

## Big Daddy's Last Dance

The reverence and revelry of a New Orleans jazz funeral procession

Drawing from West African, Haitian and European colonial influences, jazz funerals are a tradition almost entirely exclusive to New Orleans, and as culturally rich and multifaceted as the city itself. The processions generally open with a brass band performing solemn marches and dirges as family and friends accompany the deceased to a burial. Eventually, the band breaks out into more upbeat and swinging numbers, allowing mourners cathartic release in music and dance, and onlookers to form a 'second line' and join the festivities. In what director Caitlyn Greene describes as 'a love letter to New Orleans', *Big Daddy's Last Dance* captures the arc of a jazz funeral, in all its reverent, jubilant glory.

Directors: Caitlyn Greene, Jon Kasbe

10 July, 2017

The video can be viewed at the link below.

<https://aeon.co/videos/the-reverence-and-revelry-of-a-new-orleans-jazz-funeral-procession>

## Genes, Economics, and Happiness

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We explore the influence of genetic variation on subjective well-being by employing a twin design and genetic association study. In a nationally representative twin sample, we first show that ~33% of the variation in life satisfaction is explained by genetic variation. Although previous studies have shown that baseline happiness is significantly heritable, little research has considered molecular genetic associations with subjective well-being. We study the relationship between a functional polymorphism on the serotonin transporter gene (5-HTTLPR) and life satisfaction. We initially find that individuals with the longer, transcriptionally more efficient variant of this genotype report greater life satisfaction ( $n = 2,545$ ;  $p = .012$ ). However, our replication attempts on independent samples produce mixed results, indicating that more work needs to be done to better understand the relationship between this genotype and subjective well-being. This work has implications for how economists think about the determinants of utility, and the extent to which exogenous shocks might affect individual well-being.

**Keywords:** life satisfaction, twin study, genetic association, serotonin transporter gene, 5-HTTLPR, rs2020933

**Supplemental materials:** <http://dx.doi.org/10.1037/a0030292.supp>

Happiness research has become one of the liveliest subjects in economics in recent years.<sup>1</sup> Its main goal is to explain the determinants of

individual life satisfaction or subjective well-being (often loosely called happiness). Economists have mainly dealt with economic influ-

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This article expands on De Neve (2011)—a research note that reports on the genetic association result in the Add Health discovery sample. The authors thank the anonymous reviewers and Dan Benjamin, Chris Chabris, Chris Dawes, Pete Hatemi, David Laibson, Andrew Oswald, Richard Layard, Jaime Settle, Albert Vernon Smith, and Piero Stanig. De Neve benefited from the generous hospitality of the Institute for Empirical Research in Economics (IEW) at the University of Zurich and CREMA. Research was supported by National Institute on Aging Grant P-01 AG-031093 and National Science Foundation Grant SES0719404. This research

uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (<http://www.cpc.unc.edu/addhealth>). No direct support was received from grant P01-HD31921 for this analysis.

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ences; in particular, income and its distribution, labor market regulation, unemployment, and inflation. For example, Di Tella, MacCulloch, and Oswald (2001) used happiness surveys to determine the welfare costs of inflation and unemployment, showing that unemployment depresses reported well-being more than does inflation. In fact, their longitudinal study of life satisfaction self-reports enabled these authors to estimate that people would trade off a 1 percentage-point increase in the unemployment rate for a 1.7 percentage-point increase in the inflation rate. Systematic influences on life satisfaction have also been found for sociodemographic factors (age, gender, race, marital status, children, and social networks), as well as for political and cultural factors (such as democracy, decentralization, and religiosity). While variables like socioeconomic status, income, marriage, education, and religiosity are significantly associated with individual happiness, none typically accounts for more than 3% of the variation (Frey, 2008; Layard, 2005). Moreover, changes in these variables appear to yield only short-term changes to happiness. For example, the “Easterlin Paradox” (Clark et al., 2008; Easterlin, 1974) suggests that increases in real income either have no lasting effect on happiness, or only a quite small one (Stevenson & Wolfers, 2008). A reason appears to be that happiness levels tend to revert toward what psychologists describe as a “set point” or “baseline” of happiness that is partly shaped by personality and genetic predispositions (Diener & Lucas, 1999; Kahneman, Diener, & Schwarz, 1999). This in turn has implications for how economists think about the determinants of utility, and the extent to which exogenous shocks might affect an individual’s well-being.

Although previous studies have shown that baseline happiness is significantly heritable (Lykken & Tellegen, 1996), little research has considered molecular genetic associations with subjective well-being. Here, we first corroborate the earlier work showing that happiness is significantly influenced by genetic variation in a nationally representative twin sample, and subsequently, we present mixed evidence for a candidate gene association with life satisfaction. In our discovery sample, we initially found that individuals with a transcriptionally more efficient version of the serotonin transporter gene (*SLC6A4*, more commonly referred to as 5-HTT

or SERT for the protein it encodes) are significantly more likely to report higher levels of life satisfaction. However, our replication efforts on independent samples produce mixed results. This combination of economics and genetics is of rising salience (Beauchamp et al., 2011; Benjamin et al., 2012; Benjamin et al., 2007).

Before we detail our genetic association approach and results, we explore the general influence that genes may have on happiness through a twin study design. A growing number of studies use twin research techniques to gauge the relative importance of genetic and environmental influences on economic behaviors (e.g., Cesarini, Dawes, Johannesson, Lichtenstein, & Wallace, 2009; Fowler, Dawes, & Christakis, 2009). We estimate the heritability of subjective well-being at 33%, indicating that about one-third of the variance in individual life satisfaction can be attributed to genetic influences.

Although twin studies are an important step in establishing the influence of genes in subjective well-being, they do not identify the specific genes involved. The increasing availability of genotypic information now allows us to test hypotheses about targeted genes and their effects. One place to start the search for such genes is among those that have already been shown to account for variation in emotional states. Among these, 5-HTT is a prime candidate. The 5-HTT gene encodes a transporter in the cell wall that absorbs serotonin into the presynaptic neuron in parts of the brain that influence mental states (Bertolino et al., 2005; Canli & Lesch, 2007; Hariri et al., 2002; Heinz et al., 2005). 5-HTT has been studied for more than 20 years, and much is known about the way different versions of this gene influence transcription, metabolism, and signal transfers between neurons, all of which may influence personality. In particular, less transcriptionally efficient variants of this gene have been shown to moderate the influence of life stress on de-

<sup>1</sup> Books are, for example, Kahneman et al. (1999); Graham and Pettinato (2002); Frey and Stutzer (2002a), Van Praag and Ferrer-I-Carbonell (2004); Layard (2005), or Frey (2008); articles are for example, Easterlin (1974); Clark and Oswald (1996); Frey and Stutzer (2002b); Di Tella, MacCulloch, & Oswald (2003); Luttmer (2005); Di Tella and MacCulloch (2006); Rayo and Becker (2007); Dolan et al. (2008); Fowler and Christakis (2008); Urry et al. (2004) or Clark, Frijters, & Shields (2008).



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pression (Caspi et al., 2003), and the more transcriptionally efficient alleles have been linked to optimism (Fox, Ridgewell, & Ashwin, 2009). As a result, economists have specifically identified 5-HTT as a candidate gene for further study (Benjamin et al., 2007).

Using data from two independent sources, the National Longitudinal Study of Adolescent Health (Add Health) and the Framingham Heart Study (FHS), we analyze the relationship between variants of 5-HTT and life satisfaction. We find some evidence of significant association in both data sets, suggesting that the 5-HTT gene may play a role in explaining life satisfaction, yet more work needs to be done to verify and better understand the relationship between this genotype and subjective well-being. We do not claim that 5-HTT determines happiness, nor do we exclude the likely possibility that several other genes may play a role in accounting for the influence of genes on happiness.

### The Add Health Data

This research is based on genetic and survey data collected as part of Add Health. The study was initially designed to explore the health-related behavior of adolescents in Grades 7 through 12, but it has been employed widely across disciplines and has made recent contributions in economics (Alcott, Karlan, Mobius, Rosenblat, & Szeidl, 2007; Echenique & Fryer, 2007; Echenique, Fryer, & Kaufman, 2006; Norton & Han, 2008). In the first wave of the Add Health study (1994–1995), 80 high schools were selected from a sampling frame of 26,666 based on their size, school type, census region, level of urbanization, and percentage of the population that was white. Participating high schools were asked to identify junior high or middle schools that served as feeder schools to their school. This resulted in the participation of 145 middle, junior high, and high schools. From those schools, 90,118 students completed a 45-min questionnaire, and each school was asked to complete at least one School Administrator questionnaire. This process generated descriptive information about each student, the educational setting, and the environment of the school. From these respondents, a core random sample of 12,105 adolescents in Grades 7–12 were drawn, along with several oversamples, totaling more than 27,000 adolescents. These

students and their parents were administered in-home surveys in the first wave.

Wave II (1996) was comprised of another set of in-home interviews of more than 14,738 students from the Wave I sample and a follow-up telephone survey of the school administrators. Wave III (2001–02) consisted of an in-home interview of 15,170 Wave I participants. Finally, Wave IV (2008) consisted of an in-home interview of 15,701 Wave I participants. The result of this sampling design is that Add Health is a nationally representative study. Women make up 49% of the study's participants, Hispanics 12.2%, Blacks 16.0%, Asians 3.3%, and Native Americans 2.2%. Participants in Add Health also represent all regions of the United States.

In Wave I of the Add Health study, researchers created a sample of sibling pairs including all adolescents that were identified as twin pairs, half-siblings, or unrelated siblings raised together. Twin pairs were sampled with certainty. The sibling-pairs sample is similar in demographic composition to the full Add Health sample (Jacobson & Rowe, 1998). The number of identical (monozygotic) and nonidentical (dizygotic) twins who participated in Wave III was 1,098 (434 MZ and 664 DZ), with 872 twins (434 MZ and 438 DZ) in same-sex pairs. The Add Health data has been widely used for twin studies (Fowler, Baker, & Dawes, 2008; Harris, Halpern, Smolen, & Haberstick, 2006).

Allelic information for a number of genetic markers was collected for 2,574 individuals as part of Wave III. The candidate genotypes were chosen for inclusion in the study because they are known to affect brain development, neurotransmitter synthesis and reception, and hormone regulation. Details of the DNA collection and genotyping process are available at the Add Health website (Add Health Biomarker Team, 2007). These candidate genes include markers that identify alleles (variants) of the serotonin transporter gene or 5-HTT. The promotor region of 5-HTT contains a variable number tandem repeat (VNTR) sequence that influences transcriptional activity—the “long” 528 base-pair allele is associated with a higher basal activity than the “short” 484 base-pair allele. This functional polymorphism on the 5-HTT gene is commonly referred to as 5-HTTLPR (5-HTT-linked polymorphic region). Allele frequency for the short allele is 43% and for the long allele is 57%.



In 2012, Add Health released a second batch of genotypical data that extended to almost all Wave III participants and included the 5-HTTLPR genotype. The distributional frequencies of this new sample (used here for replication purposes) closely align with the earlier discovery sample.

In Wave III, subjects were asked “How satisfied are you with your life as a whole?” Answer categories ranged from very dissatisfied, dissatisfied, neither satisfied nor dissatisfied, satisfied, to very satisfied. Alternative answers were “refused” or “don’t know,” and these were discarded for the purpose of this study (<1% of interviewees gave such a response). This question-and-answer formulation is standard in the economics of happiness literature (Di Tella et al., 2001, 2003; Frey, 2008; Kahneman & Krueger, 2006). The distribution of answers to the life satisfaction question is shown in the Appendix. In line with the happiness literature, a large majority of respondents report being satisfied or very satisfied (Frey & Stutzer, 2002a). That most people, in fact, report a positive level of subjective well-being is the object of an article by Diener and Diener (1996), where the authors find this distribution to be representative in a wide cross-national analysis.

## Twin Design

### Method

Twin studies compare the traits, behaviors, and other outcomes (called “phenotypes”) of twins who share 100% of their genetic material (identical or *monozygotic* [MZ] twins) to those who share 50% of their genetic material (fraternal or *dizygotic* [DZ] twins) to estimate the relative importance of genetic and environmental influences (Ashenfelter & Krueger, 1994; Taubman, 1976). Following Benjamin et al. (2012), if we assume that the residual factors covary equally MZ and DZ twins (informally called the “equal environments” assumption), and there are no gene–environment interactions, then the variance in happiness can be decomposed into additive genetic effects (A), common or shared environmental influences (C), and unshared or unique environmental influences (E). The ACE model does not allow us to observe environmental and genetic influences directly, but it does allow us to estimate these

effects by observing the covariance across MZ and DZ twins.

Although the assumptions underlying the ACE model are strong, the method produces results that have been validated in numerous other studies. For example, studies of twins reared apart generate similar heritability estimates to those generated by studies of twins raised together (Bouchard, 1998). More recently, Visscher et al. (2006) utilize the small variance in percentage of shared genes among DZ twins to estimate heritability without using any MZ twins, and they are able to replicate findings from studies of MZ and DZ twins reared together. Moreover, personality and cognitive differences between MZ and DZ twins persist even among twins whose zygosity has been miscategorized by their parents, indicating that being mistakenly treated as an identical twin by one's parents is not sufficient to generate a difference in concordance (Kendler, Neale, Kessler, Heath, & Eaves, 1993; Scarr & Carter-Saltzman, 1979; Xian et al., 2000).

The ACE model can be formally expressed as:

$$y_{ij} = \mu + A_{ij} + C_j + E_{ij}$$

where  $y$  is the measure of the phenotype,  $j$  denotes the family,  $i$  denotes the individual twin in the family,  $\mu$  is the mean of this phenotype across all observations,  $A_{ij} \sim N(0, \sigma_A^2)$  is the additive genetic component,  $C_j \sim N(0, \sigma_C^2)$  is the shared environment component, and  $E_{ij} \sim N(0, \sigma_E^2)$  is the unshared environment component. Notice that these assumptions imply:

$$\text{Var}(y) = \sigma_A^2 + \sigma_C^2 + \sigma_E^2.$$

If we further assume that the unshared environment is uncorrelated between twins ( $\text{COV}(E_{1j}, E_{2j}) = 0$ ), that genes are perfectly correlated between MZ twins ( $\text{COV}_{\text{MZ}}(A_{1j}, A_{2j}) = \sigma_A^2$ ), and the covariance between DZ twins who share half their genes on average is half that of identical twins ( $\text{COV}_{\text{DZ}}(A_{1j}, A_{2j}) = \frac{1}{2}\sigma_A^2$ ), then we have two additional equations

$$\text{COV}_{\text{MZ}}(y_{1j}, y_{2j}) = \sigma_A^2 + \sigma_C^2,$$

$$\text{COV}_{\text{DZ}}(y_{1j}, y_{2j}) = \frac{1}{2}\sigma_A^2 + \sigma_C^2.$$



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The covariance equations reflect the fact that DZ twins share on average 50% of their genes whereas MZ twins share all of their genes. Based on these equations, we can estimate the ACE model via a random effects regression model with the  $2 \times 2$  variance-covariance matrix specified as:

$$\Omega_j = \begin{bmatrix} \sigma_A^2 + \sigma_C^2 + \sigma_E^2 & R_j\sigma_A^2 + \sigma_C^2 \\ R_j\sigma_A^2 + \sigma_C^2 & \sigma_A^2 + \sigma_C^2 + \sigma_E^2 \end{bmatrix}$$

where  $R$  is the genetic relatedness of the twin pair equaling 1 for MZ twins and  $\frac{1}{2}$  for DZ twins. We

use the variances of the random effects to generate estimates of heritability, common environment, and unshared environment.<sup>2</sup>

To generate the ACE estimates, we use the structural equation modeling program *OpenMx* developed by Neale, Boker, Xie, & Maes, (2010). In addition to estimating ACE models, we estimate all of the possible submodels to compare model fit. These include an AE model, which assumes only genes and unshared environment influence the phenotype ( $C = 0$ ), a CE model which assumes only common and unshared environment influence the phenotype ( $A = 0$ ), and an E model ( $A = 0$  and  $C = 0$ ). If a submodel fits better than the general ACE model, this suggests the parameters left out of the submodel are not significantly contributing to model fit. To compare the submodels, we use the Akaike Information Criterion (AIC) in maximum likelihood estimation, where smaller values indicate better fit.

### Twin Results

When assessing the role of genetic influences, the first step is to compare the correlation in phenotype among MZ twin pairs with that of DZ twin pairs. For life satisfaction, the correlation coefficient for MZ twins is 0.334 and for DZ twins is 0.132. The difference in correlations is significant ( $p = .013$ , one-sided). These correlations show that identical twins are significantly more similar in their level of happiness than fraternal twins, which suggests that genetic factors might play a role in this trait.

In Table 1, we report results from several variance decomposition models described earlier. Note that the ACE model yields a herita-

bility estimate of 33% ( $SE = 0.044$ ), and the estimate for unshared environment is 67% ( $SE = 0.029$ ), while a null estimate is returned for common environment. In other words, about a third of the variance in happiness in our sample can be attributed to variance in genetic factors. We also examine the submodels and find that the models with lowest AIC all include A, suggesting that the finding that happiness is heritable is robust to different model specifications.<sup>3</sup>

Compared with previous studies of happiness, our heritability estimate of 33% is on the lower end of reported estimates. In fact, the seminal paper by Lykken and Tellegen (1996) estimated heritability at about 50%, and subsequent estimates ranged from 38% (Stubbe, Posthuma, Boomsma, & De Geus, 2005) to 36%–50% (Bartels & Boomsma, 2009) to 42%–56% (Nes, Roysamb, Tambs, Harris, & Reichborn-Kjennerud, 2006). However, the Add Health study includes other questions that suggest the heritability of happiness rises as people age. The standard life satisfaction question used in this article is only asked of Add Health subjects in Wave III (2001–20002), but in other interview waves the following question is asked of participants: “How often was the following true during the past seven days? You felt happy.” Answers range from “never or rarely” to “most of the time or all of the time.” Figure 1 in Supplementary Online Material shows the MZ and DZ twin pair correlations of the time series that combines the “life satisfaction” and “You felt happy” questions. The basic heritability estimates that result from comparing MZ and DZ correlations range from 22% in Wave I (1994) to 54% in Wave IV (2008). This longitudinal analysis is consistent with a growing body of longitudinal twin research that shows that the heritability of a number of traits (e.g., intelligence) increases with age (Plomin, DeFries, McClearn, & McGuffin, 2008). It also shows

<sup>2</sup> They are defined as  $\frac{\sigma_A^2}{\sigma_A^2 + \sigma_C^2 + \sigma_E^2}$ ,  $\frac{\sigma_C^2}{\sigma_A^2 + \sigma_C^2 + \sigma_E^2}$ , and  $\frac{\sigma_E^2}{\sigma_A^2 + \sigma_C^2 + \sigma_E^2}$  respectively.

<sup>3</sup> When we split our twin sample by sex we find that there are significant differences between men and women. As in Table 1, the results given in Table 1 in Supplementary Online Material show that the AE models fit happiness best according to the AIC values. However, the heritability estimate for males is 39%, whereas for females it is 26%.



Table 1  
Summary of ACE Twin Model Results

|     | Life satisfaction |       |       | Fit statistics |        |     | Akaike Information Criterion | diff -2ll | diff df | p    |
|-----|-------------------|-------|-------|----------------|--------|-----|------------------------------|-----------|---------|------|
|     | $a^2$             | $c^2$ | $e^2$ | ep             | -2ll   | df  |                              |           |         |      |
| ACE | 0.331             | 0.000 | 0.669 | 4              | 1878.9 | 795 | 288.9                        | —         | —       | —    |
| AE  | 0.331             | —     | 0.669 | 3              | 1878.9 | 796 | 286.9                        | 0         | 1       | 1    |
| CE  | —                 | 0.257 | 0.743 | 3              | 1882.9 | 796 | 290.9                        | 4         | 1       | 0.05 |
| E   | —                 | —     | 1     | 2              | 1907.2 | 797 | 313.2                        | 28.3      | 2       | 0    |

Note. The models consist of additive genetic factors (A), shared or common environmental factors (C), and unshared environmental factors (E). The model includes 217 monozygotic and 219 dizygotic same-sex twin pairs.

that the finding that happiness is heritable is robust to a variety of measures and time periods over the life course. These findings are generally taken to mean that genes and environment can play differing roles in explaining experience at different points in the life course.

### Genetic Association

Twin studies are important because they allow us to gauge the relative influence of our genetic makeup on subjective well-being. However, twin studies do not give insight into which specific genes may be involved in explaining the heritability of traits. Because Add Health collected a number of specific genetic markers, it presents us with a unique opportunity to move beyond a twin design study. Below we introduce some basic concepts in genetics, our genetic association research design, and present discovery and replication results for our candidate gene study.

### Basic Concepts in Genetics

Human DNA is composed of an estimated 21,000 genes that form the blueprint for molecules that regulate the development and function of the human body. Genes are distinct regions of human DNA that are placed in the 23 pairs of chains, or chromosomes that make up all human DNA. Almost all human cells contain the same DNA they inherited at the moment of conception.

Individuals inherit one-half of their DNA from each parent, with one copy of each gene coming from the mother and one copy from the father. Some genes come in different versions, known as “alleles”—for example, sickle cell disease results from a particular

allele coding for abnormal rather than normal hemoglobin. Each parent has two separate copies of an allele at each “locus,” or location, on the chromosome, but each sperm or egg cell contains only one of these alleles. Thus a child has a 50% chance of receiving a particular allele from a particular parent. For example, suppose that at a given locus there are two possible alleles, A and B. If both parents are “heterozygous” at that locus, meaning they each have an A and a B allele (AB or BA—order is irrelevant), then a given offspring has a 25% chance of being “homozygous” for A (AA), a 25% chance of being homozygous for B (BB) and a 50% chance of being heterozygous (AB or BA). If an individual is heterozygous at a locus, a “dominant” allele may impose itself on the “recessive” allele, and the expression of the latter allele will not be observed.

Genes transcribe proteins that begin a cascade of interactions that regulate bodily structure and function. Many of the observable traits and behaviors of interest, referred to as “phenotypes” are far downstream from the original “genotypes” present in the DNA. While in some cases, one allele can single-handedly lead to a disease (such as sickle cell anemia, Huntington’s disease, or cystic fibrosis), the vast majority of phenotypes are “polygenic,” meaning they are influenced by multiple genes (Mackay, 2001; Plomin et al., 2008) and are shaped by a multitude of environmental forces. As a result, association models between genotypes and phenotypes are an important first step, but they are not the end of the story. It is also important to investigate the extent to which genetic associations are moderated by environmental factors and other genes.



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**5-HTTLPR, Serotonin, and Happiness**

One strategy in behavioral genetics is to start with a “candidate” gene that is thought to influence behaviors or processes in the body that are related to the phenotype of interest. For subjective well-being, this means focusing on genes that affect brain development, neurotransmitter synthesis and reception, hormone regulation, and transcriptional factors (Benjamin et al., 2007; Damberg, Garpenstrand, Hallman, & Orelund, 2001).

We choose a candidate gene that has already received a great deal of attention by biologists and social scientists for its association with mental states. The 5-HTT gene is critical to the metabolism of serotonin in the brain. As shown in Figure 1, serotonin is a chemical that is released by a neuron and sensed by a receptor on the receiving neuron, passing an electric potential across a gap called a nerve synapse (the nerve that emits the serotonin is on the “presynaptic” side of the gap). Signals are carried throughout the body by the sequential release of a neurotransmitter by one neuron after another across these synapses. The 5-HTT gene codes for the serotonin transporters that are placed in the cell wall and reabsorb the neurotransmitter serotonin from the synaptic cleft. Most serotonin is recycled after use and the serotonin transporter allows serotonergic neurons to restock. The serotonin transporter gene has been studied extensively and much is known about the way different versions of this gene influence serotonergic neurotransmission which, in turn, is found to influence personality and mental health (Canli & Lesch, 2007; Hariri & Holmes, 2006; Hariri et al., 2002).

The 5-HTT gene contains a 44 base-pair variable-number tandem repeat (VNTR) polymorphism<sup>4</sup> in the promoter region<sup>5</sup> (5-HTTLPR) that is believed to be responsible for variation in transcriptional efficiency. The “long” (528 bp) and “short” (484 bp) polymorphism produce the same protein, but the long allele is associated with an approximately three times higher basal activity than the shorter allele. As a consequence, the long variant produces significantly more 5-HTT mRNA<sup>6</sup> and protein (Canli & Lesch, 2007; Glatz, Mössner, Heils, & Lesch, 2003; Heils et al., 1996; Lesch et al., 1996; Little et al., 1998). The long polymorphism thus results in increased gene expression and more

serotonin transporters in the cell membrane. In turn, more serotonin is reintroduced into the presynaptic cell. This process is also shown in Figure 1.

Functional variation in the serotonin transporter gene is increasingly understood to exert influence on parts of the brain regulated by serotonergic neurotransmission. In particular, research shows increased amygdala activation to negative emotional stimuli among carriers of short alleles (Canli, Omura, Haas, Fallgatter, & Constable, 2005; Hariri et al., 2002; Heinz et al., 2005; Munafò, Brown, & Hariri, 2008; Pezawas et al., 2005). A morphometrical study of this genetic association reports reduced gray matter volume in short-allele carriers in limbic regions critical for processing of negative emotion, particularly perigenual cingulate and amygdala (Pezawas et al., 2005). These authors conclude that 5-HTTLPR induced variation in anatomy and function of an amygdala-cingulate feedback circuit critical for emotion regulation indicates one mechanism for a genetic susceptibility for depression (Pezawas et al., 2005). Another morphometrical study corroborates the finding that short-allele carriers show decreased volume in the affective division of the anterior cingulate and decreased gray matter density in its pregenual region (Canli et al., 2005). The same study also finds that the 5-HTTLPR polymorphism is associated with activation changes to positive stimuli, suggesting a general role in emotional regulation, rather than negative valence specifically (Canli et al., 2005).

Myriad behavioral studies also suggest that serotonin and 5-HTT play an important role in emotional regulation (Hariri & Holmes, 2006; Hariri et al., 2002; Heils et al., 1996). Specifically, variance in 5-HTTLPR was found to be associated with variation in mental health outcomes (Lesch et al., 1996), and subsequent studies report that about 10% of the variance in anxiety-related traits depends on variation in

<sup>4</sup> A VNTR polymorphism is a repeated segment of DNA that varies among individuals in a population.

<sup>5</sup> A promoter region is the regulatory region of DNA that tells transcription enzymes where to begin. These promoter regions typically lie upstream from the genes they control.

<sup>6</sup> Messenger RNA (mRNA) is a type of RNA that carries information from DNA to ribosomes. In turn, these ribosomes “read” messenger RNAs and translate their information into proteins.



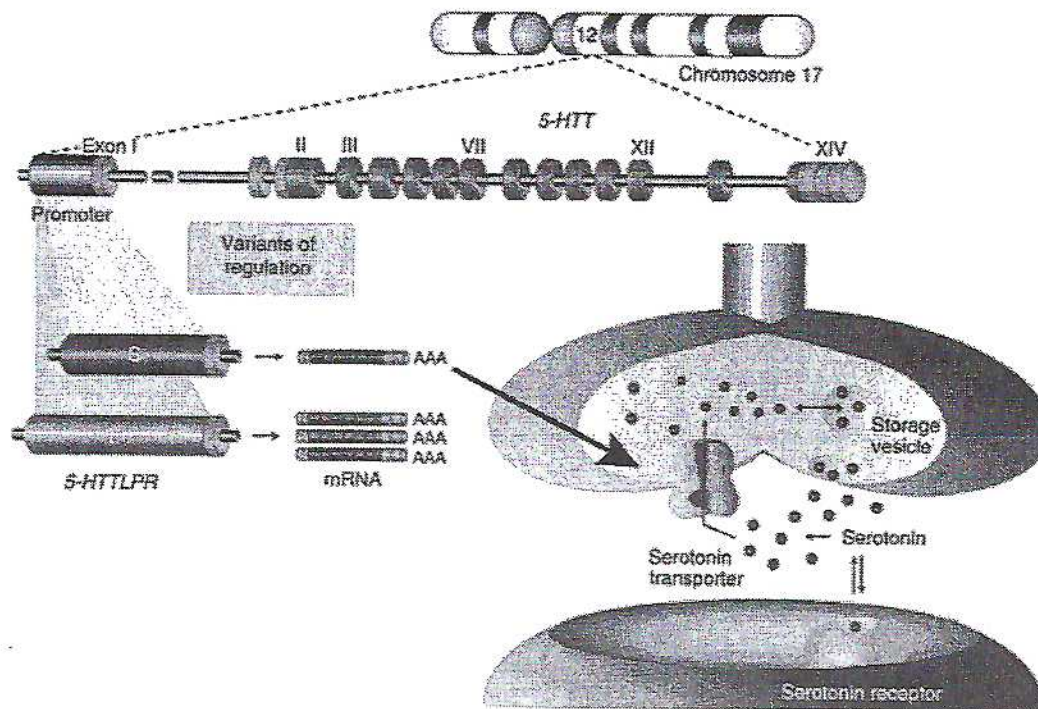


Figure 1. Representation of the long/short functional variant on the 5-HTT gene and the release, reception, and recycling of serotonin in neurons. Adapted from Canli and Lesch (2007), with permission from the Nature Publishing Group.

serotonin transporters (Munafò, Clark, & Flint, 2005; Sen, Burmeister, & Ghosh, 2004). A recent study by Fox et al. (2009) also suggests that 5-HTTLPR may influence optimism. The authors obtained DNA from about 100 participants and compared reaction times with pictures with positive, negative, and neutral emotional valence (replicating a common experiment in psychopathology research). The results show that individuals with the transcriptionally more efficient 5-HTTLPR alleles display a significant bias toward processing positive information and selectively avoiding negative information. This emotionally self-protective pattern does not obtain in individuals carrying one or both short alleles. It is important to note, however, that positive and negative emotions are not different sides to the same coin. A score of zero on a depression or anxiety scale may be indicative of the absence of such mental health issues, but it is not indicative of the presence of happiness (McGreal & Stephen, 1993).

Not all studies show a direct relationship between a gene variant and a phenotype. In-

stead, developmental or concurrent environments may moderate an association between genes and phenotypes. A study by Caspi et al. (2003) suggests a gene-environment interaction for the influence of life stress on depression. The authors find that individuals with short 5-HTTLPR alleles are more vulnerable to stress-induced depression. Among those individuals that had experienced a relatively large number of stressful life events, about 33% of the carriers of the less efficient short allele were cases of diagnosed depression compared with only 17% of the individuals that carried both long alleles. Thus, in the Caspi et al. (2003) study, the gene itself is not associated with depression. Rather, it is the combination of both gene and environment that yields a significant association. In this study we do not report on a gene-environment interaction, but the direct association between the number of long 5-HTTLPR alleles and life satisfaction.

It is important to highlight, however, that later studies often fail to replicate candidate gene discoveries. For example, a meta-analysis



by Risch et al. (2009) that incorporated 14 studies yielded no evidence for a direct effect between 5-HTTLPR and depression nor an indirect effect moderated by stressful life events.

### Association Methods

Genetic association studies test whether an allele or genotype occurs more frequently within a group exhibiting a particular phenotype than those without the phenotype. However, a significant association can mean one of three things: (a) The allele itself influences subjective well-being; (b) the allele is in “linkage disequilibrium” with an allele at another locus that influences subjective well-being; or (c) the observed association is a false-positive signal because of population stratification.<sup>7</sup>

Population stratification occurs because groups may have different allele frequencies because of their genetic ancestry. Subjective well-being in these groups may be the product of their environments, alleles other than the one of interest, or some unobserved reason. For example, two groups may not have mixed in the past for cultural reasons. Through the process of local adaptation or genetic drift, these groups may develop different frequencies of a particular allele. At the same time, the two groups may also develop divergent behaviors that are not influenced by the allele but solely by the environment in which they live. Once these two groups mix in a larger population, simply comparing the frequency of the allele to the observed behavior would lead to a spurious association.

There are two main research designs employed in association studies, case-control designs and family based designs. Case-control designs compare the frequency of alleles or genotypes among subjects that exhibit a phenotype of interest to subjects who do not. As a result, case-control designs are vulnerable to population stratification if either group is especially prone to selection effects. A typical way to control for this problem is to include controls for the race or ethnicity of the subject or to limit the analysis to a specific racial or ethnic group. Family based designs eliminate the problem of population stratification by using family members, such as parents or siblings, as controls. Tests using family data compare whether offspring exhibiting the trait receive a risk allele from their parents more often than would be

expected by chance. This design is very powerful in minimizing Type I error but also suffers from much lower power in detecting a true association. Xu and Shete (2006) show, based on extensive simulation work, that a case-control association study using mixed-effects regression analysis outperforms family based designs in detecting an association while at the same time effectively limiting Type I error.

To test for genetic association we employ a mixed-effects ordinary least squares (OLS) regression model<sup>8</sup>:

$$Y_{ij} = \beta_0 + \beta_G G_{ij} + \beta_k Z_{kij} + U_j + \varepsilon_{ij}$$

where  $i$  and  $j$  index subject and family respectively. For the 5-HTT gene,  $G = 2$  if the subject's genotype is LL,  $G = 1$  for genotypes LS or SL, and  $G = 0$  if the subject's genotype is SS (where L represents having a copy of a 528 base-pair “long” allele, and S represents having a copy of a 484 base-pair “short” allele).  $Z$  is a matrix of variables to control for the underlying population structure of the Add Health sample as well as potentially mediating factors such as age, gender, education, religiosity, marriage, job, welfare, or medication that may all influence subjective well-being. Finally, the variable  $U$  is a family random effect that controls for potential genetic and environmental correlation among family members, and  $\varepsilon$  is an individual-specific error.

To control for the effects of the underlying population structure, we include indicator variables for whether a subject self-reported as

<sup>7</sup> Given our data, we cannot differentiate between 1 and 2. To do so, we would need additional genetic information about loci in close proximity to the locus of interest. Thus, a significant association means that either a particular allele, or one likely near it on the same gene, significantly influences subjective well-being.

<sup>8</sup> The choice between OLS and ordered probit regression analysis rests on whether the categories of the life satisfaction are considered cardinal or ordinal. Economists typically consider these happiness scores as ordinal and have mainly opted for the ordered type of analysis. Psychologists and sociologists interpret happiness categories as cardinal and therefore use OLS. Ferrer-i-Carbonell and Frijters (2004) survey and test both empirical literatures to conclude that assuming cardinality or ordinality of happiness surveys makes little difference in studies where the dependent variable is measured at a single point in time. We opted for OLS, but other analyses using ordered probit reveal no meaningful differences in coefficients or significance.



Black, Hispanic, or Asian (base category is White). Following the policy of the United States Census, Add Health allows respondents to mark more than one race. Since this complicates the ability to control for stratification, we exclude these individuals ( $N = 117$ ), but a supplementary analysis including them yields substantively equal results. Population stratification is a pertinent challenge in our sample. The Hardy-Weinberg equilibrium (HWE) test indicates a significant deviation from the expected frequencies ( $\chi^2 = 7.66, p = .005$ ). On the other hand, when considering the separate HWE test statistics by ethnicity there are no longer significant deviations from the expected HWE frequencies (see Table 6 in Supplementary Online Material). As such, introducing ethnicity controls and running a Whites-only case control test (Xu and Shete, 2006) may adequately control for population stratification.

#### Association Results: Add Health Discovery Sample

Table 2 shows the results of several specifications of the model to test the hypothesis that the 5-HTTLPR long allele is associated with subjective well-being in the original Add Health discovery sample.<sup>9</sup> Each of these specifications includes variables for age, gender, and race to control for population stratification. *Model 1* shows that the long allele is significantly associated with increased life satisfaction ( $p = .012$ ). In Figure 2, we summarize the results for 5-HTTLPR by simulating first differences from the coefficient covariance matrix of *Model 1*. Holding all else constant and changing the 5-HTTLPR variant for all subjects from zero to one long allele would increase the reporting of being very satisfied with one's life in this population by about 8.5%. Similarly, changing the 5-HTTLPR variant from zero to two long alleles would increase the reporting of being very satisfied by about 17.3%.

*Model 2* includes a number of socioeconomic factors that are known to influence subjective well-being. In particular, having a job, education, marriage, divorce, religiosity, welfare assistance, and being on medication. This model also suggests that there is a statistically significant association ( $p = .005$ ) between the 5-HTTLPR long variant and the reporting of life satisfaction. Notice also that the coefficient

actually increases a bit, suggesting that the association cannot be explained by a mediation effect this genotype may have on any other variables included in the model.<sup>10</sup>

Following Xu and Shete (2006), as a robustness test for population stratification, we also include *Model 3* that is a case-control association model for those subjects that uniquely identified themselves as being white. The coefficient on 5HTTLPR and its  $p$  value ( $p = .017$ ) suggest that population stratification between self-reported racial categories is not driving the association between 5-HTTLPR and life satisfaction in the Add Health discovery sample.

#### Replication Studies

Specific genotypes usually only account for a very small amount of the variance in complex social behaviors, which means the tests often have low power. As a result, it is very important to replicate results in independent samples (Beauchamp et al., 2011; Benjamin et al., 2012). Such efforts to replicate a significant genetic association result, as well as increasing the sample sizes, are key in addressing the possibility that the original association would be a spurious result or false positive. Below we first report on our replication effort in the FHS. More recently, the release of new genotypical data for the Add Health data allowed for another replication effort which we also detail below. Although the association between functional variation on the 5-HTT gene and life satisfaction found in the Add Health discovery sample replicates in the Framingham sample it does not replicate in the new Add Health sample.

**Replication Study 1: FHS.** Here we utilize the FHS, a population-based, longitudinal, observational cohort study that was initiated in 1948 to prospectively investigate risk factors for cardiovascular disease. Since then, the FHS has come to be composed of four separate but related cohort populations: (a) the Original Co-

<sup>9</sup> This genetic association result in the Add Health discovery sample is also reported in De Neve (2011).

<sup>10</sup> We also report the results of association tests with 5-HTTLPR for each of these socioeconomic factors in the appendix. An association with medication is nearly significant ( $p = .08$ ) but loses its significance ( $p = .17$ ) when controlling for age, gender, and race. Hence, medication cannot be considered a mediating variable (Baron & Kenny, 1986).



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Table 2  
*OLS Models of Association Between 5-HTTLPR and Life Satisfaction (Discovery Sample)*

|                | Model 1     |       |      | Model 2     |       |      | Model 3     |       |      |
|----------------|-------------|-------|------|-------------|-------|------|-------------|-------|------|
|                | Coefficient | SE    | p    | Coefficient | SE    | p    | Coefficient | SE    | p    |
| 5-HTTLPR long  | 0.059       | 0.023 | .012 | 0.065       | 0.023 | .005 | 0.070       | 0.029 | .017 |
| Black          | −0.111      | 0.048 | .021 | −0.114      | 0.049 | .020 |             |       |      |
| Hispanic       | 0.198       | 0.117 | .092 | 0.216       | 0.118 | .067 |             |       |      |
| Asian          | −0.196      | 0.073 | .007 | −0.221      | 0.071 | .002 |             |       |      |
| Age            | 0.004       | 0.009 | .705 | −0.011      | 0.009 | .262 | −0.031      | 0.012 | .008 |
| Male           | 0.014       | 0.033 | .682 | 0.028       | 0.033 | .406 | 0.039       | 0.041 | .341 |
| Job            |             |       |      | 0.093       | 0.041 | .024 | 0.104       | 0.057 | .071 |
| College        |             |       |      | 0.115       | 0.033 | .001 | 0.238       | 0.042 | .000 |
| Married        |             |       |      | 0.232       | 0.041 | .000 | 0.318       | 0.050 | .000 |
| Divorced       |             |       |      | −0.313      | 0.153 | .041 | −0.310      | 0.155 | .047 |
| Religiosity    |             |       |      | 0.103       | 0.017 | .000 | 0.082       | 0.023 | .000 |
| Welfare        |             |       |      | −0.236      | 0.098 | .017 | −0.121      | 0.153 | .432 |
| Medication     |             |       |      | −0.045      | 0.032 | .162 | −0.095      | 0.041 | .021 |
| Intercept      | 4.078       | 0.208 | .000 | 4.096       | 0.210 | .000 | 4.514       | 0.262 | .000 |
| N              |             | 2,545 |      |             | 2,528 |      |             | 1,446 |      |
| R <sup>2</sup> |             | 0.01  |      |             | 0.06  |      |             | 0.08  |      |

Note. Variable definitions are in the Appendix. SEs and p values are also presented.

hort enrolled in 1948 ( $N = 5,209$ ); (b) the Offspring Cohort (the children of the Original Cohort and spouses of the children) enrolled in 1971 ( $N = 5,124$ ); (c) the Omni Cohort enrolled in 1994 ( $N = 508$ ); and (d) the Generation 3 Cohort (the grandchildren of the Original Co-

hort) enrolled beginning in 2002 ( $N = 4,095$ ). Published reports provide details about sample composition and study design for all these cohorts (Cupples & D'Agostino, 1988; Kannel, Feinleib, McNamara, Garrison, & Castelli, 1979).

The FHS makes available genetic markers for its participants. Of the 14,428 members of the three main cohorts, a total of 9,237 individuals have been genotyped (4,986 women and 4,251 men) for single nucleotide polymorphisms (SNPs). These are specific locations on human DNA where a single pair of nucleotides varies for some part of the human population. FHS makes available a data set of expected genotypes for all 2,543,887 SNPs in the European ancestry HapMap sample that was computed from the 550,000 observed SNPs from an Affymetrix array using the program MACH (for information on how this data set was constructed, see De Bakker, 2008). Although this data does not contain the same VNTR polymorphism marker for 5-HTT that we analyze in Add Health, it does contain a nearby marker called "rs2020933," and the "A" allele of this marker is known to be associated with higher transcriptional efficiency of serotonin transporters (Fahad et al., 2010; Lipsky, Hu, & Goldman, 2009; Martin, Cleak, Willis-Owen, Flint, & Shifman, 2007; Wendland, Martin, Kruse, Lesch, & Mur-

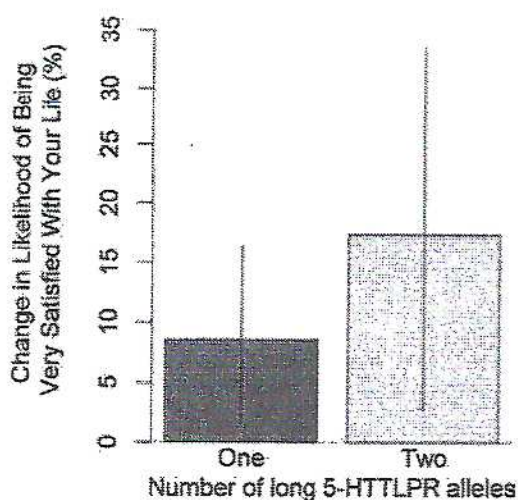


Figure 2. Increasing the number of "long," more efficient 5-HTTLPR alleles is associated with higher life satisfaction in the Add Health discovery sample. First differences based on simulations of Model 1 parameters are presented along with 95% confidence intervals. All other variables are held at their means.



phy, 2006). It is also known to be in positive linkage disequilibrium with the long allele of 5-HTTLPR (Huezo-Diaz et al., 2009). The FHS also asked 3,460 participants in the offspring cohort a variant of the life satisfaction question: "Indicate where you think you belong between these two extremes ... satisfied with job or home life OR ambitious, want change." Respondents were given a 7-point scale to choose from, and we reverse-coded the scale so that higher values indicated greater satisfaction with life ( $M = 4.7$ ,  $SD = 1.7$ ). Although this question is not exactly like the one asked in Add Health, if there is a real association between 5-HTT and happiness, we expect it to show up in spite of variations in the way the question is asked.

We merged the gene and life satisfaction data and conducted an association test using a linear regression with a general estimating equations (GEEs) approach to account for within-family correlation of errors. As shown in Model 1 in Table 3, this association is significant ( $p = .05$ ) and in the expected direction. In Model 2 we include additional controls for age and gender. We also include the first 10 principal components of a singular value decomposition of the subject-genotype matrix in the regression (see Appendix), which has been shown to effectively

control for population stratification (Price et al., 2006). Once again, the association is significant ( $p = .05$ ).

**Replication Study 2: New Add Health Data.** The staggered release of genotypical data by Add Health provides another opportunity to test the association between the 5-HTTLPR genotype and life satisfaction in an independent replication sample as well as the larger combined sample. Tables 4 (replication sample) and 5 (full sample) present results for association models that are identical to those run in the discovery sample and shown in Table 2. While all model specifications return coefficients on 5-HTTLPR that indicate a positive correlation between the long alleles of this genotype and life satisfaction, no model specification obtains statistical significance. We thus fail to replicate the original significant association result in the newly available Add Health data.

### Discussion

Our main objective here has been to provide empirical evidence that genes matter for subjective well-being and to encourage economists to consider the importance of biological differences. The results we present ad-

Table 3  
General Estimating Equations Models of Association Between rs2020933 and Life Satisfaction  
(Framingham Heart Study)

|                        | Model 1     |       |     | Model 2     |       |     |
|------------------------|-------------|-------|-----|-------------|-------|-----|
|                        | Coefficient | SE    | p   | Coefficient | SE    | p   |
| rs2020933 "A" alleles  | 0.22        | 0.11  | .05 | 0.21        | 0.11  | .05 |
| Age                    |             |       |     | 0.04        | 0.00  | .00 |
| Male                   |             |       |     | -0.00       | 0.06  | .99 |
| Principal component 1  |             |       |     | -0.88       | 1.57  | .58 |
| Principal component 2  |             |       |     | 0.04        | 6.43  | .99 |
| Principal component 3  |             |       |     | -3.32       | 2.21  | .13 |
| Principal component 4  |             |       |     | -1.08       | 2.33  | .64 |
| Principal component 5  |             |       |     | -3.30       | 2.64  | .21 |
| Principal component 6  |             |       |     | 1.13        | 2.45  | .65 |
| Principal component 7  |             |       |     | 2.21        | 1.97  | .26 |
| Principal component 8  |             |       |     | -2.10       | 2.21  | .34 |
| Principal component 9  |             |       |     | -0.52       | 2.06  | .80 |
| Principal component 10 |             |       |     | -1.82       | 2.26  | .42 |
| Intercept              | 4.68        | 0.04  | .00 | 2.90        | 0.16  | .00 |
| N                      |             | 2,843 |     |             | 2,831 |     |
| R <sup>2</sup>         |             | 0.01  |     |             | 0.05  |     |

Note. SEs and p values are also presented.



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Table 4  
Replication Sample OLS Models of Association Between 5-HTTLPR and Life Satisfaction

|                | Model 1     |        |      | Model 2     |        |       | Model 3     |       |      |
|----------------|-------------|--------|------|-------------|--------|-------|-------------|-------|------|
|                | Coefficient | SE     | p    | Coefficient | SE     | p     | Coefficient | SE    | p    |
| 5-HTTLPR long  | 0.003       | 0.012  | .823 | 0.000       | 0.012  | 0.985 | 0.003       | 0.015 | .827 |
| Black          | −0.127      | 0.022  | .000 | −0.117      | 0.023  | 0.000 |             |       |      |
| Hispanic       | −0.067      | 0.033  | .044 | −0.067      | 0.033  | 0.042 |             |       |      |
| Asian          | −0.081      | 0.034  | .017 | −0.093      | 0.034  | 0.007 |             |       |      |
| Age            | 0.002       | 0.005  | .614 | −0.010      | 0.005  | 0.030 | −0.007      | 0.006 | .249 |
| Male           | 0.010       | 0.016  | .542 | 0.024       | 0.017  | 0.154 | −0.009      | 0.023 | .680 |
| Job            |             |        |      | 0.092       | 0.021  | 0.000 | 0.115       | 0.029 | .000 |
| College        |             |        |      | 0.200       | 0.017  | 0.000 | 0.238       | 0.042 | .000 |
| Married        |             |        |      | 0.237       | 0.022  | 0.000 | 0.278       | 0.028 | .000 |
| Divorced       |             |        |      | −0.080      | 0.061  | 0.187 | −0.056      | 0.071 | .429 |
| Religiosity    |             |        |      | 0.075       | 0.009  | 0.000 | 0.068       | 0.012 | .000 |
| Welfare        |             |        |      | −0.112      | 0.043  | 0.009 | −0.214      | 0.062 | .001 |
| Medication     |             |        |      | −0.041      | 0.018  | 0.019 | −0.046      | 0.025 | .063 |
| Intercept      | 4.119       | 0.102  | .000 | 4.107       | 0.105  | 0.000 | 4.029       | 0.139 | .000 |
| N              |             | 10,163 |      |             | 10,030 |       |             | 5,335 |      |
| R <sup>2</sup> |             | 0.004  |      |             | 0.042  |       |             | 0.055 |      |

Note. Variable definitions are in the Appendix. SEs and p values are also presented.

dress one possible source of the “baseline” or “set point” for happiness that prior work has identified (Graham, 2008; Kahneman et al., 1999). The existence of a baseline does not mean that the socioeconomic influences on happiness so far identified by researchers are unimportant. Rather, our results complement

these studies and suggest a new direction for research.

As indicated by the  $R^2$  values in Table 2—where the 5-HTTLPR genotype explains less than one percent of the variation in life satisfaction—genotype effect sizes tend to be very small. Still, contrary to a SNP, a func-

Table 5  
Full Sample OLS Models of Association Between 5-HTTLPR and Life Satisfaction

|                | Model 1     |        |      | Model 2     |        |      | Model 3     |       |      |
|----------------|-------------|--------|------|-------------|--------|------|-------------|-------|------|
|                | Coefficient | SE     | p    | Coefficient | SE     | p    | Coefficient | SE    | p    |
| 5-HTTLPR long  | 0.012       | 0.011  | .249 | 0.012       | 0.011  | .267 | 0.015       | 0.014 | .287 |
| Black          | −0.127      | 0.020  | .000 | −0.120      | 0.021  | .000 |             |       |      |
| Hispanic       | −0.077      | 0.030  | .010 | −0.070      | 0.030  | .018 |             |       |      |
| Asian          | −0.102      | 0.032  | .001 | −0.114      | 0.032  | .000 |             |       |      |
| Age            | 0.002       | 0.004  | .599 | −0.011      | 0.004  | .013 | −0.012      | 0.006 | .029 |
| Male           | 0.011       | 0.015  | .440 | 0.024       | 0.015  | .114 | −0.000      | 0.020 | .984 |
| Job            |             |        |      | 0.091       | 0.019  | .000 | 0.117       | 0.026 | .000 |
| College        |             |        |      | 0.181       | 0.015  | .000 | 0.229       | 0.020 | .000 |
| Married        |             |        |      | 0.233       | 0.019  | .000 | 0.283       | 0.025 | .000 |
| Divorced       |             |        |      | −0.124      | 0.057  | .031 | −0.122      | 0.069 | .076 |
| Religiosity    |             |        |      | 0.085       | 0.008  | .000 | 0.075       | 0.011 | .000 |
| Welfare        |             |        |      | −0.136      | 0.040  | .001 | −0.198      | 0.058 | .001 |
| Medication     |             |        |      | −0.045      | 0.032  | .162 | −0.060      | 0.022 | .006 |
| Intercept      | 4.122       | 0.092  | .000 | 4.113       | 0.095  | .000 | 4.134       | 0.124 | .000 |
| N              |             | 12,391 |      |             | 12,232 |      |             | 6,639 |      |
| R <sup>2</sup> |             | 0.004  |      |             | 0.044  |      |             | 0.058 |      |

Note. Variable definitions are in the Appendix. SEs and p values are also presented.



tional polymorphism such as the 5-HTTLPR variable number tandem repeat (VNTR) covers a broader fragment of the genome and is understood to have a potentially larger phenotypical influence (Can, Coe, & Eichler, 2011; Redon et al., 2006). Following on this, we also note that because the twin analysis suggests that all genes together account for about a third of the total variance that it is therefore highly likely that many other genes, in conjunction with environmental factors, help to explain how baseline happiness varies from one person to another.

Another use of work such as this is to address the problem of omitted variable bias (OV). A missing variable might be linked to multiple parameters and thus bias the estimate of the causal effect of *X* on *Y*. To the extent that genetic attributes are a source of OV, and to the extent that they can be added to models of economic outcomes and behaviors, accounting for such variables will improve causal estimates of other attributes.

While the Add Health study presents us with a valuable opportunity to explore a genetic basis of subjective well-being, we want to emphasize a limitation of the data. The Add Health sample is restricted to individuals who are 18–26 years old during Wave III, so our results apply only to the subjective well-being of young adults and not to people in different age categories. However, the strong similarity in the distribution of answers in the Add Health data compared with other life satisfaction surveys used in the happiness literature suggests that the age limits are not likely to gravely distort our results (Di Tella et al., 2001, 2003; Frey, 2008; Kahneman & Krueger, 2006). Our analyses in the FHS, which has a much wider age range, further suggests a degree of generalizability.

A second important limitation is that we use a case-control method that is vulnerable to population stratification. Because of limited mobility, local adaptation, and genetic drift, it is possible that people from different cultures have a different incidence of certain genotypes, which could lead to a spurious association between genotype and cultural attributes. We limit this potential threat to the validity of our results by including controls for race and limiting the analysis to a specific racial or ethnic group in Add Health. Our

related association analysis in the FHS—that controls for the first 10 principal components of a singular value decomposition of the subject-genotype matrix—has been shown to effectively deal with the problem of population stratification (Price et al., 2006).

The estimates of the influence of sociodemographic, economic, and cultural covariates on life satisfaction in Table 2 corroborate the generally identified systematic effects of these variables in the literature (for a survey, see Dolan, Peasgood, & White, 2008). In particular, gender does not systematically affect happiness. Higher age has a negative, though not statistically significant effect (this is not surprising considering that our sample refers to young adults). African Americans and Asian Americans are systematically less happy than are Whites, while Latinos are somewhat happier, but not in a statistically significant way. Better educated and married individuals report having significantly higher life satisfaction, while divorced people are more unhappy. Having a job strongly raises life satisfaction. This reflects the psychic benefits of being occupied and integrated into society. At the same time it suggests that having an income raises life satisfaction. In contrast, persons on welfare are much less happy than those employed which reflects the psychic costs of unemployment. Religious individuals are significantly more happy than those without religious beliefs. Persons with less good health, as measured by the need to be on medication, are also less happy. As is the case with most research on happiness, these estimates identify correlations, not causality, given the difficulty in disentangling endogeneity. Once again, consistency with previous studies suggests that results using the Add Health data may generalize to other populations and a wider demography in terms of age.

The life satisfaction question and answer formulation used in Add Health is standard in the economics and psychology literatures (Diener & Diener, 1996; Di Tella et al., 2001; Frey, 2008; Kahneman & Krueger, 2006). This question has been cross-validated with alternative measures that gauge subjective well-being (Bartels & Boomsma, 2009; Kahneman & Krueger, 2006) and Oswald and Wu (2010) provide objective confirmation of life satisfaction as a measure of subjective well-being. Still, the life satisfaction question has been criticized for inducing a focusing illusion by drawing attention to people's rela-



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tive standing rather than moment-to-moment hedonic experience (Kahneman, Krueger, Schkade, Schwarz, & Stone, 2006).

### Conclusion

Our results corroborate prior research in suggesting that genetic factors significantly influence individual subjective well-being. Using twin study techniques we estimate that genetic variation explains about 33% of the variance in individual happiness. Moreover, using molecular genetic methods we studied the relationship between a functional polymorphism on the serotonin transporter gene (5-HTTLPR) and life satisfaction. Generally, our results provide mixed evidence for a positive association between the “long,” more efficient 5-HTTLPR alleles and self-reported life satisfaction in a discovery sample and two replication samples. By moving beyond a twin study and focusing on specific genes, our analysis is among the first to consider pathways through which genes may influence happiness levels.

Given prior research linking the “short,” less transcriptionally efficient, alleles of the 5-HTT gene to mood disorders, and the “long,” more efficient alleles to optimism bias, we hypothesized that carriers of the “long” alleles would be more likely to report being satisfied with their lives. We find some support for this intuition in both the Add Health and FHS data, but more work needs to be done to better understand the relationship between this genotype and subjective well-being.

We have stressed that genetic factors complement, rather than substitute for, the existing studies showing the influence of sociodemographic, economic and cultural variables on life satisfaction. Future work could attempt to identify other genes or gene-environment interactions that are implicated in subjective well-being. Finding out which genes they are and what physical function they have will improve our understanding of the biological processes that underlie economic outcomes like well-being and may also shed light on their evolutionary origin (Fitzpatrick et al., 2005). While the 5-HTT gene may be a good candidate gene for further study, it is important to reemphasize that there is no single “happiness gene.” Instead, there is likely to be a set of genes whose ex-

pression, in combination with environmental factors, influences subjective well-being.

More broadly, these results suggest that integrating the unique biology of each individual, in addition to studying experience and environment, may usefully complement existing models and increase their explanatory power (Caplin & Dean, 2008). We also believe that genetic association studies such as ours may be a new catalyst for two important lines of research. First, economics places a high premium on causal inference. Provided that robust genetic associations are available and that exclusion restrictions are met, genotypes could function as instrumental variables to disentangle the reverse causality in important relationships that have been plagued by endogeneity. First attempts at using genes as instruments have been tried on the link between health and educational attainment (Beauchamp et al., 2011; Fletcher & Lehrer, 2011; Norton & Han, 2008; O'Malley, Rosenquist, Zaslavsky, & Christakis, 2010; von Hinke Kessler Scholder et al., 2010). Second, integrating genetic variation and neuroscientific research may further advance our understanding of the biological underpinnings of individual behavior. For example, the work by Urry et al. (2004) presents neural correlates of subjective well-being. Some of the neurological variation they observe may result from differences in genotypes and could thus inform and stimulate new studies. Since genes are upstream from neurological processes, a better understanding of genetic variation may bring us closer to understanding the objective sources of subjective well-being.

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## Appendix

## Variable Definitions

*5-HTTLPR long* is a variable for having 0, 1, or 2 of the 528 base-pair alleles of the 5-HTT gene (as opposed to the 484 base-pair version). The *race/ethnicity* indicator variables are based on the questions “Are you of Hispanic or Latino origin?” and “What is your race? [white/black or African American/American Indian or Native American/Asian or Pacific Islander]”. *Age* is self-reported age and *Male* is an indicator taking the value of 1 if the respondent is a male and 0 for a female. *Job* is the response to the question “Do you currently have a job?” *College* is an indicator variable taking the value 1 if the respondent completed at least one year of college and 0 for no college. It is based on the question “What is the highest grade or year of regular school you completed?” *Married* and *Divorced* are dummies derived from the population subset that have married and answered “Are you still married?” *Religiosity* relies on “To what extent are you a religious person?” and takes a value between 0 and 3 for very religious. *Welfare* is a dummy for “Are you receiving welfare?” *Medication* is a dummy for “In the past 12 months, have you taken any prescription medication—that is, a medicine that must be prescribed by a doctor or nurse?” *DRD4* is the number of r7 alleles (0, 1, or 2) as opposed to r4 alleles. *DRD2* is the number of a2 alleles (0, 1, or 2) as opposed to a1 alleles. *DAT1* is the number of r9 alleles (0, 1, or 2) as opposed to r10 alleles. *MAOA* is the number of “High” alleles (0, 1, or 2) as opposed to “Low” alleles. *rs2304297* is the number of G alleles (0,

1, or 2) for this SNP on *CHRNA6* (as opposed to C alleles). *rs892413* is the number of C alleles (0, 1, or 2) for this SNP on *CHRNA6* (as opposed to A alleles). *rs4950* is the number of G alleles (0, 1, or 2) for this SNP on *CHRNA3* (as opposed to A alleles). *rs13280604* is the number of G alleles (0, 1, or 2) for this SNP on *CHRNA3* (as opposed to A alleles). *rs2020933* is the number of A alleles (0, 1, or 2) for this SNP on 5-HTT (as opposed to T alleles).

*Principal Component 1-10* is the individual loading for each individual on the 10 principal components associated with the 10 largest eigenvalues of a singular value decomposition of the subject-genotype matrix. These 10 values contain information about population structure, so including them in an association test helps to control for population stratification (Price et al., 2006). Because principal component analysis assumes independent observations, we did not use our entire (family-based) FHS sample to construct the principal components. Instead we used a subsample of 2,507 unrelated individuals to calculate the principal components of the genotypic data and then projected the other individuals in the sample onto those principal components, thus obtaining the loadings of each individual on each of the top 10 principal components.

Received February 12, 2012

Revision received August 7, 2012

Accepted August 21, 2012 ■



## "The Story of An Hour"

Kate Chopin (1894)

- 1 Knowing that Mrs. Mallard was afflicted with a heart trouble, great care was taken to break to her as gently as possible the news of her husband's death.
- 2 It was her sister Josephine who told her, in broken sentences; veiled hints that revealed in half concealing. Her husband's friend Richards was there, too, near her. It was he who had been in the newspaper office when intelligence of the railroad disaster was received, with Brently Mallard's name leading the list of "killed." He had only taken the time to assure himself of its truth by a second telegram, and had hastened to forestall any less careful, less tender friend in bearing the sad message.
- 3 She did not hear the story as many women have heard the same, with a paralyzed inability to accept its significance. She wept at once, with sudden, wild abandonment, in her sister's arms. When the storm of grief had spent itself she went away to her room alone. She would have no one follow her.
- 4 There stood, facing the open window, a comfortable, roomy armchair. Into this she sank, pressed down by a physical exhaustion that haunted her body and seemed to reach into her soul.
- 5 She could see in the open square before her house the tops of trees that were all aquiver with the new spring life. The delicious breath of rain was in the air. In the street below a peddler was crying his wares. The notes of a distant song which some one was singing reached her faintly, and countless sparrows were twittering in the eaves.
- 6 There were patches of blue sky showing here and there through the clouds that had met and piled one above the other in the west facing her window.
- 7 She sat with her head thrown back upon the cushion of the chair, quite motionless, except when a sob came up into her throat and shook her, as a child who has cried itself to sleep continues to sob in its dreams.
- 8 She was young, with a fair, calm face, whose lines bespoke repression and even a certain strength. But now there was a dull stare in her eyes, whose gaze was fixed away off yonder on one of those patches of blue sky. It was not a glance of reflection, but rather indicated a suspension of intelligent thought.
- 9 There was something coming to her and she was waiting for it, fearfully. What was it? She did not know; it was too subtle and elusive to name. But she felt it, creeping out of the sky, reaching toward her through the sounds, the scents, the color that filled the air.
- 10 Now her bosom rose and fell tumultuously. She was beginning to recognize this thing that was approaching to possess her, and she was striving to beat it back with her will—as powerless as her two white slender hands would have been. When she abandoned herself a little whispered word escaped her slightly parted lips. She said it over and over under [her] breath: "free, free, free!" The vacant stare and the look of terror that had followed it went from her eyes. They stayed keen and bright. Her pulses beat fast, and the coursing blood warmed and relaxed every inch of her body.



- 10 She did not stop to ask if it were or were not a monstrous joy that held her. A clear and exalted perception enabled her to dismiss the suggestion as trivial. She knew that she would weep again when she saw the kind, tender hands folded in death; the face that had never looked save with love upon her, fixed and gray and dead. But she saw beyond that bitter moment a long procession of years to come that would belong to her absolutely. And she opened and spread her arms out to them in welcome.
- 11 There would be no one to live for during those coming years; she would live for herself. There would be no powerful will bending hers in that blind persistence with which men and women believe they have a right to impose a private will upon a fellow-creature. A kind intention or a cruel intention made the act seem no less a crime as she looked upon it in that brief moment of illumination.
- 12 And yet she had loved him—sometimes. Often she had not. What did it matter! What could love, the unsolved mystery, count for in the face of this possession of self-assertion which she suddenly recognized as the strongest impulse of her being! "Free! Body and soul free!" she kept whispering.
- 13 Josephine was kneeling before the closed door with her lips to the keyhole, imploring for admission. "Louise, open the door! I beg; open the door—you will make yourself ill. What are you doing, Louise? For heaven's sake open the door."
- 14 "Go away. I am not making myself ill." No; she was drinking in a very elixir of life through that open window.
- 15 Her fancy was running riot along those days ahead of her. Spring days, and summer days, and all sorts of days that would be her own. She breathed a quick prayer that life might be long. It was only yesterday she had thought with a shudder that life might be long.
- 16 She arose at length and opened the door to her sister's importunities. There was a feverish triumph in her eyes, and she carried herself unwittingly like a goddess of Victory. She clasped her sister's waist, and together they descended the stairs. Richards stood waiting for them at the bottom.
- 17 Some one was opening the front door with a latchkey. It was Brently Mallard who entered, a little travel-stained, composedly carrying his grip-sack and umbrella. He had been far from the scene of the accident, and did not even know there had been one. He stood amazed at Josephine's piercing cry; at Richards' quick motion to screen him from the view of his wife.
- 18 When the doctors came they said she had died of heart disease—of the joy that kills.

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Husband

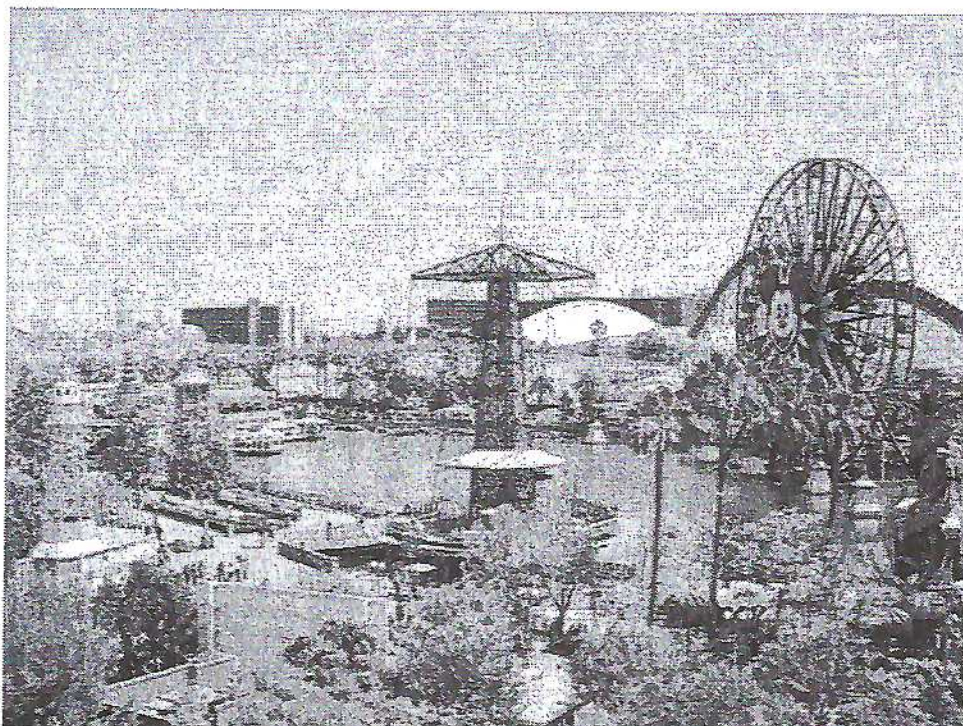


# The Happiness Project

By Andrew O'Hagan  
from *T: The New York Times Style Magazine*

July 17, 2015

*Walt Disney didn't just build a theme park for childhood fantasy. He created a world we believe in, and a journey to the land of the better self.*



Credit: Daniel Stier

*With Disneyland, Walt Disney fashioned an American nirvana in Anaheim, Calif.*

A few years ago I was driving around Hollywood in a rental car I'd inherited from the folk singer Beth Orton. We were guests under the Hollywood sign, Beth had a small baby, and it was time for her to go back to London and I kept the car. People leave stuff behind in a car; you get to know them by their parking stubs and beverage stirrers. But Beth's an original: She left me a CD compilation of the best of the Disney songs, and I cried for two weeks as I drove up Santa Monica Boulevard or over Laurel Canyon or to the beach. I cried in Griffith Park and on the freeway to a shopping mall in Sherman Oaks. I wasn't sad; I was just, well, Disneyfied — enjoying the small tearful yearnings that come with those songs. One night I stopped at a beautiful edge on Mulholland Drive and looked at the twinkling lights of the city, the years seeming to roll back with Louis Armstrong's version of "When You Wish Upon a Star."

*Fate is kind  
She brings to those who love  
The sweet fulfillment of  
Their secret longing.*

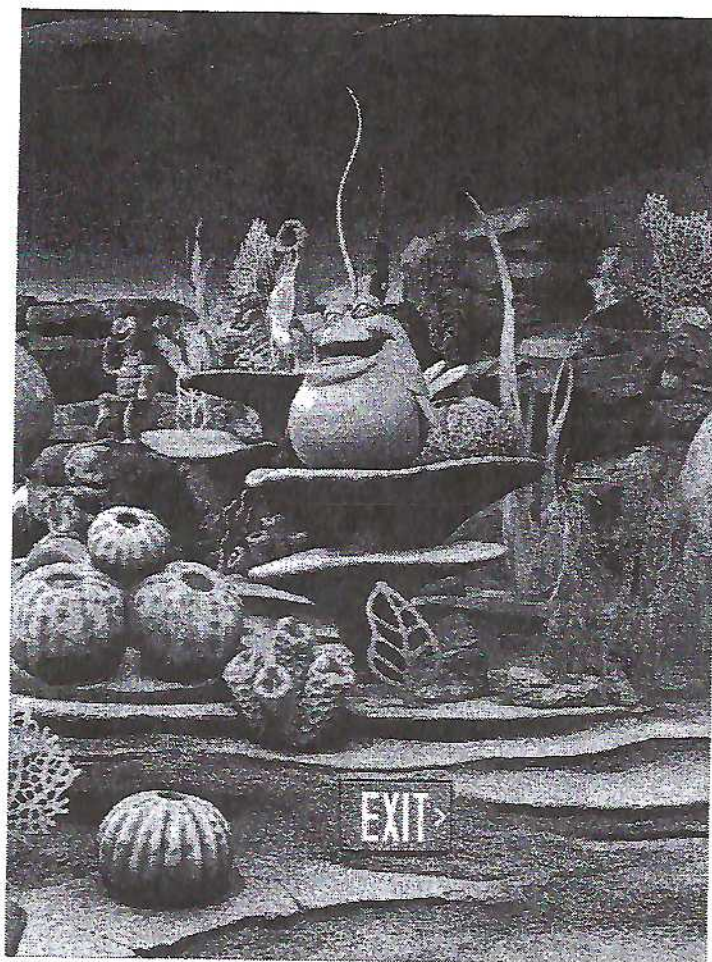


If modern sentiment has an architecture, it was Disney that built it. Disney furnished the house and chose the curtains, Disney stocked the fridge. In time, the power of Disney came to light up your hall and your landing, its vapors making their way over your vacuumed carpets and into the minds of your children. Disney offers a hand to anybody who ever dreamed that the earth is all of heaven we shall ever know. To love color, to love style, to know yourself in an element of fantasy, is to think in the terms forged by Walt Disney. Driving the roads that summer on the West Coast, I understood the significance of an ideal that had never felt wrong or tired — it was the Disney aesthetic, a delicate passion ruled by hope, a sense of necessary invention, and yet a sense of loss always embedded at the root of every extravagance.

I first saw a Disney film in the Scottish industrial town of Kilmarnock, on a typically cold and wet day in the 1970s. When foreigners think of Scotland they often think of beautiful mountains and deep shadowy glens, apple-cheeked girls wearing tartan, men in kilts drinking whisky and staring bravely into the future. In other words: They think of "Brigadoon." But it's worth remembering that when Arthur Freed, the well-known producer of musicals at MGM, was scouting locations for that film, he came back from Scotland disappointed, complaining it didn't look "Scottish enough." So the town of Kilmarnock — despite being the place from where the great poet Robert Burns published his debut poems, the famous Kilmarnock edition — was a place where dreams had traditionally come crashing down. The cinema was half-empty, and I was young enough to need a booster seat, but I'll never forget it, the return of "Snow White and the Seven Dwarfs," which seemed to me like a vision of higher possibility.

Never for a second did it appear to me implausible. Never for a moment did it look unreal or exaggerated or in any way fake. If it was fabricated or designed, then it appeared so, to me, only in the way life was fabricated or designed: Snow White and her prince and her little friends were agents in a social-realist drama, a description of society and moral life that was fundamentally true and based on observed experience. I knew it was high end. I knew it was dreamy. But it was not impossible, and it seemed we all had an obligation to live as colorfully as that, if only the gods and the reality managers would allow it. My love affair with America started when a lady from old Europe looked in a magic mirror and saw a pretty girl. My entire sense of work and play, of domestic life and love, was informed by that film, and stamped American, not because I recognized it from life, but because I wanted to. I was too young to recognize the look — the German Expressionist aspect of that film, by way of Gustave Doré — but it seeped into my unconscious and expanded for all time my wish to transport myself. My father was beside me. He believed in the innate absurdity of human effort, and the habits of the dwarfs delighted him. It's all tangled up now — it always is, and that's how Disney captures you for life, by generating tenderness mingled with regret — but the singing of "Heigh-Ho" on the car journey home will always stay with me as part of the lost essence of my family.





Credit: Daniel Stier

*Most parents have lived with the characters of "The Little Mermaid," but to travel into their "world" is to join your child in an enchantment she never knew could be realized.*

Walt Disney spent part of his childhood in Marceline, Mo., a railroad town and former prairie, 120 miles northeast of Kansas City. Its central street in the early 1900s, when Disney was a child, became a model for an idea of American happiness, and a version of it, Main Street, U.S.A., is the first thing you come across in Disneyland Park in California. Before he became king of the irresistible falsehood, Walt Disney was merely a child, and in his works we might understand what Freud viewed as the foolishness of American democracy. The Disney view of happiness — embodied in a perfect street, a cast of animals, a fairy-tale castle, a bunch of rides — might be foolish but it is also attuned to the habits of modern yearning. Freud was for bringing illusions to an end, whereas Walt Disney was for bringing them to fruition, and, in those two views of human progress, we might argue that Disney's was the more forgiving of the human condition. In the world of Disney, we feel homesick for a home that never really existed, yet everything we care about, whether being loved or feeling right or having fun or looking good, stems from a set of narcissistic compulsions that Disney embraced and built to graphic completion. That is his contribution, and, however foolish, however impossible in the end, it gives life to the notion that happiness is a creation, something made rather than



inherited, a beautiful, necessary lie. A poet once said, "There are plenty of ruined buildings in the world but no ruined stones." But who can live in a world of stones? One might argue, along with Walt Disney, that the reality we love is the reality we bought into.

I brought my daughter, Nell, and her aunt Sophia. In the British way, Sophia isn't really her aunt, but she's helped bring Nell up, and is family. I was thinking as the plane took off about modern Disney's design for living and how expensive it is. If you ask a young Russian or an Iranian who love America why they love it, they believe it is a happiness mall. They believe you can pay your way to being anything you fancy and that it represents a kind of genius, possessed and managed by corporations. They speak of Apple and Disney, Coke or Ralph Lauren, all the entertainment, technology and fashion companies, as if they harvest the atoms of well-being and sell them to the world, and this, to young non-Americans directed by such dreams, is the meaning of freedom today. Especially Disney, because Disney usually comes with a song and a doll your child can take to bed. My daughter was excited for Disneyland, she said, because it was like going on a magic journey. She asked if the shops would be open all day and all night and whether the food was free. "Nothing is free, darling," I said, "but it is free to you and that's why it's a treat. We're going to have a wonderful time."

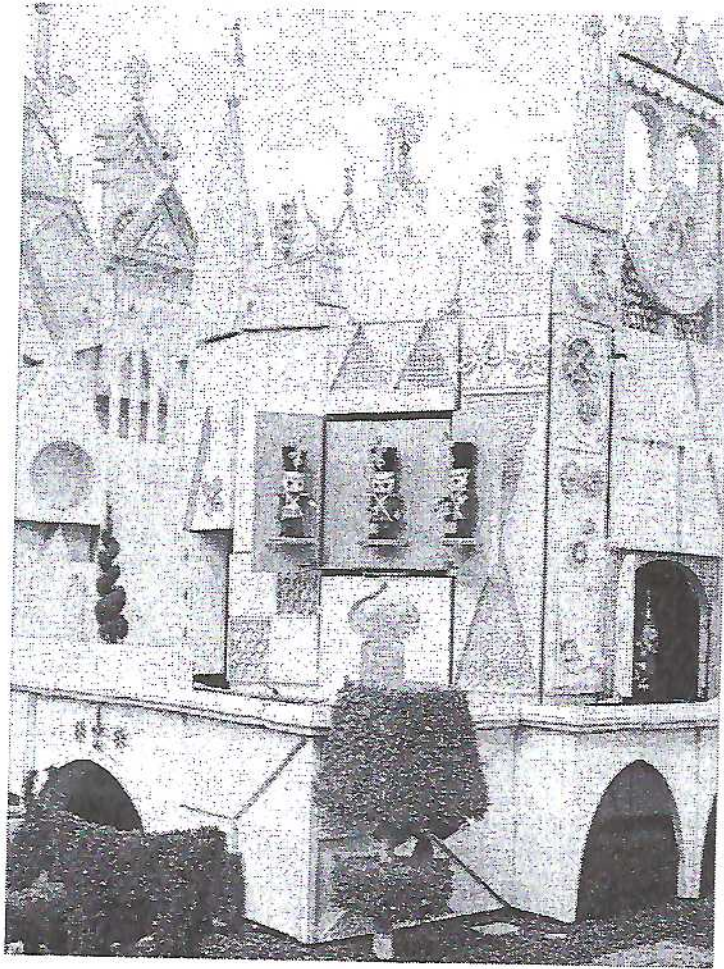
I think the gods of reality like revenge. They were watching us. Flying over the Atlantic, I woke up shivering and with swollen sinuses and a feeling of a coming flu like a gathering storm, a tempest in my happiness firmament, and I perceived from that moment a kind of Miltonic rebellion rising inside my head. A writer, you might say, is someone with an acute interest in oppositions, and our journey toward Disneyland was by necessity going to involve a certain conjuring with misery. My body did it by itself. And by the time we were standing at reception in Disney's Grand Californian Hotel & Spa, I was sneezy, I was dozy, I was happy but suffering a private existential crisis about what happiness meant. The hotel, to me, is a mountain chalet as imagined by Lewis Carroll and Charles Rennie Mackintosh. It is a riot of wooden balconies and iron flowers, which they say are inspired by the California Arts & Crafts movement. Disney music, that endless, tinkling sound of make-believe and mend, was playing through the universe — or was it just the street? — and I lay on the Disney bed and felt simply awful. And it felt like a crime. It felt like a crime against fantasy and against good times. We'd traveled all our lives to this spot of loveliness, and now I was roiling on the pillows and asking for the company doctor? As it happened, there wasn't one, and I was put through to a medical call center somewhere in Anaheim. Let me just say that a part of me will forever be lying bravely on that bed in Disneyland, with grinding bones and head of fire, while the music from "The Lion King" mocks me from the window.



Nell came back that evening levitating with gladness. It was as if she'd met the world as it really should be, and it was bliss to see her so full of faith. Walt Disney wanted to build a place that would stand against the horrors of the known world, where rockets were Golden Zephyrs, not nuclear weapons, where trains could be any color of the rainbow, made for the transport of children, not prisoners. And where every fear came wrapped in a yelp of sheer delight. With Disneyland, Walt Disney felt he was giving America a better version of itself: "The idea of Disneyland is a simple one," he wrote in a prospectus for the park. "It will be a place for people to find happiness and knowledge. It will be a place for parents and children to share pleasant times in one another's company; a place for teacher and pupils to discover greater ways of understanding and education. Here the older generation can recapture the nostalgia of days gone by, and the younger generation can savor the challenge of the future." The press opening, on July 17, 1955, was a fiasco — there was no drinking water, many of the attractions closed early and the TV broadcast was wild and amateur — and one might argue that the design and the idea of Disneyland has a fear of disaster embedded in it. Happiness, after all, is like that. We can hardly live with happiness for the fear of it suddenly ending. In the days after I left Disneyland, I walked through Los Feliz looking, as it were, for the footprints of Walt Disney, the man. Some have said he was a person with demons, and perhaps creating his own small world was a kind of compensation. In any case, what he created was a new way of thinking about life and dreaming, a kind of American Eden linked from his day to ours by an unfailing investment of common belief and a love of invention and dollars.

He found a key in memory, and built, in Disneyland, a resort that both creates memories and indulges them. By going there as adults, we are visiting a part of our belief system that experience has failed to dull, and, for our children, we are creating a memory with the hope that the same thing will happen for them. Mr. Disney had a genius for bridging the gap between childhood and adult longing and he made a universal out of that, as well as a brand. Of the thousands of reviews left on TripAdvisor, one sums this up for me. It's from a woman called Kathryn in New Zealand, left on May 31, 2015, with the headline: "I felt like the best parent in the world": "My husband and I grew up watching Disney every Sunday night. For us, going to Disneyland had always been a dream.... We always knew we wanted to take our children there, and we got the opportunity last Christmas. When we walked in, I felt like crying with happiness, and over the three days we visited, that feeling never went away."





Credit: Daniel Stier

*If the real world is in some ways a daunting prospect for most children, then It's a Small World exists at their level. Created for the 1964 World's Fair, the boat ride takes passengers past singing dolls representing youngsters of many nationalities, all apparently within reach.*

Nell came into our hotel room with a rasping love of the place that got me up from my philosophical sickbed. We went into the Disney California Adventure Park and found ourselves in a colored clamshell, entering the Little Mermaid: Ariel's Undersea Adventure, a ride in the Paradise Pier section. Lights and cold air gave us the illusion of floating underwater, and Nell looked up at me to see if I was believing. "This is awesome," I said.

"A bit awesome," she said.

"You mean, 'not really'?"

"I dunno. I like her face," she said. By this point in the ride Ariel was singing "Part of Your World" and every fiber in my sick being was saying "Yes. Yes, we are."

"I think Daddy likes it more than me," Nell said.



Reader, I am not beyond shame. But I was so happy I wanted to cry. I suddenly needed to live in this lagoon with all these fake bubbles. Nell is one of life's natural stylists. She might only be 11 but she knows what's what. When we stood in front of a giant painted billboard near Mickey's Fun Wheel, and Sophia went to take a picture, Nell started doing the Charleston and I felt that the best spirit of all the best girls resided in my daughter. She ate a corn dog and we ate popcorn and bad food never tasted so good.

My daughter responded immediately to the idea of America as a built environment and of Americans as built too, by themselves. I think we all do. I took Nell to Paradise Pier in the hope she'd feel like Dorothy in the land of Oz, and she did, seeming entitled to her own large sense of belonging in a place that she'd dreamt of. And that place, Disneyland, is then a part of parental self-creation: In America, in Disneyland, you're all the father or mother you can imagine yourself to be, creating — along with the Imagineers — a place for childhood that is larger and purer than you remember it being the first time round. So that is an evening we will always remember, the evening we looked up and imagined the sky too must be Disney.

"Less is a bore," said the American architect and writer Robert Venturi. And the year I was born he was tracing maps of desire onto parking lots and the Las Vegas Strip. During my time in Disneyland I thought about him a lot and believed his thinking was slightly like that of most 11-year-olds today, who live in a world of super graphics and aesthetics linked to shopping. On day two, Nell and I had our only argument of the trip about her constant wish to be in the Disney shop. Absurdly, I tried to argue that we didn't come all this way to buy rubbish. But of course we did. Floating over the emptiness of the parks in the minutes before they opened each morning was the pall of prepurchase. "I really want some ears," said Nell, "like really, really. It would just be weird not to have them." And not being weird is what Disneyland is all about, in a weird sort of way. The idea of buildings being symbols of what they are — of a concession stand shaped like a hot dog, a swimming pool that looks like a wave — is not only accessible to children but makes perfect sense to them. To be a child is to look at the horizon and see your desires. When Nell wanted an ice cream she simply looked for the building that looked that way. Life should be like this, she seemed to say — legible, self-representational, literal and witty. The world on her iPod Touch is like that, and her mind looks for scenographic explanations of what's going on. Communication has enslaved her generation to iconography — to symbols, logos and graphics as familiar as family. And to her and to Robert Venturi, that is always going to feel like freedom. Venturi tries to argue us out of our adult complacency. "Let us remember," he writes, "that throughout the history of architecture and urbanism, iconography has always dominated the scene, instructing and persuading us with its religious and civic content in ways no different from today's vigorous (and despised) commercial iconography. Let us acknowledge the validity of those signs as a flourishing element within that vital, generic American scene, as well as within the great tradition of architecture and urbanism! Let us today transfer the murals from the inside to the outside of the buildings!"



As an adult in Disneyland, you're subconsciously waiting for answers. But mainly you're just waiting. The boredom of waiting vies with sudden thrill, in the same way that happiness always competes with fear. The resort is full of frights and angular delights, but modern children are hard-core, and the park must cater to a certain amount of near-horror. Arguably, in the days of warty witches bearing questionable apples, it used to be more psychological, but today's screen princesses are savvier, more materialistic — as are their fans — and one can feel that fashion virtues outdo ordinary virtues in the kingdom of "Frozen." Cascading stardust must seem to be the opposite of spilled blood, but actually each is thrilling in its own way. There is something non-negotiable in how the children, especially the young girls, especially my own daughter, see access to princesshood, and beauty generally, as a power grab and a way to have everything they want and still keep singing. In one of the shops — I always lose arguments — they offer a "Frozen" makeover service for girls who want the look. Gary, the storefront operator who looked like an extra from "Glee," twirled, curled and generally outgirded the gaggle of tiny females surrounding him. Nell played it cool — she knows her beauty secrets — but her eyes widened when he showed her the book of hairstyles. "That one, please," she said.

"Princess Nell, you will be the belle of all the balls in the universe," Gary said.

"Just one ball would suffice," I muttered, under my breath.

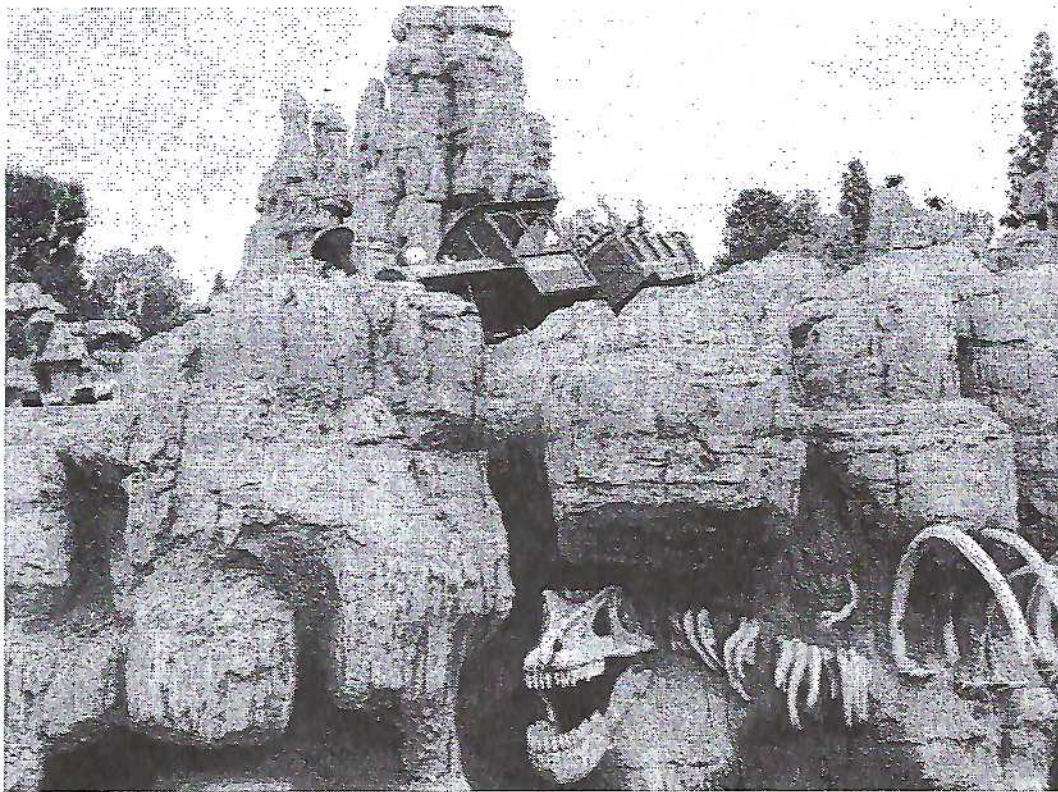
"Daddy's getting a little tired, isn't he, Princess Nell?"

"He's always like that."

"Well, we know all about tired daddies here. You just sit down, Daddy, and we'll get on with turning Nell into the princess she has always been."

It was 35 dollars. And all these girls — complete strangers to what they had — were primped, braided, gelled, glittered and generally, but equally, made to look like mini-adult punk girls with no faith in natural beauty. Nell kept making faces at me from the chair. Even though she couldn't see it yet, she knew it was rubbish, but it was "Frozen." I found it a bit strange seeing all these small girls in a row, learning how hard one has to work for every grain of enchantment. When Gary and the girls had done with Nell, they all stood back, ready to spin the chair to the mirror. "Are you ready, Princess?" he asked. And as one, the staff shouted, "Let it go!"





Credit: Daniel Stier

*Walt Disney wanted to build a place where trains could be any color of the rainbow. Big Thunder Mountain Railroad's roller coaster speeds in and out of canyons, waterfalls and tunnels in an area that spans nearly 200 feet.*

"Cool, thank you," she said, very politely, inspecting her new postapocalyptic hairdo. "Can we go to Autopia now? I'm really desperate to drive." It must be one of the secrets of Disney that so many of their stories problematize childhood, as opposed to celebrating it. Very few of the kids in Disney are just getting on with being young. They are often ersatz children, like the Seven Dwarfs, or ageless children who never grew up, like Peter Pan and the Lost Boys, or children who are made of wood, like Pinocchio, or children's favorite stand-in, animals. And the stylistics of Disneyland depend on the idea, not that childhood is an awfully great adventure, but that adulthood is, especially American adulthood, with its cars, its fantastic journeys, its fearful secrets and its love of prospecting for gold and spending it. We went on the cars a few times, just so Nell could feel like a person in control of her environment. She'd torn the princess hairdo out by now, and was free-wheelin' in some California of the mind, like a mini Joan Didion.



Since Disney took over Marvel and Pixar, it has reinvigorated itself as the world's chief purveyor of happiness narratives. But has it changed human nature? Can a \$49 billion conglomerate still keep its flair for opening up hearts and minds? I mean, if Disneyland is an alternative world, a better world than actuality allows, how can it be preserved as such, without Real World Problems climbing over the fence? My days in Disneyland helped cure my sickness, not because of any profound (or even basic) medical provision, but because it reminded me that the bloods of happiness are thicker than the waters of discontent. But when we got to Autopia, I saw that children can be what Saul Bellow called "reality instructors," perpetually animating the world around them with harsh self-interest. Nell's a mean driver, and the boy in front was nervous and slow, and he kept stopping. I wanted to bump him — I'm even meaner than she — but she got through it by turning him into a figure of derision. You can give people sunshine, you can give them Mickey wristbands linked to their parents' credit cards, as they do at Disney World, but in the end you can't stop them wishing to drive off the tracks and smash their way into an individual fantasy of their own. Disneyland, a bit like the garden in Milton's "Paradise Lost," is a place with no need for a police force, because sin doesn't exist and can barely even be imagined. But for the sociopathic among us, the greatest happiness of the greatest number will always find itself challenged by a basic wish to drive your car off the rails and go harum-scarum into the sunset. We didn't want to hurt anybody, but we did want to assert our own sense of chaos amid the uniform gladness. I might be a bad parent, but I applaud my daughter for that, for not being a happiness machine.

Enchantment is a melting pot, but we remain ourselves in the end. Disneyland is only a democracy for those who can afford to get in, and, even then, your spending power will continue to put pressure on your sense of freedom. As we swept round on the Golden Zephyr, I felt we were part of a simple factor of joy and on the Grizzly River Run we held on to each other as if meeting the thrill together was an unforgettable thing. In the Enchanted Tiki Room, when the mechanical birds opened up and sang to us, I felt much more than myself, much more than the emperor of ice cream and a lovely girl's daddy: I felt transported into her realm of astonishment as her eyes blazed with wonder. How lucky to be there. How amazing to live in a world where Disney can do that and we can pay. There are alleged to have been departmental stand-offs and a great variety of difficulties over the modernizing of Disney's resorts, and we won't be going into them here, but it seems to me that the main achievement of the MagicBand, the wristband that allows customers to enter their hotel rooms, enter the parks and gain meals, is that it adds to the heavenly unreality of the place. You feel you're not paying. In paradise, there is no need for money and no need for admonishment; one simply floats on an air of entitlement, and hopes that human nature and human insufficiency won't intrude.



The greatest ride in Disneyland, as you might see, is the ride through one's own ambivalence. And that is how it should be. Disneyland is a beautiful attempt at the impossible, and the impossible is not always beautiful. I hadn't stopped coughing by the time we made it to the first parade through Disneyland's main thoroughfare, but I'd stopped complaining about it, and at Nell's instigation I was beginning to see in Disney colors. The fireworks were part of it, the pink castle another, and the parade floats, full of waving and dancing characters, came to seem the most natural way to end a day of sweet experiences. "She looked at me," Nell said of the lady passing in the shape of a dancer from "Aladdin." "He pointed at me," she said of one of the chimney sweeps on the "Mary Poppins" float, and I believed her. In Disneyland, every child feels chosen, and why wouldn't you empty your bank account to see that happen, when the child is yours? Show-business values abounded, and only a curmudgeon, or a writer, would choose to question the authenticity of the performers' smiles or ask how much they are being paid. Night came on in a blaze of trumpets, and I simply didn't care anymore. I had joined the merry band, and fell into the happy void of my daughter's capacity for wonder, as F. Scott Fitzgerald understood it.

The structures of sentiment are nowhere better displayed. Disneyland is still in essence a 20th-century domain of imminent perfection. I feel that many a father before me, in his private self, hoped that, when the bomb comes, he would be in the teacups with his daughter, gently turning, turning gently, while believing that those odd streaks across the sky are merely the fireworks that nightly light up the windows of Main Street. "Let's go and find Mickey Mouse," Nell said. And in that moment the sky was ours and the evening too.

"Where will we find him, darling?"

And she smiled at me. "In his house, of course."

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