Mannheim/Germany or via announcements on BPD-associated Web sites. The HCs were randomly selected from the resident register of the city of Mannheim.

All participants underwent diagnostic assessment using the SCID-I [45] and the IPDE [44]. To obtain a psychopathological characterization of the patient sample, trait dissociation by the German version of the Dissociative Experience Scale (Fragebogen zu Dissoziativen Symptomen, FDS; [46]) and global BPD symptom severity by the Borderline Symptom List (BSL-95; [47]) were assessed.

Demographic and clinical characteristics (trait dissociation, BPD symptom severity, comorbid diagnoses) of the full sample can be found in Supplemental Table 1. There were no significant differences regarding age [BPD (Mean ± SD): 27.6 ± 7.5; HC: 27.8 ± 7.5], sex (all female), and ethnicity (all Caucasian). All participants were right-handed. There was a statistical trend for significant differences in level of education. As expected, both groups differed significantly in clinical measures of trait dissociation and BPD symptom severity.

From this initial sample, one HC and five BPD patients dropped out of the study before completion of the experiment. From the remaining sample, a subset of neuroimaging data had to be discarded due to movement artifacts or technical problems during MR scanning. The final neuroimaging data set comprised data from 26 HC and 27 BPD patients for the first (conditioning) session, 22 HC and 21 BPD patients for extinction recall, and 17 HC and 24 BPD patients for reacquisition. Since some participants did not show an adequate skin conductance response (SCR) > 1 μS during the scanning procedure, SCR data of 21 HC and 21 BPD from the first (conditioning) session and SCR of 15 HC and 14 BPD from the second session (extinction recall, reacquisition) could be included into the final psychophysiology analysis.

Demographic and clinical characteristics (trait dissociation, BPD symptom severity) of all subsamples can be found in Supplemental Table 2 (subsamples neuroimaging dataset) and Supplemental Table 3 (subsamples SCR dataset). There were no significant differences regarding age and educational level in any subsample. As expected, within all subsamples, BPD patients reported significantly more trait dissociation and BPD symptom severity.

Experimental design

The experimental design was an aversive differential delay conditioning procedure which is depicted by Fig. 1. Dependent variables were the blood-oxygen-level-dependent (BOLD) signal, SCR, and valence/arousal ratings. The conditioned stimuli (CS) were two neutral graphic patterns (blue square and yellow triangle). The US consisted of unpleasant, but tolerable electrical stimulation to the right thumb. Stimulus duration was 5.8 s (s), and the unconditioned stimulus (US) lasted for 2.8 s and was administered 3 s after CS onset. The inter-trial interval (ITI) lasted between 8 and 12 s, randomly generated. CS+ (CS paired with the US) and CS− (non-reinforced CS) were counterbalanced across participants and presented in pseudorandom order with the constraint of a maximum of two consecutive presentations of the CS+ (danger signal) or CS− (safety signal).

The US (electrical stimulation) was delivered through a copper electrode of an electrical stimulus generator (Digitimer, DS7A, Wewyn Garden City, UK). Before the experiment, for each participant, the level of pain stimulation was individually determined. To this end, participants were given a series of stimuli, starting with a mild stimulus, which was gradually increased to (1) detection threshold, (2) pain threshold, and (3) pain tolerance. This procedure was repeated three times. Finally, we delivered stimuli that were 80 % above pain threshold. Participants were asked to rate the intensity of pain on a Likert scale ranging from 0 = not painful to 10 = extremely painful. For the experiment, we used stimulation intensities that were rated with at least 7/10. Therefore, the experimental level of pain was objectively different, but subjectively both groups received the same level of painful stimulation. This procedure was chosen due to the fact of higher pain threshold in BPD [48, 49].

The first session (conditioning session) started with a phase in which participants were familiarized with the CS+ and CS− (*'familiarization'*: 10 CS+, 10 CS−). During the following *acquisition* phase, 18 CS+ paired with the US

Table 1 Brain activity during the different conditioning phases in healthy controls (HC) and in patients with borderline personality disorder (BPD)

Conditioning phase	Group	T contrast	MNI coordinates (*X*, *Y*, *Z*)	*K*	Hemisphere	Label (Brodmann area)	Lobe	*T* value	*Z* value	*p* value
Early acquisition	HC	CS+	0, −66, 12	110	R	Posterior cingulate	Limbic	6.12	4.71	0.037
			−27, −48, −15	49	L	Fusiform gyrus (BA37)	Occipital	6.25	4.77	0.021
			−18, −9, −12*	53	L	Parahippocampal gyrus/uncus/amygdala	Limbic	5.33	4.03	<0.05, ROI
			−24, −3, −24*		L		Limbic	4.83	3.78	
			18, −15, −15*	33	R	Parahippocampal gyrus (BA28)	Limbic	4.83	3.78	<0.05, ROI
		CS+ > CS−	No significant clusters at p(FDR) < 0.05							
	BPD	CS+	No significant clusters at p(FDR) < 0.05							
		CS+ > CS−	33, 39, 15	178	R	Middle frontal gyrus	Frontal	7.26	5.28	0.003
			27, 51, 3		R	superior frontal gyrus	frontal	4.17	3.60	0.003
			−60, 3, 3	49	L	Superior temporal gyrus	Temporal	5.29	4.30	0.022
			−66, −27, 12	61	L	Superior temporal gyrus (BA42)	Temporal	4.90	4.07	0.035
			66, −33, 18	89	R	Superior temporal gyrus (BA42)	Temporal	4.84	4.03	0.036
			−33, 15, 3*	16	L	Insula	Sub-lobar	4.32	3.70	<0.05, ROI
			−39,−3,−9*		L			3.63	3.22	<0.05, ROI
Late acquisition	HC	CS+	48, 33,−6*	34	R	Inferior frontal gyrus (BA47)	Frontal	4.80	4.00	<0.05, ROI
		CS+ > CS−	−30, −66, −51	1716	L	Inferior semilunar lobule	Cerebellum	6.32	4.84	0.004
			−33, −78, −30			declive	posterior	6.14	4.75	0.004
			−39, −63, −42			cerebellar tonsil		5.91	4.63	0.004
			45, −48, 51	606	R	Inferior parietal lobule (BA40)	Parietal	6.16	4.76	0.004
			63, −42, 24					5.59	4.46	0.004
			48, −45, 30					5.35	4.33	0.004
			54, 21, 33	178	R	Middle frontal gyrus (BA46)	Frontal	5.62	4.48	0.004
			54, 30, 18			inferior frontal gyrus (BA46)	frontal	4.27	3.67	0.008
			42, 6, 57			inferior frontal gyrus		4.26	3.66	0.008
			−60, −45, 24	216	L	Inferior parietal lobule (BA40)	Parietal	5.29	4.29	0.004
			−42, −51, 33					4.51	3.82	0.006
			−33, −51, 36					4.38	3.74	0.007
			39, 42, 30	35	R	Middle frontal gyrus	Frontal	4.95	4.10	0.005
			48, 27, −9*	85	R	Inferior frontal gyrus (BA47)	Frontal	4.93	4.08	<0.05, ROI
			−54, 21, 0*	41	L	Inferior frontal gyrus (BA47)	Frontal	4.22	3.63	<0.05, ROI
			−51, 15, −6*				frontal	4.16	3.59	<0.05, ROI
	BPD	CS+	48, 15, 45	49	R	Middle frontal gyrus (BA8)	Frontal	6.76	5.09	0.011
			39, 57, 3	98	R	Middle frontal gyrus (BA8)	Frontal	5.88	4.65	0.027
			60, −24, −24	32	R	Inferior temporal gyrus	Temporal	5.36	4.36	0.028
			−27, −66, −30	158	L	Pyramis	Cerebellum	5.15	4.24	0.030
			−57, −36, −6	38	L	Middle temporal gyrus	Temporal	4.83	4.04	0.040
			6, 39, 42	87	R	Medial frontal gyrus (BA8)	Frontal	5.06	4.18	0.040

Table 1 continued

Conditioning phase	Group	T contrast	MNI coordinates (*X, Y, Z*)	*K*	Hemisphere	Label (Brodmann area)	Lobe	*T* value	Z value	*p* value
		CS+ > CS−	48, 15, 45	208	R	Middle frontal gyrus (BA8)	Frontal	7.08	5.55	0.001
			48, 39, 30		R	Middle frontal gyrus (BA46)	frontal	4.44	3.80	0.017
			−18, −102, 3	831	L	Middle occipital gyrus (BA18)	Occipital	6.29	4.86	0.004
			39, 57, 3	120	R	Middle frontal gyrus (BA9)	Frontal	5.99	4.71	0.004
			30, 12, 3	46	R	Inferior frontal gyrus	Frontal	4.93	4.11	0.012
			33, 21, −9		R			3.89	3.42	0.027
			60, −42, 42	58	R	Inferior parietal lobule (BA40)	Parietal	4.51	3.84	0.016
			6, 36, 42	32	R	Medial frontal gyrus (BA8)	Frontal	4.44	3.79	0.017
			−45, 54, −3	45	L	Middle frontal gyrus (BA10)	Frontal	4.39	3.77	0.018
Extinction	HC	CS+	48, 48, −3*	R	18	Middle frontal gyrus	Frontal	5.57	4.45	<0.05, ROI
			−39, 15, 45*	L	31	Middle frontal gyrus	Frontal	5.01	4.13	<0.05, ROI
			9, 36, 24*	R	57	Orbito frontal gyrus	Frontal	4.86	4.04	<0.05, ROI
			33, 63, 18*					4.63	4.63	<0.05, ROI
			21, 63, 21*					3.87	3.87	<0.05, ROI
			−45, 39, −9*	L	20	Inferior frontal gyrus (BA 47)	Frontal	4.54	3.84	<0.05, ROI
			−27, 57, 24*	L	15	Middle frontal gyrus	Frontal	4.23	3.64	<0.05, ROI
			45, 48, 27*	R	14	Middle frontal gyrus	Frontal	4.13	3.57	<0.05, ROI
			51, 45, 21*	R		middle frontal gyrus	frontal	4.12	3.56	<0.05, ROI
			42, 36, 24*	R	9	Middle frontal gyrus	Frontal	4.07	3.53	<0.05, ROI
		CS+ > CS−	24, 66, 21*	R	28	Superior frontal gyrus (BA10)	Frontal	3.96	3.45	<0.05, ROI
			15, 63, 24*	R		Superior frontal gyrus	frontal	3.90	3.41	<0.05, ROI
			−45, 21, 45*	L	7	Middle frontal gyrus	Frontal	3.83	3.36	<0.05, ROI
	BPD	CS+	45, 42, −12*	R	99	Orbitofrontal gyrus (BA11)	Frontal	4.76	4.00	<0.05, ROI
			30, −6, −12*	R	5	Amygdala	Limbic	3.90	3.43	<0.05, ROI
		CS+ > CS−	No significant clusters p(FDR) < 0.05							
Extinction recall	HC	CS+	12, −63, 0	2235	R	Lingual gyrus	Occipital	10.14	6.12	<0.001
			6, −78, 15		R	lingual gyrus	Occipital	9.14	5.82	<0.001
			−12, −84, 3		L	cuneus (BA 17)	occipital	7.16	5.09	<0.001
			−36, 42, −9	17	L	Middle frontal gyrus	Frontal	5.00	4.04	0.002
			−39, −63, −39	31	L	Tuber	Cerebellum	4.79	3.92	0.003
			51, −48, 3	33	R	Middle temporal gyrus	Temporal	3.80	3.30	0.016
			63, −51, −9		R	middle temporal gyrus (BA21)	Temporal	3.76	3.27	0.017
			−27, −3, −27	10	L	Parahippocampal gyrus	Limbic	4.38	3.67	0.006
			−24, 51, 39	4	L	Superior frontal gyrus (BA9)	Limbic	3.86	3.34	0.014
			−36, −24, −15	6	L	Parahippocampal gyrus	Limbic	3.80	3.30	0.016
		CS+ > CS−	No significant clusters $p < 0.05$							

Table 1 continued

Conditioning phase	Group	T contrast	MNI coordinates (*X*, *Y*, *Z*)	*K*	Hemisphere	Label (Brodmann area)	Lobe	*T* value	*Z* value	*p* value
	BPD	CS+	12, −72, 9	449	R	Cuneus (BA17)	Occipital	6.78	4.78	0.032
			45, 9, −9*	49	R	Insula (BA38)	Sub-lobar	4.34	3.58	0.05, ROI
			−27, −3, −27	3	L	Parahippocampal gyrus	Limbic	4.38	3.67	0.009
		CS+ > CS−	24, 39, −18*	7	R	Orbitofrontal gyrus (BA11)	Frontal	4.90	3.89	<0.05, ROI
			6, 18, 51*	2	R	medial frontal gyrus (BA8)	Frontal	4.00	3.37	<0.05, ROI
Re-acquisition	HC	CS+	51, −45, 18	13705	R	Superior temporal gyrus,	Temporal,	11.53	6.01	<0.001
			−51, −24, 24		L	insular cortex (BA13)	Sub-lobar	11.09	5.91	<0.001
			−66, −27, 30		L			9.78	5.60	<0.001
			48, 12,0*					8.88		<0.05, ROI
			30, 57, 18	219	R	Superior frontal gyrus/middle frontal gyrus (BA9,	Frontal	7.78	5.02	<0.001
			27, 51, 24		R	BA10)	Frontal	6.88	4.70	<0.001
			42, 54, 12		R		Frontal	6.22	4.43	<0.001
					R					
			−39, 45, 24	74	L	Middle frontal gyrus (BA10, BA9)	Frontal	4.94	3.84	0.001
			−27, 51, 30		L			4.90	3.82	0.001
			−48, 45, 15		L			4.74	3.73	0.001
			−30, 3, −18*	45	L	Parahippocampal gyrus (BA34)	Limbic	5.10	3.92	<0.05, ROI
			−24, 0, −24*		L	amygdala/uncus	Limbic	5.14	3.94	<0.05, ROI
		CS+ > CS−	−3, 51, −6*	151	L	Medial frontal gyrus (BA10)	Frontal	6.80	4.28	<0.05, ROI
			0, 39, −18*		R			5.67	3.88	<0.05, ROI
			12, 45, −12*		R			4.98	3.60	<0.05, ROI
			−21, 42, 45*	L	19	Superior frontal gyrus	Frontal Frontal	5.18	3.68	<0.05, ROI
			−15, 42, 39*			superior frontal gyrus		5.03	3.62	<0.05, ROI
			−27, 24, 51*	10	L	Middle frontal gyrus (BA8)	Frontal	4.59	3.42	<0.05, ROI
	BPD	CS+	9, 3, 39*	19*	R*	Anterior cingulate (BA24)	Limbic	4.64	3.84	<0.05, ROI
			39, 54, −15*	16*	R*	Orbitofrontal gyrus (BA11)	Frontal	4.38	3.67	<0.05, ROI
		CS+ > CS−	36, 51, 12*	10	R	Middle frontal gyrus (BA10)	Frontal	4.28	3.61	<0.05, ROI

Clusters were determined applying an extended threshold of $p < 0.05$ FDR corrected on the whole-brain voxel-wise level. Clusters indicated by an *asterisk* were detected by region of interest (ROI) analyses with predefined anatomical masks. Sizes of subsamples were 26 for session 1 (familiarization, acquisition, extinction), 22 for extinction recall, and 17 for re-acquisition. *Note L* left, *R* right, *k* cluster size of contiguous voxels, *MNI* Montreal Neurological Institute, *R/L* right/left, *BA* Brodmann area, *CS+* conditioned stimulus that was sometimes paired with the unconditioned stimulus (US) but here only in absence of the US, *CS−* conditioned stimulus never paired with US and 36 CS− never paired with the US were presented. Furthermore, 18 CS+ were presented but not paired with the US (catch trials). Catch trials were used to differentiate between BOLD responses to the CS+ and to the pain stimulus (US). During the *extinction* phase, 18 CS+ and 18 CS− were presented. The second session (reacquisition session) took place 72 h later and consisted of the *extinction recall* phase (18 CS+ and 18 CS−) and a *reacquisition* phase (18 CS−, 9 CS+, 9 CS+/US).

Apparatus and physiological recordings

Stimulus delivery was controlled by a PC running Presentation software (Neurobehavioral Systems, San Francisco). Physiological data acquisition was controlled by an Ibook running Vitagraph version 4.61 (Becker Meditec, Karlsruhe, Germany). Physiological channels were recorded at a rate of 256 Hz in continuous mode using the Vitaport II system (Becker). SCR was obtained from 10-mm (sensor diameter) Ag/AgCl electrodes (Marquette Hellige GmbH, Freiburg, Germany) filled with an isotonic EDR jelly TDE-246 (Steffens, Berlin, Germany) and placed on the thenar and hypothenar of the non-dominant hand (constant voltage method with 0.5 V).

Procedure

The study was approved by the ethical review committee of the University of Heidelberg, Germany, in accordance with the Declaration of Helsinki. All subjects gave written informed consent after receiving a description of the study and scanning procedure. All participants were paid for study participation (10 €/h). The experiment was conducted between 2007 and 2009 at the Central Institute of Mental Health in Mannheim, Germany.

All participants underwent diagnostic assessments including the SCID-I and IPDE by trained diagnosticians and completed clinical questionnaires (FDS, BSL) as described above. The conditioning procedure took place on two different days within a fixed time interval. On a first day, the conditioning session (familiarization phase, acquisition phase, extinction phase) was conducted during fMRI. Immediately before the experiment, the level of pain stimulation was individually determined for each participant as described in detail above. The reacquisition session (extinction recall phase and reacquisition phase) took place 72 h later in the same MR scanner.

Throughout the scanning procedure, participants viewed stimuli presented on a screen by a projector via a mirror mounted on the head coil. Participants were not informed about CS–US contingencies and were told to passively view the stimuli. After each phase (familiarization acquisition, extinction, extinction recall, and reacquisition), participants rated the valence and arousal of the CSs to test awareness of the CS–US contingency. In addition, participants rated their acute dissociation on scales ranging from 1 = very calm to 9 = very arousing, 1 = very pleasant to 9 = very unpleasant, and 1 = no dissociative symptoms to 9 = very intense dissociative symptoms. Valence/arousal ratings of the CS were obtained by presenting the CS together with a visual analogue scale, and patients used a keypad to move the cursor and to choose a number.

fMRI assessment

fMRI was conducted by a Siemens MAGNETOM Vision 1.5 Tesla whole body scanner (Siemens Medical Solution, Erlangen, Germany). Head movement artifacts and scanning noise were restricted using head cushions and headphones within the scanner coil. To acquire BOLD signals (T2-weighted echo planar imaging, EPI), a standard protocol (see also [27]) with the following parameters was used: repetition time (TR): 3.77 s, echo time (TE): 45 ms; flip angle: 90°, matrix: 64 × 64, slice thickness: 3 mm, slice gap: 1 mm, FOV: 220 × 220 mm, and number of slices: 35. There were 100 images for familiarization, 320 for acquisition, and 160 for all other phases.

Data analysis

Psychometrics

Means and standard deviations of the BSL and the FDS were analyzed using t tests. For group comparisons, significance levels were set to $p < 0.05$. All analyses were conducted with SPSS (Version 22.0 for Windows; SPSS Inc., Chicago, IL).

Ratings

To compare valence and arousal ratings, repeated-measures analyses of covariance (rm-ANCOVA) were performed for the mean of each phase with group (HC, BPD) as between-subject factor, stimulus type (CS+, CS) as within-subjects factor, and present-state dissociative experience as covariate. A Greenhouse-Geisser correction was employed in case the sphericity assumption was not met. Statistical analysis was performed using SPSS 22.0 (SPSS Inc, Chicago, IL). An alpha level of $p < 0.05$ determined statistical significance.

Skin conductance response (SCR)

The electrodermal activity was determined according to published guidelines [50] with the program EDR-Para (Schäfer, Wuppertal). Phasic SCRs were defined as the response magnitude (maximum deflection) within a 1- to 4-s time frame (first interval response, FIR). SCRs lower than 0.05 µS were scored as zero. The FIR was counted as missing when the SCR was clearly initiated prior to CS onset. If such an anticipatory response was superimposed on a stimulus-related response, the scoring method (B) as described by Boucsein ([50], p. 136) was used. SCRs were log-transformed to reduce skewness [50]. To compare SCR to CS+ and CS− type × group, rm-ANCOVAs were performed for the mean of each phase with present-state dissociative experience as covariate. A Greenhouse-Geisser correction was employed if the sphericity assumption was not met. Statistical analysis was performed using SPSS 22.0 (SPSS Inc, Chicago, IL). An alpha level of <0.05 determined statistical significance.

fMRI data

Functional data were analyzed with SPM5 (Welcome Department of Imaging Neuroscience, University College London, UK, 2005). The first four images at the beginning of each trial were discarded to enable the signal to achieve steady-state equilibrium between radiofrequency pulse and relaxation.

Within preprocessing, the EPI time series were realigned and unwarped to the mean to correct for intra-subject's head movements. Mean images were normalized to an MNI (Montreal Neurological Institute, www.bic.mni.mcgill.ca) echoplanar imaging template with affine registration followed by nonlinear transformation with 25 mm cutoff, medium regularization and 16 iterations, resampled with trilinear interpolation, and written in $3 \times 3 \times 3\ \text{mm}^3$ isotropic voxels. The normalization parameters determined for the mean functional volume were then applied to the corresponding functional image volumes for each participant. Finally, images were smoothed with a Gaussian kernel of 8 mm full-width at half-maximum. The data were high-pass filtered (1/128 Hz cutoff) to remove low-frequency signal drifts.

The consecutive statistical analyses of the fMRI data relied upon the general linear model (GLM) to model effects of interest as implemented in SPM5. For each subject, the following events of interest were defined as regressors of interest: US, CS−, CS+$_{\text{paired}}$, and CS+$_{\text{unpaired}}$ for each phase of the conditioning paradigm: familiarization, acquisition, extinction, extinction recall, and reacquisition phase.

In order to assess the temporal course of brain activation, we split up the acquisition phase (of the first conditioning session) into an early and late acquisition phase. In addition, we investigated habituation effects during acquisition by modeling linear time effects for CS+$_{\text{unpaired}}$, CS+$_{\text{unpaired}}$ > CS−, and CS+$_{\text{paired}}$ (CS paired with US) using the time modulation function as implemented in SPM ($p < 0.05$).

Task-related activity (BOLD response) for each event was modeled by convolving a vector of the event onset times with a canonical hemodynamic response function (HRF). The regressors of interest were included together with time as covariate in a GLM to yield parameter estimates for brain activation related to the different event types (US, CS−, CS+$_{\text{paired}}$, CS+$_{\text{unpaired}}$). T contrast images for CS+$_{\text{paired}}$ > CS− and CS− > CS+$_{\text{paired}}$ were computed for each conditioning phase (familiarization, early acquisition, late acquisition, extinction, extinction recall, reacquisition) separately.

At the second level (group level), the resulting first level contrast images were entered into random effect models: *Within-group* (one sample) *t* tests were used to compare brain activity during CS+ to brain activity in response to CS−. *Between-group* (two sample) *t* tests were used to analyze group differences in response to CS+, CS−, as well as CS+$_{\text{unpaired}}$ > CS− during the different conditioning phases. Clusters were determined using an extent threshold of $p < 0.05$ on the voxel-wise whole-brain level. The false discovery rate (FDR) correction was applied to correct for multiple comparisons.

Region of interest analyses Based on our a priori hypotheses (stronger activation in the amygdala and insula during acquisition and reacquisition and less activation in the mPFC during within-session and between-session extinction in BPD patients than HC), regions of interest analyses (ROIs) were performed for the following brain areas: amygdala, ACC, hippocampus, insula, OFC, dlPFC, and vmPFC. Anatomically based ROIs of these regions were created using the Masks for Regions of Interest Analysis (MARINA) software program (Bertram Walter, Bender Institute of Neuroimaging, University of Giessen, Germany). These masks were then used for small volume corrections.

In a separate model, state dissociation ratings were included as a covariate in order to test the effect of this

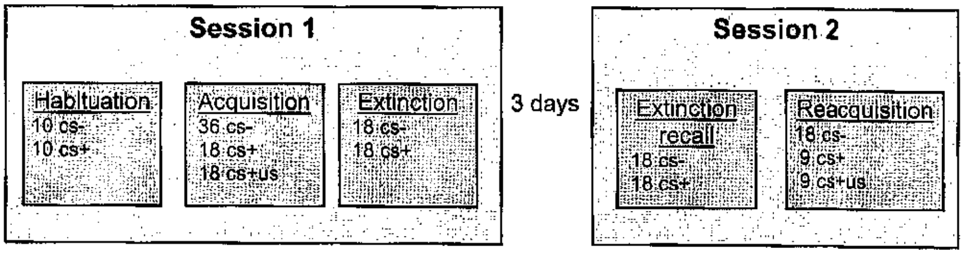


Fig. 1 Experimental design: Ratings of valence and arousal were performed after each of the five experimental phases. *CS+* conditioned stimulus paired with the US; *CS−* non-reinforced conditioned stimulus, *US* unconditioned stimulus

variable on the results. Furthermore, a subgroup analysis with BPD patients with versus without current PTSD compared to HC was computed.

Results

Ratings

Means and standard deviations of arousal (a) and valence (b) ratings during acquisition, extinction, extinction recall, and reacquisition in HC and BPD patients are depicted in Fig. 2.

For arousal ratings after acquisition, a significant effect of type [$F(1,34) = 23.929$, $p < 0.001$] but no significant interaction with group was found [$F(1,34) = 0.001$, $p = 0.973$]: All participants showed a higher rating of the CS+ compared to the CS−. For the arousal ratings after extinction and extinction recall, no significant effect of type [$F(1,30) = 0.250$, $p = 0.621$; $F(1,24) = 0.566$, $p = 0.459$] and no significant interaction with group were found [$F(1,30) = 0.009$, $p = 0.925$, $F(1,24) = 0.452$, $p = 0.508$]. After extinction, BPD patients rated both stimuli as more aversive than the HC indicated by a significant effect of group [$F(1,30) = 5.565$, $p = 0.025$].

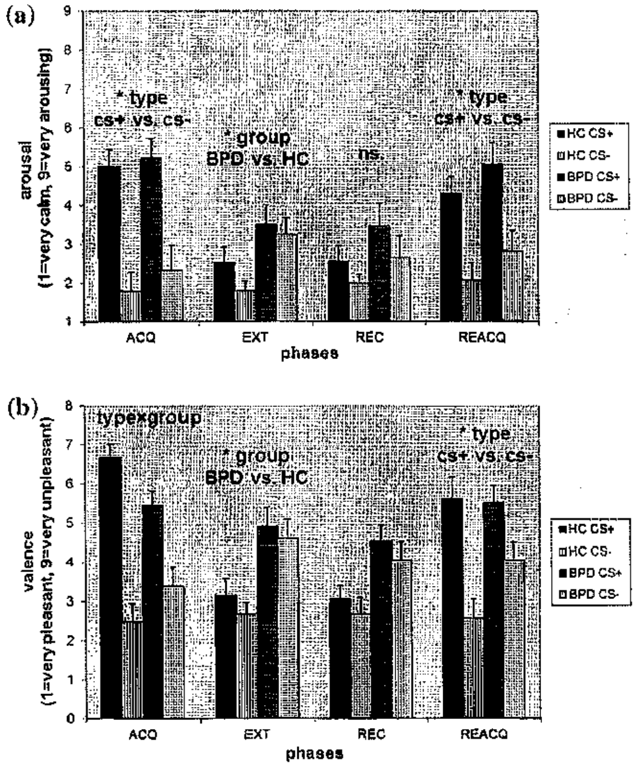


Fig. 2 Results of the arousal (**a**) and valence (**b**) ratings. *ACQ* acquisition, *EXT* extinction, *REC* extinction recall, *REACQ* reacquisition, *HC* healthy controls, *BPD* BPD patients, *CS+* conditioned stimulus paired with the US, *CS−* non-reinforced conditioned stimulus, significant at $^{*}p < 0.05$, $^{\#}p < 0.10$

For the arousal ratings after reacquisition, a significant effect of type [$F(1,24) = 18.315$, $p < 0.001$] and no significant interaction with group were found [$F(1,24) = 0.337$, $p = 0.567$]: All participants showed a more aversive rating of the CS+ compared to the CS− (see Fig. 2a).

For the valence ratings after acquisition, a significant effect of type [$F(1,34) = 15.199$, $p < 0.001$], with more aversive rating of the CS+ than the CS−, and a significant interaction effect type × group [$F(1,34) = 4.223$, $p = 0.048$] were found. For the valence ratings after extinction and extinction recall, no significant effect of type [$F(1,30) = 0.334$, $p = 0.568$; $F(1,24) = 1.189$, $p = 0.286$] and no interaction with group were found [$F(1,30) = 0.021$, $p = 0.886$, $F(1,24) = 1.156$, $p = 0.293$]. After extinction, again, a significant effect of group was found for the valence ratings [$F(1,30) = 14.944$, $p = 0.001$]: BPD patients rated both stimuli as more aversive than the HC. For the valence ratings after reacquisition, a significant effect of type [$F(1,24) = 14.138$, $p = 0.001$] but no interaction with group was found [$F(1,24) = 0.199$, $p = 0.660$]: All participants showed a more aversive rating of the CS+ compared to the CS− (see Fig. 2b).

Skin conductance response (SCR)

Means and standard deviations of SCR during acquisition, extinction, extinction recall, and reacquisition in HC and BPD patients are shown in Fig. 3. During acquisition, no significant effect of type [$F(1,34) = 2.291$, $p = 0.139$, $\eta^2 = 0.063$] and no significant interaction with group were found [$F(1,34) = 0.039$, $p = 0.844$]. During extinction, no significant interaction with group was found [$F(1,33) = 0.962$, $p = 0.334$], but a significant effect of type [$F(1,33) = 9.895$, $p = 0.003$] with participants shows stronger responses to the CS+ than to the CS−. During extinction recall, no significant effect of type [$F(1,20) = 0.209$, $p = 0.652$] and a trend for the interaction effect type x group were found [$F(1,20) = 3.194$, $p = 0.089$], with BPD patients showing descriptively a stronger response to CS+ compared to the CS−, whereas HCs showing descriptively a stronger response to CS− compared to the CS+. During reacquisition, no significant effect of type [$F(1,20) = 1.967$, $p = 0.176$, $\eta^2 = 0.090$] and no interaction with group were found [$F(1,20) = 1.768$, $p = 0.199$, $\eta^2 = 0.081$].

fMRI

Between-group t tests

No significant group differences on the whole-brain level could be detected at a threshold level of $p < 0.05$ after correction for multiple comparisons (FDR correction).

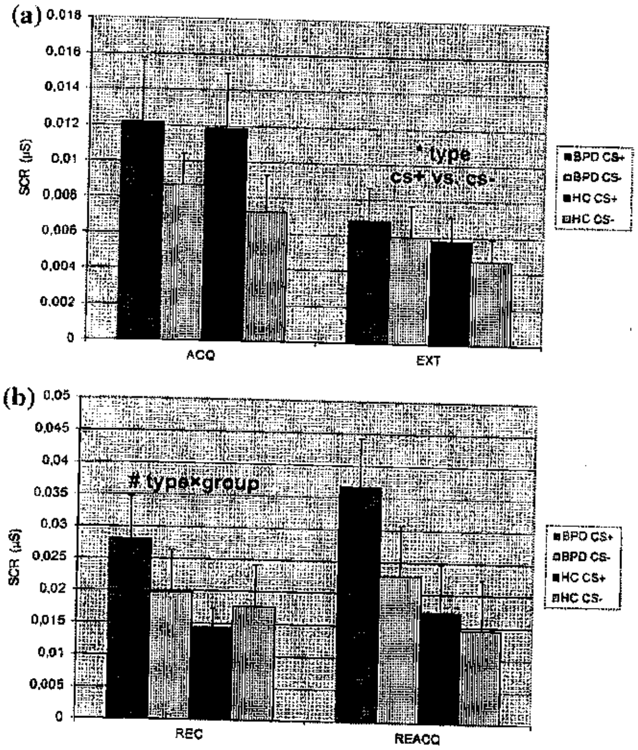


Fig. 3 Skin conductance response. *Note ACQ* acquisition, *EXT* extinction, *REC* extinction recall, *REACQ* reacquisition, *HC* healthy controls, *BPD* BPD patients, *CS+* conditioned stimulus paired with the US, *CS−* non-reinforced conditioned stimulus

Within-group t tests

Results for brain activity in response to CS+$_{\text{unpaired}}$ and CS+$_{\text{unpaired}}$ > CS− can be found in Table 1. In the following, results are reported for each group and for each conditioning phase separately [$p(\text{FDR}) < 0.05$].

Healthy control group

During *early acquisition*, no significant clusters were observed for the CS+$_{\text{unpaired}}$ > CS− contrast. During presentation of the CS+$_{\text{unpaired}}$, HC recruited the fusiform gyrus (BA37) and right posterior cingulate. Our ROI analyses further revealed significant CS+$_{\text{unpaired}}$-related activation in the bilateral parahippocampal gyrus and left amygdala.

During *late acquisition*, HC showed significant stronger activation in response to the CS+$_{\text{unpaired}}$ than to CS− in the bilateral inferior frontal gyrus (BA46 and BA47), bilateral inferior parietal lobule, right middle frontal gyrus (BA46), and left cerebellum.

During *extinction*, HC exhibited significant differential activation for CS+$_{\text{unpaired}}$ > CS− in the right superior frontal gyrus (BA10) and left middle frontal gyrus (clusters revealed by ROI analyses). In addition, our ROI analyses revealed significant CS+$_{\text{unpaired}}$-related activation in the right OFC, left inferior frontal gyrus (BA47), and bilateral middle frontal gyrus.

During *extinction recall*, no significant clusters were found for the CS+$_{\text{unpaired}}$ > CS− contrast. In response to the CS+$_{\text{unpaired}}$, HC activated the right lingual gyrus and left cuneus, the left middle frontal gyrus and superior frontal gyrus (BA9), the left parahippocampal gyrus, right middle temporal gyrus, and left tuber (cerebellum).

During *reacquisition*, our ROI analyses revealed significant clusters in the bilateral medial frontal gyrus (BA10), left superior frontal gyrus, and left middle frontal gyrus (BA8) for the CS+$_{\text{unpaired}}$ > CS− contrast. In response to the CS+$_{\text{unpaired}}$, HC showed recruitment of the bilateral superior temporal gyrus and insular cortex (BA13), bilateral middle frontal, and superior frontal. For the CS+$_{\text{unpaired}}$ contrast, ROI analyses additionally revealed significant clusters in the left amygdala and left parahippocampal gyrus.

BPD group

During *early acquisition*, BPD patients showed significant differential activation for CS+$_{\text{unpaired}}$ > CS− in the right superior and middle frontal gyrus as well as in the bilateral superior temporal gyrus. ROI analyses further revealed significant stronger activation in the left insula (see Fig. 4a) for the CS+$_{\text{unpaired}}$ > CS− contrast.

During *late acquisition*, BPD patients exhibited significant stronger activation in response to CS+$_{\text{unpaired}}$ > CS− in the bilateral middle and medial frontal gyrus (BA8, BA9, BA10, BA46), right inferior frontal gyrus, left middle occipital gyrus (BA18), and right inferior parietal lobule (BA40). In response to the CS+$_{\text{unpaired}}$, in addition, patients recruited the right inferior temporal gyrus and left middle temporal gyrus.

During the *extinction* phase, no significant clusters were observed for the CS+$_{\text{unpaired}}$ > CS− contrast. In response to CS+$_{\text{unpaired}}$, BPD patients activated the right orbitofrontal gyrus (BA 11) and right amygdala (see Fig. 4b).

During *extinction recall*, BPD patients exhibited CS+$_{\text{unpaired}}$-related activation in the right cuneus and left parahippocampal gyrus as well as in the right insula. For the CS+$_{\text{unpaired}}$ > CS− contrast, ROI analyses revealed stronger activation in the orbitofrontal gyrus (BA11) as well as medial frontal gyrus (BA8) in the patient group.

During *reacquisition*, we found significant clusters in the middle frontal gyrus (BA10) for the CS+$_{\text{unpaired}}$ > CS− contrast as well as clusters in the orbitofrontal gyrus (BA11) and ACC (BA24) for the CS+$_{\text{unpaired}}$ contrast.

Linear habituation

Complete results for linear time effects during the acquisition phase in each group can be found in Supplemental Table 4.

Healthy control group

In HC, we found a habituation effect, i.e., significant decrease in activity in the mid-cingulate gyrus in response to CS$+_{\mathrm{unpaired}}$ as well as CS$+_{\mathrm{unpaired}}$ > CS−. Our ROI analysis further revealed a significant linear decrease in activity in the right amygdala and bilateral parahippocampal gyrus for the repeated exposure to the CS in temporal contingency with the US (CS$+_{\mathrm{paired}}$) (see Supplemental Table 4).

BPD group

BPD patients showed a habituation of left insula activation and left medial frontal gyrus activation in response to CS$+_{\mathrm{unpaired}}$ as well as CS$+_{\mathrm{unpaired}}$ > CS− during acquisition. For the CS$+_{paired}$ (i.e., repeated exposure to the CS in temporal contingency with the US), we found a significant linear decrease in activation in the left parahippocampal gyrus and inferior frontal gyrus (see Supplemental Table 4). The ROI analysis with the amygdala mask revealed no significant clusters.

Subgroup analysis

Analyses with state dissociation as a covariate and for the subgroup of BPD patients with comorbid PTSD led to similar results (data not shown).

Discussion

In this fMRI study, we investigated the neural correlates of emotional associative learning in un-medicated female patients with BPD compared to female HC participants. During an aversive differential delay conditioning paradigm, brain activity (BOLD responses), subjective ratings, and SCR were assessed. In addition to within-session extinction, we investigated extinction recall (between-session extinction) and reinstatement, i.e., reacquisition of the initial CS–US contingency, after 72 h. We were further interested in the temporal graduation of brain activation. Therefore, we slit the initial acquisition phase into an '*early acquisition*' and a '*late acquisition*' phase and estimated habituation effects in terms of a linear decrease in brain activity during each conditioning phase.

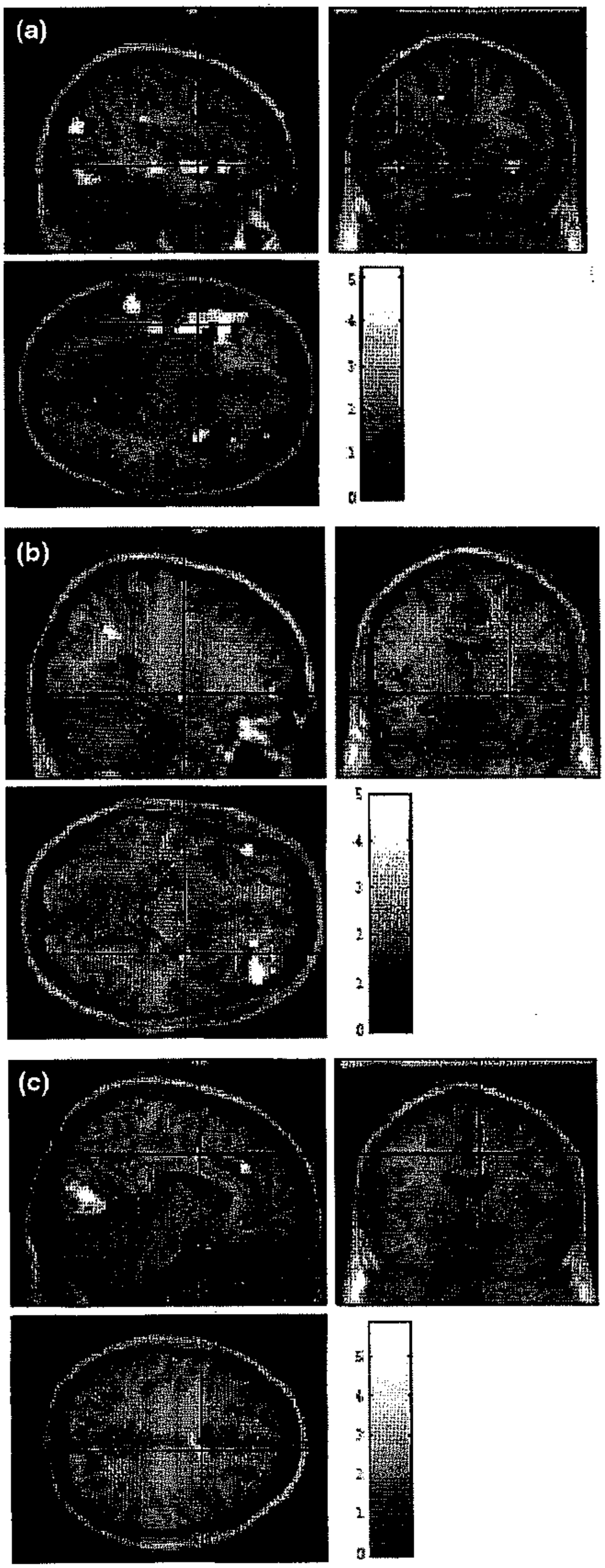
Concerning subjective ratings, both groups perceived the CS+ as more arousing than the CS− after acquisition as well as reacquisition, suggesting that they successfully learned the difference between safety and danger signal. During extinction, a significant group effect was found with higher arousal and valence ratings for CS+ but also for CS− in BPD patients than in healthy participants. This finding may point to disturbed emotional learning in terms of slower extinction processes in BPD patients compared to HC.

SCR was assessed as a more direct measure of arousal and a more proximal measure of the fear conditioning process. Differential learning effects by means of stronger responses to CS+ than to CS− were only observed for the extinction phase. However, since some participants did not show an adequate SCR (>1 µS), the sample size for our psychophysiology analysis was probably too small to detect statistically significant effects (as effect sizes were in a medium range). Interestingly, we found a trend for BPD patients responding stronger to CS+ than to CS− during extinction recall, again arguing for slower extinction learning in these patients.

On the neural level, no significant between-group differences were found after correcting for multiple comparisons. During acquisition as well as reacquisition, both groups showed significant CS$+_{unpaired}$-related activation in the amygdala, insula, and mPFC brain areas that have been identified as central parts of a 'classical fear network' (for a review see, e.g., [28, 31]).

Results of within-group comparisons and time effect analyses point to a different temporal course of brain activity in the two groups. During the early acquisition phase, BPD patients but not HC showed differential activation to CS$+_{\mathrm{unpaired}}$ > CS− in the left insula. The insula has been implicated in external (e.g., detection of salient events including threat in the environment) as well as internal attention (e.g., interoceptive awareness, monitoring of arousal states) [51, 52]. It has been proposed that the insula plays an important role in conveying a cortical representation of fear to the amygdala [20], particularly in the context of uncertain aversive events [12, 13, 23]. Early recruitment of the insula may therefore suggest increased vigilance to potential threat already at an early stage of the acquisition phase in BPD patients. During the late acquisition phase, presentation of CS$+_{\mathrm{unpaired}}$ (compared to CS−) was associated with increased activation in fronto-parietal brain regions including the dlPFC, OFC, vlPFC (inferior frontal gyrus), and inferior parietal lobule in both groups. Involvement of these brain areas in acquisition processes has also been reported in previous fear conditioning studies [16, 17, 28, 31]. It has been proposed that the dlPFC may predominantly be active when aversive (threatening) stimuli are predictable during fear conditioning [31]. The present neuroimaging findings may therefore be related to a successful differential learning of safety and danger signals at a later stage of the acquisition phase in both groups.

In our present study, we were not only interested in within-session extinction but also in *extinction recall* (between-session extinction) after 72 h. During within-session extinction, both groups recruited the OFC when exposed to the CS+. BPD patients—but not



◄**Fig. 4** Brain activity during the different conditioning phases in BPD patients: **a** Insula activation was found during the early acquisition phase, **b** right amygdala activation was found during extinction, **c** right mid-cingulate activation was observed during reacquisition

HC—additionally showed $\text{CS+}_{\text{unpaired}}$-related activation in the amygdala, whereas healthy participants additionally recruited the inferior frontal gyrus and middle frontal gyrus. Activation in the amygdala is thought to occur as an orienting response to (especially self-relevant) salient stimuli and is assumed to serve the relay of emotionally relevant information to different processing channels [9, 21, 28]. During extinction, BPD patients in our study rated both the CS+ and CS− as significantly more arousing and aversive than HC. In this context, CS+-related amygdala activation may be related to altered differential learning in these patients. While healthy participants showed significantly stronger prefrontal activation in response to CS+ > CS− during extinction, BPD patients showed increased activation for CS+ > CS− only during extinction recall (i.e., during the second extinction session after 72 h). Moreover, during extinction recall, BPD patients but not HC showed $\text{CS+}_{\text{unpaired}}$-related activity in the insular cortex. As mentioned above, we found a trend for stronger SCR to CS+ than to CS− in the BPD group. In this context, the present neuroimaging findings may point to delayed extinction learning in BPD patients.

When the original contingency of the CS+ and US was renewed after 72 h, HC showed $\text{CS+}_{\text{unpaired}}$-related activation in the insular cortex, amygdala, and parahippocampal gyrus—regions critically involved in fear acquisition [28]. After extinction, reacquisition gives a renewed meaning to the CS+. Amygdala and insula might play an important role in mediating these kinds of contingency changes and in the building of new associations [22]. During reacquisition, BPD patients but not HC activated the ACC (BA24), which has been associated with the anticipation of pain and the integration of nociceptive input with memory [6]. Both groups during reacquisition increased activity in the middle frontal gyrus (BA10) in response to the $\text{CS+}_{\text{unpaired}}$ compared to the CS−, but HC additionally recruited parts of the dlPFC (BA8). As mentioned above, the dlPFC has been proposed to play an important role in regulatory processes including emotion down-regulation [32] as well as processing aversive stimuli that are predictable [31].

In the present study, we were further interested in the temporal course of brain activation, particularly in linear habituation effects during acquisition. Interestingly, in the patient group, we found a linear decrease in activity in the left insula in response to CS+$_{unpaired}$ > CS−. Since patients showed increased activity in this area (suggesting increased vigilance) to the CS+$_{unpaired}$ during early acquisition, they may have learned the association between the CS and US relatively early and therefore reduced their attention to the CS+$_{unpaired}$ over the course of the acquisition phase. In HC, we found a linear habituation effect for the mid-cingulate cortex (BA 5) to the CS+ during acquisition. Of note, amygdala habituation to CS+$_{paired}$ during acquisition was only found in HC but not in the BPD group. This finding is in line with a recent fMRI study by Kamphausen and colleagues [43] who applied an instructed fear task. In this study, healthy participants but not BPD patients showed a habituation (i.e., decrease in activity) in the amygdala and an increase in activity in the mPFC over the course of the instructed fear task. Moreover, BPD patients previously showed a failure to habituate to the repeated presentation of aversive pictures which was associated with prolonged amygdala activity and smaller increases in amygdala–insula connectivity [53, 54].

All in all, our findings may be related to disturbed emotional learning processes in patients with BPD. Early activation in the insula in response to CS+$_{unpaired}$ > CS− suggests that patients may react more sensitively to fear conditioning in terms of increased vigilance already at the beginning of the acquisition phase. Amygdala activity during extinction and insula activity along with stronger SCR to CS+ than CS− during extinction recall—as well as the lack of this activation in HC—may point to delayed extinction processes in BPD patients. However, further fMRI studies are needed to confirm and extend these findings and may also investigate conditioning processes in a social context.

There are several limitations of this study. First of all, we lost data since several participants did not show an appropriate SCR due to dropouts or due to technical problems. The sample size for the analysis of the SCR was probably too small to find significant effects. The partial reinforcement during acquisition (CS+ not always followed by US) may have led to psychophysiological responses that were less clear-cut. Regarding fMRI assessment, we used a standard protocol with a TR of 3.77 s which is relatively long given the duration of the US of 2.8 s. Moreover, although we excluded substance dependence, psychotic disorders, and current depression as comorbid conditions, it might well have been that other comorbid conditions such as antisocial personality disorder or anxiety disorders have influenced learning processes and its neural correlates in the present study. We tested the influence of comorbid PTSD, but found no differences in any of our dependent variables between patients with and without PTSD. A clinical assessment of depressive symptoms (e.g., the BDI) would have been helpful to further characterize our sample.

Despite these limitations, we believe that our data are of importance as, to our knowledge, this is the first neuroimaging study investigating the neural correlates of acquisition, (within-session) extinction, (between-session) extinction recall, and re-acquisition within a classical delay conditioning paradigm in BPD. The alterations in the time course of neural response patterns during the different learning phases as demonstrated here—in conjunction with the influence of dissociation on conditioning as demonstrated earlier [42]—might be relevant for the understanding of disturbed learning processes in this disorder and should be considered in the context of learning-based psychotherapy. Findings may further point to the necessity of stress and emotion regulation training in these patients.

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Conflicts of interest There are no conflicts of interest.

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Abstract:
The Keynote Speaker at Winter Conference on Animal Learning and Behavior (WCALB) 2014 was Dr. Björn Brembs whose address was titled, Pavlovian and Skinnerian Processes Are Genetically

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Separable. The essence of the address, that describes the research on which Dr. Brembs based this conclusion, is described below. Articles in this issue representing the related Focus Session include: The Many Faces of Pavlovian Conditioning by Dr. Jozefowiez, Pavlov + Skinner = Premack by Dr. Killeen, Evocation of Behavioral Change by the Reinforcer is the Critical Event in Both the Classical and Operant Procedures by Dr. Donahoe, On Choice and the Law of Effect by Dr. Staddon, Response-Outcome versus Outcome-Response Associations in Pavlovian-to-Instrumental Transfer: Effects of Instrumental Training Context by Gilroy, Everett and Delamater, and The Instrumentally-Derived Incentive-Motivational Function by Dr. Weiss. As a whole, they attempt to increase our contact with, and get at the essence of, what is actually happening with these operant and classical contingencies in the laboratory and nature. The Research Seminar Session revealed the current tendency for explanations of behavior to be reduced to physiology, neuroscience, and genetics. However, anti-reductionists saw shortcomings in this approach.

They recommended an interconnected holistic approach which shifts the focus away from the structure of discrete behaviors and toward examining the environment in which the behavior occurs and the consequences produced. The distinction between structural and functional analysis points to a difficulty of integrating facts about behavior with other levels of analysis that requires our attention.

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Stanley J. Weiss &
Jesús Rosales-Ruiz, Editors
Special Issue

Introduction to the Special Issue on Operant/Classical Conditioning: Comparisons, Intersections and Interactions

The 2014 Winter Conference on Animal Learning and Behavior Focus and Research Seminar Sessions

Classical
Conditioning

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The Keynote Speaker at Winter Conference on Animal Learning and Behavior (WCALB) 2014 was Dr. Björn Brembs whose address was titled, *Pavlovian and Skinnerian Processes Are Genetically Separable.* The essence of the address, that describes the research on which Dr. Brembs based this conclusion, is described below. Articles in this issue representing the related Focus Session include: *The Many Faces of Pavlovian Conditioning* by Dr. Jozefowiez, *Pavlov + Skinner = Premack* by Dr. Killeen, *Evocation of Behavioral Change by the Reinforcer is the Critical Event in Both the Classical and Operant Procedures* by Dr. Donahoe, *On Choice and the Law of Effect* by Dr. Staddon, *Response-Outcome versus Outcome-Response Associations in Pavlovian-to-Instrumental Transfer: Effects of Instrumental Training Context* by Gilroy, Everett and Delamater, and *The Instrumentally-Derived Incentive-Motivational Function* by Dr. Weiss. As a whole, they attempt to increase our contact with, and get at the essence of, what is actually happening with these operant and classical contingencies in the laboratory and nature. The Research Seminar Session revealed the current tendency for explanations of behavior to be reduced to physiology, neuroscience, and genetics. However, anti-reductionists saw shortcomings in this approach. They recommended an interconnected holistic approach which shifts the focus away from the structure of discrete behaviors and toward examining the environment in which the behavior occurs and the consequences produced. The distinction between structural and functional analysis points to a difficulty of integrating facts about behavior with other levels of analysis that requires our attention.

Dr. Björn Brembs Professor of Neurogenetics at the Institute of Zoology at Universität Regensburg was the Keynote Speaker at the 2014 Winter Conference on Animal Learning & Behavior. The title of his address was *Pavlovian and Skinnerian Processes are Genetically Separable.* This set the stage for the Conference theme and the related Focus Session concerned with *Operant/Classical Conditioning: Comparisons, Intersections and Interactions.* Articles in this issue of the *International Journal of Comparative Psychology* have been developed from that Focus Session. The impressive Keynote Address Dr.

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Brembs delivered is sketched below. For additional information, see Brembs and Plendl (2008), Brembs (2009, 2011) and Colomb and Brembs (2010). The slides from Dr. Brembs' presentation can be found at http://www.slideshare.net/brembs/wcalb14.

In his introduction, Brembs noted that Skinner realized in the 1930s that operant conditioning was likely composed of several processes (Konorski & Miller, 1937a, 1937b; Skinner, 1935, 1937). Brembs went on to claim that his research demonstrates that Skinner was right. He described an operant conditioning situation where a food-deprived rat was reinforced with a food pellet for every lever press (a fixed ratio or FR1), and went on to explain that, although probably there are two processes operating simultaneously here, this paradigm makes it difficult to reveal them directly. Instead, one needs to use other experiments, such as those he designed with tethered flies as subjects, organized analogously to the rats' FR1 situation, wherein he could precisely measure the direction in which they were turning.

Brembs explained that these experiments used colors instead of a lever: One color (say, blue) was associated with an aversive heat-beam – much like an unpressed lever is associated with no food. In contrast, another color (e.g., green) was associated with the heat beam being switched off when the stationary flying fly attempted a turn in the proper direction – much like a depressed lever produces food.

From the way he views these paradigms, in both experiments the stimuli were under full control of the animal. When the rat presses the lever or the fly generates a particular turning maneuver, the situation changes. For the rat, food is delivered. For the fly, heat is switched off. In both cases, the animal's behavior has produced a preferred situation. If the rat stops pressing and engages in some other behavior, food ceases to be delivered. If the fly stops attempting to turn to the right and instead attempts a left turn (coloring its environment blue), the heat is switched on.

Brembs claimed that if the rat case parallels the fly case such that:

> (a) the fly turning right under green light and shutting the heat off is analogous to the rat pressing the lever down and producing food, *and*
> (b) the fly turning left under blue light and turning the heat on is analogous to the rat not pressing the lever (lever up) leading to no food,

then two things happen to rats in a Skinner box. The rat learns that the lever means food (just as the fly learns that green means heat) and that lever pressing produces food. The topography of behavior (i.e., left or right paw, nose, tail, etc.) is irrelevant as long as the lever is pressed and the microswitch is closed. In this stage, (1) the behavior is still flexible, (2) any modification of the neural circuits controlling the behavior are actively suppressed and, (3) learning about the environment is dominant – what Brembs calls *world-learning*. He reports that the biochemical processes and associated molecules involved in this type of learning are those discovered in classical conditioning, e.g., adenylyl cyclases of type I, cyclic adenosine monophosphate (cAMP), and protein kinase A or PKA (Brembs & Plendl, 2008).

Brembs went on to explain that this memory of the environment is independent of the behavior that controlled the stimuli. That is, the animals can use a different, even orthogonal, behavior to press the lever (or change the color) and still keep their performance up, compared to controls (Brembs, 2009). In the case of the flies, he can test this hypothesis in a way that is impossible in the rat because if the lever is removed, there is nothing for the rat to press - if one wants to test if the behavior has been conditioned independently of the lever. In comparison, in his paradigm with flies, turning direction preference can be tested after training, with the colors turned off. At this stage, using this paradigm, the flies show no preference (Brembs, 2009).

The second thing that happens, Brembs elaborated, is that, as conditioning continues, the suppression or inhibition that keeps the behavior flexible is overcome – making the behavior more stereotyped. This means that now the behavior is not that flexible any more so a different behavior is less effective in controlling the stimuli. In the case of the flies, when Brembs removed the colors after this extended training, now they showed a preference for the previously unpunished turning direction. With this stage of conditioning having been termed *habit formation* in vertebrates, and because the fly situation appeared quite similar, Brembs co-opted this term for invertebrates as well.

In rats, after habit formation, selective devaluation of the reinforcer no longer leads to a reduction in lever pressing rate, demonstrating the behavior's inflexibility (Balleine & Dickinson, 1998; Balleine & O'Doherty, 2010; Costa, 2007, 2011; Graybiel, 2005; Yin & Knowlton, 2006). At this stage, the biochemistry underlying the recruited neuronal circuitry is completely different. However, if the type I cyclases function is blocked, then the flies can continue to learn well. Protein kinase C (PKC), instead of PKA, is important with PKC manipulations having no effect on world-learning. This type of learning modifies the behavior of the animal itself and Brembs therefore termed it *self-learning* (Colomb & Brembs, 2010).

Brembs went on to explain that, in flies, it can be showed that it is the inhibition of world-learning that slows down self-learning. Compared to learning with colors, the same experiment without colors leads to a PKC-dependent preference for turning direction requiring only half the training sessions (Brembs & Plendl, 2008).

Besides PKC, Brembs showed that the gene *FoxP* is also necessary for self-learning. He explained that this is important because the genes *FOXP1* and *FOXP2* in humans (the closest relatives to *FoxP* in flies) are associated specifically with language acquisition and other motor skills. In fact, he asserts that the *FoxP* evidence directly contradicts Chomsky's statement that language acquisition and operant learning share only superficial similarities (Mendoza et al., 2014).

Finally, Brembs described how in the marine snail *Aplysia*, he and his colleagues have isolated the neurons involved not only in learning which behavior is rewarded/punished, but also the ones controlling when the behavior is supposed to be initiated (Brembs, 2003; Brembs, Lorenzetti, Reyes, Baxter, & Byrne, 2002; Nargeot, Le Bon-Jego, & Simmers, 2009; Nargeot & Simmers, 2010, 2012; Sieling, Bédécarrats, Simmers, Prinz, & Nargeot, 2014). This research shows how operant learning modifies both the timing and the kind of behavior that will be emitted. Moreover, it provides mechanistic insight in how behavioral variability is first generated on a neuronal level and then restricted due to reinforcement. Brembs contends that these groundbreaking findings provide a roadmap for future research on the neurobiological processes underlying all forms of operant learning in vertebrates as well as in invertebrates.

Special Issue Article Overview

The following articles in this issue, by highly regarded behavioral scientists concerned with conditioning and learning, contribute to a more complete appreciation of the intersections, interactions, and commonalities, as well as the differences between operant and classical conditioning processes. As a whole, they serve to increase our contact with, and get at the essence of, what is actually happening with these contingencies in the laboratory as well as in nature. Each of the articles speaks for itself, but there are some themes, objectives, consolidations and concerns represented therein that will be briefly touched on below.

Jozefowiez in *The Many Faces of Pavlovian Conditioning* shows how included under the term "Pavlovian conditioning" are: a procedure, the learning resulting from that procedure plus the process meant to explain that learning. He is concerned that this interferes with our adequately understanding these various phenomena. In fact he shows even if one focuses at only the *process* there is behavioral as well as neural evidence supporting that it is "extremely unlikely that a single Pavlovian conditioning process is responsible for learning in all procedures classified as Pavlovian conditioning." Jozefowiez describes how the same concerns also apply to the operant conditioning situation.

Pavlovian conditioning is the name of a procedure in which a CS is paired with a US and Jozefowiez describes a wide range of preparations exemplifying this procedure. They include: Pavlov's salivary conditioning procedure in dogs, the nictitating membrane response conditioning in rabbits, fear conditioning in rats, autoshaping in pigeons and taste aversion learning. In each case, a CS is paired with a US and the CS comes to elicit a response related to the US.

Weiss' *Instrumentally Derived Incentive Motivational Functions* (appetitive and aversive) may add "faces" to Pavlovian conditioning as a procedure as well as a process. He proposes that these functions support the contention that environmental conditions being differentially correlated with operant-behavior-produced reinforcement could be responsible for most classical conditioning and resulting incentive-motive states." There was no traditional Pavlovian CS – US pairing in any of his training conditions. This can be considered an example of how learning principles derived from the laboratory can be extended to help us better understand how they are likely operating in nature. These "states" are clearly learned, anticipate the future and energize behavior.

Donahoe *is* seeking the "essentials." He proposes "... that the critical reinforcing event in both the classical and operant procedures is more closely correlated with the evocation of a *change in behavior* induced by the reinforcing stimulus than with the presentation of that stimulus itself." He goes on to describe evidence at the behavioral, as well as the neurological level, that support this consolidation. This moment-to-moment conception of the conditioning process represents an insightful integration. Weiss proposes that most classical conditioning occurs outside the laboratory through the reinforcement differences that come to exist between environmental situations as the underlying operant contingencies take hold. Might a more molar model be necessary for the "classical conditioning" produced in this manner?

In *Pavlov + Skinner = Premack,* Killeen makes Premack's principle central to all reinforcement. Essentially, Premack translated *reinforcement* from what the operant produced *per se* to the behavior the subject engaged in, after the operant created the appropriate opportunity. According to this Premack, when one's preferred behaviors are ranked from 1 to n, a behavior is reinforced by the opportunity to engage in a lower ranked one and punished by producing conditions that require one to engage in a higher ranked behavior.

Premack originally tested his principle in rather simple laboratory situations wherein he showed, for example, that children with an initial preference for pin-ball playing over gum drops, would eat gum drops for the opportunity to play pin-ball, and vice-versa (Premack, 1959). Although confirming the process, studies of this type probably also limited and constrained its application.

Killeen takes Premack out of the laboratory and uses a hierarchy of natural action patterns, adapted from Timberlake and Lucas (1989), that applies Premack's principle to complex behavioral repertoires of animals in nature. It is Killeen's thesis "...that operations that move an animal into a particular subsystem or mode [e.g., predation, defense, escape] are *motivational.* When in that subsystem, the ability to engage in an action ... will reinforce actions that lie above it ... and be reinforced by actions that lie below it in that column."

He goes on to describe how through selective breeding the English Pointer is trapped in one link of an action chain and perpetually unable to consummate the nominal goal of that action – and is concerned that our laboratory animals have regularly been stuck at local minima. Killeen clearly helps us appreciate how (a) operant conditioning, (b) naturally occurring sequences of behavior related to particular situations and conditions plus, (c) Pavlovian conditioning can be effectively integrated for a more complete understanding of-going behavior.

Staddon, in his *On Choice and the Law of Effect* (LOE), presents a simple dynamic analysis of the molar matching law and related research. He begins with cumulative records which show behavior of individulal subjects in real time – rather than thousands of aggregated responses as most "matching" experiments do. His simple law-of-effect (LOE) model easily reproduces the post-reinforcement "burst" of responses that reliably occur when animals are first exposed to intermittent reinforcement. He shows how much can be explained just by this elementary, non-temporal process.

He goes on to explain how concurrent variable-interval schedules tend to produce alternation because of the strong negative feedback that VI schedules provide – the longer one choice is neglected, the higher its payoff probability becomes. It is perhaps not surprising that a changeover delay is usually needed to get the matching relation: "The precise correspondence between relative frequency of responding and relative frequency of reinforcement broke down when the COD was omitted." (Herrnstein, 1961, p. 271).

Staddon points out that concurrent random-ratio schedules have no such feedback. Data from random-ratio schedules suggest a law-of-effect-based cumulative-effects model which assumes: (a) that reinforcement-linked response tendencies compete; (b) that the highest-strength response is the one that occurs; (c) other responses are "silent," their strength unchanged until they occur; and (d) response strengths depend on relatively long-term reinforcement history. He shows that LOE models make several interesting predictions where matching either fails or does not apply.

Staddon's discussion of VI schedules in the matching research led the author (SJW) to recall that some years ago he ran a few undergraduates on a VI schedule for several 30-min sessions as a baseline for an anticipated subsequent manipulation. When interviewed after these sessions, every student said, to the effect, "Well Professor Weiss, you stumped me. A few times I thought I figured out the sequence, like two fast and three spaced responses, but ultimately I couldn't." That humans are accustomed to solving problems could severely limit the application of findings based on paradigms without a "logical solution".

Gilroy, Everett and Delamater, in their *Response-Outcome versus Outcome-Response Associations in Pavlovian-to-Instrumental Transfer: Effects of Instrumental Training Context*, report that training two instrumental Response-Outcome (R-O) relations in distinct contexts disrupted the ability of those responses to be modulated by Pavlovian stimuli in a sophisticated multiple response, outcome and context Pavlovian-to-Instrumental (PIT) paradigm. They interpret this to support the view that instrumental learning results in the development of highly specific Response-Outcome (R-O) associations that are used in the backward direction during a specific PIT test.

Gilroy et al. go on to discuss their results in the context of the potential associations that could be learned (context-outcome, response-outcome, and outcome-response) during instrumental learning and caution that although "... we have emphasized associative mechanisms at the intersection of Pavlovian and instrumental learning that would permit for an interaction at the levels of learning and performance, it is important to realize that there very well may be important differences, as well, in the underlying associative circuitries of Pavlovian and instrumental learning that may prevent interactions from taking place in some circumstances." (It should be noted that PIT is the current manner of describing what has traditionally been referred to as the transfer-of-control (TOC) paradigm. See Weiss' article in this issue for a functional analysis of the single response, outcome and context TOC design.)

Research Seminar Session: Conditioning, Learning, Behavior and Levels of Explanation

The status of behavioral research as a scientific subject matter is well established. Yet, even though behavioral analysis is a prominent feature of various disciplines (e.g., behavioral genetics, behavioral neuroscience, behavioral biology and behavioral pharmacology), it too often continues to be regarded as relevant only because of what it tells us about other processes considered, more important such as physiology and brain function. Skinner (1938) wrote, "Facts about behavior are not treated in their own right, but are regarded as something to be explained or even explained away by the prior facts of the nervous system." (p. 4). This reductionistic tradition continues to be favored today. West and King (2001) summarized it well when they said, "… the study of behavior seems to be something someone does to get to somewhere else. Behavior affords a gateway to physiology, neuroscience, and genetics." (p. 587).

This issue of reductionism was raised at the WCALB 2014 Research Seminar Session. In many ways, the points discussed paralleled current debate in the scientific community. For some participants, it was already all said and done that behavioral phenomena could be explained completely by physiology, neuroscience, and genetics and by reducing behavior to neural networks. The behavioral explanation was questioned, since it was assumed that the more complete explanation of behavior must be found inside the black box. This position was opposed by some who asserted instead that an explanation outside the black box at the behavioral level was valid and the way to go.

However, those who have searched for explanations at the behavioral level have been accused of focusing only on behavior and ignoring the role of physiology, neuroscience, and genetics. This is unfortunate, because without those behavioral facts, physiology, neuroscience, and genetics would not have much to study. As Skinner said, "Valid facts about behavior are not invalidated by discoveries concerning the nervous system, nor are facts about the nervous system invalidated by facts about behavior. Both sets of facts are part of the same enterprise, and I have always looked forward to the time when neurology would fill in the temporal and spatial gaps which are inevitable in a behavioral analysis." (1988, p. 128). Thus, physiology, neuroscience and genetics supplement a behavioral analysis at the level of the whole organism interacting with the environment. From this perspective, facts about the organism (i.e., physiology, brain and genes) are considered part of the conditions necessary for the occurrence of behavior rather than explanations of behavior on their own. The explanation lies in the environmental contingencies, but facts from neuroscience and physiology help fill in the physical details that connect behavior to biology.

One alternative to reductionism discussed at WCALB 2014 was to acknowledge that there are different causes of behavior as proposed by Aristotle (triggers, functions, substrate and formal representation), all valid in their own right. In this view, the understanding of any phenomenon requires the understanding of all of its causes and how they interconnect. This position is in keeping with the modern anti-reductionism stance. For instance, in recent years biological scientists have moved away from the idea that biological systems can be fully explained by molecular biology and have instead favored some sort of holism or interconnected system (Van Regenmortel, 2004).

In the study of development, West and King (2001) have also recommended a systems approach that integrates the molecular and the non-molecular. Similarly, in recognition that drug-taking cannot be reduced to drug actions, those who study addictions have also called for a systems approach (see Kalant, 2009; and commentaries). A systems approach, while not easy, has the advantage of letting different disciplines pursue explanations at different levels as parts of a bigger picture. Complete understanding of behavior then comes from the coordination of all of these explanations (see, Killeen, 2001). An integrative science of behavior depends, of course, on the integrity of the levels. Errors at one level would necessarily produce errors in the explanatory system (Ruff, 2003).

West and King (2001) caution that the linkage between levels is compromised when the behavioral phenomena linked are devoid of important ecological and social variables relevant to the occurrence of the behavior in question. For example, they found that despite an elegant and thorough analysis of song-related neural structures, this analysis alone could not predict copulatory success. Instead, they discussed that they "... have taken a more direct approach to understanding the task of song development by focusing on its goal, successful courtship." (p. 597). As researchers move toward looking at behavior in its ecology, the focus is shifting away from the structure of discrete behaviors and instead toward examining the environment in which the behavior occurs and the consequences produced.

The distinction between structural and functional analysis points to one difficulty of integrating facts about behavior with other levels of analysis. Before behavior can be linked to other levels of observation or even reduced to one of those levels, there needs to be agreement about what is being reduced or integrated. Physiology, neuroscience and genetics have traditionally relied on a structural analysis of behavior rather than a functional one. A bird's song no doubt has a biological structure that can be mapped to other structures and functions of the nervous system. However, focusing only on structure takes away from the environmental variables that are part of the behavior. For example, a bird song could be one behavior when it is directed toward females, yet the same song could be a different behavior when directed toward males or when emitted in the absence of conspecifics. Although the song remains the same, the interaction with the environment produces different consequences. From a behavior analytic point of view, explanations are concerned with stating functional relations between behavior change and changes in the antecedent and consequent environment. These functional relations between behavior and environment create analytic units that make possible the study of behavior.

At WCALB the issue of structural and functional analysis of behavior was also discussed but at a more basic level: The level of the stimulus and response. Participants debated whether it is possible to talk about a stimulus without reference to a response or a response without reference to a stimulus. This question was new to the neuroscientists in the discussion, who were more familiar with defining stimuli and responses with respect to their structural and physical characteristics only. From this approach, the properties of the response can be defined completely by the field of biology and the properties of the stimulus by the field of physics. For the behavior scientists, however, the stimulus and response are two factors that interact to make up their subject matter: behavior. The response by itself does not constitute a behavioral event, even though it can be defined as a more or less fixed pattern of neural, muscular and glandular events all the way down to its molecules. The stimulus must also enter into the definition of behavior and it is this connection between stimulus and response that gives rise to behavioral phenomena. While behavior scientists begin with the physical description of the stimulus and response, just as biologists and physicists do, these descriptions are insufficient to characterize the interactions that behavior scientists study.

Another question raised during the discussion was what to call a stimulus that is not connected to a response or a response that is not connected to a stimulus. It was suggested to call a response not connected with a stimulus an *action*. This led to the next question, is there such a thing as a response without a stimulus? For the determinists in the room, that could not be the case. If there was a response, they would search for the stimulus. This was considered an error by the neuroscientists because it would prevent the search for internal mechanisms of action. However, the determinists objected to dissecting the stimulus from the response. Behavioral phenomena are continuous. A response leads to a stimulus which in turn leads to another response and so on, as Killeen's article in this issue describes. This continuous nature of behavior is central to the analysis of behavior.

The discussions at WCALB were very productive and stimulating. They reflected the scientific scene in general, in that an integration of the various disciplines is a continuous and ongoing challenge. It became clear that in order to have an integrative science of behavior more discussions are needed regarding the basic assumptions of the various disciplines concerned with explaining behavior. What is a stimulus, what is a response, what is the nature of the interactions that the different disciplines consider important? The mingling of behavior scientists and neuroscientists at WCALB was a good step toward addressing these questions. The meeting ended with a reminder that "criteria" is plural. We all need to be mindful of the other's perspective.

The video of the Research Seminar Session can be found at:

https://www.youtube.com/watch?v=usCYJy9Pl9Q&list=UUIq9EAG5h9yc25rYSjywNMw.

We want to thank Scott Cohn for recording this video.

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Behaviorism

Learning Theories: Behaviorism

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In its simplest form, learning is defined as gaining knowledge through study, teaching, instruction, or experience.[1] Interestingly, learning is described and viewed differently by theorists, researchers, and practitioners who have spent time investigating and experimenting in the educational psychology field.[1,2] The differences in how educational theorists believe individuals acquire, retain, and recall knowledge resulted in the development of multiple learning theories.[1-3] Based on the context of the theorists' work and other factors at the time of investigation, these theories explain how learning occurs, what internal or external factors influence learning, how memory affects learning, and how transfer of knowledge occurs.[1-3] In addition, the roles of the instructors and learners are described according to each theory of learning. A basic understanding of the various learning theories is essential for educators who strive to lead a classroom that is conducive to learning and success.

The ideas of behaviorism date back to the late 19th and early 20th centuries when John Watson, an American psychologist, believed the general public would accept and recognize the new philosophy of psychology as a true science only if it involved processes of objective observation and scientific measurement.[1] This notion of detailed observation and measurement became central to the work of behaviorists.[1]

Behaviorism emphasizes that learning occurs when an individual responds favorably to some type of external stimuli.[1-4] Behaviorism sometimes is referred to as the *stimulus-response theory*.[1] For example, when presented with a math flashcard showing the equation 6 × 8, the learner responds with the answer 48. The equation is the stimulus, and the answer is the associated response.[2] Essential elements with behaviorism include the stimulus, the response, and the association between these 2 elements.[2] Of particular importance is how the association between the stimulus and the response is made, strengthened, and maintained.[2]

Behaviorists define learning as nothing more than the acquisition of new behaviors. Behaviorists do not emphasize thinking or other mental activities as a part of the learning process because such variables are not observable behaviors.[1-4] Although the behaviorism theory discounts any mental activity, other educational theorists considered these processes to be a vital part of learning and cognition, which resulted in the development of other theories of learning.[1,4] Behaviorists do not address memory and how new behaviors or changes in behaviors are stored or recalled for future use.[2] Behaviorists refer to this type of learning, where a reaction is made to a particular stimulus, as *conditioning*.[1] Two main types of conditioning include Pavlov's classical conditioning and Skinner's operant conditioning.

Classical Conditioning

Ivan Pavlov, a Russian physiologist, noticed that dogs salivated every time they ate or saw food and believed he could condition the dogs to salivate at the sound of a bell.[1] Initially, Pavlov sounded a bell at the time food was presented to the dogs and repeated this process frequently.[1] Eventually, the sound of the bell became an indication to the dogs that food was about to be presented, and they responded by salivating at the sound of the bell regardless of whether food was presented.[1] This type of reinforcement of a natural reflex or some involuntary behavior that occurs as a response to a particular stimulus is called *classical conditioning*.[1] Pavlov was able to condition the dogs to salivate in response to the sound of the bell.

Pavlov identified 4 stages of classical conditioning: acquisition, extinction, generalization, and discrimination.[1] The acquisition stage is the initial learning of the conditioned response (the dogs salivating at the sound of the bell).[1] Pavlov believed the conditioned response would not remain indefinitely, so he used the term *extinction* to describe the disappearance of a conditioned response.[1] Pavlov demonstrated extinction by repeatedly sounding the bell without presenting food to the dogs.[1] The final 2 stages, *generalization* and *discrimination*, are opposites and explain how behaviorists believe knowledge is transferred within learners.[2] The generalization stage implies that a conditioned response might occur with similar stimuli without further training (the dogs salivating at the sound of something similar to a bell).[1] In contrast, the discrimination stage indicates that a conditioned response might occur with 1 stimulus but not with another (the dogs not salivating at the sound of something similar to a bell).[1]

Operant Conditioning

BF Skinner, a psychologist working in the United States in the 1930s, established the theory of operant conditioning: a process of reinforcing a voluntary behavior by rewarding it.[1,3] Studying the behaviors of rats, Skinner used a device (now called a *Skinner box*) that contained a lever.[1] Whenever the rats pressed the lever (an action Skinner considered normal, random, and voluntary), a pellet of food was presented.[1] As the food rewards continued during the repetition of the action, the rats learned that they had to press the lever to be fed.[1] Skinner also used reinforcement techniques to teach pigeons to dance and to roll a ball down a mini bowling alley.[1] Skinner made generalizations about his findings with rats and pigeons to humans.[1] In addition, he noted that operant conditioning also worked in a negative way: stopping a behavior from occurring by punishing it.[1,3]

Reinforcement and Punishment

Key aspects of operant conditioning include reinforcement and punishment, both of which can be positive or negative. Reinforcement refers to anything that has the effect of strengthening a particular behavior for it to occur again.[1,3] Positive reinforcement is the addition of a rewarding stimulus to get the behavior to happen again (eg, rewarding learners for making a high grade on an exam in hopes they study harder for future assessments and score high again). Negative reinforcement is the removal of an unpleasant stimulus to get the behavior to continue (eg, students learning the rules to solve a particular problem so their instructor quits nagging them about the importance of it). The unpleasant behavior of the instructor's nagging is removed when students learn the rules, solve the problem correctly, and continue the action so the nagging does not return.

Conversely, punishment refers to anything that has an effect of lessening or discouraging a particular behavior so that it does not occur again.[1,3] Positive punishment is the addition of an unpleasant stimulus to get the behavior to stop; any type of disciplinary action is considered positive punishment. Negative punishment is the removal of a rewarding stimulus to get the behavior to stop (eg, not offering extra credit opportunities in hopes the behavior stops so that the learners can receive these beneficial opportunities in the future). Skinner maintained that rewards and punishments control most human behaviors.[1-3]

In addition to Watson, Pavlov, and Skinner, other theorists were associated with the behaviorist movement. The **Table** summarizes their contributions to the theory of behaviorism.

Implications in Teaching and Learning

Behaviorists believe learning begins when a cue or stimulus from the environment is presented, and the learner reacts to the stimulus with some type of response.[1-3] Those responses are reinforced or punished, Table

Key Theorists and Their Contributions to Behaviorism[1]

Theorists	Contribution
Ivan Pavlov	Classical conditioning
Edward Thorndike	Connectionism (emphasized the role of experience in the strengthening and weakening of stimulus-response connections)
John Watson	Scientific objectivity; Law of frequency (the more frequent a stimulus and response occur in association with each other, the stronger the habit will become)
Edwin Guthrie	Contiguity (the same response to a stimulus most likely will occur over and over again during repeated exposures)
BF Skinner	Operant conditioning

and this process is repeated so that the responses become automatic.[3] Ultimately, the change in behavior indicates learning has occurred.[3] As revealed, behaviorism has little regard for mental processes or understanding and, therefore, does not prepare learners for problem-solving or critical-thinking skills.[1-3]

The instructor plays a dominant role in behaviorism by leading the learning environment, using positive and negative reinforcement to shape learners' behaviors, and presenting the content.[1] With behaviorism, learners are described as passive individuals who voluntarily respond to external stimuli.[1] Other behaviorist implications in teaching and learning include[1]:

- creating procedures and expectations to manage the classroom
- using rewards as incentives for learners to work hard and behave
- using punishments (eg, loss of privileges or withholding of rewards) effectively and sparingly to change learners' behaviors

Critics of behaviorism argue that rewards can belittle or demean a learning experience and, therefore, should be used with caution.[1] Often, rewards can evoke feelings of unfairness or competition, and some learners might become distracted from the real issue involved in completing a task or learning new material.[1] Using a rewards system or giving 1 learner increased attention might have a detrimental effect on others in the class or cause them to feel isolated.[1] Not surprisingly, rewards do not always lead to higher-quality work; however, using a behaviorist approach, rewards can result in the reinforcement of appropriate classroom behaviors, which can create a more orderly classroom environment that is conducive to learning and success for all.[1]

Learning Activities

Classroom learning activities connected to the behaviorism theory include[1-3]:

- lecturing
- recalling facts
- defining and illustrating concepts
- applying explanations
- participating in rote learning (ie, memorization based on repetition)
- completing drill and practice exercises
- establishing classroom management policies
- using rewards and punishments

Implications in Medical Imaging Education

In medical imaging education, lecturing is a dominant approach to presenting information because of the complexity of the content. Considering time management issues and restrictions in higher education, lecturing affords instructors an opportunity to present a large amount of information to a large audience. Often, medical imaging students memorize some of the content presented and recall that knowledge during an exam. The role of repetition aids in the learning of new and challenging content. Medical imaging students benefit from drill and practice exercises when working with formulas, including the Inverse-Square Law, the milliampere-seconds–distance compensation formula, and the grid conversion formula, as well as calculations involving skin dose. Medical imaging instructors benefit from using a behaviorist approach by implementing a classroom management plan to lead a classroom conducive to learning and success.

Conclusion

The theory of behaviorism can be illustrated by the adage, "practice makes perfect." Behaviorists see learning as an observable change in behavior as a result of experience and repetition. This stimulus-response theory makes no attempt to assess the mental processes necessary for learners to acquire, retain, and recall information. The change in behavior is simply achieved through a conditioning process using reinforcement and punishment. Even though little importance is placed on mental activity, concept formation, or understanding, there is a place for behaviorism in today's classrooms, especially in medical imaging education, in the areas of rote learning and classroom management.

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Theoretical Behaviorism, Economic Theory, and Choice

John Staddon

Behaviorism

Psychology is not a single science. There are—alas—many psychologies. Behaviorism is one of them, and "theoretical" is one kind of behaviorism.[1] Economics also is far from unified. To understand the relation between psychology and economics, it might be best to begin with one psychology and one economics, rather than attempt a review of two highly various fields. I compare two ways to look at choice behavior: *theoretical behaviorism* and *prospect theory*, a popular approach to behavioral economics as it has been presented in Daniel Kahneman and Amos Tversky's seminal 1979 paper and later writings.

What is *choice*? Charles Darwin wrote copious notes, two months before his engagement to his cousin Emma Wedgwood, listing the pros and cons of marriage.[2] A sample:

> *Marry* [pros]:
> Children—(if it Please God)—Constant companion,
> (& friend in old age) who will feel interested in one,—

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[1] For a review of the behaviorisms, see Staddon 2014a. For one attempt to apply theoretical behaviorism to the financial system, see Staddon 2012. An early effort to bring together psychology, biology, and economics is a collection of papers in Staddon 1980.

[2] See www.darwinproject.ac.uk/darwins-notes-on-marriage.

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> object to be beloved & played with.—better than a
> dog anyhow.—Home, & someone to take care of
> house—Charms of music & female chit-chat.—These
> things good for one's health.—but *terrible loss of*
> time . . .
> *Not Marry* [cons]: Freedom to go where one liked—choice of Society
> & little of it.—Conversation of clever men at clubs—
> Not forced to visit relatives, & to bend in every
> trifle.—to have the expense & anxiety of children—
> perhaps quarrelling—Loss of time.—cannot read in
> the Evenings—fatness & idleness . . .

There is much more, but you get the idea. This is choice indeed! No one would imagine that animals choose like this. Indeed, they do not, and for the most part, people do not either. So, I begin with the simplest possible choice situation. The two-armed bandit is popular not just in Las Vegas but among operant conditioners, those who study learned instrumental behavior. The subjects are usually animals—more easily, and ethically, subjected to experimental conditions than human beings and probably easier to understand. Pigeons and rats often seem to follow elementary economics. They are *rational*, in the sense that they maximize the rate of reward. For example, a hungry pigeon in a Skinner box randomly paid off on average for one in every ten pecks (termed a *random-ratio* schedule) on a response key on the left and on average for one in every five pecks for pecking on the right—such a pigeon will soon peck only on the right. It *maximizes.*

Indeed, optimality theory can explain many characteristics of the operant behavior of animals, of the ways they adapt to different kinds of *reinforcement schedules*. But animals also fail to act rationally, even in some strikingly simple situations.[3] The failures are more interesting than the successes. Sticking with ratio schedules, consider a *fixed*-ratio schedule that requires exactly one hundred pecks for each food reward. A pigeon will soon learn to get food this way. But he will always wait a second or two after each reward before beginning to peck, thus delaying food delivery unnecessarily. Fatigue? No; on a comparable *random* ratio, he will respond steadily, not waiting after food.

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[3] See, e.g., Staddon 2007 and references therein. For a comprehensive treatment, see Staddon 2016.

The reason he waits is that he has a built-in, automatic timing mechanism that responds to the minimum time between food deliveries enforced by the fixed (but not the random) ratio by pausing after each food. It is easy to show this by comparing two kinds of time-based schedules. Suppose you start a sixty-second timer after each food delivery and do not reward a peck until sixty seconds have passed (a fixed-interval schedule). As you might expect, once the animal has learned, he waits perhaps thirty seconds after each reward before beginning to peck. If his time sense were perfect, no variability, he would presumably wait exactly sixty seconds so as not to waste pecks. The pigeon's timing is a bit variable, so he usually starts early. Perfectly rational: do not waste pecks, but do not delay food unnecessarily. Now suppose you modify the procedure slightly by starting the sixty-second timer only after the first peck. The rational adaptation is simple: respond immediately after food, and then wait as before. But animals do not do this. If the interval duration is *T* seconds and the typical wait time is *T*/2, then on this response-initiated-delay schedule they will wait not *T*/2 but *T* seconds, delaying reward quite unnecessarily.

The message: optimality theory is of limited use in understanding what these animals are doing. Animals never, and humans almost never, are *explicit maximizers*, computing marginals and choosing accordingly, as in some interpretations of rationality. They adapt via built-in processes, like timing, that sometimes yield "rational" behavior and sometimes do not. Behavioral ecologists call them "rules of thumb" or (a familiar term to decision theorists) heuristics.

The time dimension adds complexity, and the way in which time is incorporated into learned behavior is still not perfectly understood. So, let us stick with random-ratio schedules, where time is irrelevant. A choice situation where there is no rational strategy or, to put it a little differently, all strategies are rational is choice between two *identical* random ratios. What do pigeons do and how might theoretical behaviorism explain it? The explanation follows the Darwinian consensus: instrumental learning is selection and variation. There is a repertoire of response tendencies—candidates. Candidates could be simple responses, heuristics, or what Gerd Gigerenzer calls an "adaptive toolbox" (Gigerenzer and Selten 2001). But only one response occurs at a time. In the example I am about to give, these candidates compete in a nonlinear, winner-take-all way, so that only the strongest tendency, the *active* response occurs. The other responses are *silent*—like recessive genes. The active response is either strengthened (if it is followed by reward) or diminished in strength (if there is no reward). Notice that the variation-selection approach allows for individual differences. Different people will come to a given situation with different repertoires and different active responses.

Many dynamic processes have been proposed to describe how response strength increases after reward and decreases after nonreward. The one we have found that best combines simplicity and generality incorporates the organism's history of both reward and responding. It is not the last word.[4] Nevertheless, it shows how a simple moment-by-moment process can yield complex and often adaptive behavior. I show later that prospect theory, although not a theory in the same sense as the CE model—it is classification of the data, not a predictive model—nevertheless implies a similar underlying framework.

The assumptions of the *cumulative effects (CE) model* are as follows:

1. In a given choice situation, there is a repertoire of *candidates* (responses).
2. Each response has a certain *strength*, which is given by the following, equation 1:

$$X_i = \frac{\Sigma R_i + R_i(0)}{\Sigma x_i + x_i(0)}$$

where X_i is the strength of response i, R_i, and x_i are the rewards received and i-responses made; $R_i(0)$ and $x_i(0)$ are initial conditions. In words, the strength of each response tendency is just the cumulated payoff probability for that response, biased by prior reward and response totals. The learning rule is very simple: a response not followed by reward increases the denominator and thus reduces X_i; a reward increments the numerator proportionately more than the denominator and thus increases X_i.

3. Response tendencies compete to become active in winner-take-all fashion.

This simple model behaves rationally in choosing between two options that pay off with different probabilities: it will eventually fixate on the higher-probability choice.

More surprising is that it can duplicate pigeons' performance on successive-reversal learning (Davis et al. 1993; Staddon 2014b). In these

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[4] For one thing, it is deterministic, not stochastic. The randomness in my example is provided by the schedule. On fixed-ratio schedules, the result is usually indifference, which is probably not correct—although the necessary experiment does not seem to have been done.

experiments the pigeon chooses between two keys, as in the previous example. On day 1, pecks on the left key (say) are paid off randomly with probability 1/8; the right key is not paid off. On day 2, this is reversed: right *p*(reward) = 1/8, left *p*(reward) = 0.

And so on for many days: left and right rewarded on alternate days.

Unsurprisingly perhaps, pigeons improve over days, switching their preference faster and faster each day. This is called "reversal learning set," and many cognitive explanations have been offered for it. But the CE model can duplicate the effect because, over successive reversal days, the *X* values of the two choices get closer and closer so that switching from one to the other (because of the winner-take-all rule) gets faster and faster. No need to postulate a "set" or anything beyond simple learning.[5] The same model also predicts, correctly, that reversal performance will improve less if reversals take place every four days rather than every day.

So what does the CE model (and many models like it) predict should happen when the animal must choose between two *identical* ratio schedules? Maximization makes no prediction because the animal's choice has no effect on payoff. A version of H. A. Simon's satisficing[6] might predict random fixation on one side, given that switching entails some small cost, but that is not what happens.

Figure 1 shows a simulation of the CE model in this situation and some data (Horner and Staddon 1987).[7] The model predicts, and the data show, that the animal's behavior depends on the absolute value of the payoff probability. If it is high, the animal tends to fixate (randomly) on one or other choice; if it is low, it tends toward indifference.

Gordon Tullock (1971), who was one of the first economists to look at biological problems from an economic viewpoint, proposed something like this in his nifty little note "The Coal Tit as a Careful Shopper": "Assume that the bird, like any other food-consuming animal, explores a number of different food sources [pine cones with grubs, in this case], and then settles on the one which provides the most food with the least energy expended. Since it would tend to exhaust the food supplies in that particular area, it should be 'programmed' in such a way that it periodically investigates different areas." The CE model adds dynamics plus the prediction that the bird turns sooner to "periodical investigation" when it pecks a

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[5] Pigeons never show *spontaneous reversal*—switching on the first response each day. They always require a few unsuccessful responses to yesterday's "hot" key before switching.

[6] See en.wikipedia.org/wiki/Satisficing.

[7] For an extensive theoretical analysis of this and related situations, see Staddon 1988.

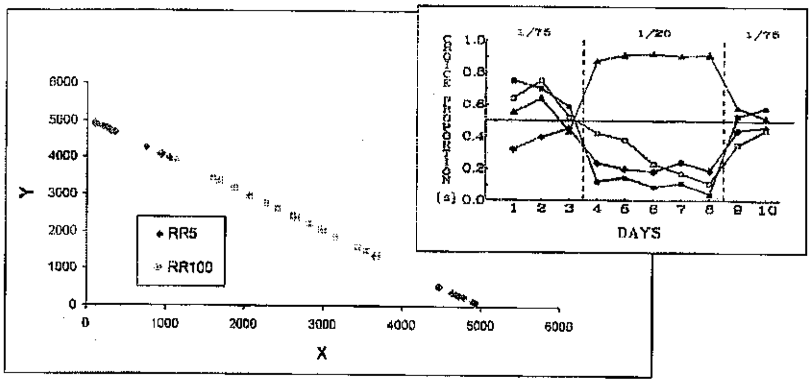

Figure 1. The pattern of choice between identical schedules as a function of absolute reinforcement rate. *Note*: CE model simulation: Two identical random ratios, 5 and 100. Preferences after twenty runs each of 4,993 time steps. Graph plots total responses: X vs. Y for two ratios: 5 and 100. Exclusive choice is favored at the smaller ratio (diamonds), indifference at the large value (squares). Initial values in Equation 1: $R_L = R_R = 5, x = y = 10$. Data: The figure plots the proportion of choices of the right-hand alternative across daily sessions for each of four pigeons for two equal-random-ratio conditions, $p = 1/75$ and $p = 1/20$, in ABA sequence (Horner and Staddon 1987, fig. 1). In both simulation and data, exclusive choice is favored at the high probability, indifference at the low.

relatively grub-free pine cone. This in outline is how one theoretical school of psychology thinks about the processes that underlie choice. How does prospect theory treat problems like this?

Human Choice

Animals must actually experience the different payoffs to arrive at a stable preference. Not so for people: you can just ask them (Kahneman 2011).[8] Consequently, the study of human choice behavior has developed along very different lines from the study of choice in animals. Nevertheless, I show that both can be conceived in a similar way.

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[8] But of course what they do when they actually experience outcomes may be very different from what they say in advance they will do. See, e.g., Ludvig, Madan, and Spetch 2014. Portions of this section, by the way, appear in Staddon 2016.

The study of human choice differs from animal choice in four main ways:

1. People begin with a stock of *wealth*. There is no real equivalent in animal experiments.[9]
2. There is no animal equivalent to *loss*.
3. Individual differences: *There is rarely unanimity in choice experiments with people*. Even when there is a statistically significant preference, 30–40 percent of subjects may deviate from the majority choice. In most animal choice experiments the effects are reversible (see figure 1), so the result can be replicated with the same subjects and no statistics are required. In practice, close to 100 percent of subjects give the same result.
4. Human choice patterns can be changed by experience: risk aversion can be changed to risk seeking by appropriate training or prompting,[10] for example. In most animal studies, choice is stable.

Because people will give immediate answers to many hypothetical choice questions, and because their decisions can often be accurately anticipated, hypotheses can be tested quickly. This strategy was adopted by two of the leaders in this field, Daniel Kahneman and the late Amos Tversky. They posed questions to themselves, and based on their own answers (which were later checked with groups of human subjects), they quickly found flaws in *utility theory*, the standard explanation in economics.

Prospect Theory

Daniel Bernoulli in the eighteenth century pointed out that the utility of a given increment in wealth is inversely related to the amount of wealth you already have. A hundred dollars is worth much more to a pauper (net wealth: $5) than to a millionaire.[11] Anticipating a now well-known principle in psychophysics, the Weber-Fechner law, Bernoulli proposed that the relationship between wealth and utility is logarithmic, so that equal

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[9] One experiment has tried, with some success, to induce monkeys to treat a stock of tokens as wealth: see Lakshminarayanan, Chen, and Santos 2011. This effect fits in with the more general analysis I present here, but it does suggest that monkeys are smarter than pigeons.

[10] Experienced traders show fewer biases than novices; for example, see Kahneman 2011.

[11] Comparing utility between individuals is epistemologically flawed, of course, even though it is the basis for the idea of social justice. We can verify that person A prefers X to Y and person B the reverse. We cannot say whether A values X more than B does. After all, the millionaire may be one just because he values a dollar, any dollar, more than does the pauper. Bernoulli's assumption is verifiable for individual utility but not for interpersonal comparison.

ratios correspond to equal value. A pay raise of \$1,000 is worth as much to an employee whose base pay is \$10,000 as a raise of \$10,000 to one who makes \$100,000.

The standard utility function is negatively accelerated, which means diminishing marginal utility. Because each increment of value adds a smaller increment of utility, people will generally be *risk averse* with respect to gains because doubling a reward less than doubles its utility. For example, in a typical experiment (Problem 3 in Kahneman and Tversky 1979),[12] ninety-five people were asked to choose between two outcomes: 4,000 with probability 0.8, versus 3,000 for sure. Eighty percent chose the sure thing, even though it is less than the expected value of the gamble: $3{,}000 < 3{,}200$. Apparently, 0.8 times the utility of 4,000 is less than the utility of 3,000, $0.8 \times U(4{,}000) < U(3{,}000)$, because the utility function is negatively accelerated.

Yet expected value alone explains Problem 4, where 65 percent of the same ninety-five people preferred a 0.2 chance of 4,000 to a 0.25 chance of 3,000: in expected values, 800 was now preferred to 750—no sign of diminishing returns there. Evidently *certainty* has an effect that goes beyond the probability of one.

There is another way to look at Problem 3. The "3,000 for sure" option is guaranteed. It increments your current state of wealth, *unless* you choose the risky option, in which case, with probability 0.2 (the chance you *do not* win the 4,000), your wealth could go down by 3,000, a *loss*. So the issue becomes the following: is $|0.2 \times U(-3{,}000)| > U(4{,}000 - 3{,}000)$, that is, is 0.2 times the cost of losing 3,000 (i.e., actual expected value 600), greater than the benefit of a net gain of 1,000? And the answer is yes if the origin of the utility curve is shifted up by 3,000 on the utility axis (i.e., 3,000 is subtracted from each utility). In this case the disutility of a loss of 600 is greater than the utility of a gain of 1,000. But such a shift requires a further assumption about how people *perceive* a choice situation. The shift of origin is not really required for Problem 3, since the all-positive utility curve works as well, but it is necessary to account for other results.

Data like these led Kahneman and Tversky to propose *prospect theory* as a replacement for standard utility theory. Prospect theory is a

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[12] The example is Problem 3; amounts are in Israeli currency. This version of prospect theory was elaborated in Tversky and Kahneman 1992, and there is a vast secondary literature. See also Kahneman's readable book *Thinking Fast and Slow* (2011) and his Nobel Prize address: www.nobelprize.org/mediaplayer/?id=531.

hybrid. The "front end" is what they term *editing*: "Prospect theory distinguishes two phases in the choice process: an early phase of editing and a subsequent phase of evaluation" (Kahneman and Tversky 1979, 274). Editing is their name for cognitive processes that set up the problem for the *evaluation* part of the theory. Editing is a process, but it is not defined in any calculable way. Terms such as framing, combination, segregation, isolation, coalescing, and cancellation have been used to label operations that the decision maker can use to simplify and structure the choice problem so that it can be evaluated. Exactly when these processes are invoked and precisely how they work are not specified by any economic theory.

The core of prospect theory is the second phase: *evaluation*, which is a modified version of expected utility theory. Evaluation is a teleological/functional theory; it describes an outcome but not the process the system uses to achieve it. Evaluation, according to prospect theory, modifies utility theory in three ways:

1. The idea of a *reference point* or adaptation level, which is set by the subject's current state of wealth.[13] The reference point moves the origin of the utility curve to a point representing the subject's current wealth. A choice option that yields a state of wealth less than the reference point is perceived as a loss. The idea that people are sensitive to changes rather than absolute values is a core principle of perceptual psychology.
2. Thus, the upper-right quadrant of the utility graph is the same as before. But the lower left part is modified, to deal with the following results involving losses. There are three steps; the last two require changes in the standard form. First, most people will choose a 50 percent chance of losing $100 only if the other possibility is a win of $200 or more: "People tend to be risk averse in the domain of gains and risk seeking in the domain of losses" (Kahneman 2011, 344). Thus, the first part of the southwest quadrant must be steeper than the first part of the northeast. This requires no big change in the shape of the standard Bernoulli utility graph, which gets steeper as it approaches the origin.

But, second, 65 percent of subjects preferred a 0.2 chance of winning 4,000 over a 0.25 chance to win 3,000 (expected values: 800 > 750;

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[13] See also Markowitz 1952.

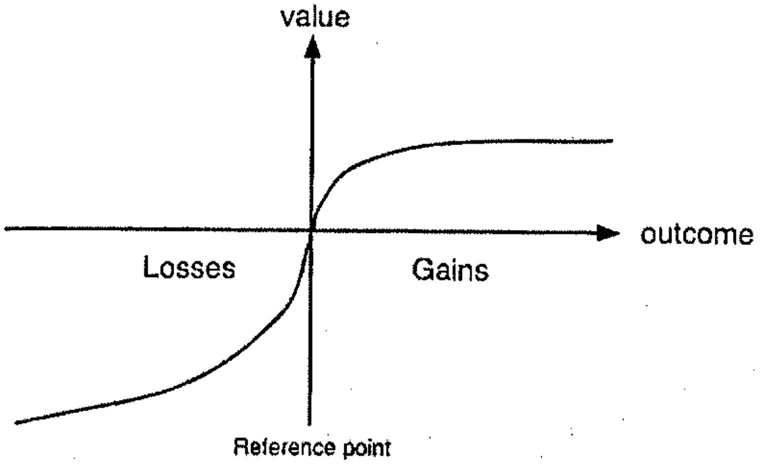

Figure 2. Prospect theory utility function

Problem 4), that is, they were in line with expected value theory and not risk averse. So the curve must straighten out at intermediate probabilities. And finally, 86 percent of people preferred a 0.9 chance to win 3,000 over a 0.45 chance to win 6,000 (Problem 7), even though both gambles have the same expected value. Now subjects showed risk aversion. Hence the curve in the southwest quadrant must begin steep, then straighten, and then flatten out. This is the iconic prospect theory utility graph (see figure 2: Kahneman and Tversky 1979, fig. 3).

3. Gambles at extreme odds do not fit even this modified utility graph. For example, 73 percent of people prefer a .001 chance to win 6,000 over a .002 chance to win 3,000 (Problem 8), even though the expected values are the same, that is, back to risk seeking.

Another exception is provided by something called probabilistic insurance. Suppose that you are indifferent about whether or not to insure your house against earthquakes. A creative salesman then makes you this offer: we are willing to insure you for just 45 percent of the original premium if we are allowed to toss a coin after an incident to decide whether we pay you or just refund your premium. Obviously this is a better deal than the original one, which you were on the point of accepting. Yet people will usually reject it. Not that they are against probabilistic insurance on principle. As Kahneman and Tversky point out, the decision to quit a risky habit like smoking is a form of probabilistic insurance.

These problems cannot be solved through changes in the utility curve alone. To fix them, Kahneman and Tversky (1979, fig. 4) introduce a somewhat untidy transformation of probabilities ("π is not well-behaved near the end-points"), termed *decision weights*, that parallels Bernoulli's transformation of value. Evidently both the utility curve and the probabilities must be transformed to accommodate data.

A Common Framework

Attempts to quantify all these modifications require four or even five fitted parameters.[14] That is just too many for a useful scientific theory. John von Neumann, genius, computer pioneer, and coinventor of game theory, famously remarked: "With four parameters I can fit an elephant, and with five I can make him wiggle his trunk." Prospect theory has evolved into a set of descriptions, not a predictive system. Nevertheless, as I will show, the underlying concepts can be related in an interesting way to choice in nonhuman animals.

Human choice behavior is affected by many things: not just the numbers you are presented with but your state of wealth; your experience, both recent and historical; the kind of goods on offer—money or things, for example—the context in which the task is presented; and your willingness to think through the options.

Kahneman and Tversky tackle this complexity by pointing out that prospect theory deals only with decisions that are made quickly. They distinguish between fast and slow cognitive processing,[15] what Kahneman calls System 1 (fast, thoughtless) and System 2 (slow, reflective). Presumably, the rapid process is simpler to understand than the slow one.

Their scheme is not as different from the analysis of animal choice behavior that I presented earlier as you might think. Consider three aspects of prospect theory: the role of consciousness, framing, and the fast-slow distinction.

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[14] See, e.g., Wilkinson and Klaes 2012, 163.

[15] I think that the fast-slow distinction is actually due to Kahneman, but because Tversky would probably have agreed had he lived, and because prospect theory is identified with both of them, I use both names.

Prospect theory is usually considered a cognitive, as opposed to a behavioristic, system. Yet the role of consciousness, especially in the fast system, is minimal: "The mental work that produces impressions, intuitions, and many decisions goes on in silence in our mind"; and "Studies of priming effects have yielded discoveries that threaten our self-image as conscious and autonomous authors of our judgments and our choices" (Kahneman 2011, 4, 55). Compare this with the following comment by a behaviorist: "Astrophysicists now tell us that more than 80 percent of the matter in the universe, so-called *dark matter*, cannot be observed. The unconscious is the dark matter of psychology. Its processes are responsible for all creative activity and most recollection" (Staddon 2014a, 9). The autonomy of the unconscious is not even a very new idea. Well before Sigmund Freud, the eccentric genius Samuel Butler (1835–1902) referred frequently to the unconscious in his posthumously published novel *The Way of All Flesh*, which is in effect an autobiographical study of his own psychological development.

Cognitive psychologists are admittedly more interested than behaviorists in consciousness. But the contemporary view, like Butler's, is that consciousness is not an active agent but something like a workspace, which "allows the novel combination of material" (Baddeley 2007). Let us agree, therefore, that cognitivists and behaviorists no longer differ greatly on the role of the unconscious.

Framing is the term Kahneman and Tversky give to the effect of context and the way a problem is presented on the response that is made. The term does not occur in the 1979 paper but, with a number of other labels such as nonlinear preference and source dependence, was made necessary by new data that did not fit the original formulation. An example of framing is "The statement that 'the odds of survival one month after surgery are 90%' is more reassuring than the equivalent statement that 'mortality within one month of surgery is 10%.' Similarly, cold cuts described as '90% fat-free' are more attractive than when they are described as '10% fat.' The equivalence of the alternative formulations is transparent, but an individual normally sees only one formulation, and what she sees is all there is" (Kahneman 2011, 88). The way in which a question is asked (this is also sometimes termed *choice architecture*) can have a huge effect on the subject's response.

There is a counterpart to framing in animal choice. It grows out of the consensus that learning in animals is best conceived as a process of variation and selection, that is, selection by consequences, and variation constrained by the situation:

> For example, Pavlovian conditioning, which allows a neutral stimulus to acquire signal properties, will itself give rise to a repertoire of reinforcer-related activities from which operant reinforcement can then select. A stimulus associated with food, or food by itself, will induce a wide range of food-related activities in a hungry animal—activities from which operant contingencies can select. Pigeons peck, chickens peck and scratch, raccoons manipulate. Pavlovian conditioning, with (say) a food US [unconditioned stimulus], in effect frames or *labels* the context as food-related. The label then limits the emitted behavior to a food-related repertoire, which is defined partly by past history but also by the organism's evolutionary history. (Staddon 2014a, 92)

The situation-induced behavioral repertoire of animals is constrained—framed—in much the same way as human choice behavior. Sometimes "Pavlovian framing" will facilitate learning—if the desired response is within the induced repertoire; but sometimes it will not—as when the induced behavior interferes with the trainer's target behavior (so-called instinctive drift).

Note that framing in this sense means that all rewards are not equivalent—as most economists seem to assume. The repertoire induced by money will be different from the repertoire induced by love of ideas or a wish to cure patients. Consequently, paying teachers or doctors more will not necessarily produce better teaching or medical care.

Finally, let us look at Kahneman's fast-slow distinction. He contrasts the quick answers he and Tversky got to their choice questions—answers that were often "irrational" in the sense that they did not maximize gain—with the slower and more "correct" answer that most people arrive at after deliberation. The quick system responds with answers that are more "available" or "accessible" than other responses that may in fact be better. Kahneman (2011, 415) also tells his readers to "remember that the two systems do not really exist in the brain or anywhere else." They are ways to talk about the fact that people may respond one way at first and another way after given some time for reflection.

But there is no difference between Kahneman's System 1 and System 2 and what I earlier called "active" and silent" responses. The active response is the first thing to occur in a given situation. But if the consequences are unsatisfactory, the active response is eventually supplanted by another response from the "silent" repertoire—Kahneman's "slow" system. Individual differences reflect available response repertoires of different individuals—differences that reflect different histories and propensities. Human responses are more complex than key pecks, of course. They are heuristics, rules of thumb and analytic strategies, the "adaptive toolbox." But there is no reason to doubt that they are emitted and selected by consequences, just like the simpler response of pigeons.

The economic approach has been looking for utility functions and decision weights that best fit a set of quite variable choice data. Since these data usually depend on historical factors that are not part of the analysis, this is likely to be an endless quest. A symptom of the difficulties this causes is that many very clever people have puzzled over the fact that human behavior often violates logical axioms such as transitivity and independence.

Transitivity is simple: if A is preferred to B and B to C, then logic says that A must be preferred to C—but often people disagree.

Independence is a bit more complicated. Look at table 1, which shows two pairs of gambles, A versus B and C versus D (Kahneman and Tversky 1979, Problem 1). Boldface shows which one of each pair is generally preferred (again, there are always individual differences): B over A, but C over D. There are two things to notice about this pattern: first, people pick the (slightly) lower expected value in the first pair but the (slightly) higher EV in the second. Second, the only difference between these two pairs of gambles is that the payoff .66*2400 (italics) has been added to both choices in A and B. Otherwise, the choice between A and B is the same as between C and D. Thus, most people's choices violate the independence assumption. The prospect theory explanation is the primacy of the certain outcome (Gamble B), which describes the result but does not explain it.

But why on earth should violations of logical axioms like this be worrisome and consume so much mental energy? Behavior is not physics or predicate calculus. Even human beings rarely maximize in an explicit way, following all the rules of optimality theory—especially when they must decide quickly. They do not consider all the options in an unbiased, neutral, and history-independent way, and then compute the costs and benefits and choose accordingly. There is therefore *no reason whatever* that human (and animal) choice should fit any set of simple axioms.

Table 1 Violation of the Independence Assumption:**Preferences** Change When a Constant Is Added to Each Choice

	Probability			Expected Value
Gamble	0.33	0.01	0.66	
A	2500	0	2400	2409
B	2400	2400	2400	2400
C	2500	0	0	825
D	2400	2400	0	816

Conclusion

The problem these examples illustrate goes beyond prospect theory. It is a fundamental problem for any outcome-based theory like expected utility. EUT is a functional/optimality theory and not a mechanism. It says that behavior will adjust so that marginal utilities are matched (or whatever), but provides no causal account for how this is accomplished. But there must be a causal account, a mechanism, and that mechanism will fail to optimize—to "act rationally"—under some conditions. Hence EUT will also fail sometimes—as Kahneman and Tversky eventually recognized. They did not, I think, understand the causal-functional distinction. They thought prospect theory was what I call a real (i.e., causal) theory and so were surprised when they had to keep adding kludges to make it work with a wider range of data.

Functional—mechanism-free—theories are not without value, but they will always run up against limits. Theoretical behaviorism, based on the simpler problems posed by learning in animals, suggests that it may be more profitable to look at the historical factors that determine people's response repertoire. Rather than rely on some form of utility theory as the ultimate explanation for economic behavior, future research might better examine the historical and cultural—*causal*—factors that explain why a particular question format drives people to frame a problem in a certain way, a way that allows them to pick "best in repertoire" even if it is not optimal.

Exactly what are the "editing" processes that cause people to perceive logically identical problems quite differently? What kind of training affects how decision makers perceive different kinds of problems? The optimality approach—marginal utility theory—can help us understand a particular choice problem. It will never tell us how people actually choose.

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ORIGINAL PAPER

Trust and Deception in Children with Autism Spectrum Disorders: A Social Learning Perspective

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Social
Learning
Theory

Abstract Previous research has demonstrated abnormal trust and deception behaviors in children with Autism Spectrum Disorders (ASD), and we aimed to examine whether these abnormalities were primarily due to their specific deficits in social learning. We tested 42 high-functioning children with ASD and 38 age- and ability-matched typically developing (TD) children in trust and deception tasks and a novel condition with reduced social components. Results indicated that while TD children improved their performance with more social components, children with ASD lacked this additional performance gain, though they performed similarly as TD children in the condition with reduced social components. Our findings highlight that deficits of ASD in trust and deception are primarily associated with failure of use of social cues.

Keywords Autism Spectrum Disorder · Trust · Deception · Social learning

Introduction

Humans can benefit from social information during social interaction, such as eye gazes and facial cues, to learn social standards (e.g., Bushwick 2001; Hareli et al. 2015), rules (e.g., Jones et al. 2013; Wang et al. 2015), and language (e.g., Baldwin 1993; Dominey and Dodane 2004; Tomasello and Farrar 1986; Yu and Ballard 2007). Social learning is a cumulative and cyclic process, which begins almost at birth and takes several years of training and practice (Bushwick 2001). However, individuals with autism spectrum disorder (ASD), who have limited attention to and understanding of social cues, may be less likely to benefit from social learning. This impairment has been proposed to contribute to core symptoms of ASD, such as defective social communication (Bushwick 2001). In the current study, we investigated two kinds of social behaviors, trust and deception, under the framework of social learning.

Trust and deception are two types of social behaviors that children learn as they grow up. Preschoolers develop selective trust to determine whether others are trustworthy based on their past reliability, motives, and past deceiving behaviors (e.g., Corriveau and Harris 2009; Koenig and Harris 2005; Vanderbilt et al. 2011). Children with ASD, however, tend to show a trust bias towards other people's testimony, and unconditionally give credence to information provided by them (Yi et al. 2013, 2014). They also experience difficulty using facial cues to selectively trust others (Ewing et al. 2015). Closely related to trust behaviors, deception, a milestone of children's social cognitive development, also experiences

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remarkable development in the preschool years. As children grow up, they deceive others more frequently with more sophisticated strategies (Chandler et al. 1989; Russell et al. 1991; Sodian et al. 1991). However, children with ASD tend to have difficulties with lying, deceiving, and manipulating beliefs of other people (Baron-Cohen 1992; Li et al. 2011; Russell et al. 1991; Sodian and Frith 1992; Talwar et al. 2012; Yirmiya et al. 1996).

Typically, children's trust and deception performance was assessed in the context of social learning, where children need to rely on social cues provided by another person (Yi et al. 2014). Yi et al. (2014) found that children with ASD display slower learning rates than TD children regarding how to distrust and deceive an adult after being repeatedly deceived. Given the complexity of the tasks, it remains an open question whether these learning impairments primarily stem from deficits in general learning abilities as opposed to social learning per se. The present study aimed to examine whether similar learning impairments persist with reduced social components when cueing information is provided by physical markers as opposed to by a human actor. We compared the trust and deception behaviors between ASD and TD groups in a social cue condition, adopted from tasks in Yi et al. (2014), as well as in a non-social cue condition in which children had to rely on physical cues. In the social cue condition, children had to find a hidden prize, with an informant always providing misleading information about its location; then they switched the roles: the informant tried to find the prize and children were given a chance to help or misguide her. The non-social cue condition followed a similar procedure as the social cue condition except that the informant, an actual human in the social cue condition, was replaced with a physical marker in the non-social cue condition. Compared to the social cue condition, all the executive steps are still present in this non-social cue condition: children need to learn whether the cueing information is false, inhibit impulsiveness to follow the false information, and suppress the action to indicate the truth. However, they do not need to rely on social cues provided by another person (e.g., verbal instructions and gestural information), which requires understanding others' intention to distrust and manipulate others' mental states to deceive. Thus, if children with ASD have relatively intact general learning, they should have similar performance as TD children in the non-social cue condition. On the other hand, based on their specific deficit in social learning, we would expect a group difference in the social cue condition. On the contrary, if they were impaired in general learning, they should display poorer performance in comparison to TD children in both conditions.

Method

Participants

Forty-two Chinese children with ASD (age range 4.31–8.22 years, $M=5.95$, $SD=0.84$) and 38 age- and ability-matched Chinese TD children (age range 4.00–7.00 years, $M=5.73$, $SD=0.71$) were recruited for this study. This age range was chosen based on the previous study (Yi et al. 2014) and our pilot results, to ensure that most children, especially children with ASD, could understand the task instructions and complete the task. To determine the minimum sample size required in the current study, we have conducted a prior power analysis based on effect sizes discovered in a previous study with similar paradigms (Yi et al. 2014). Our sample size was adequate for obtaining a power over 0.9. None of the participants had participated in our previous studies or relative research projects.

Children in the ASD group were recruited from a special school for ASD, while children in the TD group were recruited from a normal primary school in China to participate in the study. All children with ASD included in our study were previously diagnosed by two experienced pediatric clinicians strictly according to the diagnostic criteria for ASD in the DSM-IV-TR (American Psychiatric Association 2000). Since the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2000) and the Autism Diagnostic Interview-Revised (ADI-R; Lord et al. 1994) have not been officially validated and widely used in China, we confirmed the diagnosis of children with ASD using the Chinese version of Autism Spectrum Quotient: Children's version (AQ-Child[1]), the Social Responsive Scale (SRS[2]), and the Social Communication Questionnaire (SCQ[3]). All questionnaires were scored based on the parents' reports.

Children with ASD were recruited first and randomly assigned to either the social cue or the non-social cue condition. TD children were then recruited to match the ASD group by their (a) chronological age, (b) non-verbal IQ, measured by Combined Raven Test (CRT-C2), and (c) verbal ability, measured by Peabody Vocabulary Test-Revised (PPVT-R) (see Table 1). TD children were then also randomly assigned to the social cue or the non-social cue condition.

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[1] The AQ was translated by R.W.S. Chan, W.S. Liu, K.K. Chung, C.S. Sheh & E.K.F. Woo (2008), Hong Kong Working Group on ASD, cprc@hkusua.hku.hk.

[2] The SRS is the Chinese version translated from the Constantino and Gruber (2002) version.

[3] The SCQ is the Chinese version translated from Rutter et al. (2003) Copyright© 2003 by Western Psychological Services (Fourth Printing, April 2012).

Table 1 Sample sizes, age, CRT scores, and PPVT scores of the four groups of participants

Group	Social cue condition		Non-social cue condition	
	TD	ASD	TD	ASD
N (female)	17 (5)	22 (4)	21 (6)	20 (1)
Age range	4.00–7.00	4.62–7.62	4.15–6.96	4.31–8.22
Mean age	5.72 (0.69)	6.09 (0.76)	5.73 (0.75)	5.78 (0.91)
CRT (raw scores)	25.41 (8.85)	27.27 (9.01)	25.24 (5.77)	25.95 (9.65)
CRT (standardized scores)	100.69 (13.51)	100.59 (16.25)	100.00 (9.25)	100.69 (18.22)
PPVT	81.29 (11.21)	74.14 (12.35)	83.33 (14.50)	74.55 (15.29)
VMA[a]	6.29 (0.59)	6.05 (0.65)	6.48 (0.60)	6.00 (0.80)

[a] *VMA* verbal mental age, standardized scores calculated from PPVT scores

Procedure

Distrust and Deception Tasks

A 2 (participant group) * 2 (condition) between-subject design was adopted for the experiment. We asked children to randomly participate in either the social cue or the non-social cue condition, instead of completing both conditions, considering the potential carry-over effect, practice effect, and order effect. Each child completed the distrust and the deception tasks sequentially.

The child, Experimenter 1 (E1), and Experimenter 2 (E2) each sat at one side of a table where three identical boxes were placed along a line. The child was asked to participate in a hide-and-seek game to win prizes with the experimenters, and was encouraged to find "as many prizes as possible." The experimenters were trained sufficiently beforehand on performing the tasks: they were asked not to show any expressions while they interacted with children. We used the same experimenters throughout the whole experiment to minimize the potential impact of different experimenters on children's behavior. The whole procedure was illustrated by the schematic drawing in Fig. 1.

Social Cue Condition

In the distrust task, E1 showed three identical boxes and explained the rules to the child: (a) E1 hid a prize in one of the three boxes and the child had to guess where it was; (b) E2 then examined all the boxes and showed the child where the prize was by pointing at one box; (c) the child could keep the prize if he successfully found it, otherwise E2 would keep it.

After confirming that the child understood the rules, E1 announced that the game began: (a) both the child and E2 turned their backs to the table, then E1 kept one box empty, and placed a prize in each of the other two boxes; (b) the child and E2 faced the table again, then E2 examined all the boxes, pointed at the only empty box, and said to the child "the prize is here"; (c) E1 asked the child to pick one box for the prize; (d) E1 opened the box chosen by the child and provided the feedback (i.e., whether or not the child received the prize for his behavior on each trial) accordingly; (e) if the child did not choose the empty box, E1 gave him the prize; otherwise, E1 took a prize out of either one of the two remaining boxes and gave it to E2. The task was repeated for ten times. Finding the prize in five consecutive trials was considered successfully passing the task. Then the session stopped, and the remaining trials were marked as correct.

Following the distrust task, the child proceeded with the deception task, in which the child and E2 essentially exchanged their roles. First, E1 explained the new rules of the deception task to the child: (a) the child should hide a prize in one box for E2 to look for; (b) E2 could keep the prize if she was correct; otherwise, the child could keep it; (c) before taking a guess, E2 asked the child about the location of the prize, and the child should respond by pointing at one box. After confirming that the child understood the rules, E1 announced that the game began: (a) E2 turned her back to the table, and the child hid a prize in one box; (b) E2 turned around to face the table and asked the child about the whereabouts of the prize, and the child responded; (c) E2 "guessed" that the prize was hidden in the box that was just pointed to by the child; (d) E1 opened the pointed box and provided feedback (i.e., whether or not the child received the prize for his behavior on each trial) accordingly; (e) the child received the prize if he pointed at an empty box; if the child pointed at the box with prize, E2 received the prize. Similar to the distrust task, the procedure was repeated for ten times. Children were considered as passing the task if they obtained the prize in five consecutive trials.

Non-social Cue Condition

In the non-social cue condition, we attempted to reduce social components of the distrust and deception tasks. Children no longer played a zero-sum game against E2. Instead, in the distrust task, they tried to win the prize by guessing

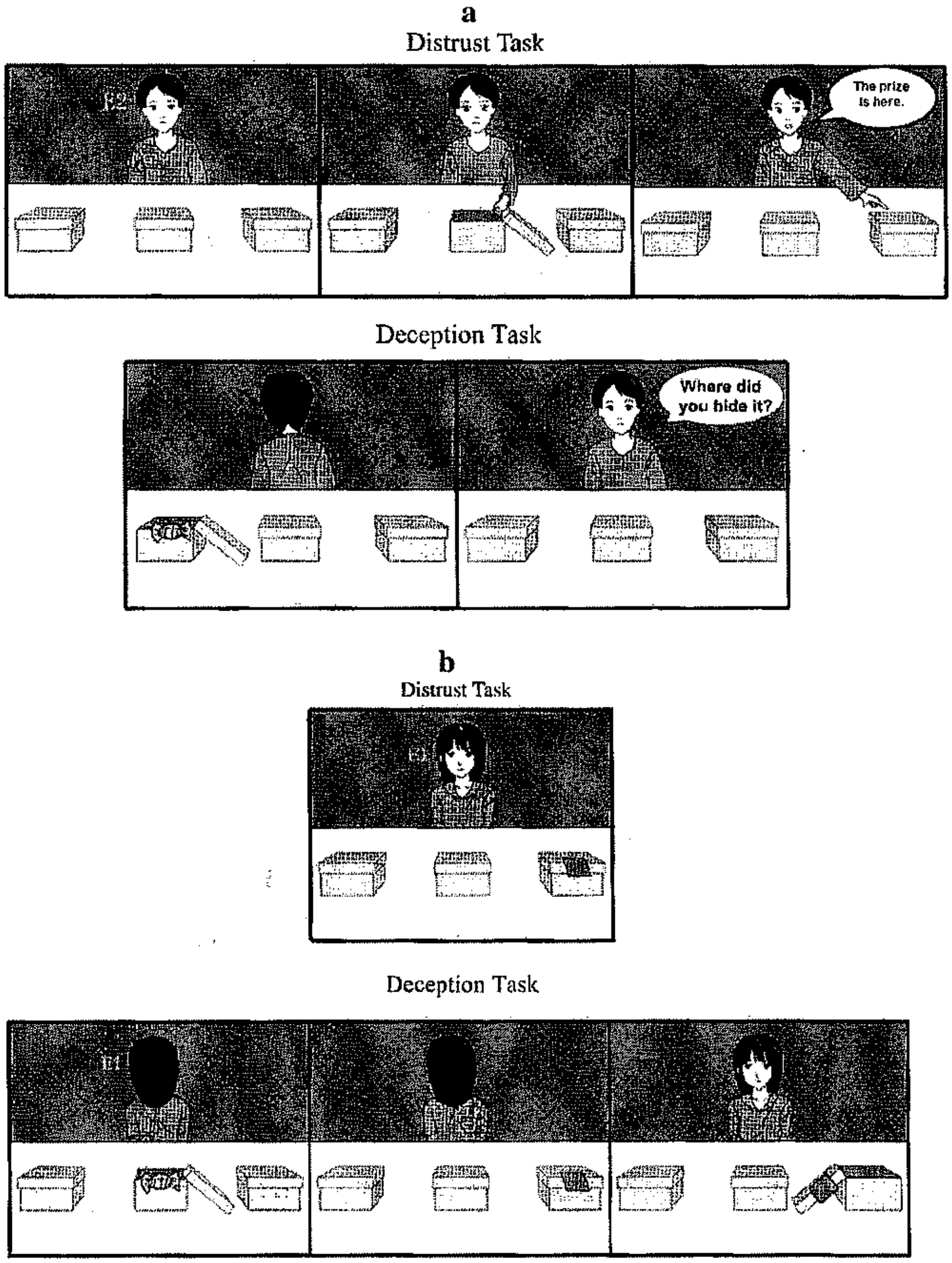

Fig. 1 Schematic drawing of the experimental procedures of the distrust and the deception tasks in the social cue (**a**) and the non-social cue (**b**) conditions

the location of the prize according to a removable sticker attached to one of the three boxes; in the deception tasks, they tried to win the prize by attaching a sticker to an empty box. Since E2 did not participate in the non-social cue condition, the child and E1 sat opposite to each other at a table.

In the distrust task, E1 showed three identical boxes to the child and explained the rules to him: there was a prize in one of the three boxes and the child had to guess where the prize was; he could keep the prize if he guessed right.

The remaining procedures were similar to those of the social cue condition, except that: (a) when the child turned his back to the table, E1 kept one box empty and placed a sticker on it, and placed a prize in each of the other two boxes; (b) right after the child faced the table again, E1 asked him to pick one box for the prize; (c) if the child chose the unbaited box, E1 gave him the prize; otherwise, if the child chose the baited box, E1 took a prize out of either one of the remaining boxes, put it aside, and stated that the prize belonged to neither the child nor E1.

Following the distrust task, the child proceeded with the deception task. E1 explained the new rules to the child: (a) the child should hide a prize in one box and then attach a sticker to one box; (b) the child could keep

Table 2 The average score over ten trials of the social cue and non-social cue conditions in the distrust and deception tasks

Task	Condition	TD	ASD
Distrust	Social cue	9.12 (0.70)	6.32 (2.32)
	Non-social cue	7.90 (1.37)	7.75 (1.65)
Deception	Social cue	9.71 (0.47)	6.95 (3.54)
	Non-social cue	9.00 (1.45)	6.85 (3.36)

the prize if he placed the sticker correctly; otherwise, neither the child nor E1 could keep the prize.

The remaining procedures were similar to those of the social cue condition, except for the following steps: (a) when E1 turned her back to the table, the child hid a prize and placed a sticker to one of the boxes; (b) E1 never asked the child about the whereabouts of the prize; (c) if the child placed the sticker on a box without the prize, he could keep the prize; otherwise, E1 put the prize aside and stated that it belonged to neither the child nor E1.

For the distrust task, if the child chose the box pointed by E2 or baited with a sticker, the trial was scored as 0; otherwise, it was scored as 1. For the deception task, if the child pointed or put a sticker on the box in which he hid the prize, the trial was scored as 0; otherwise, it was scored as 1. Hence, task performance was indicated by an array of 0 and 1 scores.

Data Analysis

We first averaged the task performance across ten trials and conducted ANCOVAs to compare these average scores between groups and conditions, with IQ and PPVT scores as the covariates. We used ANCOVAs since children's IQ and PPVT were correlated with overall trust and deception performance.[4] In our design, children "learned" from the feedbacks whether they received the prize on a trial-by-trial basis. To characterize their learning process, we also compared the two groups with two types of trial-by-trial analyses. One is to compare the two groups regarding the percentage of children who won the prize at each trial. The statistical test appropriate for this type of data is Fisher's Exact test. The other is the survival analysis that quantifies the percentage of children who eventually passed the task (i.e., winning the prize in five successive trials) and the speed of the learning process (i.e., how many trials needed to pass the task). Log-rank tests were used for group comparisons of the learning speed. Finally, we tested the correlations between the trust and deception performance (averaged scores) and their social ability in children with ASD.

Results

Distrust Task

Overall Performance

An ANCOVA was performed on the average scores of the distrust task (see Table 2) with experimental condition and participant group being independent variables, and IQ and PPVT scores being covariates. Results showed a significant main effect of the participant group: children with ASD were more likely to choose the baited box than TD children in the distrust tasks, $F(1,74)=14.58$, $p<0.001$. The main effect of the experimental condition was not significant, $F(1,74)=0.14$, $p=0.71$. However, the interaction between the participant group and the experimental condition was significant, $F(1,74)=14.73$, $p<0.001$. The simple main effect analysis indicated that in the social cue condition children with ASD were more likely to choose the baited box than TD children, $F(1,74)=28.51$, $p<0.001$, but there was no significant difference between the two groups in the non-social cue condition, $F(1,74)=0.02$, $p=0.88$.

Trial-by-trial Analysis

Trial-by-trial analyses were performed based on each group's performance in each trial (Fig. 2). The binary distribution (score 1 or 0) for each trial was compared between groups. In the social cue condition, participants in both groups performed similarly in the first trial, $p=0.22$ (Fisher's Exact test, two-tailed), but TD children outperformed children with ASD on the second trial, $p=0.01$ (Fisher's Exact test, two-tailed), and in most of the following trials. In the non-social cue condition, the two groups of children had similar performance in all trials, p's>0.05. So, such a group difference in learning to distrust disappeared in the non-social cue condition when the social components of the tasks were reduced.

Survival Analysis

We conducted a survival analysis to examine how children's trust performance would be affected by feedback over the ten trials. The curves in Fig. 3 represent the percentages of children who failed to distrust E2 at specific time points, and the shaded areas represent 95% confidence intervals. Results of the log-rank tests revealed a group difference of the learning curves in the social cue condition:

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[4] Specifically, the overall distrust performance was correlated with the IQ and PPVT scores, p's<0.05; the overall deception performance was correlated with the PPVT score, $p<0.01$.

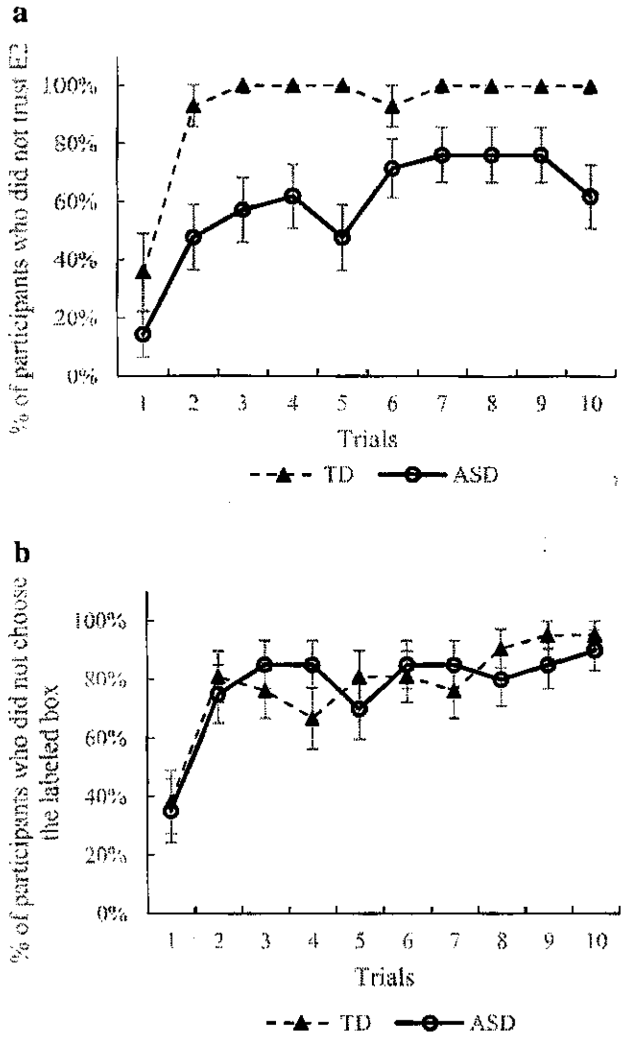

Fig. 2 Percentage of children with ASD and TD children who successfully found the prize as a function of trial numbers in the social cue (**a**) and the non-social cue (**b**) conditions of the distrust tasks. *Error bars* represent 95% confidence intervals

TD children learned faster than children with ASD in the social cue condition, $\chi^2(1)=14.3$, $p<0.001$. More specifically, the learning curve of the TD group showed a steep downward trend: on average 85.71% TD children began to choose the unbaited box in the first two trials, and 92.86% TD children successfully learned to choose the unbaited box at the end of the game. The ASD group had a less steep learning curve than the TD group, and only 47.62% children with ASD successfully learned to choose the unbaited box by the end of the game. In contrast, this group difference disappeared in the non-social cue condition: the two groups had similar learning rates, $\chi^2(1)=0.2$, $p=0.687$. At the end of the game, 51.74% TD children and 65% children with ASD successfully learned to find the prize. Assessing the difference between the conditions for each group separately, we found that TD children learned faster in the social cue condition than in the non-social cue condition, $\chi^2(1)=9$, $p=0.003$, while children with ASD had similar learning rates in both conditions, $\chi^2(1)=1.5$, $p=0.213$.

Deception task

Overall Performance

An ANCOVA was performed on the average scores of ten trials (see Table 2) of the deception task, with the experimental condition and the participant group as independent variables and IQ and PPVT scores as covariates. Results showed a significant main effect of the participant group: children with ASD were less likely to point to the empty box than TD children in the deception tasks, $F(1,74)=12.97$, $p<0.001$. The main effect of the condition, $F(1,74)=0.53$, $p=0.47$, and the interaction between the group and the condition were not significant, $F(1,74)=0.37$, $p=0.54$.

Trial-by-trial Analysis

Similar to the distrust task, a trial-by-trial analysis was performed to test the group difference in the performance of the deception task for each trial. As shown in Fig. 4, in the social cue condition, the TD group performed significantly or marginally better than the ASD group in most of the trials. In the non-social cue condition, the TD group outperformed the ASD group in the first trial, $p<0.001$ (Fisher's Exact test, two-tailed), and 90% TD children placed the sticker on the empty box in the first trial. However, only 35% children with ASD succeeded in the first trial. The group difference attenuated in the following trials and disappeared in the last three trials, p's > 0.30 (Fisher's Exact test, two-tailed), as children with ASD learned quickly from the trial-by-trial feedback. In the social-cue deception condition, 71% TD children pointed to the empty box in the first trial, marginally more than the ASD group (38%), $p=0.086$ (Fisher's Exact test, two-tailed).

Survival Analysis

The proportions of participants who failed to learn the strategy at the end of each trial were computed for each group and each condition. As illustrated in Fig. 5, the survival analysis showed a similar trend as in the distrust task: in the social cue condition, 92.86% TD children learned to point to the empty box in the first two trials, while only 66.67% children with ASD learned at the end of the ten trials. In the non-social cue condition, when the social components of the tasks were reduced, 80.95% TD children and 66% children with ASD learned to point to the empty

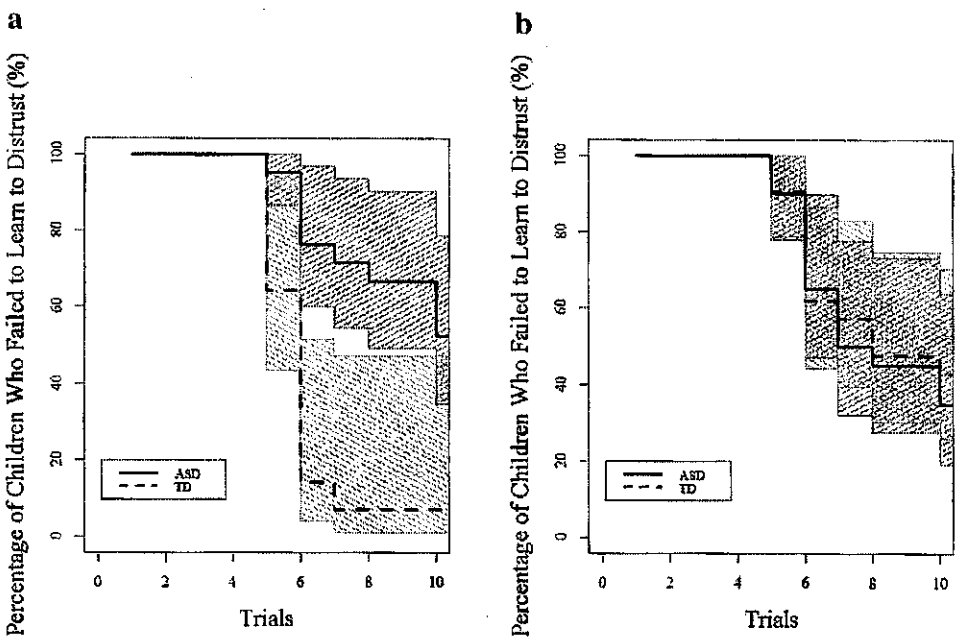

Fig. 3 Survival analyses. Percentages of children who failed to learn to choose the labeled box over trials in the social cue (**a**) and the non-social cue (**b**) conditions of the distrust tasks. The *shaded areas* represent the 95% confidence intervals

box at the end of the game. Log-rank tests showed that TD children learned faster than children with ASD in the social cue condition, $\chi^2(1)=13.4$, $p<0.001$, but the two groups had similar learning rates in the non-social cue condition, $\chi^2(1)=2.4$, $p=0.12$. When comparing the conditions within each group by the log-rank tests, we found that TD children learned faster in the social cue condition than in the non-social cue condition, $\chi^2(1)=3.9$, $p=0.049$, while children with ASD had similar learning rates in both conditions, $\chi^2(1)=0$, $p=0.97$.

Correlational Analyses

We also conducted correlational analyses to examine the possible associations between average scores of the distrust and deception tasks and the AQ, SRS, and SCQ scores in the ASD group. We did not find any significant correlation between the AQ, SRS, and SCQ scores and the trust and deception performance in ASD, p's > 0.05.

Discussion

The current study examines whether trust and deception behaviors in children with ASD are primarily affected by specific deficits in learning based on social cues. We recruited children with ASD and TD children aged from 4- to 8-years old, who had never been involved in our previous studies or any relevant research projects. Results of our social cue condition confirm the findings of Yi et al. (2014) regarding the atypical trust and deception behaviors in children with ASD: they were less likely and slower to learn to distrust and deceive the informant who repeatedly deceived them than TD children. Our novel findings came from the non-social cue condition when the social components of the distrust and deception tasks were controlled to reduce their impact: we observed a comparable learning ability in children with ASD relative to TD children. In other words, children with ASD had no difficulty learning these behaviors when the social cues were replaced by physical cues. These findings support the proposition that the abnormality of the trust and deception behaviors in children with ASD was mainly due to their defective learning in the social context (Bushwick 2001).

An alternative explanation of the specific difficulty in ASD children is that the social cue condition is cognitively more demanding simply because more information is available for processing. Compared to physical cues, the human actor brought in verbal instructions and gestural information in the tasks. This is a possible explanation for our results. The two tasks involve qualitatively similar cognitive processes, including general learning that cues are misleading based on trial-by-trial reward feedback, ignoring or inhibiting these cues, deciding to choose the alternative box and so on. The core difference is different ways to deliver the misleading information by using different cues (a person vs. a sticker). Interestingly, we found that TD children learned faster in the social cue conditions than in the non-social cue conditions despite extra available information. Thus, the presence of social cues facilitates rule understanding and problem solving in TD children, who are highly sensitive to social information. We speculate that this social strength or preference in TD children may facilitate their learning in the social context. In contrast,

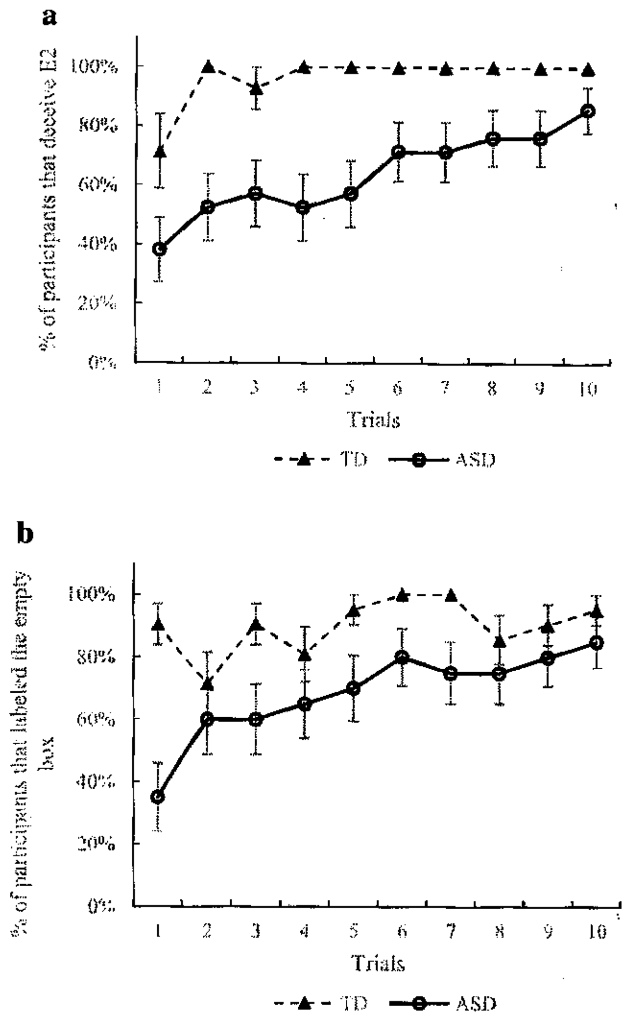

Fig. 4 Percentage of children with ASD and TD children who deceived E2 or labeled the empty box as a function of trial numbers in the social cue (a) and the non-social cue (b) conditions. *Error bars* represent 95% confidence intervals

children with ASD performed similarly in the social cue and the non-social cue conditions. Thus, the fact that they did not perform *worse* in the social cue condition suggests that extra cueing information (e.g., verbal instruction and gestural information) do not hamper the learning process. It is more likely that social strength or preference, manifested in the behaviors of TD children, is absent in children with ASD due to their diminished awareness and sensitivity to social cues. Future studies are warranted to test this hypothesis directly.

Another alternative way to explain our results of the non-social cue condition is that the two groups may interpret the non-social cues in different ways. Although there was no E2 in the non-social cue condition to interact with the child, the presence of E1 may make the TD child attribute social meanings to the physical marker as it was put by a person. At the same time, it is possible that children with ASD are less likely, compared to TD children, to attribute social meanings to physical cues in the non-social cue condition. In other words, children with ASD may interpret the physical cues in a mechanical way, while TD children may interpret it in a more social way. However, with behavioral paradigms we could not reverse engineer the exact strategies the two groups of participants used. Future research using brain imaging measures such as functional near-infrared spectroscopy (fNIRS) to explore the underlying mechanism of the tasks is therefore recommended.

Besides the findings above, we also discovered a carry-over effect for TD children in the deception task from the previous distrust task. As shown in Fig. 4, TD children in the deception task started with the better performance thus their learning curve was flatter than the distrust task. The carry-over effect by itself is an interesting observation. It might indicate some sort of learning generalization elicited by the previous distrust session, suggesting that social learning of TD children is flexible and generalizable to different conditions. However, such a generalization was not found in children with ASD in the same task, which confirms previous findings that the learning of children with ASD was less flexible and more constrained to specific circumstances relative to TD children (Yi et al. 2014).

Several limitations of the current study should be considered when interpreting our findings. First, limited by the characteristics of our tasks, the social components were not completely removed in our non-social cue condition. As discussed above, children may interpret physical markers in a social way. Despite this concern, removing the role of E2 has remarkably reduced the group differences in both distrust and deception tasks. This result suggests, in the social cue condition, that direct interaction with a person (E2) can cause significant difficulty for ASD to learn distrust and deception behaviors. Second, the cognitive load and complexity of these two conditions are not exactly the same. For example, in the social-cue condition E2 examined the boxes before the child made her judgment, while in the nonsocial cue condition this extra cue was absent. Future studies could introduce a mechanical device to attach the sticker before the child in order to make the available cues better matched between conditions. Third, we did not find any association between the trust and deception performance and the autism scales (AQ, SRS, and SCQ) in children with ASD. Future studies could use more specific social measures (e.g., pragmatic language, Theory of Mind) to explore a possible association between trust and deception and social abilities. Fourth, it is also noteworthy that our sample of ASD children is high-functioning, with an average IQ of approximately 100, which limits

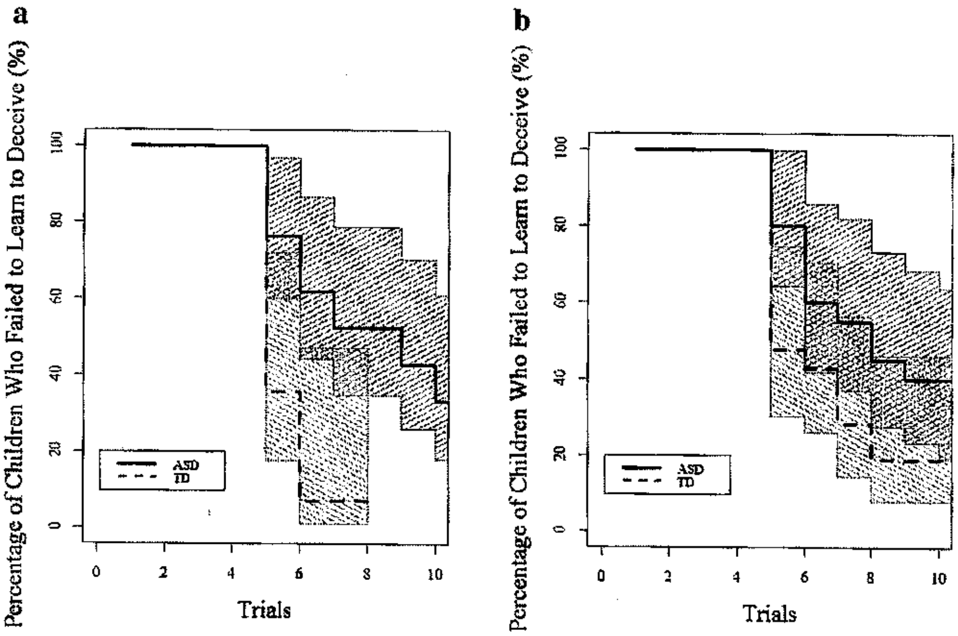

Fig. 5 Survival analyses. Percentages of children who failed to learn to deceive over trials in the social cue (a) and label the *empty box* in the non-social cue (b) conditions of the deception tasks. The *shaded areas* represent the 95% confidence intervals

the generalizability of these findings to lower-functioning children with ASD. Besides the social learning deficit, cognitive impairments may play a major role in both the social and non-social learning processes. Thus, the non-social learning could also be impaired in lower-functioning children with ASD. Our findings should also be interpreted with caution when extrapolated to children with ASD at different ages, as age plays an important role in children's development of trust and deception behaviors (e.g., Vanderbilt et al. 2011). Future research should include children with ASD with a broader age and IQ range, to investigate the developmental trajectory of social and non-social learning and the effect of cognitive dysfunction in this population. Fifth, our conclusions are based on a sample of Chinese children with ASD and TD children, so that cultural influences need to be taken into consideration when interpreting our findings. Generally speaking, Eastern and Western children exhibit highly similar distrust and deception behaviors (Fu et al. 2007; Xu et al. 2010), since both cultures generally encourage honesty and discourage deceit (Fu et al. 2010). However, subtle cultural variations of children's distrust and deception behaviors may exist, as a result of the emphasis on group harmony and collectivism in the East Asian culture (Rothbaum et al. 2000; Xu et al. 2010). For example, individualistic and collectivistic values influence the acceptability of lying, thus Chinese children prefer to lie to help the collective rather than the individual, while Canadian children do the reverse (e.g., Lau et al. 2013; Mealy et al. 2007; Sweet et al. 2010). Last, we used the between-subject design to minimize the learning generalization across conditions by randomly assigning the participants into the two conditions. With appropriate experimental designs, future research could compare one's performance in understanding the social vs. non-social cues using a within-subject design.

In summary, the current study aims to investigate whether reducing social cues could help ASD children overcome their deficits in trust and deception tasks. If so, this would provide evidence that their deficit in these tasks is more associated with their specific deficit in social learning than impairments in general learning ability. We found that the abnormalities in the distrust and deception behaviors in ASD are limited to the social situation, while their non-social learning is intact. Moreover, we found a social strength effect for TD children; their learning is benefited from the presence of social information. This social strength effect was absent in children with ASD due to their insensitivity to the social information. Besides, we also found a carry-over effect for TD children's non-social learning, which was absent in children with ASD. Overall, our findings support the social learning account of ASD (Bushwick 2001) in trust and deception behaviors. Our study has important implications for understanding the nature of ASD and interventions for individuals with the disorder. For example, when we train ASD children in social interactive tasks with similar complexity as our distrust and deception tasks, we should start with simple social cues in simple situations. After ASD children master the simple tasks, the training can incrementally include more social cues that are relatively challenging for them. Further work is needed to determine whether these findings could be generalized to a larger population of children with ASD.

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Author Contributions YY was responsible for data collection, data analysis, and manuscript preparation; YT was responsible for data analysis; JF was responsible for data collection; HL was responsible for data analysis and paper revision; KW was responsible for study design and manuscript preparation; LY was responsible for study design, data analysis, and manuscript preparation. All authors read and approved the manuscript.

Compliance with Ethical Standards

Conflict of interest All authors have no conflict of interest to declare.

Ethical Approval All procedures performed in this study were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all children included in the study and their parents.

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Philosophy

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The Limits of Learning: Habermas' Social Theory and Religion

Maeve Cooke

Abstract: Habermas' view that contemporary philosophy and social theory can learn from religious traditions calls for closer consideration. He is correct to hold that religious traditions constitute a reservoir of potentially important meanings that can be critically appropriated without emptying them of their motivating and inspirational power. However, contrary to what he implies, his theory allows for learning from religion only to a very limited degree. This is due to two core elements of his conceptual framework, both of which are key features of his account of postmetaphysical thinking. The first is the requirement of ethical agnosticism; this requires philosophy and social theory to refrain from offering guidance on questions of the good life. The second is his language-immanent conception of truth in the domain of practical reason; this follows from his rejection of any source of validity beyond human communication in this domain. I make the case for a more robust account of learning from religious traditions and metaphysical worldviews, arguing that for this purpose Habermas must modify his requirement of ethical agnosticism and relinquish his language-immanent conception of truth.

Social
Learning
Theory

Learning—learning from history and learning from other cultures—has long been a central motif in Habermas' critical social theory (Habermas 2001: 38–57; Habermas 1992: 138). In recent years, he has begun to underscore the importance of learning from religion: philosophy and social theory are encouraged to learn from religious traditions and religious believers and non-believers are urged to engage in processes of mutual learning. His new emphasis on learning from religion may come as a surprise to those familiar with his writings of the 1980s and 1990s, in which his attitude to religion is, at best, distanced. To all appearances, at least, *The Theory of Communicative Action*, first published in 1981, subscribes to the Weberian view that religious belief will die away as modernity progresses. While in his subsequent writings over the next two decades he occasionally suggests that religion might continue to have a place within modernity, the tone of his remarks is hesitant and the language he uses is cautious. In light of this it is interesting to see that the theme of learning from religion is already present in his writings of the 1980s. Thus in an essay first published in 1988, Habermas calls on postmetaphysical thinking to appropriate critically the 'old truths' of metaphysics in general, and of religion in particular (Habermas 1992: 14–5). Evidently, therefore, postmetaphysical philosophy and social theory have something to learn from religion. But learning requires what

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Habermas here calls 'critical appropriation' (in later texts he speaks of 'translation'). The need for such appropriation is attributed to the modern plurality of worldviews: the inhabitants of modernity lack a shared evaluative basis that would permit the truths of metaphysics and religion to be accepted by everyone on the basis of good reasons. This idea of critical appropriation anticipates his more recent reflections on learning from religion, in which he emphasizes religion as a reservoir of meaning that can serve as a semantic resource for postmetaphysical philosophy and social theory, as well as for the citizens of modern secular states (Habermas 2008). As before, the aim of critical appropriation is to salvage the truths of religious traditions and recast them in a generally accessible language so that everyone could accept them on the basis of good reasons, irrespective of particular religious beliefs or conceptions of the good.

Habermas' claim that religious traditions provide a valuable semantic resource for postmetaphysical philosophy and social theory calls for closer consideration. Before examining it, let me make clear that I agree both with his view that religious traditions constitute a reservoir of potentially important meanings and with his view that these meanings can be critically appropriated—translated—without emptying them of their motivating and inspirational power. In short, I agree that contemporary philosophy and social theory may learn from religion by way of (what Habermas now calls) translation. I want to argue, however, that in Habermas' critical social theory the possibility of learning from religion is highly restricted. Indeed, it is much more restricted than his recent writings give us to understand (Habermas 2008, 2006, 2010). This is due to core elements of his conceptual framework. I see two conceptual obstacles preventing him from developing a more robust conception of learning from religion. Both are key features of his idea of postmetaphysical thinking. The first is his insistence that postmetaphysical thinking must abstain from offering guidance with regard to questions of the good life and good society. In the essay referred to above, when distinguishing postmetaphysical from metaphysical thinking, he initially identifies the core issue as the 'enlightening role' of philosophy with regard to life practices as a whole.[1] Whereas metaphysical thinking offers orientation in matters relating to the conduct of life and to human flourishing in general, postmetaphysical thinking is abstinent in this regard (Habermas 1992, 14–5). I refer to this as the thesis of ethical agnosticism. The second is his insistence that in the domain of practical reason postmetaphysical thinking may not affirm any notion of validity that has a source beyond the human world of linguistic communication (Habermas 2003b: 11–5). This is part of his thesis of 'innerworldly transcendence' or 'transcendence from within' (Habermas 1996: 5, 17; Habermas 2003b: 10–1). He proposes a deflationary interpretation of the 'unconditioned' or 'absolute', according to which the transcending power of reason is contained *within* the forms of communication through which human beings reach an understanding with one another. As he puts it, 'the transcendental tension between the ideal and the real, between the realm of the intelligible and the realm of appearances, enters into the social reality of situated interactions and institutions' (Habermas 2008: 25). I refer to this as his language-immanent conception of practical validity or truth (I have more to say about what I mean by truth below). I argue that his ethical agnosticism leads to an impoverished conception of learning from religion; moreover, one that sits uneasily with his remarks on the importance of the critical appropriation of the truths of religious traditions. I then show that a more adequate account of learning from religion would require him to modify his sharp distinction between morality and ethics and, in the domain of practical reason, to give up his language-immanent conception of validity.

Let us look more closely at what Habermas means by critical appropriation of the 'old truths' of metaphysics and religion. As already mentioned, this theme is to the fore in his recent writings, where he emphasizes the importance of religion as a semantic resource for postmetaphysical philosophy and social theory (and also for the citizens of secular states, although I leave this question aside in the following[2]). Thus, in an essay on Kant, he describes religious traditions as possible reservoirs of meaning that are capable of exercising an inspirational force on society *as a whole* (Habermas 2008: 142). He criticizes Kant for according a purely instrumental function to positive religion and ecclesiastical faith, proposing instead that religious traditions, with their striking models, their vivid exemplary figures, their inspirational stories of the lives of the saints and prophets, their promises, their miracles, their suggestive images and edifying narratives, can stimulate the imagination of secular as well as religious citizens, motivating them, for instance, to work collectively towards realizing on earth a secular version of the promised kingdom of God (ibid: 223–47). As he sees it, secular translations of religious projections of successful forms of life can continue to inspire us, and encourage us to make tentative efforts at cooperation with a view to bringing about social change for the better, even without the certainty of divine assistance (ibid: 227; Habermas 2010: 18–9). For this purpose we require a form of critical appropriation or, as he now tends to say, translation, that salvages the substance of a term without deflating or exhausting it (Habermas 2006: 45). Examples include the translation of the concept of 'man [made] in the image of God' into the idea that the equal dignity of human beings deserves unconditional respect (ibid), Walter Benjamin's idea of anamnetic solidarity with past injustice, which Habermas describes as a concept 'manifestly trying to fill the gap left by the lost hope in a Last Judgment' (Habermas 2008: 241) and Marx's idea of the emancipated society, which he sees as a secular version of the kingdom of God (ibid: 231). Recall that translations recast the contents of religion in a generally accessible language that would permit everyone to accept them on the basis of good reasons, irrespective of religious belief and irrespective of their particular conceptions of the good. Recall, too, that the point of translation is semantic regeneration. By this I take Habermas to mean the provision of meanings that human beings can draw on in their various life-practices. Such meanings help us to make sense of the situations in which we find ourselves, they give us reasons for acting and they inspire us to explore new paths in life. In other words, they offer us ethical orientation: they position us in space and time, point us in certain directions and open up new possibilities.

However, as we have also seen, postmetaphysical thinking is supposed to be ethically agnostic: it is supposed to abstain from offering guidance in matters relating to the good life and good society. Moreover, it is supposed to articulate its views so that they are in principle *rationally* acceptable to everyone. How are we to make sense of what appear to be two conflicting demands? On the one hand, translation is required for the purposes of ethical orientation; on the other hand, postmetaphysical thinking is required to refrain from offering ethical orientation. How can Habermas deal with this apparent conflict? How can he allow for semantic regeneration by way of translation without compromising the ethical agnosticism of postmetaphysical thinking and the need for the translated contents to be acceptable to everyone on the basis of good reasons?

Habermas himself does not mention this difficulty, much less propose a solution to it. This may be because he believes he can avoid it with the help of his distinction between ethics and morality. And, in fact, this *is* one way of dealing with it. The conflict can be avoided by restricting the learning process to what he calls strictly moral matters, understood as matters relating to the just regulation of interpersonal conflicts from the point of view of the universalizability of interests. Postmetaphysical thinking can draw on the reservoir of meanings offered by religious traditions without violating the requirement of ethical agnosticism if it limits the possibility of learning from religion to questions of justice, leaving the domain of ethics completely to one side.

As is well known, in Habermas' conception of practical reason, moral questions are distinguished sharply from ethical ones (Habermas 1993). Ethics is concerned with questions of individual and collective self-realization, which are raised by individuals and groups in specific, local contexts; it deals with questions of the good life and good society. By contrast, morality is concerned with self-determination; it deals with matters of justice, by which he means it serves to regulate interpersonal conflicts through reference to the principle of universalizability of interests. Postmetaphysical thinking does not preempt the answers to moral questions. Nonetheless, it provides a framework for deliberation on such questions in the form of an account of communicative rationality; when applied specifically to moral questions, communicative rationality yields a formal moral theory.[3] This moral theory offers a framework for reflection on moral matters that permits them to be assessed from the point of view of a truth-analogous conception of validity, which, as such, is binding on everyone, irrespective of historical and social context.

Unlike moral questions, which raise claims to validity in a truth-analogous sense, ethical questions are held to be context-specific, not just in the sense that they emerge as questions only in specific historical and social contexts, but also in the sense that they admit of answers that hold only for particular individuals and groups and are thus not universally binding. In line with this, ethical discourses are described as intersubjective deliberations concerned with the hermeneutic explication of individual and collective value orientations and self-understandings, as opposed to seeking an outcome that is universally valid. In contrast to its contribution in the domain of morality, postmetaphysical thinking provides no framework for thinking about the validity of ethical questions: it leaves the question of ethical truth to one side. If we take seriously Habermas' distinction between morality and ethics, therefore, his call for critical appropriation of the truths of religion must be taken to refer only to moral matters; he must envisage these truths as feeding into processes of *moral* as opposed to ethical deliberation.

Some of his examples clearly support this reading of his position. Think of his example of the translation of the religious view that human beings are made in God's image into the postmetaphysical view that human beings have an inherent equal dignity, entailing the requirement of equal respect. Postmetaphysical moral theory builds this insight into its account of moral deliberation in the form of a discourse theory in which all persons affected are entitled to participate fully in the deliberation and to have their interests equally taken into account (Habermas 1990).[4] On the level of postmetaphysical discussion of concrete moral questions, this insight guides the deliberative process in a tangible way, calling on participants in discussion publicly to justify any apparent inequalities relating either to the conduct of the deliberation or to its outcome.

This reading is further supported by remarks he makes in an essay discussing what he describes as Kierkegaard's postmetaphysical approach to ethics. He writes that Kierkegaard's 'postmetaphysical abstention runs up against its limits in an interesting way as soon as questions of a 'species ethics' arise. As soon as the ethical self-understanding of language-using agents is at stake *in its entirety*, philosophy can no longer avoid taking a substantive position' (Habermas 2003b: 14–5). Habermas holds that this is the situation in which we currently find ourselves due to the advance of the biological sciences and biotechnological developments. He continues: '[these developments raise] moral questions of an *altogether different kind*; questions that touch on the ethical self-understanding of humanity as a whole'. He then observes that such questions take us *beyond* postmetaphysical thinking. This clearly points towards the reading of his position I have offered in the foregoing, according to which postmetaphysical thinking leaves questions of ethical validity to one side. At the same time, however, it shows the need for an alternative conception of postmetaphysical thinking as it relates to ethics. Habermas acknowledges this implicitly for, in an essay in the same volume, he intervenes directly in the debate over genetic engineering.[5] He justifies his intervention by expressing his concern for how biotechnological developments may deeply affect our ethical self-understanding as members of the human species, with far-reaching consequences for our self-understanding as moral beings. In my view he is right to intervene, moreover to do so as a philosopher and social theorist (as opposed simply to a concerned citizen or public intellectual). For, as I see it, philosophy and social theory have valuable contributions to make to reflection on, and discussion of, matters of such profound importance for human flourishing. If questions of human flourishing and the conduct of life were to be removed from the realm of philosophy and social theory, the result would be a very restricted conception of their tasks. We might add that seeing such questions as beyond the remit of philosophy and social theory is at odds with the self-understanding of earlier critical theorists in the Frankfurt School tradition, from which Habermas' theory has emerged; these theorists consistently emphasized critical theory's concern with human emancipation. Max Horkheimer famously described critical theories as seeking to 'liberate human beings from the circumstances that enslave them' (Horkheimer 1982: 246).[6] Evidently, Horkheimer is here advocating a mode of theorizing that would help us to attain a social condition in which we could flourish as human beings.

I have suggested that Habermas' insistence on the need for ethical agnosticism leads to a restricted conception of learning from religion and also to an impoverished conception of critical social theory. In addition, using the example of genetic engineering, I pointed out that he proves unable to maintain the ethically agnostic stance he himself advocates. His difficulty in consistently upholding the thesis of ethical agnosticism is evident, too, in the examples he gives of successful translation. For while some of these, as mentioned, clearly relate to moral matters, others seem to belong to the domain of ethics. Think, for instance, of Marx's translation into secular terms of the religious idea of the Kingdom of God; the social condition projected by Marx is one in which human beings have been emancipated from their servitude and are now free to lead a 'good life' in which they develop their particular capacities and, in general, flourish as human beings. Indeed, the overall tone of Habermas' recent writings on religion, in which he places emphasis on regeneration of the semantic fabric of society *as a whole*, suggests that what he has in mind is not just *moral* regeneration, in the narrow sense of providing resources for regulating interpersonal conflicts, but also ethical regeneration, in the sense of providing resources for life-practices that foster human flourishing.

The obvious conclusion to draw from the foregoing is that Habermas should relinquish or modify the requirement of ethical agnosticism and open up philosophy and social theory to the possibility of learning from religion in ethical as well as moral matters. But this is not an easy option for Habermas. Were he to do so, he would have to make fundamental changes to the conceptual framework of his theory. For one thing, he would have to relax his sharp distinction between morality and ethics, which is the basis for his justification of a universalist moral theory.[7] For another, he would have to give up his language-immanent conception of (practical) truth, since, as we shall see, it is incompatible with learning from religion in ethical matters.

The discussion so far has helped to clarify what it might mean for philosophy and social theory to *learn* from religion. There are, of course, different kinds of learning.[8] One kind of learning is aimed primarily at the acquisition of certain techniques or skills. But, clearly, this is not what Habermas has in mind in the present instance. A second kind of learning is aimed primarily at improving self-knowledge on the part of individuals or groups. This appears to be part of what Habermas has in mind when he refers to the need for mutual learning processes on the part of both religious and non-religious citizens (Habermas 2008). Learning in this case seems to mean a self-reflexive attitude regarding one's own view of the world, be it secular or religious. For non-believers it means, for example, self-conscious awareness that secular thinking has its historical roots in religious beliefs and traditions, that religion historically has been a force for world-disclosure and semantic renewal, that it has at times also been a force for progressive social change, and that today it retains an inspirational force for some people. For religious believers it means coming to terms with the fact of religious pluralism, with the emergence of modern science and with the spread of positive law and secular morality; it could also mean self-conscious awareness of the entwinement of religion with violence, domination and repression, though Habermas does not emphasize this especially. But this is not necessarily *mutual* learning. Indeed, in Habermas' account, the envisaged work of self-reflection on the part of secular and religious citizens seems to take place independently of one another: what he describes is not mutual learning but two complementary learning processes that run on separate tracks.[9]

The kind of learning involved in critical appropriation of the 'old truths' of religion (and of metaphysics in general) is different to this. When Habermas calls for critical appropriation or translation he apparently envisages not just improved self-knowledge but, over and above this, a kind of learning akin to a Gadamerian fusion of horizons. The latter is his model for intercultural learning. Drawing on Hans-Georg Gadamer's idea of the fusion of interpretative horizons, he sees intercultural understanding not as an assimilation, but as a convergence of perspectives, in which it is necessary for each of the culturally diverging parties to attempt to grasp things from the perspective of the other (Gadamer 1979). In this conception even profound cultural disagreements are contexts of possible learning. As Habermas makes clear, the conception rests on the presupposition that competing forms of life share an orientation towards culture-transcending ideas of truth and moral rightness; it is these culture-transcending, universalist ideas that allow individuals and groups with opposing conceptions of the good to enter into unrestricted dialogue with one another with a view to reaching agreement, instead of persisting in their claims to exclusive validity in a fundamentalist way.

Habermas does not describe learning from religion as a form of intercultural learning. Nonetheless, his conception of critical appropriation fits well with this model. Like intercultural learning, it is a process of *engagement*, in which the quest for understanding is coupled with an attitude of critical evaluation. Recall, however, that in the intercultural case, such engagement is possible only because the process of critical evaluation is guided by ideas of truth and moral rightness that are culture-transcending. If, as I have proposed, learning from religion is to include ethical as well as moral matters, Habermas will have to supply a culture-transcending idea of practical validity that is not restricted to moral questions in the narrow sense but broad enough to encompass ethical ones as well.

As things stand, this is not an option for Habermas. He himself is reluctant to move in this direction, citing a concern to respect the modern plurality of worldviews. As he sees it, the inhabitants of modernity lack a common framework that would enable them to reach agreement on substantive matters of the good life and good society no matter how long they discussed these matters and no matter how satisfactory the argumentative conditions under which they did so. In my view, however, the difficulty is not just the *factual* plurality of worldviews in modernity and the *unlikelihood* that it will give way to ethical homogeneity; rather it is a deeper, conceptual one. The difficulty is that his language-immanent account of practical validity cannot accommodate the experientially based, disclosive moment of ethical validity claims. When it comes to questions of the good life and good society, in many cases we will fail to be convinced by the arguments of others until we have undergone an epistemically (and existentially) significant shift in perception. Such shifts in perception involve truth as disclosure, whereby the world, or some aspect of it, appears in a different light and we gain a new way of looking at things. While the required shift in perception may be brought about argumentatively, as when we are swayed by the arguments of others, it typically depends on non-argumentative experiences, which make us receptive to these arguments or, indeed, substitute for them.[10] As I aim to show, truth as experientially based disclosure finds no place within the framework of postmetaphysical thinking, with its thesis of innerworldly transcendence and corresponding language-immanent conception of practical validity (truth).

This difficulty arises in the domain of practical reason in general but is especially relevant to our present discussion. For, as we have seen, learning from religion calls for critical appropriation of ethical contents that stimulate the imagination and are inspirational.[11] I want to suggest that their inspirational force depends on truth as disclosure.[12]

In order to give a sense of what I mean by truth as disclosure, I consider what it might mean critically to appropriate the meaning of an exemplary figure or action. Adopting Habermas' recent terminology, I treat this as a question about translation.[13] Our question is whether stories of exemplary figures and actions can be translated in such a way that their exemplary validity is potentially rationally acceptable to the inhabitants of modernity.[14] It seems clear that in such cases, translation is not simply a matter of rephrasing or reformulating propositional contents in a different idiom. My contention is that it should be understood as a matter of creatively rearticulating the inspirational contents in a way that produces subjective experiences in which truth is manifested anew. My hunch is that an adequate account would show that a successful translation of this kind does at least three things. It re-presents the truth of the original, it resonates with the subjectivity of its addressees and it opens their eyes to new ways of seeing themselves and the world. Translation of this kind is an aesthetic activity in the Kantian sense of the term: it involves opening up new spaces of the imagination in which truth can appear in a new time. The space it opens is one in which by way of new interpretations of self and world, and by way of a new interplay of linguistic expressions and modes of articulation, truth is re-presented: it appears to us anew.

I have chosen to focus on the translation of stories of exemplary figures and actions because critical appropriation seems particularly difficult in such cases.[15]

Indeed, we might wonder whether it is at all possible to translate stories of exemplary figures and actions in a way that does not deflate them or empty them of their truth content. Take, for instance, the biblical story of Abraham's willingness unconditionally to obey God's command to sacrifice his beloved son, Isaac.[16] On some readings of this story, Abraham is intended as an exemplary figure.[17] But even for religious believers in the Judaeo-Christian tradition, it is not at all clear why one should regard him as such; how much harder, then, for non-believers to feel the power of the example he allegedly sets. The difficulty, in my view, is that the meaning of exemplary figures and acts has an irreducible experiential dimension that is contextually and subjectively mediated. In other words, understanding exemplarity depends on a subjective response that has an affective as well as cognitive element and is, in turn, dependent on multiple factors relating to the historically specific, socio-cultural context in which the subjects addressed find themselves, as well as to the biography and psychic constitution of these subjects. This experiential component significantly complicates the translation exercise. However, as we shall see, it renders it neither impossible nor unproductive.

To speak of exemplarity is to speak of the normativity of the example: its power to inspire and to motivate us in our thinking and action. There have been some recent attempts among philosophers to offer an account of exemplary validity in terms of the Kantian paradigm of reflective judgment (e.g. Ferrara 2008). What I find missing from such accounts is consideration of the mode of truth involved in such judgment. Drawing on the work of Paul Ricoeur, I want to suggest that it is truth as disclosure: the example imaginatively opens up a space in which truth appears (Ricoeur 1995).[18] At the same time, again drawing on Ricoeur, I want to emphasize that the exemplary power of figures and actions is always mediated by contexts of interpretation. Even the most seemingly immediate experience of truth is mediated by the subjectivity of the person experiencing it. This subjectivity is in turn influenced by the historical and socio-cultural context inhabited by the person in question, and by the linguistic practices of that context. We could say: truth is always mediated truth, even when its operative mode is disclosure: even disclosed truth is *articulated* truth (ibid: 48–67).

Given the mediation of disclosed truth by contexts of interpretation and its dependence on experiential factors of a subjective and contextual kind, how is it possible critically to appropriate stories of exemplary figures and acts without emptying them of their meaning and robbing them of their motivating and inspirational power? Let us consider the exemplary act presented in the following story.

The story is taken from Orhan Pamuk's novel, *My Name is Red*, which is set in Istanbul in the 1590s (Pamuk 2001). The practice of manuscript illumination is at the centre of the book. One of the main characters is Master Osman, the master illuminator in charge of the workshop in which miniaturists are trained to illuminate manuscripts for the Sultan's palace in Istanbul. Master Osman is inspired by stories of the great master illuminators who have preceded him in the tradition, in particular by the master illuminators who produce their best work when blind, guiding their brushes from memories acquired after years of thought, contemplation and reflection. Some of these masters went blind as a result of the fervour of their work, but some of them are supposed to have blinded themselves voluntarily (ibid: 393). Part of the plot of the novel entails Master Osman being granted access to the Sultan's otherwise locked and closely guarded treasury, where he is free to examine the most precious manuscripts of the greatest master illuminators. One of these is the Great Master Bihzad, who was reputedly blind in the last years of his life. Master Osman is convinced that the Great Master Bihzad blinded himself deliberately (ibid: 391). When examining one of the manuscripts in the treasury, Master Osman finds a reference to the 'turquoise- and mother-of-pearl-handled golden plume needle which [...] Master of Master Illuminators, Bihzad, used in the act of blinding his exalted self' (ibid: 392). Master Osman finds that very needle in the treasury and uses it to blind himself, smiling as he presses the needle into the pupil of first his right eye and then his left (ibid: 393–4).

The Great Master Bihzad's act of blinding himself inspires Master Osman and motivates him to perform *his* act of self-blinding. Clearly, it offers him ethical orientation. We might also say that Bihzad's act of self-blinding has *exemplary validity* for Master Osman. The crucial question, however, is whether it can be translated in such a way that it has exemplary validity *for us*, the inhabitants of a quite different cultural context, with quite different ideas of what makes an action ethically desirable or repugnant.

I have emphasized that truth as disclosure is always mediated truth. To feel the full force of the example in the Bihzad-Osman case, we would have to inhabit a complex interpretative framework in which the purpose of illumination is to evoke and honour the divine, in which the hand of the master illuminator is merely a vehicle for the divine, in which blindness is a gift of God bestowed on those masters who, by arduous discipline and training, come so close to the divine that they no longer need physical eyesight for their works of illumination, and so on. Thus, it is nearly impossible for us, the inhabitants of a quite different historical and socio-cultural context, to feel the full force of the example in this case. Nonetheless, with the help of a translation, we may be able to feel its power in some measure, to a greater or lesser degree; moreover, by feeling its power in some measure, we gain initial access to a context of meaning that was previously alien to us; this may enable us eventually to grasp it and, perhaps, value it more deeply. Two observations may be helpful here.

First, contexts of interpretation, expression and articulation are rarely so foreign to us that we are unable to gain any imaginative access to them whatsoever. That is to say: truth's mediations are rarely completely unfamiliar to us; some points of connection are almost always available. In the Bihzad-Osman case, we may be able to gain imaginative access to the space in which truth appears by connecting, for instance, with the idea that vision is not just a matter of eyesight but a function of bodily memory as a whole and/or with the idea that the love of beauty can inspire us to act courageously and/or the idea that

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the excellence we gain through self-discipline and training brings us to a higher level of consciousness, perhaps even that it brings us closer to God. In other words, the force of the example can be experienced more or less immediately, but is rarely completely inaccessible to us.

Second, a powerful—poetically gifted—writer/translator can enable us to feel the normative force of exemplary acts even in contexts that are not readily accessible to us. It testifies to Pamuk's poetic talent as a writer of stories that his readers are likely to find Master Osman's emulation of Great Master Bihzad's act of self-blinding powerful; Pamuk invites us into an interpretative context that is alien to us in many respects; yet he succeeds in making us feel sufficiently at home there to be able to experience the exemplarity of the figure of Bihzad, not with full force but at least to some degree. In using the word 'powerful' I do not want to give the impression that I see the writer/translator as determining or controlling the transfer of meaning; rather, the genius of the writer/translator consists in her or his role as facilitator. Indeed, I wish to emphasize that the activity of critical appropriation depends crucially on the subjective activity of the readers or, more generally, receiving subjects. In other words, the writer as translator facilitates the appropriation of contexts of meaning by his or her receiving subjects in a process that involves active engagement of their subjectivity.[19]

But why should we speak of truth at all in this context? It could be objected that I have simply assumed that the power of the example to inspire and motivate is connected with a culture-transcending, truth-analogous claim to validity. This objection is warranted. I have provided no argument in support of my claim that truth appears in exemplary figures and actions. This is mainly because I have not yet worked out the requisite argument to my satisfaction. Until I do so, I can merely point to its guiding intuition. The intuition is that we could not adequately account for the inspirational and motivating power of ethical and religious experiences unless we assumed that something intrinsically valuable was disclosed to the subjects of these experiences. Nor could we adequately account for the transformative aspect of the disclosure—its power to bring about an existentially and epistemically significant shift in perception—unless we assumed that something intrinsically valuable was involved. 'Truth' seems an appropriate name for the intrinsically valuable substance of disclosures of this kind. Furthermore, in a Habermasian vein, I maintain that we would be unable to engage critically with the exemplarity of the figure or action without situating it in a cultural context with whose inhabitants we share a concern for validity in a culture-transcending, truth-analogous sense.

I wish to stress the epistemic unreliability of disclosures of this kind. This is due to the mediated nature of all experience: a subjective experience alone does not amount to reliable *knowledge* of the validity of what is disclosed. The subjects in question make claims on behalf of the intrinsic value of what appears to them, which (subject to contextual considerations) have to be opened to critical scrutiny in public processes of argumentative evaluation.[20] I want to emphasize, too, that truth as I conceive it is *inherently* culture-transcending: it constitutes a reference point for inquiry that always escapes our attempts to capture it; thus, complete knowledge of truth is not available to human beings.[21] The thesis of the culture-transcending nature of truth, coupled with the thesis that truth is disclosed in experiences of exemplary figures and actions, means that the *religious* dimension of the story of Bihzad/Osman (or, indeed, Abraham) is not crucial for the purposes of the present discussion. In other words, if we experience these figures as exemplary, it is because something of intrinsic value appears to us when we encounter them. While making sense of its intrinsic value involves a culture-transcending idea of validity, this idea of validity is not specific to any religion and shares only certain features with traditional religious conceptions of the divine.

Do translations of inspirational figures and actions offer ethical orientation in a way that respects the modern plurality of worldviews? Whereas Habermas takes respect for the plurality of worldviews to require ethical agnosticism, such agnosticism, as we have seen, unacceptably limits the possibilities of learning from metaphysical and religious traditions and is out of tune with Habermas' own emphasis on the importance of semantic regeneration. This is why I argue that he should give up his ethically agnostic position. At the same time, I share his concern to respect the plurality of worldviews. But it seems to me that such respect requires not ethical agnosticism, but a normative commitment to individual autonomy and rational accountability—two normative principles at the heart of Habermas' theory of communicative action. In other words, respecting the plurality of worldviews does not prohibit philosophy and social theory from translating the contents of metaphysical and religious traditions for the purposes of ethical orientation: it merely requires them to acknowledge that those they address must work out for themselves, on the basis of good reasons, the ethical validity of the translated contents. In my view Pamuk's translation respects the plurality of worldviews in the sense of upholding the normative principles of individual autonomy and rational accountability. An in-depth discussion of the story, in particular of the techniques it employs to provoke reflexivity in the reader, would show that an aspect of the good life (in this case, self-blinding) is not imposed on those who hear or read it;[22] rather it is left up to those it addresses to accept or reject the validity of Master Osman's action, moreover to do so for reasons of their own that they could attempt to justify to others if called upon to do so. I emphasized that *whether* we feel the force of the example is contingent on a host of complex factors. But if we do feel its force, at least to some degree, it will not be because we have been compelled unquestioningly to accept the truths of the metaphysical tradition to which both Bihzad and Osman belong; rather, it will be because Pamuk has helped us to gain imaginative access to this metaphysical tradition and enabled us to make connections between what we find there and our own experiences, actions and judgments. In other words, Pamuk will have enabled the *critical* appropriation by the inhabitants of modernity of an inspirational and motivating content.

All this suggests that Habermas does not need the thesis of ethical agnosticism, understood as the requirement that philosophy and social theory abstain from offering guidance in matters of the good life and good society. Since relinquishing this version of it would enrich his account of learning from religion (and metaphysics in general), I take the view that he should give it up. However, as mentioned, an enriched account, which would allow for ethical learning from religion, would have profound implications for the conceptual framework of his theory. As already indicated, it would require him to modify his strict distinction between morality and ethics, which would have significant implications for his account of moral validity in terms of the universalizability of interests. In addition, it would require him to give up his language-immanent conception of practical validity. I will conclude by outlining why this would be necessary.

Habermas' language-immanent conception of practical validity is most clearly evident in his discourse ethics. His rejection of anything outside of, or beyond, human practices of linguistic communication leads him to propose what we may call an *epistemic-constructivist* conception of moral truth. In his view, moral reality—the moral world—is constructed. It is 'made by us' (Habermas 2003a: 266–75). The realm of morality is itself generated in discourse. As he puts it: in the case of moral validity, the very domain of validity has to be *produced* (ibid: 262).[23] Moral validity is defined as the outcome of an idealized discursive procedure, universal in extent, in which participants reach agreement that a particular norm or principle is equally in everyone's interests. This discursively generated agreement is conceived of as the 'single right answer' to the question at hand; moreover, as an answer that is arrived at by everyone for the same reasons. This is what lends moral validity its unconditional, universally binding character and makes it analogous to truth (Habermas 2003a: 247–9).

I have a number of objections to this epistemic-constructivist account of practical validity. For one thing, as already indicated, it is unable to accommodate the experientially based, disclosive moment of practical reasoning. As I have argued in more detail elsewhere, in the domain of practical reason the rational acceptability of validity claims may depend on shifts in perception that are not attributable primarily to the exchange of arguments (Cooke 2013a; Cooke 2014). Think, for example, of trying to convince someone by reasoned argument that she should become a vegetarian. In many cases no amount of reasoning will work until the person in question has come to see the world in a way that make her receptive to the reasons for being a vegetarian (perhaps as a result of an existentially significant experience such as a visit to an abattoir). But as soon as we allow for subjective experiences in specific contexts as possible co-determinants of rational acceptability, the Habermasian conditions for generating unconditionally valid knowledge (truth) no longer obtain. No matter how perfect the argumentative conditions, in any given instance the rational acceptability of a particular argument might depend on argumentation-external factors absent in that instance. This would mean in turn that even perfect argumentative conditions would not guarantee a consensus and, hence, that an argumentatively achieved consensus could not *produce* truth. In other words, if the experientially based, disclosive moment of practical reasoning is given its due, there can be no guarantee that

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participants in deliberation would agree on the rational acceptability of moral norms or principles *even under ideal discursive conditions.*

In addition, I have some more general reservations. In a nutshell, my general objections are that Habermas' epistemic-constructivist account of moral validity is hubristic, counter-intuitive and finalistic. In his account, a rational consensus reached under idealized communicative conditions *defines* validity in a truth-analogous, unconditional sense. It is important to notice that moral validity is construed as a matter of *constructing* the single right answer to a moral problem, as opposed to *finding* it. Participants in deliberation under idealized communicative conditions are given the powers of understanding and imagination required not for recognizing the single right answer but for *producing* it. This is why perfection of the procedure of intersubjective deliberation, combined with application of the principle of universalizability, is held to result in perfect moral knowledge. I see this thesis of the coincidence of human knowledge and (moral) truth as worryingly hubristic. I also think it runs counter to some of our deepest intuitions, for it eliminates the moment of receptivity to the unanticipated and to the new that we associate with the best human achievements. Furthermore, and connected with this, I regard it as finalistic, in the sense that it postulates a possible endpoint to the process of historical learning and creative human thinking, thereby denying the finitude of human knowledge, the contingency of human life and history and the creativity and freedom of human will. For, while Habermas' discourse ethics certainly allows for the thematization and critical assessment of new experiences, new needs, new interests, new ideas and so on, it rules out reflection on the *moral* validity of the procedural framework within which such matters are thematized and critically examined. In other words, it rules out moral objections to the definition of justice as the outcome of an idealized deliberative procedure in which participants agree that the norm or principle in question is equally in everyone's interests. This opens it to the accusations that it immunizes a historically and culturally specific conception of justice against attempts to rethink it in light of new experiences, events and ideas, and implies that no further learning as regards the idea of justice is necessary (Cooke 2005: 397; Cooke 2006: 129–60).

My specific objection for our present purposes is a variation of these accusations. For, just as it rules out moral criticism of the particular conception of moral validity it proposes, his language-immanent, epistemic-constructivist account of practical validity cannot accommodate the possibility that moral truth is *disclosed*, in the sense that a space opens up in which truth appears, giving rise to experiences that cause us to see ourselves, our relationships to others and our relations to the world in new, morally relevant, ways.

I have contended that truth as disclosure is the mode of truth at stake in stories of exemplarity, suggesting that translations of stories of exemplary figures and actions enable us to feel their inspirational and motivational power by opening up new spaces of the imagination in which truth appears in a new time. The space they open is one in which truth is re-presented: it appears to us anew. I have also suggested that the language-immanent—epistemic-constructivist—

conception of truth proposed by Habermas is incompatible with an understanding of truth as disclosure. If this is so, in order to develop the rich account of learning from religion towards which he gestures, and from which his theory could, in my view, profit, he will not only have to relinquish, or at least significantly modify, his thesis of ethical agnosticism, and the distinction between morality and ethics on which it is premised; he will also have to give up the language-immanent conception of truth that is the cornerstone of his account of postmetaphysical thinking.

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NOTES

[1] Later in the essay, Habermas offers a fuller account of postmetaphysical thinking. Habermas 1992: 28–52.

[2] See my discussions in Cooke 2007, Cooke 2013a and in Cooke 2011.

[3] For an account of communicative rationality, see Cooke 1994.

[4] See also Cooke 1994, chapter 2.

[5] 'The Debate over the Ethical Self-understanding of the Species' in Habermas 2003b: 16–100.

[6] Translation altered. Cf. Horkheimer 1968: 263, who writes: '[Die kritische Theorie] zielt auf die Emanzipation des Menschen aus versklavenden Verhältnissen'.

[7] As we shall see Habermas defines moral validity as the outcome of an idealized procedure of deliberation in which all agree for the same reasons that a given norm or principle is equally in everyone's interests. The modern plurality of worldviews means that such rational agreement is conceivable only if participants abstract from their particular ethical conceptions, focusing instead on those interests that are universalizable. See Habermas 1993: 116–94.

[8] In Cooke 2002 I distinguish between three levels of learning: On a first level learning refers to gains in knowledge for a pre-defined purpose. Learning in this sense is essentially technical learning. On a second level it refers to beneficial changes in the self-understandings of the participants, and in their assignments of meaning and value. Learning on this level is essentially personal learning. On a third level learning refers to beneficial changes in the prevailing standards of what constitutes beneficial change on the second level. Learning on this level is essentially socio-cultural learning.

[9] See Cooke 2011: 481–2.

[10] See, for example, Cooke 2014. It is important not to confuse the question of how we come to find an argument rationally acceptable (a question of biography) with the question of what makes it rationally acceptable (a question of justification). It is also important to distinguish two positions as regards validity claims and argumentation. One holds that the rational acceptability of validity claims often depends in significant part on non-argumentative experiences. This is my position. It is what I mean when I refer to subjective experiences in specific contexts as possible co-determinants of rational accept-

ability. The other is that the experiential basis of rational acceptability renders critical evaluation of validity claims in argumentation impossible or unnecessary. This is *not* my position.

[11] I do not claim that *all* religious contents stimulate the imagination and are inspirational; equally, I do not claim that disclosure is the *only*—or even the primary—mode of religious truth.

[12] In earlier writings, I have referred to the mode of truth involved in ethical and religious experiences as 'truth as manifestation' (see, for example, Cooke 2011). The advantage of this terminology is that it highlights the way in which truth is *presented*, how it *appears* or is *shown* to us. The appearing of truth is also implied by the term 'disclosure'; this terminology has the advantage that it foregrounds the way in which the appearing of truth *opens our eyes* and brings about a *transformation* of our perceptions. Since it is this aspect that is most relevant in processes of critical evaluation, I prefer to speak of 'truth as disclosure' in the following.

[13] For the sake of simplicity, I leave aside the question of whether the terms 'critical appropriation' and 'translation' are equivalent in this context and take over Habermas' apparently interchangeable use of the terms.

[14] The following passages draw on my discussion in Cooke 2011.

[15] In the following, I pass over the specific difficulties involved in translating *stories* of exemplary figures and acts. In my view, every articulation of exemplarity is a re-presentation, in which truth contents are mediated in a particular way. In each case, the experiential component of exemplarity complicates translation. Stories are a particular mode of articulation (re-presentation). The distinctive difficulties they pose for translation are not relevant for our present purposes. It should be noted, nonetheless, that the exemplary validity of figures in literature and film is always *doubly* mediated: by the way in which it is presented in a particular literary text or film as well as by its reception by a particular reader or film-goer in a particular context (see Cooke 2013b).

[16] *Book of Genesis* 22: 1–19.

[17] This is how I interpret Søren Kierkegaard's reading of the story in Kierkegaard 2008.

[18] Ricoeur refers to truth as *manifestation* (see n. 12 above). A further important point made by Ricoeur is that the meaning of a religious work has a global shape that is engendered by the interplay between the multiple modes of articulation that produce it; thus, the meaning of the Christian New Testament emerges by way of a complex interaction between modes of articulation such as parables, proverbs, wise sayings and so on, each of which has its own distinctive features.

[19] Recall my observation that the exemplary validity of figures in literature and film is always *doubly* mediated (see n. 15 above).

[20] The argument for public evaluation in argumentation of ethical and religious claims to validity does not follow necessarily from the epistemic unreliability of ethical and religious experience, but requires a separate justification. Elsewhere I outline such a justification: see Cooke 2006: 129–33 and Cooke 2013a.

[21] I hold, however, that we can improve our knowledge of truth. For this, participation in public processes of argumentative evaluation of claims to truth is necessary, though not sufficient (Cooke 2006: 129–60).

[22] In order to show this convincingly a detailed textual analysis identifying Pamuk's use of a variety of distancing techniques would be necessary.

[23] Habermas writes: '[The] unconditional nature of moral validity claims can be accounted for in terms of the universality of a normative realm that has to be produced'. Translation altered. Cf. Habermas 1999: 301, where he speaks of *'eines herzustellenden Geltungsbereichs'*.

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