



Faith & Genetics

Behavioural Genetics

Genes and Their Environment

Is there a gene for bungee-jumping? Is alcoholism a genetic trait just like blood type? Are there genes for schizophrenia? Is a person's personality a series of chemical reactions in the brain determined by their genes? Scientists who study these kinds of questions are called behavioral geneticists. Most of these geneticists would say the data suggest that personality traits are influenced by but not determined by genes.

Most human characteristics are not determined by a single gene. The simple Punnett square that can be used to determine blood type won't work for most characteristics. Many human characteristics are influenced by several genes working together. And frequently, a human characteristic is the result of the interaction of one or more genes and the environment. When we say environment here, we are not talking about just the outside world—where you went to school, what you had for lunch, whether or not you exercise. The environment of a gene includes the other genes in the cell, the hormones and other chemicals to which the cell is exposed, even the interactions of cells and tissues, and also the environment outside the body.

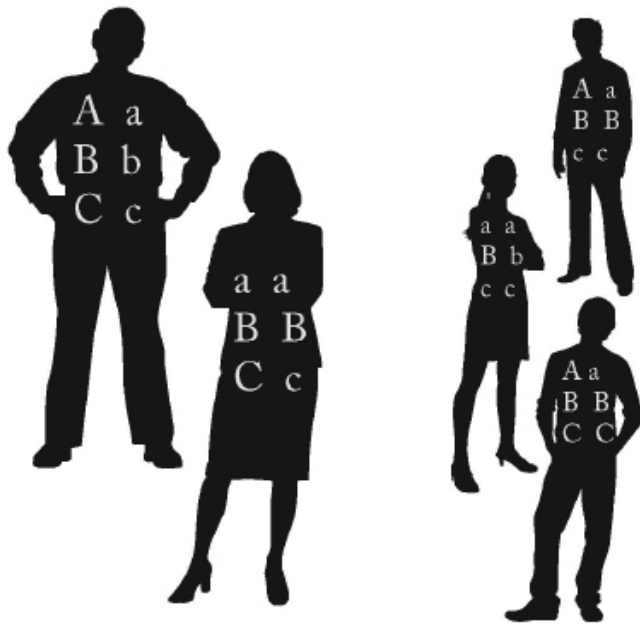
Inheritance Involving More than One Gene

Let's look first at traits that are determined by several genes, a phenomenon known as polygenic inheritance. Traits that are influenced by several genes often show a similar pattern. A good example is height. Height is a continuously varying characteristic. That is, it is not the case that humans are all either 5 feet tall or 6 feet tall. Rather, human heights are distributed through a range. Multiple genes contribute to height. In the simplest case, each gene would have two alleles: additive and nonadditive. Additive alleles would result in greater height while nonadditive alleles would not. If (and this is not really the case), height were controlled by 5 genes, each with equally acting additive and nonadditive alleles, someone with 10 additive alleles would be among the tallest humans.



Let's look at the inheritance of polygenic traits. Suppose three genes interact to influence a person's height. Because every child inherits one allele of each gene from each parent, you can look at all three genes and even prepare a Punnett square for each gene. As can be seen here, a couple who each have three dominant alleles (as indicated by A, B, and C in the figure) and three recessive alleles (indicated by a, b, c) among the three genes could end up with children who were the same height as they are, but could also be significantly shorter or taller.

You may be thinking, doesn't environment affect height? Yes, but negligibly. The vast majority of the characteristic of height is genetically determined. If my parents had fed me protein shakes as a child and sent me to a Montessori school, I still wouldn't be 7 feet tall. Environment will result in minor adjustments to the genetic underpinnings that determine a person's height.



Many characteristics are determined polygenically, including skin colour, weight, blood pressure, and blood cholesterol levels. Of course, these characteristics also have an environmental component. Determining whether a trait is determined by genes or an interaction between genes and the environment is difficult, but not impossible. In some cases, it is actually quite simple, as when a trait is determined by only one or two genes interacting with the environment.

For example, the risk of stroke by a blood clot can be related to certain alleles for two genes: prothrombin and clotting factor V. The names are not important. What is important is that the risk of stroke due to a blood clot is nearly 150 times higher in women who have the risky alleles and who are taking oral contraceptives (the environmental effect). Taking oral contraceptives if you don't have the risky genes only raises the risks 3 times. Here is a clear interaction between two easily identified genes and a single environmental factor.



Genetic Variation and the Environment: Complex Interactions

Most of the time, however, the interactions are more complicated. The number of genes involved is unknown as is the percentage of variation in the trait due to genetic variation and the percentage of variation in a trait due to environmental variation. Nowhere is this more true than with behavioural genetics.

Behavioural traits include abilities, feelings, moods, personality, intelligence, and how a person communicates, copes with rage, and handles stress. Disorders with behavioural symptoms are wide-ranging and include phobias, anxiety, dementia, psychosis, addiction and mood alteration. Very few medical conditions with behavioural components can be traced to a single gene. Huntington disease is a rare example of such a condition.

Huntington's disease is a fatal, progressive, neurodegenerative disease caused by a dominant mutated allele. Individuals who are heterozygous for Huntington disease usually develop symptoms in their late 30's or 40's. Some early symptoms of HD are mood swings, depression, and irritability or trouble driving, learning new things, remembering a fact, or making a decision. As the disease progresses, concentration

on intellectual tasks becomes increasingly difficult and the affected person may have difficulty feeding himself or herself and swallowing. Angry outbursts are the hallmark characteristic of this disease.

Since a mutation in a gene can result in behavioural traits, it is clear that genes can be involved in human behaviours. Unlike Huntington's disease, however, most behavioural disorders are not the result of a single mutated gene.

Investigating the genetics of behavior is more difficult than understanding a disorder such as sickle cell disease or Huntington's disease in which an abnormal protein clearly disrupts physiology in a particular way. One of the reasons why such investigations are difficult is that many behavioral disorders share symptoms, which can complicate diagnosis. For example, poor concentration may be a symptom of attention deficit disorder (ADD), major depressive disorders, or post-traumatic stress disorder, to name a few. Further to this, many symptoms including poor concentration can be considered part of normal behavior – surely everyone from time to time has a hard time concentrating or experiences mood swings when under some degree of stress for a time.



Another challenge to understanding the relationship between genes and behaviours is the highly subjective nature of studies that rely on self-reporting of symptoms by study subjects. A person can also, unintentionally, copy someone's unusual behavior, because he or she does not realize it's unusual. Such sources of confusion do not occur with diseases such as cystic fibrosis where a strictly physical symptoms such as shortness of breath and cough are characteristic manifestations of the disorder.

Although it is necessary to be cautious when assigning a genetic cause to a behavior, it is still possible to examine genes that contribute to a particular behavior. Typically, scientists attempt to identify behaviors that appear to be inherited, then focus on identifying and describing candidate genes (these behavioural disorders under genetic influence will be discussed in more detail in an upcoming section).

How are the experiments performed to determine whether a candidate gene is actually involved in a behavioral trait? Let's look at the gene for the serotonin transporter. Serotonin is a neurotransmitter, a chemical that carries messages from one nerve cell to another. One cell produces and releases the serotonin and a nearby cell binds the serotonin and responds in a certain way. The longer the serotonin is present, the more signaling occurs. A certain protein, called a serotonin transporter) binds to serotonin when it is between the two cells

and returns it to the releasing cell. This process shuts off the signal and thus prevents the signal from becoming continuous. Just as a car alarm can be an important signal, a car alarm that doesn't shut off soon becomes a problem. Certain drugs called selective serotonin reuptake inhibitors (SSRIs), like the antidepressants Prozac and Paxil, slow down the rate at which the serotonin is returned to the releasing cell. Having a slightly longer time for signaling appears to relieve depression in many people. Therefore, the serotonin transporter is a good candidate gene to examine for depression, anxiety, neuroses, and so on.



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It turns out that there are two alleles for the gene associated with the serotonin transporter, called the long and short alleles. The long form is more active and more quickly mops up serotonin from the space between neurons. That is, people with the long form of the gene have a shorter signaling time than people with the short form of the gene. The question then is, can we detect a behavioral difference between people with these two forms of the gene? Researchers at the National Institutes of Health conducted a study in which people's transporter genes were examined. The participants also took a standardized



test that measures neuroticism. Each individual was then given a neuroticism score. Not surprisingly, when the scores of all the people were plotted, the scores formed a generally bell-shaped curve. Some people were extremely neurotic, some were extremely tranquil, but the majority of people were somewhere in between. When the neuroticism scores of people with the two allelic forms of the serotonin transporter were plotted separately, we see that both sets of people formed a somewhat normal distribution. But if one examined the graph carefully, it could be seen that the average neuroticism score of individuals with the long form was slightly higher than the average neuroticism score of individuals

with the short form. Statistical analysis of the results suggested that approximately 1% of the variation in neuroticism scores among humans was due to a variation in the gene for the serotonin transporter. This difference is small, but real. Clearly, other genes are involved as well as the environment. The tricky part is trying to figure out how many genes may be involved and how they interact.