

# Do People Make Environments or Do Environments Make People?

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**ABSTRACT:** This article discusses the influence of people's genetic make-up on their mental states of happiness and depression. Contrary to the conventional wisdom, great fortune does not guarantee happiness; neither does great misfortune assure depression. Emotional states are surprisingly immune to "objective" social circumstances. A biological basis for this relative immunity is that people possess biological *set points* for these emotional states, rendering the effects of most life events transitory. Genotypes also have indirect effects. People react differently to psychological stressors depending on their genotypes. A susceptible person may succumb to depression, whereas a resilient person may remain unaffected. People also expose themselves to different social environments. Exposure to controllable life events is partly a result of genetic predispositions. Consilience requires that this biological individuality be considered in any understanding of human behavior, including the pursuit of happiness.

**KEYWORDS:** Set points; Emotional states; Genotype; Depression; Happiness

My thesis is simple. People inherit genes. Genes form the recipes for their nervous systems. People with different nervous systems respond to the world differently. One might say that they have different tastes and preferences. People with different tastes and preferences also create different circumstances for themselves. So in the end, people's behavior is strongly genetically determined and influenced by the social environment only in the short term. I should admit that this thesis is not part of the conventional wisdom of the general public, nor does it fit the beliefs of many behavioral scientists in fields such as sociology.<sup>1</sup> This article presents a collection of observations that are supportive of this way of thinking about behavior.

Consider two people, one who is happy, another who is sad and depressed. One way to explain their emotional states is through life events. One might assume that the Mr. Happy makes has a big income, owns a second home, is married with two children, is an athlete, and just won \$500,000 in a state lottery. Mr. Sad, by contrast, could be poor, childless, and, having purchased his 50th lottery ticket, is again a loser. We can also imagine contrasting childhoods for Mr. Happy and Mr. Sad. Mr. Happy has outgoing and emotionally warm parents who loved him dearly. Mr. Sad had

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subdued and introverted parents who criticized him harshly. He remembers his childhood as a painful time and has little desire to visit with his father.

Thus, an attribution is commonly made that life events are the cause of a person's levels of happiness versus distress. For example, it is not unusual for a death of a relative to lead to a depressive episode. One thus makes an inference that loss of a loved one is a cause of depression.

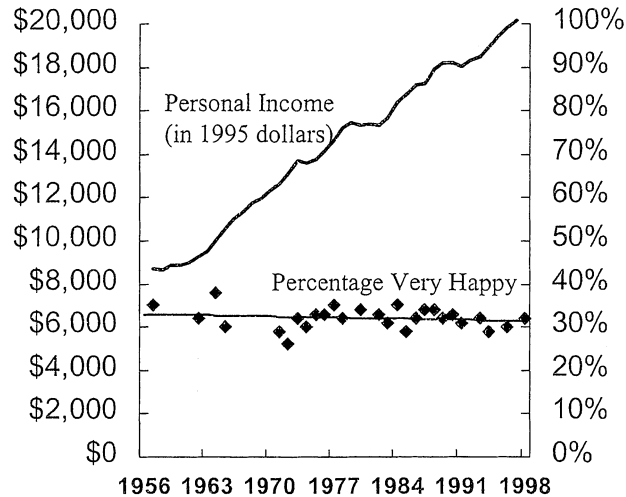
Other attributions are made for chronic levels of depression, or its opposite, happiness. In a study of 5,000 adults,<sup>2</sup> happiness showed remarkable stability over 10 years. People at the top of the happiness rank order tended to stay at the top; those people at the bottom tended to stay miserable. When depression, or happiness, are considered as chronic conditions—although psychiatric diagnoses are only given for the former—attributions of cause are typically made to early life experiences. Someone who is happy or sad in a trait-like way got their sunny or despondent dispositions from early childhood experiences in the families. Sadness might be a legacy of abusive parenting; happiness, of parenting that gave more to a child than is usually expected. These scenarios are widely accepted by the general public and child development experts alike.

#### **CURRENT LIFE CIRCUMSTANCES ARE WEAKLY RELATED TO HAPPINESS**

In the film *Casablanca*, the police inspector character tells his subordinates to “round up the usual suspects.” The usual suspects for the production of happiness are good life circumstances—Mr. Happy's high income and loving and supportive family. Yet, in studies that have been done, these persons' objective circumstances explain only a relatively small part, if any, of the variation in happiness.<sup>3,4</sup> FIGURE 1 shows the growth in wealth in 1995 dollars and the average level of happiness reported by persons on large surveys of the United States population.<sup>3</sup> Roughly *one-third* of people were “very happy” from the 1956 to 1998; during the same period, inflation-adjusted income just about doubled! The population of the United States got much richer but not much happier.

Income gains in the United States population were gradual. What about something more dramatic, like winning a million 1978 dollars in the Illinois State Lottery. Some psychologists at Northwestern University interviewed 22 major lottery winners and 22 controls living in the same neighborhoods.<sup>5</sup> The lottery winners and controls rated themselves as equally happy in the present. They also expected to be equally happy in the next couple of years. Lottery winners looked worse than controls on another measure—they reported seven ordinary activities as less pleasurable than did controls (e.g., talking with a friend, hearing a funny joke, and getting a compliment). Neither getting rich suddenly nor getting there slowly does much to increase the net level of happiness.

Karl Marx thought that the worker was exploited under capitalism, with its winner-take-all mentality. Would we find that the downtrodden are more emotionally miserable than the well-to-do? In the Minnesota Twin Family Study<sup>6</sup> 2,186 twins distributed from poor to rich gave ratings of contentment on a five-point scale. The scale range was from 1, indicating “as content as the lowest 5% of the population,” to 5, indicating “as content as the highest 5% of the population.” There was little re-



**FIGURE 1.** Happiness and national wealth since 1956. (Reprinted from Myers<sup>3</sup> with the permission of the American Psychological Association.)

duction in happiness as one moves from class 6, “unskilled labor,” and 7, “unemployed,” to 1, which indicated the professional class. Rather than miserable, the unskilled and unemployed reported almost as much psychological contentment as the middle and upper classes. Most people report they are pretty happy; the scale mean fell between 3 and 4 on the 5-point response scale. In percentages, that means that 86% of the twin sample saw themselves as in the top 35% of the population in level of contentment. On this scale, only about 24% of people admitted to being relatively unhappy.

My last example of unexpected contentment is one reflecting racial differences.<sup>7</sup> If we use self-esteem as another measure of happiness and contentment, we can discover that African-American people, as a group, have *better* self-esteem than European-heritage people (i.e., whites) in the United States. Their advantage is small but noticeable, about 0.15 standard deviations. In practical terms, imagine that we call the top 5% of whites falling above a cut-off score, “extremely happy with themselves.” We would discover that 7% of African-Americans would exceed the same cut-off score as “extremely happy.” It is not a large difference, but looked at another way, a 2% difference constitutes 40% more extremely happy African-Americans than extremely happy whites.

What is surprising about this finding is that a group thought to suffer from racial discrimination and greater poverty than other groups, on average, possesses greater self-esteem than other racial or ethnic groups in the United States. This observation accords with the earlier findings that, in a country that was getting richer over time, people were not becoming happier and people who win lotteries do not suddenly find an inner contentment. The objective circumstances of people have a relatively weak effect on how happy people are. Thus, we need another explanation for why most people were happy, why some 20–30% of the population was relatively unhappy

across all social class levels, and why among them some people suffer serious bouts of depression.

### THE SET POINT THEORY

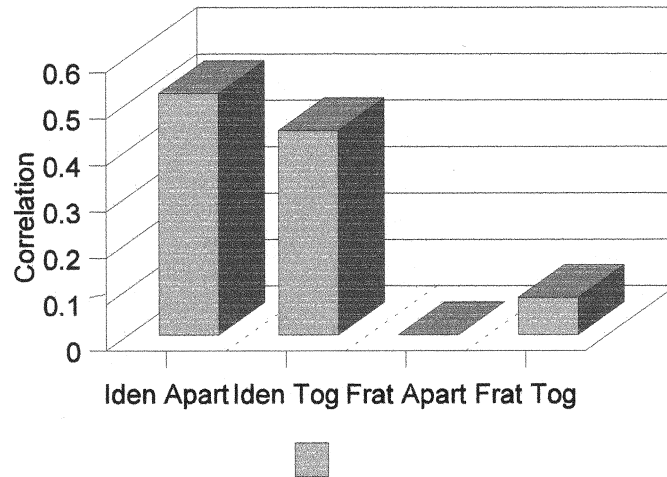
Let me offer a genetic explanation for these unexpected findings based on a *set point* theory. The idea is that our nervous system has a set point, a basal level to which it likes to return to. We know the idea of homeostasis—for example, our bodies maintain an average temperature of 98.6° Fahrenheit. When the body is diverted from that core temperature, it tries to restore its basal temperature; it does this by shivering when it's too cold and sweating when it's too hot. Of course, for body temperature, everyone has the identical set point (or nearly identical)—the normal temperature, 98.6°. For other characteristics, however, set points differ among individuals.

Weight is a good example. A person's weight is a highly heritable characteristic. Even when on comparable diets, one person can be thin and another heavy because of how easily food calories are converted into fat. The difference is caused by having a different genetic set point for weight. One can demonstrate this set point using a most unpleasant intervention: making people eat more than they want.<sup>8</sup> This intervention was conducted with six pairs of male identical twins living in a college dormitory. As they overate, each identical twin gained weight. A pair of identical twins tended to gain the same amount of body fat—showing a genetic influence on weight gain. After the force-feeding ended, most twins easily lost the weight they had gained. Each person returned to a *set point*. The same phenomenon happens to dieters, but in the opposite direction. After a diet, lost weight is quickly regained as the body returns to its set point.

The set point idea applies to happiness and sadness. Mr. Happy has a set point, shared by 60–80% of the population, which makes him routinely happy. Mr. Sad has a set point in the bottom quarter of the population. These set points are genetically imposed. Identical twins, who share all their genes, have about the same set point. Data from the Minnesota Study of Twins Reared Apart<sup>6</sup> can be used to illustrate the strong genetic influence on happiness. In this study, 1,491 twin pairs took one measure of happiness, the Well-Being Scale of the Multidimensional Personality Inventory. FIGURE 2 illustrates the similarity of twin pairs on the Well-Being Scale. Identical twins, whether the twins are reared apart or together, tend to score very much alike. About 45% of the individual differences in the Well-Being Scale can be attributed to genetic differences in “set points” among individuals. A technical note for these findings: only about 16% of the variation is attributable to the additive effects of genes, because the fraternal twin correlations were less than one-half those of the identical twins. The 29% remainder of the genetic effect is attributable to interactions among genes, including genetic dominance.

These findings demonstrate a genetic set point. The way the set point works is that daily events are likely to push individuals above and below their set points, to which they return over time. Winning a lottery, making a team, and getting a promotion at work all induce a boost in happiness, but only a temporary one. As with force-feeding, once the stimulus is gone, a person returns to his set point. Once the joyful

## Twin Resemblance



**FIGURE 2.** Twin pair resemblance on the Well-Being Scale of the Multidimensional Personality Inventory. Iden Apart: identical twin pairs raised apart ( $n = 75$ ); Iden Tog: identical twin pairs raised together ( $n = 647$ ); Frat Apart: fraternal twin pairs raised apart ( $n = 36$ ); Frat Tog: fraternal twin pairs raised together ( $n = 733$ ).

event fades from memory, a person returns to his set point for happiness. Similarly, negative life events, like a poor grade in a course, an embarrassment, or not receiving an expected raise may reduce well being, but only temporarily.

We can understand this by considering that what is stable over time in a person's level of happiness or depression is the genetic set point. If I want to know how unhappy or happy someone will be 10 years in the future, I do not ask about the person's current or future life events, except perhaps for extreme bodily injuries. Instead, I use the best available information about that person's set point, the score of his identical twin today. Because identical twins have about the same set points, I can make a projection of a co-twin's future well-being from that of the other member of the twin pair.

FIGURE 3 presents the relevant findings from the twin studies done in Minnesota.<sup>6</sup> One bar in the figure shows the 10-year test-retest association for well-being. This association was 0.50 in a group of twins tested first at age 20 years and again at age 30 years. How well can future well-being be estimated from the set point on the basis of the co-twin's score? The other bar answers this question. It shows the association of Twin A at age 20 years with Twin B, the other member of the same twin pair, at age 30 years and vice versa. Its value is 0.40. These values imply that 80% (i.e., 0.40/0.50) of the stability in well-being is due to the genetic set point. The remainder is due to any of a myriad of environmental differences between individuals; the possibilities would include prenatal developmental effects as well as nonbiological envi-

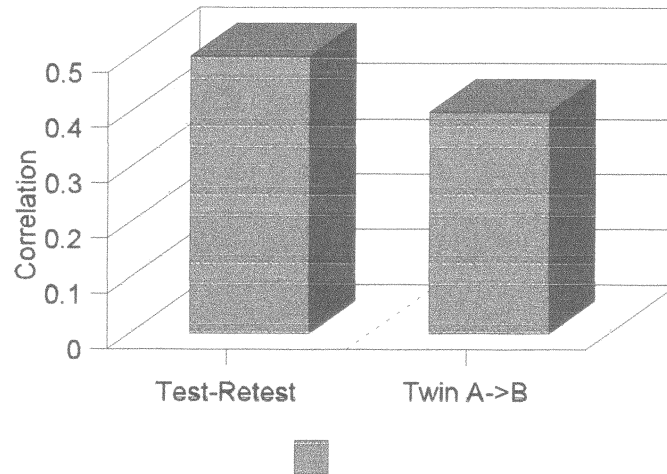


FIGURE 3. Stability of well-being over 10 years.

ronments. The main point, though, is that the majority of a person’s happiness is attributable to this stable, genetic set point. People with different set points exist at every income level and in different racial groups. That is why the overall prevalence of unhappiness is less dependent on environmental circumstances than many people would suppose based on their own intuitions.

**THE GENETIC BASIS OF DEPRESSION**

Episodes of major depression are not uncommon. In most birth cohorts, 2–9% of people develop a major depression at some point in their lives.<sup>9</sup> The prevalence of an episode of major depression increases over the life span because the first episode may occur at any age. The prevalence is also higher for the more recent birth cohorts than for earlier ones; for example, those persons born from 1935 to 1944 have a higher prevalence than do those persons born from 1905 to 1914. No one knows the exact reason for the increase by birth cohort.

Major depression also runs in families. When a characteristic is familial, it should have a higher rate among the biological relatives of affected cases (i.e., probands) than is found in the general population. Many studies demonstrated such familial transmission of depression. In the study by Klerman and Weissman,<sup>9</sup> the prevalence of depression among biological relatives was plotted against the age of onset of the probands’ depression. Except for very late-onset cases of depression, the rates were greater among the biological relatives than in the general population. Rates for relatives of depressed persons were about 45% among female relatives and 25% among male relatives versus a 2–9% rate in the general population.

The reason that major depression runs in families is because it is heritable. This has been demonstrated in both twin and adoption studies. Consider findings for a large sample of female twins in a study by one of the foremost psychiatric research-

ers, Kenneth Kendler.<sup>10</sup> Not surprisingly, Kendler found that the 444 identical twin pairs were more concordant (i.e., alike) for a diagnosis of major depression than were the 296 fraternal twin pairs. Thus, major depression is a heritable characteristic.

The surprise in Kendler's twin study came when he estimated the heritability of major depression controlling for unreliability of measurement. People are not perfect reporters of their own thoughts and behaviors. If I asked you what you wore last Thursday, what you ate for lunch, and whether you felt happy or sad, you might not give totally accurate answers for any of these questions. Kendler had the twins recall their symptoms of depression at both a time 1 assessment and a time 2 assessment. These interviews took place about a year apart. At both interviews, the twins were asked about symptoms of depression for the period of their lives starting before the time 1 assessment. If people were perfectly accurate reporters of their life time histories (LTH) of depression, they should have mentioned exactly the same symptoms of depression both times. Instead, only 55% of people who reported a LTH of depression episode at time 2 also reported a LTH at time 1. About one-third of people reported an episode of major depression at both assessment times, but they were not always the same individuals. Using a statistical method, Kendler corrected his estimate of the lifetime heritability for the inaccuracy of peoples' symptom reports on any one occasion: the estimate was a heritability of about 70%. Depression is a highly heritable disorder that is hard to diagnose.

### DEPRESSION AND THE ENVIRONMENT

The title of this article poses the question, "do environments make people, or do people make environments?" There is no simple answer to this question, because some truth clearly exists on both sides. Nevertheless, I want to focus on the case that people make their environments to a greater extent than many people recognize. We have already seen, by the set point idea, that a person's emotional life can be quite detached from his "objective circumstances." An outsider may have viewed the movie star Marilyn Monroe as glamorous and happy; but she was, though glamorous, actually chronically depressed.

Behavioral geneticists describe a couple of ways in which individuals' genotypes transact with their environments. One is that people may react differently to the same experiences because of their genotypes. Consider the case of color blindness. A man insensitive to the color red because of a mutation in a photoreceptor gene will fail to recognize that his red and green socks do not match, whereas a man with normal color vision has no trouble with the distinction. The stimulus—a particular frequency of light wave—results in a world experienced differently by normal versus color-blind men.

The same kind of coloring of perception may occur in people who are depressed. Let me give you two different examples. The first comes from the common occurrence that depressed girls are more estranged from their families. In our study of depression in the National Longitudinal Study of Adolescent Health (Add Health),<sup>11</sup> girls reported on depressed mood and on family connectedness, by means of such items as "Your family pays attention to you" and "Your family has fun together." The correlation coefficient between depressed mood and family connectedness was  $-0.37$ , negative be-

cause greater connectedness is associated with a lower level of family connectedness. (In Jacobson and Rowe<sup>11</sup> the scores on depressed mood are reversed, so this association is a positive one in their article.) This association between depressed mood and family connectedness is moderate, relative to associations typically found in the behavioral sciences.

One way to interpret this correlation is that parental treatment causes adolescents' symptoms of depression. As noted above, Mr. Sad's chronic unhappiness is often attributed to early experiences in the family; the blame is put on his parents. In the Add Health study, the questions were about current experiences, but one could easily imagine that an emotional distance between a depressed daughter and her parents did not develop overnight; therefore, her experience with family discord extended back into childhood.

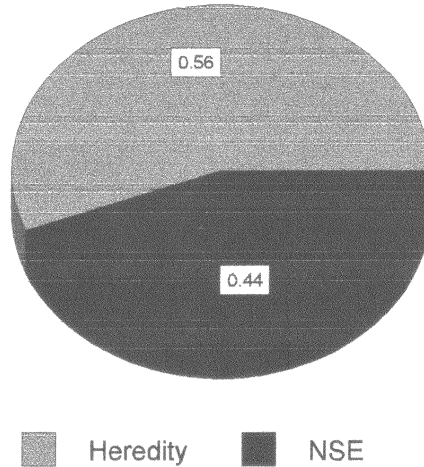
Two genetic interpretations exist for an association between parental treatment and depression, however. One reverses the direction of the causal arrow: Parental treatment may be responsive to the children's behavior. For example, a depressed daughter, being emotionally nonresponsive, may discourage parents from paying attention to her or having fun with her. Another interpretation is that genetic dispositions influence how a parent's behavior is interpreted, just as a gene making one color blind change the perception of light. Thus, a parent may be appropriately warm to a child, but a child who is genetically predisposed toward depression may not notice the warmth in the parental treatment. In either case, genetic variation can show up in the association between parental treatment and depression.

This idea is supported by a genetic decomposition of the correlation of  $-0.37$  between depressed mood and family connectedness. We decomposed this correlation into genetic and nonshared environmental components using the identical twins, fraternal twins, full siblings, half-siblings, and unrelated siblings in the Add Health sample. The exact methods are too technical for discussion in this short article. I can describe, though, part of the procedure for detecting genetic influence. This is a calculation of the *cross-sibling correlation*. That is, sibling A's family connectedness score is correlated with sibling B's depression score. When the sibling correlation is greater for siblings who are more closely related genetically, a genetic influence is inferred. Thus, a genetic influence implies a greater cross-sibling correlation in full siblings (related by 0.50) than in half siblings (related by 0.25).

The pie chart shown in FIGURE 4 presents the decomposition of the  $-0.37$  association into its genetic and environmental components. The majority (56%) of this association is due to genetic influences that are common to depressed mood and family connectedness. That does not mean that in any simple sense a gene exists for "family connectedness." What it does mean is that girls with a gene that predisposes them towards depressed mood react to parental treatments differently from those girls without these genes; they may also evoke different parental treatment. Parental treatment is not, in any simple way, a cause of the daughters' depressed mood (see also, Neale *et al.*<sup>12</sup>).

Results for boys were different: Most of the association between boys' depressed mood and family connectedness was due to nonshared environments, with little genetic effect. Boys also experienced less depressed mood than girls overall. The reason why boys are different from girls—if I can even pretend to know the answer to *that* question—would take me well beyond the scope of my topic.





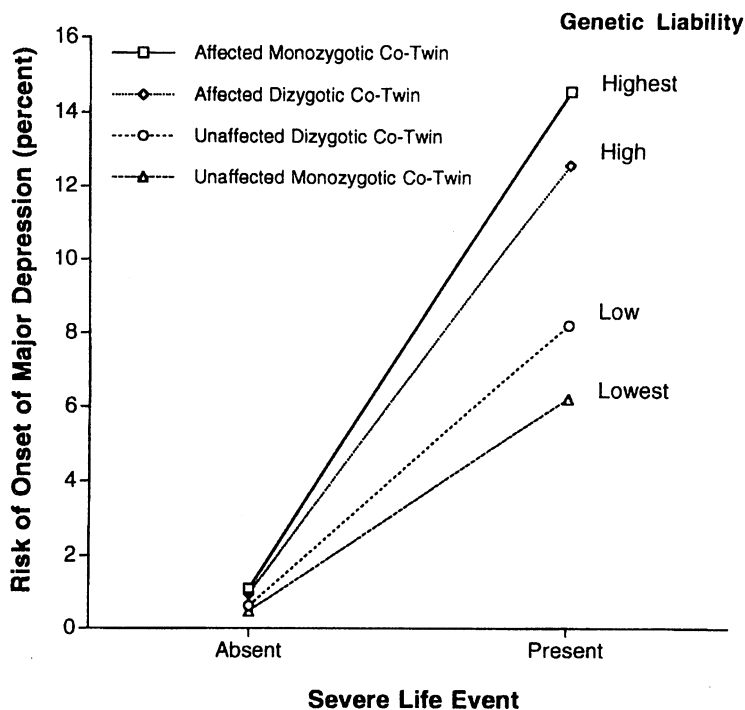
**FIGURE 4.** Decomposition of correlation of  $-0.37$  between depressed mood and family connectedness in adolescent girls. NSE = nonshared environment.

**TABLE 1.** Life events surveyed in female twins

| Event   |
|---|
| Assault   |
| Serious marital problems                          |
| Divorce/breakup                                   |
| Job loss  |
| Loss of confidant                                 |
| Serious illness                                   |
| Major   |
| Financial problem                                 |
| Death of close relative                           |
| Serious trouble getting along with close relative |
| Serious illness of close relative                 |

Among female twins, an interaction of psychological stressors by genetic predisposition is also demonstrable.<sup>13</sup> The psychological stressors were serious life events that took place a few months before an interview. TABLE 1 lists the life events given in an interview to female twins. The more serious life events, such as the death of a close relative, serious illness of a close relative, or an assault, were unlikely to be caused by any action of the twin. Life events did lead to episodes of depression. The odds of a depressive episode were 60% greater after a serious life event than if no life event had occurred. Genetic influences were also important. By coincidence, an affected biological relative also made the odds of a depressive episode 60% greater.

The most interesting result from this twin study was an interaction of genetic predisposition and a severe life effect. The genetic disposition (called a liability in



**FIGURE 5.** Risk of onset of major depression per person-month as a function of genetic liability and the presence or absence of a severely stressful life event in that month among 2,060 female twins. (Reprinted from Kendler *et al.*<sup>13</sup> with the permission of the American Psychiatric Association.)

FIG. 5) was assessed by the presence of a major depression in the co-twin. A severe life event in the same month increased the percentage of individuals who reported an episode of major depression. When the life event was absent in the month, under 2% of the 2,164 individuals in the study reported onset of depression. When the life event was present in the month, the range was from about 6.2 to 14.6%. The risk of depression was conditioned on the genetic predisposition. When a identical twin had an affected co-twin—strong evidence for a biological disposition—the risk of depression was the highest, 14.4%. When an identical twin had an unaffected co-twin, the rate was lowest, 6.2%. The rates for fraternal twins with affected and unaffected relatives fell in the middle. Thus, life events are most likely to lead to major depression in persons who are primed for depression by their biological inheritance.

**GENETIC INFLUENCES ON THE “ENVIRONMENT”**

My previous example dealt with the influence of life events. We saw that the experience of loss can trigger depression, especially in susceptible persons. Some life

events are definitely uncontrollable—except in the old-time Mafia, the death of a close relative is unlikely to be a consequence of another relative’s behavior. Other life events are more controllable. Marital conflict is partly an external stressor, but a person can also provoke a fight with a spouse. Auto accidents can be blameless, or a consequence of a history of reckless driving and road rage. Daily hassles can be a source of stress, but persons who are rude and self-centered may provoke greater hostility from others than do agreeable persons.

What my few examples show—and I could generate many more—is that people, in many ways, *make their own environments*. Perhaps we need a distinction between the “behavioral environment” and other kinds of environments, such as the physical environment and the ecological environment. The environment of greatest interest to a working behavioral scientist is usually made by other people. Office conflict might raise cortisol levels in office workers, but the “environment” is not something independent of their gossip and back-biting. The family environment is just the way parents treat their children; it is manufactured by parental behaviors. Because what we label “the environment” is really peoples’ behaviors, environmental variation can show genetic influence just as individual characteristics like height and weight do.

This is exactly what is found when we look for genetic influences in a life events scale.<sup>14,15</sup> In Plomin’s study, twins reported on their life events using a multi-item life events scale.<sup>15</sup> Life events were divided into two types: controllable life events (e.g., hassles in the office) and uncontrollable life events (e.g., a death in the family). Genetic influences were estimated using the resemblance of twins in four groups: identical twins raised together, identical twins raised apart, fraternal twins raised together, and fraternal twins raised apart. The sample sizes ranged from 45 identical twin pairs raised apart to 127 fraternal twins raised together. FIGURE 6 presents the heritability estimates for the two kinds of life events; they were 0.43 and 0.18, for controllable and uncontrollable life events, respectively. What is called in behavioral

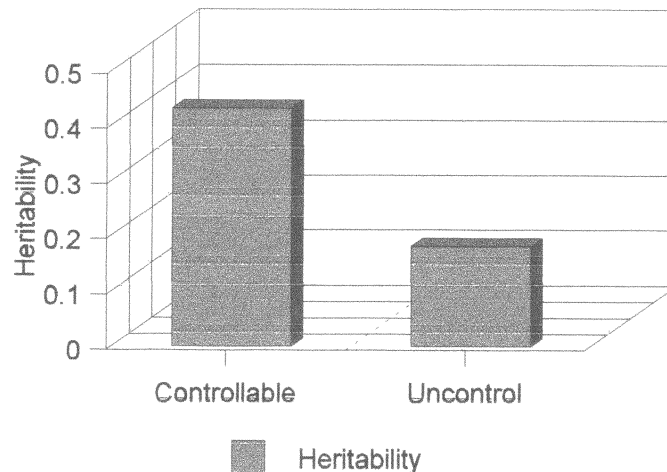


FIGURE 6. Heritability of controllable and uncontrollable life events.

science the environment, because it is a result of peoples' behavior, is not always innocent of genetic influence.

### PROSPECTS

The theme of this conference is the consilience of knowledge. One form of consilience is integrating knowledge from different levels of analysis. Science has been successful by pursuing a reduction of complex phenomena into simpler parts. Complex functions in cell biology have been reduced to biochemical pathways: protein products to the transcription of genes and genes to a genetic code shared by all living species.

In behavioral science, most fields have assiduously avoided this process of looking down from a complex phenomenon to its component parts. Child development and sociology, to identify two fields, have not always embraced a biological level of analysis that creates lasting and recognizable differences among individuals. No one, of course, can ignore biologically based differences in height, weight, or complexion. Biological differences in the brain, brought about by inheriting different genes, are not directly visible to an observer; their effects must be inferred, as I have shown using twin and adoption study designs. I have shown that the reason some people are persistently happy, and others persistently miserable, is that they have different biological set points for these emotional states. I have shown that people who differ genetically may experience different social environments—environments are not random, but are experienced according to biases that come with different genetic inheritances. I have also shown that, although even stressful life events leave most people unscathed, people with a biological predisposition towards depression are more responsive to these events than those without this predisposition. To understand human individuality, social science must be *bio-social* in its outlook.

One future prospect is to look to drugs to change behavior. The general public is already embracing the biology of mental states, perhaps too much so. A cartoon about the book *Listening to Prozac*<sup>16</sup> shows a woman reaching for a detergent box labeled, "Prozac—mood brightener." Drugs make possible a manipulation of biological individuality that was heretofore impossible. They offer reduction in the symptoms of serious mental illnesses that are debilitating for their sufferers. However, drugs may also offer enhancements of personality. Should the unhappy try to brighten their moods? Is the pursuit of happiness, or happiness itself, the birthright of every child born in the United States?

A second future prospect is tying complex traits to single genes. This article has not covered work in molecular psychiatry relating specific genes to depression or happiness. One reason is that any single gene will explain only a small part of the variation in a behavioral trait. It will take many years to acquire enough genetic information to make predictions about individuals' likely mood from their genotypes, although the information will be more useful at a group level earlier. When genotypes can be linked to predispositions toward both happiness and depression, behavioral science will develop a deeper understanding of how people make their own environments.

Consilience is uniting different analytic levels. To try to understand individual differences in emotional response without reference to biology is fruitless. To try to

understand the historical changes in depression rates without reference to history and culture is also bound to fail. Scientific advance depends on a relentless effort to reduce complex phenomena to their less complex elements and to build back and see how the elements contribute to the whole. Modern research on happiness and depression is a good illustration of this principle of consilience between the natural and social sciences.

### ACKNOWLEDGMENTS

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