Sample Paper

This paper was written by one of my students and provides a general idea of what I am looking for in your paper for this class. I believe that it is a very good paper in that it reflects the format that is required, and a high level of writing and thinking ability. Note that the paper consists of three parts: a summary of a psychology related article, a critical analysis of the article, and the article itself. Note also the general length of the student’s paper and of the article the student chose to write about.

Professor Stewart
Psy 300
Summary/Analysis Paper

“The Character Code”

Summary:

In the article “The Character Code” found in the Scientific American Mind, Turhan Canli writes about a certain “anxiety gene” that scientists have recently been studying. This gene seems to be connected with the increased likelihood of people who have this gene to becoming depressed when something upsetting occurs in their life. Canli stresses that this gene alone doesn’t make people depressed—environment also has an impact—but it does seem to make them more susceptible to mental problems. This discovery offers an intriguing possibility that a person’s genes might play a large part in determining their personality. However, this topic can be controversial, as it seems to imply that people have less control over their reactions to life than they might like to believe.

This controversial “anxiety gene” that Canli writes about has to do with serotonin. Serotonin has many functions, including affecting mood. Neurons within the nervous system have special serotonin transporters that affect the reuptake of serotonin after it has been secreted to excite the next neuron. These serotonin transporters seem to be very important. In 1996, Lesch and colleagues from the NIMH and the National Cancer Institute did a study, and found that there are two possible lengths of the serotonin reuptake gene that people can have: short or long. Lesch discovered that the long gene gives a neuron more transporters than a short gene does. After finding this, he tested people for anxiety and found that those with the short gene showed more tendencies towards anxiety that those with the long gene. Lesch’s conclusion was that this gene “accounts for 3 to 4 percent of the total variation...in anxiety-related personality traits.”

Later studies have built upon these findings about the impact of the length of this serotonin transporter gene. Researchers have since discovered that people who have a short gene have more excitable amygdalas. This is significant because the amygdala has to do with how the brain deals with strong emotions. People with more
excitable amygdalas (and the short transporter gene) have stronger emotional responses—fear, anger, depression—than do people with less excitable amygdalas (and long transporter genes).

Canli, the author, has done his own research on this possibility and found that it does seem as though these transporter genes do affect how people react to negative stimuli. But he also has found that it seems as though the length of these genes affects how dynamic the brain is in general, even in a so-called “resting-state”. Canli conducted a test in which he had subjects first look at a screen with negative words, and then at a blank screen. The brains of those people who had short transporter genes had more activity in the amygdala, and other parts of the brain as well, at all times as compared to the brains of the subjects who had long genes. Because of these findings, Canli suggests that “this chronic arousal may lead to anxiety, fearfulness and, possibly, a predisposition to mood disorders such as depression” for people who have the short serotonin transporter gene.

But does having a short serotonin transporter gene actually make someone depressed? Studies have found that this gene alone does not have the power to make someone depressed. The environment also plays a large part. A 2003 study by Caspi of King’s College London found that this gene does not affect a person’s risk of depression under normal, non-stressful conditions. But this gene does affect a person’s risk for depression when they are under high stress. The higher the levels of stress, and the more tragic or distressing circumstances a person suffers, the more this gene seems to increase the likelihood of the person becoming depressed. In this particular study, after experiencing at least four extremely distressing events in their lives, 33 percent of the subjects with short transporter genes became depressed, while only 17 percent of the subjects with the long transporter genes became depressed. Further studies by other researchers have seemed to support these findings, as they show that people with these short genes are more likely to undergo bouts of depression after painful or traumatic life events than are people with long transporter genes.

Canli and Lesch, along with a few colleagues, did more research on this topic and found that the length of a person’s serotonin transporter gene also seems to affect how likely they are to dwell on negative events in their life. In this study, subjects with short genes had more activity in the resting state of their brains in both their amygdalas and hippocampuses. The hippocampus is responsible for memory, so when combined with the emotions from the amygdala, it would seem as though these subjects were more likely to recall and continue to respond to past events (memories) even when they were no longer undergoing stress. In contrast, subjects with long genes had lower resting levels of activity in their amygdalas and hippocampuses. If fact, the more traumatic events they experienced in their lives, the lower the activity levels were in those areas of their brains. This seems to suggest that these people didn’t dwell on their past traumas, and were therefore less likely to become depressed about
them. It could be that the more active brains caused by short transporter genes make subjects think too much about their problems and therefore become depressed.

Despite these significant findings from the research done on the serotonin transporter gene, at the end of the article the author warns that this specific gene is just one of at least 15 genes that affect how a person responds to stress. And of course environment also plays a part. Much more research is still needed to fully understand genetic dispositions towards actual behavior, but the author suggests that in the future these findings might lead to better treatment for patients that are at a high risk for stress.

Analysis:

This article focuses on the impact one tiny little serotonin transporter gene can have on how likely a person is to become depressed under stressful circumstances. On a small scale, this article focuses on this gene as being only one factor—one gene among at least 15—that affects a person’s sensitivities to life. But on a large scale, these discoveries open the door to much larger possibilities, and have much larger implications.

First, it seems as though the discovery of this serotonin transporter gene and its effects on people’s reactions to stress could be very helpful in the field of medicine. Once doctors can scientifically understand and explain a problem, they can look for more effective ways to treat it. This can easily be seen in the cases of more “physical” ailments and diseases where antibiotics and other treatments have been discovered. In the past, it seems as though mental problems and feelings didn’t fit into the categories of true illnesses. But in today’s world, there are countless recognized mental disorders as scientists begin to discover that there are actual biological factors that can account for such illnesses. Problems such as depression are no longer considered to be just in someone’s head. Instead, depression is considered a legitimate illness that is treated with drugs as well as therapy. This is where discoveries such as the impact of the serotonin transporter gene seem as though they will come in handy. Maybe scientists will be able to come up with a drug meant specifically for people with short transporter genes that will be able to benefit them in the largest way possible. It follows to reason that as more and more biological factors are discovered to account for depression, the more specialized, and presumably more successful, drugs for this problem might become. I believe that continued research in this field could greatly help the countless people who are suffering from depression, and whose current treatment is not as helpful as it could be.

Yet there may be implications beyond just new medicines, the ability to treat illnesses before they occur, and the capability to set up different treatments depending on gene sequence. Will scientists also be able to alter faulty genes, or maybe even create a “perfect” person? Who knows? New advances in gene therapy and gene cloning
certainly seem to be heading in this direction. But as far as the possibility of creating humans with the ideal genetic code, or altering faulty or undesirable genes, what is within our rights as humans to do? With such limitless possibilities and power come much responsibility and many potential problems. As far as maybe having one’s genotype read—as the Human Genome Project shows is now becoming possible—is it really a healthy idea to know what one’s genes might imply? As we pondered in class, is it really beneficial for someone to know they will probably become depressed, or have a certain disease, or be likely to die young? Knowing such likelihoods may actually make a person unwittingly act in such a way as to bring about a self-fulfilling prophecy. Increasing genetic discoveries could certainly give rise to many ethical issues with no clear or simple answers.

Yet even in the face of discoveries concerning the powers of genetic codes, the environment can’t be completely forgotten. Consider again the case of the serotonin transporter gene and its relationship with depression. As the author of the article was careful to point out, environment plays a part as to whether or not this specific serotonin transporter gene will actually make it likely for someone to become depressed. I think that it is important to remember that biological factors alone do not completely determine the outcomes in a person’s life. I fear that with such incredible advances in science, there is a danger of this being forgotten. For as scientists do more research, and continue to unlock the human genetic code, they might begin to compare the relationship of this genetic code and how a person functions to that of a program telling a computer how to run. But do people really want to think of themselves as nothing more than extremely sophisticated robots, born and hardwired with a specific genetic code that serves as the program running their life? Do people really want to believe they were born to behave a certain way, and that there is nothing they can do about it? If so, this could be used as an excuse for ill behavior and as a means to dodge responsibility for one’s actions, including criminal actions.

Although the specific focus of this article was on how the length of just one tiny serotonin reuptake gene can affect how likely a person is to become depressed, taking this discovery and applying this concept to other genes leads to larger implications. It seems as though this human program outlined in the human genetic code just might determine our personality and, therefore, how we live our lives. The implications of this mysterious genetic code might seem very promising, especially to scientists, researchers and doctors. Once unlocked, it could give a nice neat little explanation of how to expect a person to behave, and how their behavior can be treated and modified. But I wonder if this is just a little bit too scientific and cold. Can human beings really be broken down into a simple code in their DNA and be completely defined by it? If this is the case, we may be destined to either fail or succeed from birth, depending on what genes we were born with.

But if the environment is indeed important, as the author suggests, we are not completely predestined to either
fail or succeed. In this case, the experiences a person undergoes affects how likely they are to become depressed, even if they do have certain predispositions determined by their biological makeup. I would like to think that our environment and how we choose to respond to it also determines our personality and whether or not we are going to be depressed. But perhaps it is just that I don’t want to think that we all born either destined to be happy or destined to be depressed, destined to succeed or destined to fail. Maybe I just would like to think that we have a certain amount of control over how our lives turn out, while in reality we all have far less control than we would like to believe. But I certainly hope that is not the case. I believe that it is a combination of biological factors, environment, and our own choices and actions that make us who we are, and which ultimately decide how we view life.

I know that in my own life, I can see how these factors all interact. When I was in my early teens, I went through a time where I could have been considered to be depressed. I haven’t a clue whether or not I have a long or a short serotonin transporter gene, but I do know that I have always had a tendency to think about both past, present, and future issues more than is probably healthy. I “ruminate”, as the author of the article would say. Does this make me more predisposed to depression? Maybe so. At the time I was depressed, however, there were environmental issues going on as well as problems with my own outlook. I found that eventually, after I mentally worked through my issues and tried to make actual changes in my actions and how I responded to events in my life, my depression gradually faded away. This experience has led me to believe that there are many choices we have in our own lives, and that we are not completely ruled by our biological factors. All factors are important and deserve consideration.

So am I correct in my beliefs? Perhaps there is no real answer to this question. The exact importance and impact of these different factors is certainly under debate, but as shown in the article, genetic factors are certainly under increasing focus. Science is such an ever-changing field, with discoveries being made every day that would once have been beyond the wildest of imaginations. Genetic discoveries can offer countless possible medical advances that are full of promise, but also raise many ethical questions and questions of self-will. Although it may not seem like it at first, the implications of even one small gene concerning serotonin and the likelihood of depression can potentially unlock a Pandora box of unanswerable questions and issues. Who knows what the future will hold?

Article: Scientific American Mind, February/March, 2008

The Character Code
By Turhan Canli
You are diagnosed with a crippling illness. You lose your job. Someone close to you suddenly dies. Some people recover rapidly from life’s calamities and disappointments, whereas others are devastated.

The roots of such emotional differences have fascinated psychologists and nonspecialists alike. Environmental factors, such as a person's upbringing, exert a tremendous influence on his or her resilience in the face of misfortune or failure. But as biologists (and parents) have long suspected, genes lay much of the groundwork for individual personality traits. Studies that compare the traits of identical twins, who have all the same genes, with those of fraternal twins, who share just half their DNA, suggest that genes account for 40 to 60 percent of the individual variation in anxiety levels and susceptibility to depression.

Recently scientists have begun to identify specific genes that shape facets of human personality. Based on an early understanding of the chemical underpinnings of mood and mood disorders, they have pinpointed genetic quirks that may contribute to curiosity, attention deficits and impulsive violence.

The roots of anxiety and emotional resilience reside partially in a gene that affects brain levels of serotonin—a chemical messenger that influences sleep, thought and mood, among other functions. The anxiety-provoking form of this gene is very common; more than half of the Caucasian population has inherited it from at least one parent. Recent work has not only connected this gene with anxiety-related personality traits but has also established a basis in the brain for the gene's effects on anxiety.

This "anxiety gene" raises the risk of depression, however, only in the wake of very difficult life circumstances, the latest data show, illustrating the importance of an interaction between genes and particular life experiences in molding personality. Revealing such molecular tics to anxiety, along with their partners in the environment, may lead not only to a new understanding of human behavior but also to better treatments for—and possibly ways to prevent—mood disorders.

**Anxiety Gene**

Scientists have long speculated that problems with serotonin signaling underlie much of the pathology of mood disorders. A key molecule in this process is a protein called the serotonin transporter, which pumps serotonin from the space outside neurons, the synapse, back into neurons.

Indeed, research reported in the 1980s and 1990s hinted that people with depression and certain anxiety disorders bore either fewer or less efficient serotonin transporters than normal. Meanwhile scientists discovered an association between heightened anxiety levels in animals and people and increases in serotonin-induced communication between neurons. (Paradoxically, Prozac and similar antidepressants reduce anxiety and depression by inhibiting serotonin reuptake, and thus boosting levels of serotonin outside neurons—something scientists are still struggling to explain.)

Such observations led clinical psychiatrist Klaus-Peter Lesch of the University of Wuerzburg and his colleagues at the National Institute of Mental Health (NIMH) and the National Cancer Institute to wonder whether variations in the gene, or molecular blueprint, for the serotonin transporter might influence a person's anxiety level and possibly his or her susceptibility to depression. Lesch and his colleagues discovered that the gene came in two lengths—long and short. Both produced functional proteins, but as the researchers reported in 1996, the long form of the gene causes a neuron to churn out more of the transporter than the short one does.

This quantitative difference does affect anxiousness, Lesch's team found. Among 505 people who took a test for anxiety-associated traits, those who had inherited at least one copy of the short version of the serotonin transporter gene received higher scores than did those who inherited the long version of the gene from both parents. Lesch and his co-workers concluded that the serotonin transporter gene accounts for 3 to 4 percent of the total variation—and 7 to 9 percent of the inherited variation—in anxiety-related personality traits.

**Angst in the Brain**

Researchers have since identified a neurological basis for this effect: having a short serotonin transporter gene boosts the excitability of the amygdala, an almond-shaped group of neurons deep in the brain that processes fear and other emotions. In 2002 psychiatry researcher Ahmad R. Hariri, then at the NIMH, and his colleagues reported showing 28 healthy volunteers faces conveying fear or anger or bearing neutral expressions while they scanned their brains using functional magnetic resonance imaging (fMRI). They found that in the 14 people who had inherited at least one copy of the short transporter gene the amygdala was especially enlivened by the emotive faces. It was less active in the individuals with two long forms of the gene.

Additional studies have buttressed the theory that this genetic variant has consequences for the emotional
brain. In a 2004 study a team led by psychologist Tomas Furmark of Uppsala University in Sweden showed that patients with social phobia who carried the short form of the serotonin transporter gene showed more activity in the amygdala during a public speaking task than did those with two long versions of the gene. Other researchers found that a part of the prefrontal cortex charged with the processing of risk and fear was also more aroused in response to negative images in bearers of the short transporter gene.

Research from my laboratory suggests, however, that the effect of this gene on brain activity may be more general; rather than controlling the response to negative stimuli, it may instead fine-tune the background level of neural activity in the emotional brain. Lesch and I, along with several colleagues, measured activation levels in the amygdala and other brain regions in 41 people while they viewed negative, neutral and positive words—or just stared at a spot on a computer screen.

Corroborating Hariri's work, we found that the people with at least one short transporter gene showed higher activity in the amygdala in response to negative stimuli—words, in this case—than did the individuals carrying two long forms of the gene. More surprisingly, however, as we reported in 2005, the amygdala of those who had the short gene was unusually dynamic while the subjects were simply staring at the computer screen, and we discovered that this resting-state dynamism could account for the amygdala's enhanced response to negative stimuli. We also observed greater neural activation in response to positive stimuli in other brain regions in the individuals carrying the short transporter gene.

Our data thus suggest that the amygdala and other parts of the emotional brain are naturally more aroused in people who have inherited the short serotonin transporter gene. We hypothesize that this chronic arousal may lead to anxiety, fearfulness and, possibly, a predisposition to mood disorders such as depression.

**Surviving Stress**

But carrying this genetic variant is unlikely to beget depression unless your environment also conspires against you. Studies show that the gene variant boosts depression risk only in the presence of significant stress from misfortune or failure. In 2003 psychiatry researcher Avshalom Caspi of King's College London and his colleagues reported analyzing the serotonin transporter gene in 847 New Zealanders whom they also surveyed about stressful life events such as illness, financial difficulties and romantic disappointments that had occurred between ages 21 and 26.

Although a person's transporter gene did not budge depression risk in the absence of stress, it did influence his or her tendency toward gloominess in response to adversity. The risk of depression and suicidal thoughts rose as the number of stressful events mounted—but only in those with at least one short copy of the transporter gene. And after four or more traumatic occurrences, 33 percent of the subjects who carried at least one short transporter gene became depressed as compared with just 17 percent of those who bore two copies of the lengthier blueprint, suggesting that the long gene protects against depression in the wake of acutely negative experiences.

In 2005 psychiatric geneticist Kenneth S. Kendler and his colleagues at Virginia Commonwealth University replicated this finding in 549 twins whom they interviewed about signs of major depression and anxiety within the past year, along with the occurrence—dated to the nearest month—of 15 types of stressful life events, including divorce, job loss, robbery and illness in the family. The researchers found that individuals with two short forms of the serotonin transporter gene were more likely to become depressed after mild stressors than were those with one or two long forms. The next year geneticist Peter R. Schofield of the Prince of Wales Medical Research Institute in Sydney and his co-workers reported that serious adversity was more likely to produce a bout of major depression within five years in those carrying two short transporter genes, as compared with those who had at least one long transporter gene.

Lesch and I, along with several colleagues, observed these differing vulnerabilities to life's misfortunes in the brain. We surveyed 48 healthy volunteers to determine how many times they had experienced significant tension from, say, work, relationships, finances or illness; some of them were also assessed for their tendency to ruminate, a risk factor for depression. We then used noninvasive imaging techniques such as fMRI to measure brain activity while the subjects focused on various facial expressions or just stared at a spot on a computer screen.

In the individuals carrying a short serotonin transporter gene, life stress was not associated with a boost in brain activation in response to moody facial expressions but did lead to a higher resting level of activity in the amygdala and hippocampus—a memory-processing region that is vulnerable to stress—and to a greater tendency to ruminate. The opposite was true for people who carried two long forms of the gene: the more crises these people had faced, the lower the background level of activity in their amygdala and hippocampus—and the less they dwelled on things, we reported in 2006. These results support the theory that stress interacts with a short serotonin transporter gene to produce a highly vigilant emotional brain that predisposes a person toward depression.

Stress that is combined with a deficit in the serotonin transporter protein perturbs the emotional brains of mice as well. In a study published in 2007 geneticist Andrew Holmes of the National Institute on
Alcohol Abuse and Alcoholism and his colleagues deleted the serotonin transporter gene in mice, creating a lack of the transporter protein meant to mimic the less pronounced deficit in people who inherit the abbreviated transporter gene.

The genetically altered mice showed signs of depression—they stood still more than normal mice—after several stressful swim tests. The animals also had unusual difficulty getting over a fear of a sound the researchers had instilled in them—by pairing the sound with a shock to the foot—and then subsequently tried to extinguish. The DNA disruption also affected the rodents’ brains; in the genetically manipulated mice, the researchers found structural oddities in the amygdala and in an area of the prefrontal cortex that plays a role in stress and fear.

Molecules of the Mind

Despite such findings, variation in the serotonin transporter gene is thought to explain only a small part of people's differing sensitivities to the stresses of life. Geneticists estimate that at least 15 genes, along with various environmental factors, most likely contribute to anxiety and a person's susceptibility to stress. Only if researchers can complete this genetic and environmental mosaic will they be able to confidently gauge the propensity to develop anxiety and depression in each of us—or to pinpoint the causes of such disorders in those who have them.

Moreover, with a more complete picture, those of us at high risk might be able to act to prevent the emergence of such mood disorders. And an ability to home in on a molecular cause of anxiety or depression in psychiatric patients might enable doctors to select the treatment most likely to work for each patient.

Meanwhile the unraveling of the entire human genetic code under the auspices of the Human Genome Project and of human genetic variation in the so-called Human HapMap Project is expected to accelerate the outing of genes that work in the brain to shape our personalities. As these genes come to light, psychologists will have to increasingly incorporate these molecules of inheritance into their explanations of human behavior, mental illness and temperament.

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