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GENES, ECONOMICS, AND HAPPINESS[1]

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A major finding from research into the sources of subjective well-being is that individuals exhibit a "baseline" level of happiness. We explore the influence of genetic variation by employing a twin design and genetic association study. We first show that about 33% of the variation in happiness is explained by genes. Next, using two independent data sources, we present evidence that individuals with a transcriptionally more efficient version of the serotonin transporter gene (*SLC6A4*) report significantly higher levels of life satisfaction. These results are the first to identify a specific gene that is associated with happiness and suggest that behavioral models benefit from integrating genetic variation.

KEYWORDS: Subjective Well-Being, Neuroeconomics, Twin Design Study, Genetic Association.

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> *We all know from comparing siblings that people are born different, and these differences are then amplified by subsequent experience. So our happiness depends on our genes and our experience (past and present). Any social reformer has to be mainly interested in the role of experience since that is all that we can change. But we will never understand that bit unless we understand the complete reality, and the complete reality includes a strong role for the genes.*
>
> —Prof. Lord Richard Layard, Lionel Robbins Memorial Lecture, LSE, February 27, 2003.

1. INTRODUCTION

Happiness research has become one of the liveliest subjects in economics in recent years[1]. 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 influences, 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 socio-demographic factors (age, gender, race, marital status, children, and social networks) as well as for political and cultural factors

[1]Books are e.g. Kahneman, Diener, and Schwarz (1999), Graham and Pettinato (2002), Frey and Stutzer (2002a), Van Praag and Ferrer-I-Carbonell (2004), Layard (2005), or Frey (2008); articles are e.g. Easterlin (1974), Clark and Oswald (1996), Frey and Stutzer (2002b), Di Tella, MacCulloch, and Oswald (2003), Luttmer (2005), Di Tella and MacCulloch (2006), Rayo and Becker (2007), Dolan, Peasgood, and White (2008), Fowler and Christakis (2008), Urry, Nitschke, Dolski, Jackson, Dalton, Mueller, Rosenkranz, Ryff, Singer, and Davidson (2004) or Clark, Frijters, and Shields (2008).

(such as democracy, decentralization, and religiosity). While variables like socio-economic status, income, marriage, education, and religiosity are significantly associated with individual happiness, none typically accounts for more than 3% of the variation (Layard, 2005; Frey, 2008). Moreover, changes in these variables appear to yield only short term changes to happiness. For example, the "Easterlin Paradox" (Easterlin 1974, 2004, Clark, Frijters and Shields 2008) suggests that increases in real income either have no lasting effect on happiness, or only a quite small one (Stevenson and Wolfers, 2008). The reason appears to be that happiness levels tend to revert toward what psychologists describe as a "set point" or "baseline" of happiness that is influenced by personality and genetic predispositions (Kahneman, Diener, and Schwarz, 1999; Diener and Lucas, 1999).

Although previous studies have shown that baseline happiness is significantly heritable (Lykken and Tellegen, 1996), none has so far identified a specific gene associated with subjective well-being. Here, we replicate the earlier work showing that happiness is significantly influenced by genetic variation in a nationally-representative sample, and then we present evidence of a specific gene that is associated with life satisfaction. We find that individuals with a transcriptionally more efficient version of the serotonin transporter gene (*SLC6A4*, also known as 5-HTT) are significantly more likely to report higher levels of life satisfaction and we replicate this association on an independent data set. This combination of economics and genetics is of rising salience (Benjamin, Chabris, Glaeser, Gudnason, Harris, Laibson, Launer, and Purcell, 2007; Beauchamp, Cesarini, Johannesson, van der Loos, Koellinger, Broenen, Fowler, Rosenquist, Thurik, and Christakis, 2010).

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, and Wallace (2009), Fowler, Dawes, and 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, *SLC6A4* is a prime candidate. The *SLC6A4* gene encodes a transporter in the cell wall that absorbs serotonin into the presynaptic neuron in parts of the brain that influence mental states (Hariri, Mattay, Tessitore, Kolachana, Fera, and Goldman, 2002; Bertolino, Arciero, Rubino, Latorre, Candia, and Mazzola, 2005; Heinz, Braus, Smolka, Wrase, Puls, Hermann, and et al., 2005; Canli and Lesch, 2007). *SLC6A4* has been studied for more than twenty 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 depression (Caspi, Sugden, Moffitt, Taylor, Craig, Harrington, McClay, Mill, Martin, Braithwaite, and Poulton, 2003); and the more transcriptionally efficient alleles have been linked to optimism (Fox, Ridgewell, and Ashwin, 2009). As a result, economists have specifically identified *SLC6A4* as a candidate gene for further study (Benjamin, Chabris, Glaeser, Gudnason, Harris, Laibson, Launer, and Purcell, 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 *SLC6A4* and life satisfaction. We find evidence of significant association in both data sets, suggesting that the *SLC6A4* gene may play a role in explaining subjective well-being. While we do not claim that *SLC6A4* determines happiness, nor do we exclude the possibility that several other genes may also play a role, we do think that the results suggest at least one possible causal pathway able to account for the influence of genes on happiness. And to our knowledge, this is the first study to identify a specific gene involved in the process. This in turn has implications for how economists think about the determinants of utility, and the extent to which exogenous shocks might affect and individual's well-being.

2. THE ADD HEALTH DATA

This research is based on genetic and survey data collected as part of the National Longitudinal Study of Adolescent Health (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 (Echenique, Fryer, and Kaufman, 2006; Echenique and Fryer, 2007; Alcott, Karlan, Mobius, Rosenblat, and Szeidl, 2007; Norton and Han, 2009). 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 percent 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-minute 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 over-samples, 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%.[2] Participants in Add Health also represent all regions of the United States: the Northeast makes up 17% of the sample, the South 27%, the Midwest 19%, and the West 17%.

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 and Rowe, 1998). The number of identical (monozygotic) and non-identical (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 (Harris, Halpern, Smolen, and Haberstick, 2006; Fowler, Baker, and Dawes, 2008).

Allelic information for a number of genetic markers were collected for 2,574 individuals as part of Wave III. The genes chosen for inclusion in the

[2] A breakdown for those providing DNA samples is presented in the Appendix.

study are known to affect brain development, neurotransmitter synthesis and reception, and hormone regulation. Allelic information includes markers that identify alleles (variants) of the serotonin transporter gene or *SLC6A4*. The promotor region of *SLC6A4* (called 5-HTTLPR) contains a variable number tandem repeat (VNTR) sequence that influences transcriptional activity—the "long" 528 base-pair allele is associated with a much higher basal activity than the "short" 484 base-pair allele. Allele frequency for the short allele is 43% and for the long allele is 57%. Details of the DNA collection and genotyping process are available at the Add Health website (Add Health Biomarker Team, 2007).

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 (less than 1% of interviewees gave such a response). This question and answer formulation is standard in the economics of happiness literature (Di Tella, MacCulloch, and Oswald, 2001, 2003; Kahneman and Krueger, 2006; Frey, 2008). The distribution of answers to the life satisfaction question is shown in Appendix. In line with the happiness literature, a large majority of respondents report being satisfied or very satisfied (Frey and Stutzer, 2002a). That most people, in fact, report a positive level of subjective well-being is the object of a paper by Diener and Diener (1996), where the authors find this distribution to be representative in a wide cross-national analysis.

3. TWIN DESIGN

3.1. *Methods*

Twin studies compare the traits, behaviors, and other outcomes (called "phenotypes") of twins who share 100% of their genetic material (identical

or *monozygotic* twins) to those who share 50% of their genetic material (fraternal or *dizygotic* twins) in order to estimate the relative importance of genetic and environmental influences (Taubman, 1976; Ashenfelter and Krueger, 1994). If we assume that the influence of the environment on the phenotype is the same for monozygotic (MZ) and dizygotic (DZ) twins (the "common 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, Medland, Ferreira, Morley, Zhu, Cornes, Montgomery, and Martin (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 ones parents is not sufficient to generate a difference in concordance (Scarr and Carter-Saltzman, 1979; Kendler, Neale, Kessler, Heath, and Eaves, 1993; Xian, Scherrer, Eisen, True, Heath, Goldberg, Lyons, and Tsuang, 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, μ 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:

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

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

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

$$COV_{DZ}(y_{1j}, y_{2j}) = \frac{1}{2}\sigma_A^2 + \sigma_C^2$$

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 2×2 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.[3]

To generate the ACE estimates, we use the structural equation modeling program *OpenMx* developed by Neale, Boker, Xie, and 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.

3.2. *Twin results*

When assessing the role of genetic influences, the first step is to compare the correlation in phenotype among MZ twin pairs to that of DZ twin pairs. For life satisfaction, the correlation coefficient for MZ twins is 0.345 and for DZ twins is 0.129. The difference in correlations is significant ($p = 0.032$, 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 I we report results from several variance decomposition models described above. Note that the ACE model yields a heritability estimate of 33%, while the estimate for common environment is 0% and the estimate for unshared environment is 67%. 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

[3] 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.

include A, suggesting that the finding that happiness is heritable is robust to different model specifications.[4].

TABLE I

SUMMARY OF ACE TWIN MODEL RESULTS.

	Life satisfaction			*Fit statistics*						
	a^2	c^2	e^2	ep	-2ll	df	AIC	diff -2ll	diff df	p
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 MZ and 219 DZ same-sex twin pairs.

Compared to 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, and De Geus, 2005) to 36–50% (Bartels and Boomsma, 2009) to 42–56% (Nes, Roysamb, Tambs, Harris, and 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 paper is only asked of Add Health subjects in Wave III (2001–02), 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 3 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

[4]When we split our twin sample by sex we find that there are significant differences between men and women. As in Table I, Table III in the Appendix shows 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%.

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, and McGuffin, 2008). It also shows 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.

4. 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 results for our candidate gene study.

4.1. *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, Huntingtons disease, or cystic fibrosis), the vast majority of phenotypes are "polygenic", meaning they are influenced by multiple genes (Mackay, 2001; Plomin, DeFries, McClearn, and McGuffin, 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.

4.2. SLC6A4, *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 (Damberg, Garpenstrand, Hallman, and Oreland, 2001; Benjamin, Chabris, Glaeser, Gudnason, Harris, Laibson, Launer, and Purcell, 2007).

We choose a candidate gene that has already received a great deal of attention for its association with mental states. The *SLC6A4* 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 "pre-synaptic" 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 *SLC6A4* gene codes for the serotonin transporters (5-HTT or SERT) 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 (Hariri, Mattay, Tessitore, Kolachana, Fera, and Goldman, 2002; Hariri and Holmes, 2006; Canli and Lesch, 2007).

The *SLC6A4* gene contains a 44 base-pair variable-number tandem repeat (VNTR) polymorphism[5] in the promoter region[6](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

[5] A VNTR polymorphism is a repeated segment of DNA that varies among individuals in a population.

[6] 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.

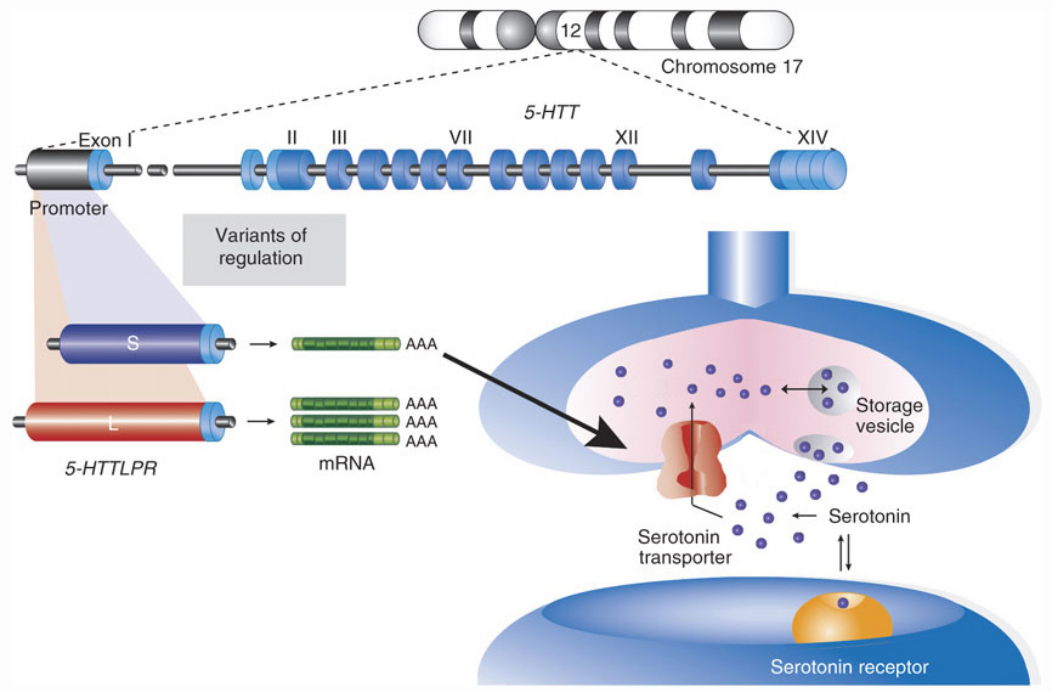

FIGURE 1.— Representation of the long/short variant of the *SLC6A4* gene and the release, reception, and recycling of serotonin in neurons. Adapted from Canli & Lesch (2007), with permission from the Nature Publishing Group.

the long allele is associated with an approximately three times higher basal activity than the shorter allele. Consequently, the long variant produces significantly more 5-HTT mRNA[7] and protein (Lesch, Bengel, Heils, Sabol, Greenberg, Petri, and et al., 1996; Little, McLaughlin, Zhang, Livermore, Dalack, and McFinton, 1998; Glatz, Mössner, Heils, and Lesch, 2003; Canli and Lesch, 2007). 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 pre-synaptic cell. This process is also shown in Figure 1.

[7]Messenger ribonucleic acid (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.

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 (Hariri, Mattay, Tessitore, Kolachana, Fera, and Goldman, 2002; Heinz, Braus, Smolka, Wrase, Puls, Hermann, and et al., 2005; Munafò, Brown, and Hariri, 2008; Pezawas, Meyer-Lindenberg, Drabant, Verchinski, Munoz, Kolachana, Egan, Mattay, Hariri, and Weinberger, 2005; Canli, Omura, Haas, Fallgatter, and Constable, 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, Meyer-Lindenberg, Drabant, Verchinski, Munoz, Kolachana, Egan, Mattay, Hariri, and Weinberger, 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, Meyer-Lindenberg, Drabant, Verchinski, Munoz, Kolachana, Egan, Mattay, Hariri, and Weinberger, 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, Omura, Haas, Fallgatter, and Constable, 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, Omura, Haas, Fallgatter, and Constable, 2005).

Myriad behavioral studies also suggest that serotonin and *SLC6A4* play an important role in emotional regulation (Heils, Teufel, Petri, Stober, Riederer, Bengel, and Lesch, 1996; Hariri, Mattay, Tessitore, Kolachana, Fera, and Goldman, 2002; Hariri and Holmes, 2006). Specifically, variance

in 5-HTTLPR was found to be be associated with variation in mental health outcomes (Lesch, Bengel, Heils, Sabol, Greenberg, Petri, and et al., 1996) and subsequent studies report that about 10% of the variance in anxiety-related traits depends on variation in serotonin transporters (Sen, Burmeister, and Ghosh, 2004; Munafò, Clark, and Flint, 2005). A recent study by Fox, Ridgewell, and Ashwin (2009) also suggests that 5-HTTLPR may influence optimism. The authors obtained DNA from about 100 participants and compared reaction times to 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 towards processing positive information and selectively avoiding negative information. This emotionally self-protective pattern does not obtain in individuals carrying one or both short alleles.

Not all studies show a direct relationship between a gene variant and a phenotype. Instead, developmental or concurrent environments may moderate an association between genes and phenotypes. A study by Caspi, Sugden, Moffitt, Taylor, Craig, Harrington, McClay, Mill, Martin, Braithwaite, and Poulton (2003) suggests a gene-environment interaction for the influence of life stress on depression. The authors find that individuals with short 5-HTTLPR alleles gene 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 as compared to only 17% of the individuals that carried both long alleles. Thus, in the Caspi, Sugden, Moffitt, Taylor, Craig, Harrington, McClay, Mill, Martin, Braithwaite, and Poulton (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 inter-

action, but the *direct* association between the number of long 5-HTTLPR alleles and life satisfaction. Future research may produce new insights from exploring how environmental factors moderate the association between 5-HTTLPR and happiness.

4.3. *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: (1) The allele itself influences subjective well-being; (2) the allele is in "linkage disequilibrium" with an allele at another locus that influences subjective well-being; or (3) the observed association is a false positive signal due to population stratification.[8]

Population stratification occurs because groups may have different allele frequencies due to 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

[8]Given our data, we cannot differentiate between 1 and 2. In order 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.

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.

Hence, to test for genetic association we employ a mixed-effects OLS regression model:[9]

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

where i and j index subject and family respectively. For the *SLC6A4* 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

[9]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.

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 ϵ 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 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.

4.4. *Association results*

Table II shows the results of several specifications of the models to test the hypothesis that the 5-HTTLPR long allele is associated with subjective well-being. 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 ($p = 0.012$). 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 satisified by about 17.3%.

TABLE II

OLS MODELS OF ASSOCIATION BETWEEN 5-HTTLPR AND LIFE SATISFACTION.

	Model 1			*Model 2*			*Model 3*		
	Coeff.	*SE*	P-value	Coeff.	*SE*	P-value	Coeff.	*SE*	P-value
5-HTTLPR long	0.059	0.023	0.012	0.065	0.023	0.005	0.070	0.029	0.017
Black	-0.111	0.048	0.021	-0.114	0.049	0.020			
Hispanic	0.198	0.117	0.092	0.216	0.118	0.067			
Asian	-0.196	0.073	0.007	-0.221	0.071	0.002			
Age	0.004	0.009	0.705	-0.011	0.009	0.262	-0.031	0.012	0.008
Male	0.014	0.033	0.682	0.028	0.033	0.406	0.039	0.041	0.341
Job				0.093	0.041	0.024	0.104	0.057	0.071
College				0.115	0.033	0.001	0.238	0.042	0.000
Married				0.232	0.041	0.000	0.318	0.050	0.000
Divorced				-0.313	0.153	0.041	-0.310	0.155	0.047
Religiosity				0.103	0.017	0.000	0.082	0.023	0.000
Welfare				-0.236	0.098	0.017	-0.121	0.153	0.432
Medication				-0.045	0.032	0.162	-0.095	0.041	0.021
Intercept	4.078	0.208	0.000	4.096	0.210	0.000	4.514	0.262	0.000
N	2545			2528			1446		
R^2	0.01			0.06			0.08		

Note: Variable definitions are in the Appendix. Standard errors (SE) and P-values are also presented.

Model 2 includes a number of socio-economic 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 ($p = 0.005$) 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.[10]

[10] We also report the results of association tests with 5-HTTLPR for each of these socio-economic factors in the appendix. An association with medication is nearly significant ($p = 0.08$) but loses its significance ($p = 0.17$) when controlling for age, gender, and race. Hence, medication cannot be considered a mediating variable (Baron and Kenny, 1986).

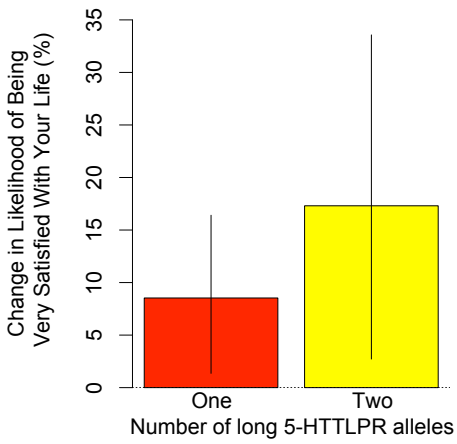

FIGURE 2.— Increasing the number of "long," more efficient 5-HTTLPR alleles yields significantly higher life satisfaction. First differences, based on simulations of *Model 1* parameters, are presented along with 95% confidence intervals. All other variables are held at their means.

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 5-HTTLPR and its p-value ($p = 0.017$) suggest that population stratification between self-reported racial categories is not driving the association between 5-HTTLPR and life satisfaction.

5. REPLICATION: THE FRAMINGHAM HEART STUDY

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. Here, we utilize the Framingham Heart Study (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: (1) the Original Cohort enrolled in 1948 (N=5,209); (2) the

Offspring Cohort (the children of the Original Cohort and spouses of the children) enrolled in 1971 (N=5,124); (3) the Omni Cohort enrolled in 1994 (N=508); and (4) the Generation 3 Cohort (the grandchildren of the Original Cohort) enrolled beginning in 2002 (N=4,095). Published reports provide details about sample composition and study design for all these cohorts (Cupples and D'Agnostino, 1988; Kannel, Feinleib, McNamara, Garrison, and Castelli, 1979).

The Framingham Heart Study makes available genetic markers for its participants. Out 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 *SLC6A4* 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 (Martin, Cleak, Willis-Owen, Flint, and Shifman, 2007; Wendland, Martin, Kruse, Lesch, and Murphy, 2006; Lipsky, Hu, and Goldman, 2009; Fahad, Vasiliou, Haddley, Paredes, Roberts, Miyajima, Klenova, Bubb, and Quinn, 2010). It is also known to be in positive linkage disequilibrium with the long allele of 5-HTTLPR (Huezo-Diaz, Rietschel, Henigsberg, Marusic, Mors, Maier, Hauser, Souery, Placentino, Zobel, Larsen, Czerski, Gupta, Hoda, Perroud, Farmer, Craig, Aitchison, and McGuffin, 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 (mean=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 *SLC6A4* 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 (GEE) approach to account for within-family correlation of errors. As shown in Model 1 in Table III, this association is significant ($p = 0.05$) and in the expected direction. In Model 2 we include additional controls for age and gender. We also include the first ten 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, Patterson, Plenge, Weinblatt, Shadick, and Reich, 2006). Once again, the replicated association is significant ($p = 0.05$).

6. 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 address one possible source of the "baseline" or "set point" for happiness that prior work has identified (Kahneman, Diener, and Schwarz, 1999; Graham, 2008). The existence of a baseline does not mean that the socio-economic 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 value in Table II, the *SLC6A4* gene explains less than one percent of the variation in life satisfaction, but our twin analysis suggests

TABLE III

GEE MODELS OF ASSOCIATION BETWEEN RS2020933 AND LIFE SATISFACTION.

	Model 1			*Model 2*		
	Coeff.	*SE*	P-value	Coeff.	*SE*	P-value
rs2020933 "A" alleles	**0.22**	**0.11**	**0.05**	**0.21**	**0.11**	**0.05**
Age				0.04	0.00	0.00
Male				-0.00	0.06	0.99
Principal Component 1				-0.88	1.57	0.58
Principal Component 2				0.04	6.43	0.99
Principal Component 3				-3.32	2.21	0.13
Principal Component 4				-1.08	2.33	0.64
Principal Component 5				-3.30	2.64	0.21
Principal Component 6				1.13	2.45	0.65
Principal Component 7				2.21	1.97	0.26
Principal Component 8				-2.10	2.21	0.34
Principal Component 9				-0.52	2.06	0.80
Principal Component 10				-1.82	2.26	0.42
Intercept	4.68	0.04	0.00	2.90	0.16	0.00
N	2843			2831		
R^2	0.01			0.05		

Note: Variable definitions are in the Appendix. Standard errors (SE) and P-values are also presented.

that *all* genes together account for about a third of the total variance. Therefore, there are probably many other genes which, in conjunction with environmental factors, help to explain how baseline happiness varies from one person to another. The association with *SLC6A4* is probably the first of a number of associations that will likely be identified over the course of the next few years.

Another use of work such as this is to address the problem of omitted variable bias (OVB). 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 OVB, 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 as compared to other life satisfaction surveys used in the happiness literature suggests that the age limits are not likely to gravely distort our results (Di Tella, MacCulloch, and Oswald, 2001, 2003; Kahneman and Krueger, 2006; Frey, 2008). Moreover, our successful replication in the Framingham Heart Study, 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. Moreover, we successfully replicate a related association in the Framingham Heart Study that controls for the first ten principal components of a singular value decomposition of the subject-genotype matrix, which has been shown to effectively deal with the problem of population stratification (Price, Patterson, Plenge, Weinblatt, Shadick, and Reich, 2006).

The estimates of the influence of socio-demographic, economic, and cultural covariates on life satisfaction in Table II corroborate the generally identified systematic effects of these variables in the literature (for a survey, see Dolan, Peasgood, and White 2008). In particular, gender does not systematically affect happiness. Higher age has a negative, though not sta-

tistically 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 and Diener, 1996; Di Tella, MacCulloch, and Oswald, 2001; Kahneman and Krueger, 2006; Frey, 2008). This question has been cross-validated with alternative measures that gauge subjective well-being (Kahneman and Krueger, 2006; Bartels and Boomsma, 2009) 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 focussing illusion by drawing attention to people's relative standing rather than moment-to-moment hedonic experience (Kahneman, Krueger, Schkade, Schwarz, and Stone, 2006).

7. CONCLUSION

Our results suggest that genetic factors significantly influence individual subjective well-being. Using twin study techniques we estimate that genetics explains about 33% of the variance in individual happiness. Moreover, using alternative methods we have identified one particular gene—*SLC6A4*—as having a positive association with self-reported life satisfaction in two independent samples. By moving beyond a twin study and focusing on specific genes, our analysis is able to suggest potential causal pathways through which genes influence happiness levels. A significant body of research has shown that the serotonin transporter gene influences the human psyche via its impact on neurological processes, thereby establishing a potential causal chain leading from this genotype to self-reported life satisfaction. Given prior research linking the "short," less trancriptionally efficient, alleles of the *SLC6A4* 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 happy, and this intuition is supported in both the Add Health and Framingham Heart Study data. The causal structure must be further studied once additional data reporting the genetic endowment of individuals coupled with data on their subjective well-being become available.

We have stressed that genetic factors complement, rather than substitute for, the existing studies showing the influence of socio-demographic, 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, Ben-Shahar, Smid, Vet, Robinson, and Sokolowski, 2005). While the *SLC6A4* gene may ex-

plain a significant portion of the variation in happiness, it is important to re-emphasize that there is no single "happiness gene." Instead, there is likely to be a set of genes whose expression, 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 and 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 (Fletcher and Lehrer, 2009; Norton and Han, 2009; von Hinke Kessler Scholder, Smith, Lawlor, Propper, and Windmeijer, 2010; Beauchamp, Cesarini, Johannesson, van der Loos, Koellinger, Broenen, Fowler, Rosenquist, Thurik, and Christakis, 2010; O'Malley, Rosenquist, Zaslavsky, and Christakis, 2010). We foresee this to be a promising avenue in economic research. 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, Nitschke, Dolski, Jackson, Dalton, Mueller, Rosenkranz, Ryff, Singer, and Davidson (2004) presents neural correlates of subjective well-being. Some of the neurological variation they observe may result from differences in genotype and could thus inform and stimulate new candidate gene association studies. Since genes are upstream from neurological processes, understanding them may bring us closer to understanding the objective sources of subjective well-being.

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REFERENCES

Add Health Biomarker Team (2007): "Biomarkers in Wave III of the Add Health Study," http://www.cpc.unc.edu/projects/addhealth/files/biomark.pdf.

Alcott, H., D. Karlan, M. Mobius, T. Rosenblat, and A. Szeidl (2007): "Community Size and Network Closure," *American Economic Review*, 97(2), 80–85.

Ashenfelter, O., and A. B. Krueger (1994): "Estimates of the Economic Return to Schooling from a New Sample of Twins," *American Economic Review*, 84(5), 1157–73.

Baron, R., and D. Kenny (1986): "The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations," *Journal of Personality and Social Psychology*, 51(6), 1173–1182.

Bartels, M., and D. I. Boomsma (2009): "Born to be Happy? The Etiology of Subjective Well-Being," *Behavior Genetics*, 39(6), 605–615.

Beauchamp, J., D. Cesarini, M. Johannesson, M. van der Loos, P. Koellinger, P. Broenen, J. Fowler, J. Rosenquist, A. Thurik, and N. Christakis (2010): "Molecular Genetics and Economics," Mimeo: Harvard University.

Benjamin, D. J., C. F. Chabris, E. L. Glaeser, V. Gudnason, T. B. Harris, D. I. Laibson, L. Launer, and S. Purcell (2007): "Genoeconomics," in *Biosocial Surveys*, ed. by M. Weinstein, J. W. Vaupel, and K. W. Wachter. The National Academies Press: Washington, D.C.

Bertolino, A., G. Arciero, V. Rubino, V. Latorre, M. D. Candia, and V. Mazzola (2005): "Variation of human amygdala response during threatening stimuli as a function of 5HTTLPR genotype and personality style," *Biological Psychiatry*, 57(12), 1517–1525.

BOUCHARD, T. (1998): "Genetic and Environmental Influences on Adult Intelligence and Special Mental Abilities," *Human Biology*, 70(2), 257–279.

CANLI, T., AND K. LESCH (2007): "Long story short: the serotonin transporter in emotion regulation and social cognition," *Nature Neuroscience*, 10(9), 1103–1109.

CANLI, T., K. OMURA, B. W. HAAS, A. FALLGATTER, AND R. T. CONSTABLE (2005): "Beyond affect: a role for genetic variation of the serotonin transporter in neural activation during a cognitive attention task," *Proceedings of the National Academy of Sciences USA*, 102, 12224–12229.

CAPLIN, A., AND M. DEAN (2008): "Dopamine, Reward Prediction Error, and Economics," *Quarterly Journal of Economics*, 123(2), 663–701.

CASPI, A., K. SUGDEN, T. MOFFITT, A. TAYLOR, I. CRAIG, H. HARRINGTON, J. MCCLAY, J. MILL, J. MARTIN, A. BRAITHWAITE, AND R. POULTON (2003): "Influence of Life Stress on Depression: Moderation by a Polymorphism in the 5-HTT Gene," *Science*, 301, 386–389.

CESARINI, D., C. T. DAWES, M. JOHANNESSON, P. LICHTENSTEIN, AND B. WALLACE (2009): "Genetic Variation in Preferences for Giving and Risk-Taking," *Quarterly Journal of Economics*, 124(2), 809–842.

CLARK, A. E., P. FRIJTERS, AND M. A. SHIELDS (2008): "Relative Income, Happiness, and Utility: An Explanation for the Easterlin Paradox and Other Puzzles," *Journal of Economic Literature*, 46(1), 95–144.

CLARK, A. E., AND A. J. OSWALD (1996): "Satisfaction and Comparison Income," *Journal of Public Economics*, 61(3), 359–381.

CUPPLES, L., AND R. D'AGNOSTINO (1988): "Survival following initial cardiovascular events: 30 year follow-up," in *The Framingham Study: An epidemiological investigation of cardiovascular disease*, ed. by W. B. Kannel, P. A. Wolf, and R. J. Garrison, pp. 88–2969. National Heart, Lung and Blood Institute, Bethesda, MD.

DAMBERG, M., H. GARPENSTRAND, J. HALLMAN, AND L. ORELAND (2001): "Genetic mechanisms of behavior: don't forget about the transcription factors," *Molecular Psychiatry*, 6(5), 503–510.

DE BAKKER, P. (2008): "Imputation in the Framingham Heart Study," Mimeo: Harvard Medical School.

DI TELLA, R., AND R. MACCULLOCH (2006): "Some Uses of Happiness Data in Economics," *Journal of Economic Perspectives*, 20, 25–46.

DI TELLA, R., R. MACCULLOCH, AND A. J. OSWALD (2001): "Preferences over Inflation and Unemployment: Evidence from Surveys of Happiness," *American Economic*

Review, 91(1), 335–341.

——— (2003): "The Macroeconomics of Happiness," *Review of Economics and Statistics*, 85(4), 809–827.

Diener, E., and C. Diener (1996): "Most People Are Happy," *Psychological Science*, 7(3), 181–185.

Diener, E., and R. Lucas (1999): "Personality and subjective well-being," in *Well-being: The foundations of hedonic psychology*, ed. by D. Kahneman, E. Diener, and N. Schwarz. Sage, New York, NY.

Dolan, P., T. Peasgood, and M. White (2008): "Do we really know what makes us happy? A review of the economic literature on the factors associated with subjective well-being," *Journal of Economic Psychology*, 29(94-122).

Easterlin, R. (1974): "Does Economic Growth Improve the Human Lot? Some Empirical Evidence," in *Nations and Households in Economic Growth: Essays in Honour of Moses Abramowitz*, ed. by P. David, and M. Reder. Academic Press.

Echenique, F., and R. G. Fryer (2007): "A Measure of Segregation Based on Social Interactions," *Quarterly Journal of Economics*, 122(2), 441–485.

Echenique, F., R. G. Fryer, and A. Kaufman (2006): "Is School Segregation Good or Bad?," *American Economic Review*, 96(2), 265–269.

Fahad, A. R., S. A. Vasiliou, K. Haddley, U. M. Paredes, J. C. Roberts, F. Miyajima, E. Klenova, V. J. Bubb, and J. Quinn (2010): "Combinatorial interaction between two human serotonin transporter gene variable number tandem repeats and their regulation by CTCF," *Journal of Neurochemistry*, 112(1), 296–306.

Ferrer-i-Carbonell, A., and P. Frijters (2004): "How Important is Methodology for the Estimates of the Determinants of Happiness," *The Economic Journal*, 114(July), 641–659.

Fitzpatrick, M., Y. Ben-Shahar, H. Smid, L. Vet, G. Robinson, and M. Sokolowski (2005): "Candidate genes for behavioural ecology," *Trends in Ecology and Evolution*, 20(2), 96–104.

Fletcher, J., and S. Lehrer (2009): "Using Genetic Lotteries within Families to Examine the Causal Impact of Poor Health on Academic Achievement," Prepared for the Annual Meeting of the American Economic Association.

Fowler, J. H., L. A. Baker, and C. T. Dawes (2008): "Genetic Variation in Political Participation," *American Political Science Review*, 101(2), 233–248.

Fowler, J. H., and N. A. Christakis (2008): "Dynamic Spread of Happiness in a Large Social Network: Longitudinal Analysis Over 20 Years in the Framingham Heart

Study," *British Medical Journal*, 337(2338), 1–9.

Fowler, J. H., C. T. Dawes, and N. A. Christakis (2009): "Model of Genetic Variation in Human Social Networks," *Proceedings of the National Academy of Sciences USA*, 106(6), 1720–1724.

Fox, E., A. Ridgewell, and C. Ashwin (2009): "Looking on the bright side: biased attention and the human serotonin transporter gene," *Proceedings of the Royal Society B*, 276, 1747–1751.

Frey, B. S. (2008): *Happiness: A Revolution in Economics*. MIT Press.

Frey, B. S., and A. Stutzer (2002a): *Happiness and Economics: How the Economy and Institutions Affect Well-Being*. Princeton University Press.

——— (2002b): "What Can Economists Learn from Happiness Research?," *Journal of Economic Literature*, 40(2), 402–435.

Glatz, K., R. Mössner, A. Heils, and K. Lesch (2003): "Glucocorticoid-regulated human serotonin transporter (5-HTT) expression is modulated by the 5-HTT gene-promotor-linked polymorphic region," *J. Neurochem.*, 85(5), 1072–1078.

Graham, C. (2008): "Economics of Happiness," in *The New Palgrave Dictionary of Economics*, ed. by S. N. Durlauf, and L. E. Blume. Palgrave Macmillan.

Graham, C., and S. Pettinato (2002): *Happiness and Hardship: Opportunity and Insecurity in New Market Economics*. Brookings Institution Press.

Hariri, A., and A. Holmes (2006): "Genetics of emotional regulation: the role of the serotonin transporter in neural function," *Trends Cognitive Science*, 10, 182–191.

Hariri, A., V. Mattay, A. Tessitore, B. Kolachana, F. Fera, and D. Goldman (2002): "Serotonin transporter genetic variation and the response of the human amygdala," *Science*, 297(5580), 400–403.

Harris, K. M., C. T. Halpern, A. Smolen, and B. C. Haberstick (2006): "The National Longitudinal Study of Adolescent Health (Add Health) Twin Data," *Twin Research and Human Genetics*, 9(6), 988–997.

Heils, A., A. Teufel, S. Petri, G. Stober, P. Riederer, D. Bengel, and K. Lesch (1996): "Allelic Variation of Human Serotonin Transporter Gene Expression," *J. Neurochem.*, 66(6), 2621–2624.

Heinz, A., D. Braus, M. Smolka, J. Wrase, I. Puls, D. Hermann, and et al. (2005): "Amygdala-prefrontal coupling depends on a genetic variation of the serotonin transporter," *Nat Neurosci*, 8(1), 20–21.

Huezo-Diaz, P., M. Rietschel, N. Henigsberg, A. Marusic, O. Mors, W. Maier, J. Hauser, D. Souery, A. Placentino, A. Zobel, E. R. Larsen,

P. M. Czerski, B. Gupta, F. Hoda, N. Perroud, A. Farmer, I. Craig, K. J. Aitchison, and P. McGuffin (2009): "Moderation of antidepressant response by the serotonin transporter gene," *British Journal of Psychiatry*, 195, 30–38.

Jacobson, K., and D. Rowe (1998): "Genetic and Shared Environmental Influences on Adolescent BMI: Interactions with Race and Sex," *Behavior Genetics*, 28(4), 265–278.

Kahneman, D., E. Diener, and N. Schwarz (1999): *Well-being: The Foundations of Hedonic Psychology*. Russel Sage, New York.

Kahneman, D., and A. B. Krueger (2006): "Developments in the Measurement of Subjective Well-Being," *Journal of Economic Perspectives*, 20, 3–24.

Kahneman, D., A. B. Krueger, D. Schkade, N. Schwarz, and A. A. Stone (2006): "Would You Be Happier If You Were Richer? A Focusing Illusion," *Science*, 312(5782), 1908–1910.

Kannel, W., M. Feinleib, P. McNamara, R. Garrison, and W. Castelli (1979): "An investigation of coronary heart disease in families," *American Journal of Epidemiology*, 110, 281–290.

Kendler, K. S., M. C. Neale, R. C. Kessler, A. C. Heath, and L. J. Eaves (1993): "A Test of the Equal-Environment Assumption in Twin Studies of Psychiatric Illness," *Behavior Genetics*, 23, 21–27.

Layard, R. (2005): *Happiness: Lessons from a New Science*. Penguin.

Lesch, K., D. Bengel, A. Heils, S. Sabol, B. Greenberg, S. Petri, and et al. (1996): "Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region," *Science*, 274, 1527–1531.

Lipsky, R. H., X.-Z. Hu, and D. Goldman (2009): "Additional Functional Variation at the SLC6A4 Gene," *American Journal of Medical Genetics B Neuropsychiatric Genetics*, 150B(1), 153.

Little, K., D. McLaughlin, L. Zhang, C. Livermore, G. Dalack, and P. McFinton (1998): "Cocaine, ethanol, and genotype effects on human midbrain serotonin transporter binding sites and mRNA levels," *American Journal of Psychiatry*, 155, 207–213.

Luttmer, E. F. (2005): "Neighbors As Negatives: Relative Earnings And Well-Being," *Quarterly Journal of Economics*, 120(3), 963–1002.

Lykken, D., and A. Tellegen (1996): "Happiness is a Stochastic Phenomenon," *Psychological Science*, 7(3), 186–189.

Mackay, T. (2001): "The genetic architecture of quantitative traits," *Annu Rev Genet*,

35, 303–339.

Martin, J., J. Cleak, S. Willis-Owen, J. Flint, and S. Shifman (2007): "Mapping regulatory variants for the serotonin transporter gene based on allelic expression imbalance," *Molecular Psychiatry*, 12(5), 421–2.

Munafò, M. R., S. M. Brown, and A. R. Hariri (2008): "Serotonin transporter (5HTTLPR) genotype and amygdala activation: a meta-analysis," *Biological Psychiatry*, 63, 852–857.

Munafò, M. R., T. Clark, and J. Flint (2005): "Does measurement instrument moderate the association between the serotonin transporter gene and anxiety-related personality traits? A meta-analysis.," *Molecular Psychiatry*, 10, 415–419.

Neale, M., S. Boker, G. Xie, and H. Maes (2010): "OpenMx - Advanced Structural Equation Modeling (http://openmx.psyc.virginia.edu/)," .

Nes, R. B., E. Roysamb, K. Tambs, J. R. Harris, and T. Reichborn-Kjennerud (2006): "Subjective well-being: genetic and environmental contributions to stability and change," *Psychological medicine*, 36(7), 1033–1042.

Norton, E., and E. Han (2009): "How Smoking, Drugs, and Obesity Affect Education, Using Genes as Instruments," Prepared for the Annual Meeting of the American Economic Association.

O'Malley, A., J. Rosenquist, A. Zaslavsky, and N. Christakis (2010): "Estimation of Peer Effects in Longitudinal Models Using Genetic Alleles as Instrumental Variables," Mimeo: Harvard University.

Oswald, A. J., and S. Wu (2010): "Objective Confirmation of Subjective Measures of Human Well-Being: Evidence from the U.S.A," *Science*, 327, 576–579.

Pezawas, L., A. Meyer-Lindenberg, E. M. Drabant, B. A. Verchinski, K. E. Munoz, B. S. Kolachana, M. F. Egan, V. S. Mattay, A. R. Hariri, and D. R. Weinberger (2005): "5-HTTLPR polymorphism impacts human cingulate-amygdala interactions: a genetic susceptibility mechanism for depression," *Nature Neuroscience*, 8, 828–834.

Plomin, R., J. C. DeFries, G. E. McClearn, and P. McGuffin (2008): *Behavioral genetics*. Worth Publishers, New York, NY, 5th ed edn.

Price, A. L., N. J. Patterson, R. M. Plenge, M. E. Weinblatt, N. A. Shadick, and D. Reich (2006): "Principal components analysis corrects for stratification in genome-wide association studies," *Nature Genetics*, 38, 904–909.

Rayo, L., and G. S. Becker (2007): "Habits, Peers, and Happiness: An Evolutionary Perspective," *American Economic Review*, 97(2), 487–491.

Scarr, S., and L. Carter-Saltzman (1979): "Twin Method: Defense of a Critical Assumption," *Behavior Genetics*, 9, 527–542.

Sen, S., M. L. Burmeister, and D. Ghosh (2004): "Meta-analysis of the association between a serotonin transporter promoter polymorphism (5-HTTLPR) and anxiety related personality traits," *Am. J. Med. Genet. B*, 127, 85–89.

Stevenson, B., and J. Wolfers (2008): "Economic Growth and Subjective Well-being: Reassessing the Easterlin Paradox," *Brookings Papers on Economic Activity*, 2, 1–87.

Stubbe, J., D. Posthuma, D. Boomsma, and E. De Geus (2005): "Heritability of life satisfaction in adults: a twin-family study," *Psychological Medicine*, 35(11), 1581–8.

Taubman, P. (1976): "The determinants of earnings: Genetics, family, and other environments: A study of white male twins.," *American Economic Review*, 66, 858–870.

Urry, H. L., J. B. Nitschke, I. Dolski, D. C. Jackson, K. M. Dalton, C. J. Mueller, M. A. Rosenkranz, C. D. Ryff, B. H. Singer, and R. J. Davidson (2004): "Making a Life Worth Living: Neural Correlates of Well-Being," *Psychological Science*, 15(6), 367–372.

Van Praag, B., and A. Ferrer-i-Carbonell (2004): *Happiness Quantified: A Satisfaction Calculus Approach*. Oxford University Press.

Visscher, P., S. Medland, M. Ferreira, K. Morley, G. Zhu, B. Cornes, G. Montgomery, and N. Martin (2006): "Assumption-free estimation of heritability from genome-wide identity-by-descent sharing between full siblings," *PLoS Genetics*, 2:e41.

von Hinke Kessler Scholder, S., G. D. Smith, D. A. Lawlor, C. Propper, and F. Windmeijer (2010): "Genetic Markers as Instrumental Variables: An Application to Child Fat Mass and Academic Achievement," University of Bristol, Centre for Market and Public Organisation Working Paper 10/229.

Wendland, J., B. Martin, M. Kruse, K. Lesch, and D. Murphy (2006): "Simultaneous genotyping of four functional loci of human SLC6A4, with a reappraisal of 5-HTTLPR and rs25531," *Molecular Psychiatry*, 11(3), 224–6.

Xian, H., J. F. Scherrer, S. A. Eisen, W. R. True, A. C. Heath, J. Goldberg, M. J. Lyons, and M. T. Tsuang (2000): "Self-Reported Zygosity and the Equal-Environments Assumption for Psychiatric Disorders in the Vietnam Era Twin Registry," *Behavior Genetics*, 30, 303–310.

Xu, H., and S. Shete (2006): "Mixed-effects Logistic Approach for Association Following Linkage Scan for Complex Disorders," *Annals of Human Genetics*, 71(2), 230–237.

APPENDIX A

Variable Definitions

5-HTTLPR long is an variable for having 0, 1, or 2 of the 528 base-pair alleles of the *SLC6A4* 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 *CHRNB3* (as opposed to A alleles). *rs13280604* is the number of G alleles (0, 1, or 2) for this SNP on *CHRNB3* (as opposed to A alleles). *rs2020933*

is the number of A alleles (0, 1, or 2) for this SNP on *SLC6A4* (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, Patterson, Plenge, Weinblatt, Shadick, and Reich, 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.

TABLE IV

ACE TWIN MODELS OF LIFE SATISFACTION, BY GENDER

Life satisfaction (females)				*Fit statistics*						
	a^2	c^2	e^2	ep	-2ll	df	AIC	diff -2ll	diff df	p
ACE	0.205	0.050	0.745	4	919.8	388	143.8	-	-	-
AE	0.263	-	0.737	3	919.8	389	141.8	0.05	1	0.83
CE	-	0.205	0.795	3	920.3	389	142.3	0.53	1	0.47
E	-	-	1	2	928.0	390	148.0	8.24	2	0.02

Life satisfaction (males)				*Fit statistics*						
	a^2	c^2	e^2	ep	-2ll	df	AIC	diff -2ll	diff df	p
ACE	0.389	0.000	0.611	4	951.3	400	151.3	-	-	-
AE	0.389	-	0.611	3	951.3	401	149.3	0	1	1
CE	-	0.308	0.692	3	955.0	401	153.0	3.73	1	0.05
E	-	-	1	2	972.4	402	168.4	21.11	2	0

FIGURE 3.— Longitudinal cross-twin correlations

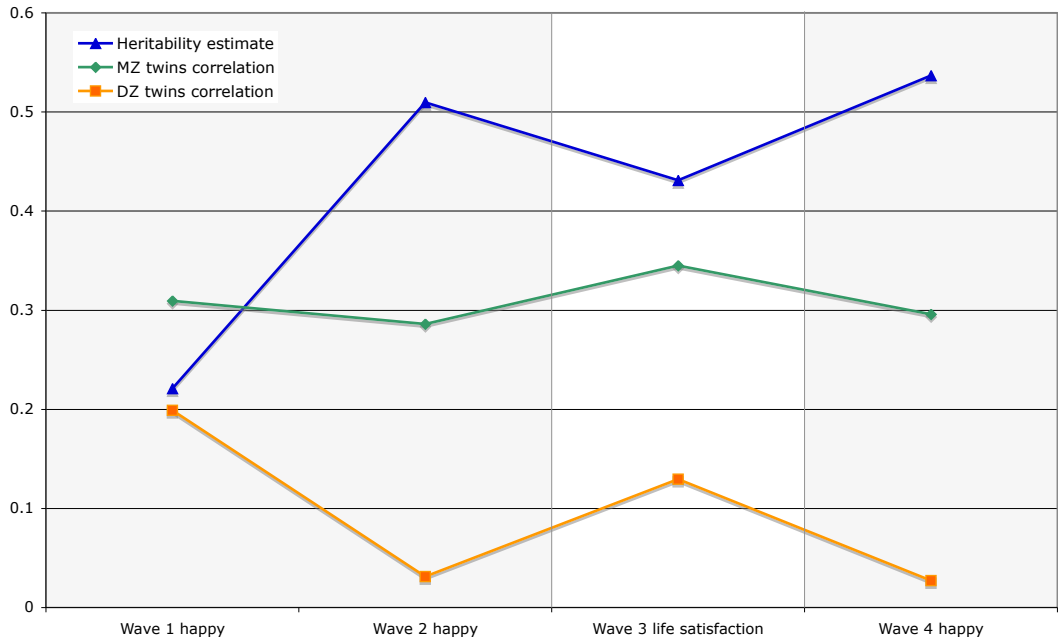

TABLE V

OLS MODELS OF ASSOCIATION BETWEEN 5-HTTLPR AND LIFE SATISFACTION THAT INCLUDE AVAILABLE ADD HEALTH GENETIC MARKERS.

	Model 1			*Model 2*			*Model 3*		
	Coeff.	*SE*	P-value	Coeff.	*SE*	P-value	Coeff.	*SE*	P-value
5-HTTLPR: long	0.061	0.026	0.021	0.066	0.025	0.009	0.080	0.032	0.011
MAOA: high	-0.014	0.022	0.518	-0.020	0.021	0.336	-0.017	0.027	0.528
DRD4: r7	-0.000	0.033	0.993	0.001	0.032	0.970	0.024	0.039	0.548
DRD2: a2	0.008	0.030	0.777	-0.000	0.029	0.991	0.048	0.036	0.243
DAT1: r10	0.043	0.032	0.169	0.045	0.030	0.135	0.047	0.036	0.191
rs2304297: G	-0.028	0.061	0.647	-0.012	0.059	0.842	0.018	0.085	0.837
rs892413: C	-0.010	0.051	0.837	-0.024	0.050	0.628	-0.024	0.073	0.744
rs4950: G	-0.042	0.065	0.520	-0.039	0.062	0.528	0.006	0.077	0.939
rs13280604: G	0.066	0.065	0.035	0.036	0.061	0.555	0.013	0.073	0.863
Black	-0.138	0.065	0.035	-0.150	0.065	0.020			
Hispanic	0.294	0.160	0.067	0.256	0.147	0.081			
Asian	-0.205	0.083	0.014	-0.250	0.079	0.002			
Age	0.006	0.010	0.581	-0.009	0.010	0.390	-0.027	0.013	0.033
Male	0.046	0.037	0.212	0.061	0.037	0.097	0.039	0.046	0.384
Job				0.108	0.046	0.019	0.104	0.057	0.071
College				0.154	0.037	0.000	0.245	0.048	0.000
Married				0.218	0.048	0.000	0.259	0.057	0.000
Divorced				-0.285	0.154	0.065	-0.265	0.159	0.096
Religiosity				0.160	0.020	0.000	0.131	0.026	0.000
Welfare				-0.205	0.112	0.068	-0.100	0.165	0.546
Medication				-0.072	0.037	0.053	-0.129	0.046	0.005
Intercept	3.960	0.246	0.000	3.932	0.243	0.000	4.227	0.304	0.000
N	1939			1910			1110		
R^2	0.015			0.087			0.102		

Note: Variable definitions are in the Appendix. Standard errors (SE) and P-values are also presented.

Summary Statistics

TABLE VI

SAMPLE MEANS.

	Mean	Std Dev	Min	Max
Life satisfaction	4.20	0.79	1	5
5-HTTLPR long	1.14	0.72	0	2
Age	21.9	1.7	18	26
Religiosity	1.43	0.92	0	3

TABLE VII

PERCENTAGE OF SUBJECTS EXHIBITING THESE CHARACTERISTICS.

	Percent
White	70.9
Black	19.0
Hispanic	14.7
Asian	8.2
Male	47.8
College	54.9
Married	17.3
Divorced	1.4
Welfare	4.2
Medication	61.2

FIGURE 4.— Distribution of life satisfaction

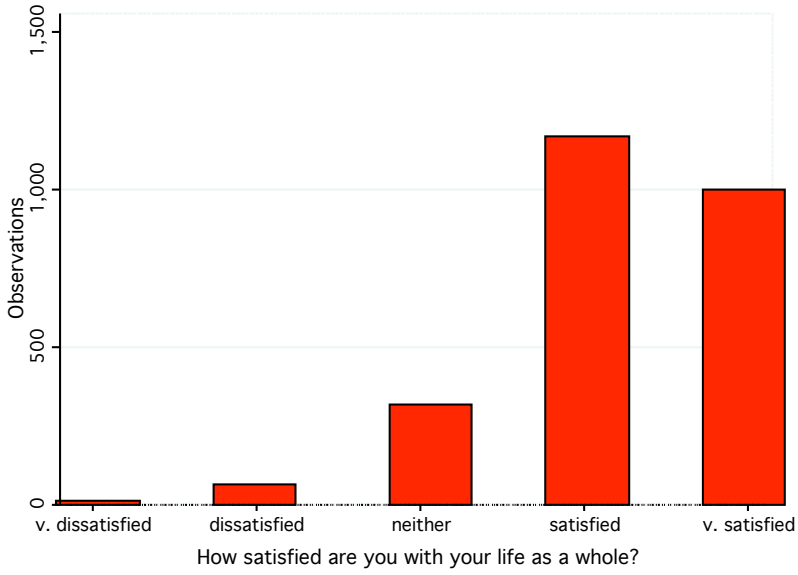

FIGURE 5.— Distribution of life satisfaction, by zygosity

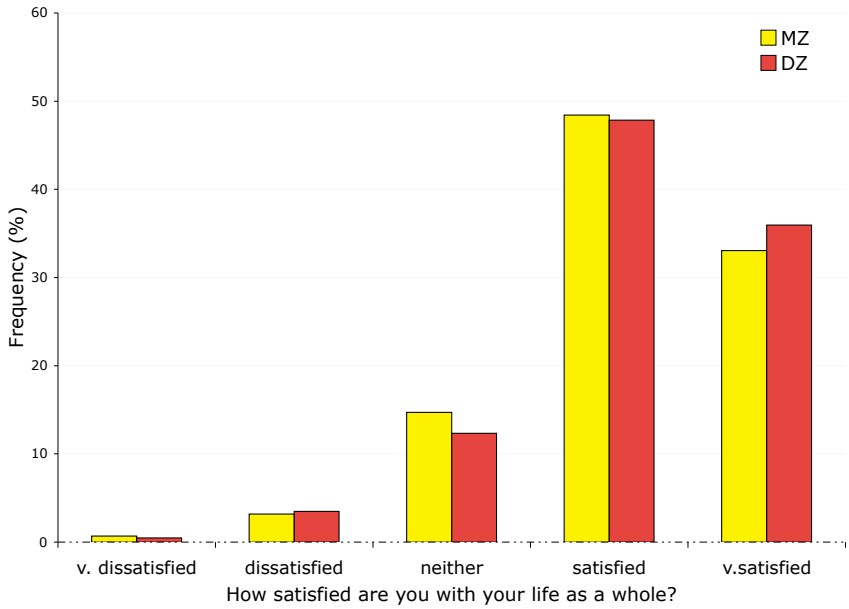

TABLE VIII

CROSS-TABS

	5-HTTLPR long			
Life satisfaction	0	1	2	Total
Very dissatisfied	4	4	5	13
	(0.8%)	*(0.3%)*	*(0.6%)*	*(0.5%)*
Dissatisfied	17	35	13	65
	3.3%	*2.9%*	*1.6%*	*2.6%*
Neither	72	149	97	318
	14.2%	*12.6%*	*11.3%*	*12.4%*
Satisfied	226	544	394	1,164
	44.4%	*45.9%*	*45.7%*	*45.6%*
Very satisfied	190	453	353	996
	37.3%	*38.2%*	*41.0%*	*39.0%*
Total	509	1,185	862	2,556
	100%	*100%*	*100%*	*100%*
	(20%)	*(46%)*	*(34%)*	

TABLE IX

LIFE SATISFACTION AND GENOTYPE, BY RACE

Race		Mean
White		
	Life satisfaction	4.24
	5-HTTLPR long	1.12
Black		
	Life satisfaction	4.13
	5-HTTLPR long	1.47
Hispanic		
	Life satisfaction	4.22
	5-HTTLPR long	0.93
Asian		
	Life satisfaction	4.01
	5-HTTLPR long	0.69

TABLE X

POTENTIAL MEDIATORS

DV	*5-HTTLPR long* $p-value$
Job	0.17
College	0.99
Married	0.33
Divorced	0.16
Religiosity	0.48
Welfare	0.25
Medication	0.08

Note: Table presents p values for 5-HTTLPR long in models with job, college attendance, married, divorced, religious, welfare, and medication as dependent variables. Regressions also include race, age, and gender controls.