Genes and Psychology in The News

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Abstract

This article critiques the way that news articles report the relationship between genes and psychological processes. The news frequently reports that genes cause various psychological processes. However, these claims rest upon unscientific data; selective use of data (ignoring contradictory data); failure to consider just how the physical properties of genes and their by-products (e.g., neurotransmitters) could or could not control psychological phenomena; misconstruing the nature of psychological phenomena; specious arguments; and unwarranted leaps of faith. Consequently, the causal conclusion is unwarranted, and news reports misinform the public about this vital issue. I delineate an alternative theory of the relation between biology and psychology which explains why genes may correlate with psychological processes without causing, or predisposing, them.

Recently a spate of science articles in newspapers has reported evidence that a single gene controls psychological phenomena such as language, antisocial behavior, schizophrenia, and depression. These news articles are important because they shape the public's scientific knowledge. Consequently, it is important to evaluate the reported evidence and conclusions in order to ascertain whether the public is being educated or misled.

Let us first consider the gene for language. News articles around August 14, 2002 announced that a gene for language has been discovered. The research originally appeared in the Oct. 4, 2001 issue of Nature. The discovery was that a few individuals (mostly from a family called KE) with a particular genetic defect have impaired speech and grammar recognition.

This does not imply that a gene for language exists. The deficits of KE are much broader than grammar. They include low IQ (mean of 75), dyslexia, and the inability to repeat simple single words. Dr. Vargha-Khadem, a cognitive neuroscientist who has studied the subjects for 10 years, and first identified the mutant gene FOXP2 stated: "The inherited disorder does not affect morphosyntax exclusively, or even primarily; rather, it affects intellectual, linguistic, and orofacial praxic functions generally. The evidence from the KE family thus provides no support for the proposed existence of grammar-specific genes" (Vargha-Khadem, et al., 1995, p. 930, emphasis added). In other words, the abnormal FOXP2 gene interferes with a wide range of psychological capacities. It is not a gene which generates grammar or language, per se.

Reports of the language gene blur this critical distinction. They assume that a mutant gene which affects language is a gene for language. However, the genetic impairment of grammar is only one partial effect in a

much broader pattern. This fact negates the claim that FOXP2 determines a specific psychological phenomenon. It leads to an entirely novel conception of how genes affect psychology. A gene generates a broad physiological substratum upon which psychological phenomena can be constructed. As we shall see, the construction process itself is not directed by genes. It is organized by cultural and mental processes. Genes are necessary to generate the general capacity for psychological phenomena; however, this is quite different from genes determining that the phenomena, i.e., the competencies, will occur and what their specific content will be. Genes only have codes for physiological matters such as "association neurons;" genes do not have codes for psychological phenomena such as grammar, love, problem solving, or syllogistic reasoning.

Mutant genes may produce deformed physical substrata which cannot support psychological functions. Vargha-Khadem, et al. (1995) believe that this is the way in which the mutant gene produced the KE family's psychopathologies. This is quite different from a gene laying down a faulty program or circuitry, for grammar for example.

That genetically defective individuals manifest grammatical incapacities does not indicate or prove that a gene causes, or even pre-disposes, grammar. It simply indicates that the gene was necessary for grammar. There is a key difference between being necessary and being a cause.

Genetic defects may impair your ability to program your computer, play the trombone, chess, or monopoly. A normal genotype, like a normal brain and endocrine system, is necessary for these abilities; but it does not cause them, nor is it directly responsible for them. Normal biology is a

necessary foundation for our cognitive skills; however, it does not cause them.

In the same way, I need the floor in my room to be intact in order to place the furniture in a certain location. If the floor collapses the position of the furniture can't be sustained. But the floor doesn't cause the furniture to be located where they are. As long as the floor is intact, I can place them wherever I want.

Thus, the fact that something is a necessary foundation for something does not mean it causes it. Obviously, language requires a normal genetic substratum. A defective genome undermines the ability to use language – just as it undermines the ability to play monopoly. But this does not mean that a gene causes or predisposes language, any more than it causes or predisposes me to play monopoly.

One cannot extrapolate from the causal power of a defective element to the causal power of a normal element. The fact that a defective element prevents some act does not imply that a normal element causes the act.

The conclusion that language is caused by a single gene rests upon logical errors plus a selective reporting of data. The conclusion is based on conflating genetic impairment to broad psychological capacities with genetic determination of specific psychological competencies.

These weaknesses, and others, plague reports about genetic causation of other psychological phenomena.

On Sept. 18, 2003, The New York Times (p. A21) science writer Nicholas Wade reported research on monkeys which claimed to have demonstrated a genetic basis of fairness. Monkeys were taught to exchange pebbles for pieces of food. If one monkey was given a grape in return for her pebble, but another received a less desirable piece of food,

the shortchanged monkey sometimes refused to hand over the pebble, sometimes refused to eat the food, and sometimes became agitated. From these reactions, the researchers and reporter concluded a) that monkeys have a sense of fairness and respond negatively when it is violated; b) monkeys' sense of fairness is akin to humans'; and c) fairness in both species, is genetically determined.

Let us examine the basis of these conclusions. The first point is pure speculation. No argument or evidence is adduced to substantiate that the reactions express a sense of fairness. The second and third conclusions surface in the statements: "The fact that we find the sense of fairness in nonhuman primates implies it is an evolved behavior." "Protesting unfair treatment of oneself, in other words, probably has a genetic basis in capuchins (monkeys) and so presumably in all social primates, including people." The first proposition is preposterous because it implies that any nonhuman primate behavior has evolved through genetic mutation. This is obviously false. Many of their behaviors are acquired, not evolved (including rudimentary language skills). In fact, the whole transaction of trading pebbles for food was taught to the monkeys in this project! It did not evolve through natural selection of genes. In the second quotation, Wade hyperbolizes that the monkeys' reactions are a "protest" against unfair treatment of themselves. This is pure editorializing. Worse, he uncritically parrots the authors' view that monkeys' "sense of fairness" has a genetic basis which extends to human fairness. Instead of observing that this conclusion is speculative and illogical, he concludes that the march of science has discovered yet another behavior under genetic control.

The conclusion is refuted by the obvious fact that fairness in humans is a cultural-historical construct. What people believe is fair varies with the

culture. Americans believe it is fair for Bill Gates to own hundreds of millions of dollars while millions of his countrymen live in poverty. Americans believe it is fair for wealthy corporate managers to give millions of dollars to legislators for passing favorable laws, while poor people are excluded from this process. Other cultures would reject these conditions as unfair. Even the notion of equal exchange is not necessarily the universal criteria of justice. All societies recognize the fairness of subsidizing the young, weak, and infirm despite their inability to produce equal value.

Fairness thus has variable cultural content which is humanly constructed. It is not a fixed, automatic, natural, universal idea that could be produced by genetic processes. Sociobiologists and evolutionary psychologists ignore and obfuscate the cultural construction and variation of fairness in an attempt to fit it into a genetic mold where it does not belong.

The same errors plague genetic explanations of anti-social behavior. On August 3, 2002, the San Francisco Chronicle reported that anti-social behavior is linked to a single gene. The first sentence reads "The likelihood that an abused child will become anti-social or violent as an adult hinges partly on a gene that influences brain chemistry."

The first problem is a conceptual confusion between antisocial and violent behavior. Most anti-social acts are not violent. Thus, what is true for antisocial behavior may have nothing to do with violence. Yet the article sometimes claims genetic influence on one and sometimes the other, or both. This makes the article suspect because it's not clear exactly what behavior the single gene is supposed to explain.

Anti-social behavior includes smoking dope, not talking to one's parents, becoming a gang member or a recluse, setting forest fires or

playing computer chess all day long, refusing to help others in need, (in some places) not attending church, and sometimes violence such as fighting, raping, and car-bombing government buildings. Now a response-tendency to violate social norms -- which would underlie and encompass all these diverse anti-social behaviors -- would be very general and abstract. It would predispose one to violate any norm by engaging in any of a wide range of acts. Such a response-tendency presupposes a sense of what a social norm is, what the various norms of one's own society are, and what kinds of behaviors would violate them. But this kind of abstract knowledge and abstract response-tendency -- whose content varies with the culture -- cannot be encoded in DNA. Genes only control simple, fixed, automatic behavior (cf., Bernard, 1924, 1926, pp. 123-141).

Even when an association between genes and behavior is found, this does not imply causation. Genetically based height is associated with playing basketball, yet the association is due to the fact that the rules of the game lead coaches to select (genetically based) tall players, and the rules of the game attract tall people to play because of financial and social benefits. The genes for tallness do not themselves produce basketball players. Any possible genetic marker associated with anti-social and/or violent children undoubtedly has a similarly indirect, socially-determined relation to the behavior.

The model of gene-brain biochemistry-behavior is additionally undermined because the purported chemical-behavior link does not exist. The gene for antisocial and/or violent behavior is said to work by reducing neurotransmitters such as serotonin and dopamine. Yet these chemicals are not specifically linked to any particular behaviors. They are involved in virtually all behavior (Ratner, 2000, pp. 24-33). They transmit electrical

impulses across billions of synapses throughout the brain. Thus, there is no way that their amount could selectively control antisocial and/or violent behaviors.

This same point refutes the claim that schizophrenia is caused by genes which disrupt the binding of neurotransmitters in synapses – reported in the New York Times, Dec. 13, 2002. Any genes which affect the amount of neurotransmitters, or the receptivity of neurons to neurotransmitters, would have general effects throughout the nervous system. The process would be analogous to interference in your phone line which makes all telephone messages difficult to understand. Irregularities in neurotransmitters would cause a general confusion, disorientation, and irritation because all information would be degraded or distorted.

Psychophysiologists, psychiatrists, and pharmaceutical corporations dearly believe that schizophrenia is this kind of mechanical problem in "the wiring" of the brain. The Diagnostic & Statistical Manual objectifies this impression by defining schizophrenia as abstract symptoms such as delusions, hallucinations, and flat affect. This abstract conception of schizophrenia is compatible with biochemical causation and treatment. Deterioration in broad psychological competencies such as attention, symbolic processing and understanding, emotion and motivation are possible effects of biochemical disturbances.

However, schizophrenia is not a mechanical failure in wiring which degrades all communication and causes general disorientation. Schizophrenia is a psychological phenomenon that consists of specific psychological symptoms. Laing, in his classic The Divided Self (1969), found that schizophrenics create a false, public, socially acceptable self that masks an inner self filled with imaginary ways of withdrawing from

odious social circumstances. Laing & Esterson's Sanity, Madness, and the Family (1964) documents the untenable social situations which schizophrenics face. Family members denigrate and confuse the schizophrenic-to-be and then deny this mistreatment. This prevents the subject from understanding it clearly. He is forced to invoke distorted interpretations and becomes delusional (cf. Ratner, 1970).

Delusions are not an endogenous general disorientation, or broad incapacity to process information. They are culturally induced ways of coping with particular kinds of stressful situations. Escaping from untenable situations by constructing a dualistic self and resorting to distorted/delusional interpretations cannot be determined by neurotransmitters. Thus, what schizophrenia really is cannot be explained by genetic, biochemical causation; and what the genetic-biochemical model explains is not schizophrenia.[1]

Heinrichs (1993) enumerates other conundra in the link between schizophrenia and biochemical processes. These include the failure to identify consistent biochemical differences between schizophrenics and normals, the failure to identify pathways by which biochemical agents could cause specific schizophrenic symptoms, the ambiguity of the schizophrenic syndrome which manifests greatly diverse symptoms, the inability of hypothesized neurological causes to actually cause these diverse symptoms, and the lack of a drug that consistently counteracts schizophrenia, per se.

These conundra plague biochemical explanations of depression as well. Depression has been explained in terms of genes which cause insufficient serotonin. Most recently, newspapers around July 11, 2003 trumpeted a study by Caspi, et al. (2003) which found that Ss with a short allele of

gene 5-HTT had a higher incidence of depression than Ss with a long allele. The increased incidence of depression only occurred when Ss had confronted 3 or more stressful events (between their 21st and 26th birthdays). Ss with the short allele who encountered 1 or 2 stressful events had a lower incidence of depression than Ss with the long allele. The authors conclude that there is an interaction between genes and stress. Genes do not generate depression alone; only when 3 or more stressful events occur.

For example, confronted by 4+ stressful events, 33% of Ss with a short allele became depressed while 17% of Ss with a long allele did so. However, confronted by 1 stressful event, 11% of Ss with a short allele became depressed while 16% of Ss with a long allele did so. (The authors, who claimed to have located a gene that predisposes toward depression, do not elaborate on the fact that the short allele is associated with decreased depression in the case of 1 and 2 stressors. The newspapers, which also trumpeted the "gene for depression" also studiously avoided mentioning that the short gene allele could be construed as a preventative for depression in the face of 1 or 2 stressful events. Since people experience 1 or 2 stressors more than 3+, the "prophylactic effect" of the gene is more newsworthy than its "disabling effect." To their credit, most newspapers did mention the fact that only a minority of individuals with the short allele became depressed after 3+ stressful events.)

The authors claim that the short allele produces depression under high stress by reducing serotonin levels. Thus, the gene and serotonin directly produce depression, although only under unusual conditions and only among a minority of people. Serotonin is therefore not a sufficient cause of depression. The authors, and science reporters, also acknowledge that low

serotonin is only one cause of depression, and only in a minority of people. Low serotonin is not a necessary cause of depression any more than it is a sufficient cause.

These caveats on the influence of genes on depression are a welcome correction to the usual sweeping claims that genes cause depression. However, even the limited claim for a negative relation between depression and serotonin, caused by a short allele of 5-HTT, under a large number of stressors, and among a minority of people is dubious for the following reasons.

a) The formula relating depression to stress, genes, and serotonin does not translate well into everyday life. Translating Caspi, et al.'s conclusions into real life terms leads to the following scenario: you witness your mother's murder; then you lose your job, yet you do not become depressed because your serotonin level is normal. Your car is then stolen, and because this is your third stressor, your 5-HTT gene (short allele) triggers a lowering of serotonin and you become depressed. If you had had a long allele you would not have become depressed. Placed in real-life terms, Caspi's diathesis-stress model is dubious (Ratner, 1991, pp. 283-291). Are we really to believe that confronted by 3+ extremely traumatic stressors, 84% of the population would escape depression if they had a lot of serotonin in their brains? The rate of depression has been on the increase for the last 60 years and the age of onset is getting lower, with 15-24 year olds being most likely to have experienced a depressive episode. Are we to believe that these demographic changes have occurred primarily among individuals with short alleles of 5-HTT and low serotonin?

b) Why would a short allele reduce serotonin during the third stressor but not during the first or second? Why would a longer allele maintain an even serotonin level during the third stressor?

c) Sociological research contradicts the authors' findings. It demonstrates a monotonic relationship between social stress and depression. In one study, 100% of Ss who experienced 4+ stressful events became depressed (Ratner, 1991, p. 287). Anthropological research demonstrates that depression is culturally specific, not universal (Ratner, 1991, pp. 264-278; cf. Ratner, 2000).

d) The relationship between 5-HTT, serotonin, and depression is purely speculative. Serotonin levels of the Ss were not measured so we do not know whether short and long allele Ss actually differed in serotonin levels. We thus have no idea what the mediating link between 5-HTT and depression is. Nevertheless, we shall take the authors' claim seriously. Below, we shall examine whether serotonin causes depression.

e) The authors, and news reporters, overlook the psychology of depression. Indeed, they treat depression as a biochemical by-product devoid of psychological nature.

The authors construe depression as the product of a mechanical failure in the brain. Consequently, it appears not based upon particular interpretations of particular situations; nor is it a particular way of coping with them. Depression is construed as the natural result of a biochemical deficiency, much like diabetes is the result of an insulin deficiency. Of course, sugar may trigger diabetes just like high stress may trigger depression; however the primary cause of both diseases -- i.e., what produces the characteristics of the symptoms -- is internal biochemical deficiencies. According to the diathesis-stress model, X number of

stressors + a short allele of gene 5-HTT triggers low serotonin levels that produce depression in Y % of individuals.

This model regards depression as a disorder of physical organs, with natural, universal features, in the mold of physical diseases. Depression seems to have nothing whatsoever to do with psychology! Even stressful events are defined numerically (1, 2, 3, 4) without reference to the kind and severity of the psychological stress which is experienced. The content of stressors is dissolved and replaced by abstract numbers of events devoid of content.

The person is entirely left out of the account. Variables interact on their own without people doing anything. This is the crudest type of correlation research which avoids conceptual issues that are necessary for scientific understanding.

The deeper we consider the psychological quality of depression the more implausible it is that genetically-controlled serotonin could cause it. Depression, like all emotions, is a complex psychological state. One only becomes depressed after interpreting a situation in a particular way. For example, one becomes depressed upon losing a job because of all the negative consequences that it implies for one's standard of living, self-esteem, and social interactions. If one doesn't care about these issues one will not become depressed. Depression involves caring, expecting, reasoning, self-esteem, and social values. Depression is <u>about</u> something. It involves intentionality directed at comprehending and coping with things. It is a particular way of reacting to particular things. It draws upon previous experience. It has content.

Research demonstrates that depression includes pessimism about life improving. It also often includes guilt that one is responsible for one's

unfortunate situation (Ratner, 1991, pp. 265-268, 282-292). Now, guilt is a cultural value. It only occurs in particular cultures. This is one reason for the anthropological finding that depression is culturally specific.

If one does not live in a culture that fosters them, one will not feel guilty and depressed, no matter how much serotonin is concentrated in the brain. And if one does live in a culture which fosters guilt and depression, one will only become depressed if one invokes cultural values as interpretative filters of situations. Serotonin does not dictate whether a person does so.

f) There is no evident, or even plausible, way that human neurophysiology could directly produce the concrete experience of depression. On the contrary, human neurophysiology is unique in being substantially organized by experience and transmitting that experience to psychological phenomena.

The limbic system which processes emotions is penetrated and controlled by cortical structures. These cortical structures transmit symbolic information from culture to the limbic system. The limbic system thus imparts cultural, cognitive information to emotions. The limbic system is not a primitive, autonomous, automatic system for generating depression on its own in some "purely biological" fashion (Armstrong, 1999; Ratner, 1989a, 2000, 2004).

The conclusion that a gene + serotonin cause depression is further challenged when we consider just what serotonin does. It determines how much electricity is transmitted across synapses. It does not determine any content to the electrical impulse, nor does it selectively transmit certain impulses with particular information content. It simply regulates the amount of electricity across synapses. Serotonin connects with 14

different kinds of receptors which project onto many areas of the brain. Some serotonin receptors inhibit responses while others facilitate/excite them. Serotonin's effects also vary with concentrations of other neurotransmitters including the neuropeptides, dopamine, noradrenaline, and even insulin. Serotonin mediates virtually all behavior.

This complex picture does not match the biochemical model of depression. Given serotonin's vast effects on the nervous system, it is unlikely that it could reliably produce one particular psychological effect, such as depression, in some algorithmic relation to its concentration. How could a single monoamine, whose sole function is to conduct electrical impulses across billions of synapses throughout the nervous system, make you experience a particular emotion such as feeling despondent about life events? It is as implausible as it would be for serotonin to cause you to become interested in studying French or selling your car. There is no way that greater or less transmission of electricity throughout the brain could reliably produce a particular feeling.

Indeed, psychophysiologists never attempt to explain what the process might be by which serotonin causes depression. They buttress their model with extensive descriptions of neuroanatomy, showing pathways by which serotonin reaches the limbic system which is known to "be involved with" emotions. But we are never told just how the limbic system produces depression. It is as though the limbic system automatically produces depression when its supply of serotonin is insufficient. Psychophysiologists content themselves with identifying a neurophysiological correlate of depression and then speciously claim to have discovered a basis of depression. They ignore the fact that any neurophysiological correlate of depression also correlates with many

physical and psychological reactions having nothing to do with depression. Low serotonin is found in anti-social personality disorder, alcoholism, panic, and more (Thase & Howland, 1995, p. 228).

The biochemical model imagines that serotonin has a direct, algorithmic relation to depression, like insulin has to diabetes. Serotonin seemingly turns depression on and off, much like a dimmer switch regulates the brightness of a light. However, depression is not a fixture with fixed characteristics in a fixed location waiting to be switched on or off. There is no depression center waiting to be turned on or off. Depression draws upon memory, perception, thoughts, and feelings with neural correlates all over the brain. It is constructed by people out of complex experiences and images. It is a psychological phenomenon formed by interpretations, intentionality, and cultural values. It is these factors which determine whether depression will occur and what it's characteristics/qualities will be. It is these factors that the diathesis-stress model ignores and obscures.

Research on drugs and depression confirms doubts about the biochemical model. Despite all the hype from physiological psychologists, psychiatrists, and pharmaceutical companies, drugs are not very effective in treating depression. Most studies - including those recently reviewed by British and American health regulators - have found that S.S.R.I.'s are no more effective in fighting teenage depression than sugar pills. Even in adults, S.S.R.I.'s have been found to offer only modest benefits. In about half of all adult tests, the drugs prove no more effective than placebos. In a thorough review of the efficacy data submitted to the U.S. Food and Drug Administration for approval of the 6 most widely prescribed antidepressants approved between 1987 and 1999, Kirsch, et al. (2002) conclude that "our data suggest that the effect of antidepressant drugs

are very small and of questionable clinical significance." The mean difference between drug and placebo was approximately 2 points on the 62-point Hamilton Depression Scale.

The hypothesis that low serotonin causes depression is further weakened by additional pharmaceutical results:

a) Drugs are more effective when they control a number of biochemical agents than when they focus upon one, such as serotonin. Drugs which regulate noradrenaline as well as serotonin (serotonin and noradrenaline re-uptake inhibitors, or SNRIs) are more effective than SSRIs.
b) Anti-depressive drugs can work without increasing serotonin or its metabolites.
c) There is a large cross-over effect, where
    (1) drugs which affect other biochemical processes besides serotonin, and alleviate non-depressive symptoms, help depression as well.
    (2) SSRIs designed to target depression, alleviate other unrelated disorders such as anti-social behavior, alcoholism, and bulimia.
d) Any reported psychological changes in depression occur two to four weeks after anti-depressive drugs are administered and neurotransmitter levels have normalized (Thase & Howland, 1995, pp. 228-229).

Although serotonin cannot cause depression, it is possible that a correlation exists. In the same way, a short allele of gene 5-HTT does not cause depression but it may be associated with a differential incidence in

depression of 17% (33% of individuals with the short allele vs. 16% of individuals with the long allele became depressed after 4+ stressful events). Such correlations could exist for the following reasons.
1) A low level of serotonin, intermingled with other neurotransmitters, has broad affects on many bodily functions (energy level, sleep, eating) and general psychological competencies (concentration and memory). Certain of these general disturbances may aggravate the individual and make it difficult to cope with stress. When this person encounters stress, she may develop any number of psychological disorders. These depend upon the way she has been treated and prevalent cultural values she adopts. She may feel bad about herself, gloomy about her prospects, and depressed. She may also, or alternatively, become anxious, compulsive, delusional, act out, dissociate, or form multiple selves. Which of these symptoms develop is not a function of a particular biochemical mechanism.

Since individuals interpret the effects of the low serotonin complex in different ways, only a small percentage of people with the low serotonin complex become depressed (only 33% of Caspi et al.'s short-allele Ss became depressed after confronting 4+ stressors). Another reason for the low percentage is that low serotonin will have different effects on the body and on general psychological competencies at different times as surrounding conditions change (e.g., as concentrations of other neurotransmitters change). At certain times, low serotonin will not produce bothersome psychophysiological effects, and the person will be able to handle stress adequately and not become depressed. Individuals also can feel depressed about stressful events when they have normal serotonin levels. This situation further weakens the correlation between the amine and the emotion.

This cognitive appraisal model was pioneered by Schachter. It has subsequently been elaborated by Nisbett, Lazarus, Mandler, and others (Ratner, 1989a). It explains how alcohol, narcotics, psychedelic drugs, energy level, hormones, and electrical stimulation of the brain affect emotions. They do not directly produce concrete emotions. Rather, they generate general moods which become defined by the individual through cultural scripts.

How the complex effects of medication alleviate symptoms of psychological depression in some individuals has eluded psychophysiologists. It is likely that drugs counteract a complex of biochemical effects which disturb bodily functioning and undermine psychological competencies such as concentration and memory. Alleviating these effects makes people feel better in general. It enables people to cope with stressors without interference from physiological distractions and degraded general psychological competencies. The person may then (depending upon social and psychological processes) view himself and the world more positively, and overcome depression along with many other negative reactions. Drugs do not attack depression, per se, the way fungicide kills fungus. Their effects are general, and mediated by psychological processes of interpretation (Ratner, 1991, chaps. 5, 6).

Medications which affect neurotransmitters are somewhat effective in treating depression for the same reason that electro-convulsive shock was. ECT disrupted general functioning of the brain and psychology. Sometimes depressive thoughts were affected. ECT did not directly or specifically attack depression, although a side effect was to relieve depression, occasionally.[2] (This "shot gun" approach is being resurrected in a new form, "transcranial magnetic stimulation." TMS utilizes magnets to alter

electrical signals in the brain. Sometimes this general alteration will disrupt depressive thoughts and feelings. TMS will soon be proclaimed the next "anti-depressive treatment.")

Of course, one may negatively interpret his personality and external situations for many reasons having nothing to do with low serotonin. In this case serotonin will not alleviate depression.[3]

2) Biochemical processes (including genes and neurotransmitters), affect the largely instinctual, non-psychological reactions of infants. A short allele of gene 5-HTT may incline some infants to react strongly to fearful stimuli (Caspi, p. 387). Some parents of these infants may come to label and treat them as weak-willed or hypersensitive. Some of these children may adopt these labels and treatments as their self-definition. Later, when they encounter stress, they regard themselves as incapable of coping with it, they feel overwhelmed, become pessimistic, reproach themselves and feel guilty, and ultimately become depressed. They are more prone to feel this way after repeated encounters (3+) with stressors, which they interpret as demonstrating their incompetence.

In addition, the stress one experiences as a result of low self-confidence in difficult situations, leads the body to cope by reducing serotonin levels.[4] Thus, depression and low serotonin are both a function of how the individual was treated and what he has internalized from this treatment.

3) A correlation exists serotonin and depression because the effects of low serotonin on the body and on general psychological competencies such as memory, attention, and motivation are construed as the symptoms of depression. In other words, depression is whatever the effects of a low serotonin complex are. This definition guarantees that depression will

correlate with a low serotonin complex (low serotonin + particular concentrations of other neurotransmitters and biochemical agents).

Defining depression in terms of biomedical theory is what the DSM does. It defines depression as at least five of the following nine symptoms within a two-week period: a) significant weight loss or weight gain, or decrease or increase in appetite nearly every day; b) psychomotor agitation or retardation (lethargy); c) fatigue or loss of energy; d) diminished ability to think or concentrate ; e) insomnia or hypersomnia (sleeping more than 10 hours per day); f) markedly diminished interest or pleasure in activities; g) depressed mood; h) recurrent thoughts of death, suicidal Ideation, or suicide attempt; i) feelings of worthlessness, excessive or inappropriate guilt. The first five of these symptoms are direct psychophysiological effects of biochemical processes. And these five symptoms are enough to warrant a certification of depression. One need not have significant psychological symptoms -- such as feeling depressed -- in order to be categorized depressed!

Defining depression as the DSM does makes it appear to have biochemical origins. However, a fuller description of the psychology of depression renders this link implausible. When we emphasize that depression involves guilt feelings, low self-esteem, a pessimistic view of life, expectations which are not fulfilled, dissatisfaction with particular situations and people (including unhappiness about seasonal changes as in "seasonal affective disorder"), and can include hallucinations, anxiety, catatonia, mania, and even positive moods (as in the case of "depression with atypical features"), it becomes dubious that monoamines could produce this complex psychology of concrete thoughts, feelings, and motives.

DSM's constricted definition of depression also fosters the impression that medication effectively alleviates the symptoms of depression. If depression is defined as the effects of biochemical processes, then chemicals which alter these processes will naturally alter their "depressive" effects. Drugs which relieve disturbances in energy, sleep, and concentration will be declared to have treated depression. Of course, concrete psychological aspects of depression are omitted from this account. We have seen above how ineffective drugs are in relieving the real psychological issues of depression. And when they do help, they act as a general palliative, not a specific treatment.

Conclusion

Reports in the media and journals that psychological phenomena are genetically caused are dubious. They rest upon unscientific data; selective use of data (ignoring contradictory data); specious arguments; unwarranted leaps of faith which are unwarranted given the biochemistry of genes and neurotransmitters, and the neuroanatomy of the cortex; and misconstruing psychological phenomena -- as singular, simple, fixed, automatic, natural, universal acts; localized in a single, circumscribed brain area; abstract capacities instead of concrete competencies; abstract functions such as "processing information," "thought disorder," "disorientation," or "emotional imbalance;" physical reactions with unknown psychological significance.

News reporters and science editors uncritically parrot conclusions that psychology is genetically caused. They fail to point out obvious logical and empirical errors in the research which they report. They rarely present

critiques by dissenting social scientists. News reporters headline any suggestion of genetic determination of psychology regardless of how preposterous and undocumented it is. Cultural explanations of psychology rarely find space in the media. News reporters are doing a poor job of educating the public about the basis and nature of psychological phenomena.

The genetic model, endorsed by psychobiologists, sociobiologists, evolutionary psychologists, and psychiatrists, fails to understand that human culture has produced a transformation in the way biology influences behavior. Advanced human culture replaces the strict biological control of behavior found in lower animals (Ehrlich, 2000). Culture is not incongruously "added onto" the same biological substrate which exists in animals. Biological processes lose their determining power over human behavior. This is why behavior can change rapidly, as is now happening under the globalizing impact of market economics.

Genes produce a cellular substratum which is conducive to psychological development. The cellular substratum that genes generate is the capacity to develop psychological functions such as language, emotions, memory, logical reasoning, personality, perception, learning, motivation, and imagination. However, genes and their cellular products do not determine that psychological functions will develop, or what their characteristics or content will be. Geertz (1973, p. 50) expressed this eloquently when he said "We live in an `information gap.' Between what our body tells us and what we have to know in order to function, there is a vacuum we must fill ourselves, and we fill it with information (or misinformation) provided by our culture." Genes lay out the potential capacity for psychological development, however psychological

competence to reason, recall, learn, imagine, and feel in particular ways is only actualized if the biological capacity is socially stimulated and canalized (cf., Donald, 1991; Ratner, 1989a, b; Ratner, 1991, chaps. 1, 4, 5, 6; Ratner, 1998, 2000, 2004, forthcoming; Tomasello, 1995).

Genes may directly determine simple physical characteristics such as eye color. However, they do not directly determine psychological phenomena. In the latter case, genes produce a potentiating substratum rather than particular phenomena. The substratum is like a Petri dish which forms a conducive environment in which bacteria can grow, however, it does not produce, bacteria.

Genes relate to speech in an analogous manner that the tongue relates to speech. The tongue is necessary for speech, however, the tongue is not a specifically speech organ, nor does it directly cause speech to occur. It is only utilized for speech if the individual learns language and desires to speak. Genes involved in speech function analogously. They are not specifically or exclusively for speech, nor do they cause speaking to occur the way the gene for eye color produces blue eyes.

If genes did determine competence, psychological phenomena would have a fixed content that was automatically and universally invoked under given conditions. There would be no social content, no variation, and no volition in psychology. Psychological phenomena would be like instinctual responses.

Genetic determination of human psychological phenomena would also require a single, circumscribed, functionally-fixed cortical area exclusively associated with a single, well-defined, circumscribed psychological function. Yet these conditions do not obtain in the case of complex psychological phenomena. “Even the simplest cognitive process is of enormous

informational complexity, involving perception, memory, decision making, emotion, set, and virtually any other mental activity." “The more complex the psychological process, the less likely it is that a narrowly circumscribed [cortical] region uniquely associated with that process will be found.” (Cf. Uttal, 2001, pp. 214, 13). Diffuse, inseparable, volitional psychological functions distributed over wide areas of the cortex which are multifunctional and plastic, are unlikely to have specific genetic determinants.

Genetic determination of psychology is also impugned by the great plasticity of the cortex. The human cortex is organized by experience, it is not innately hard-wired. Experience stimulates the growth of new neural components, and it organizes synapses that are endogenous to the cortex. Experience affects the size of neurons, the extent of dendritic branching, myelinization, the number of synapses, and which naturally formed synapses will be retained or degenerate. Importantly, synaptogenesis in the cortex during the first postnatal years produces a large pool of connections which are initially unspecified for function. Experience organizes these into specific functions (Huttenlocher, 1994). If the neural substratum of psychological functions is organized by experience, it is hardly likely that the functions themselves are genetically determined.

Cortical regions are remarkably malleable to take on a variety of psychological functions. An example of this "equipotentiality" is that when the “speech areas” in the dominant hemisphere are destroyed (before the critical age of 8), other areas in the non-dominant hemisphere take over this function and recovery of language is nearly complete (Huttenlocher, 1994). These latter areas were not genetically programmed for language yet they assumed this function.

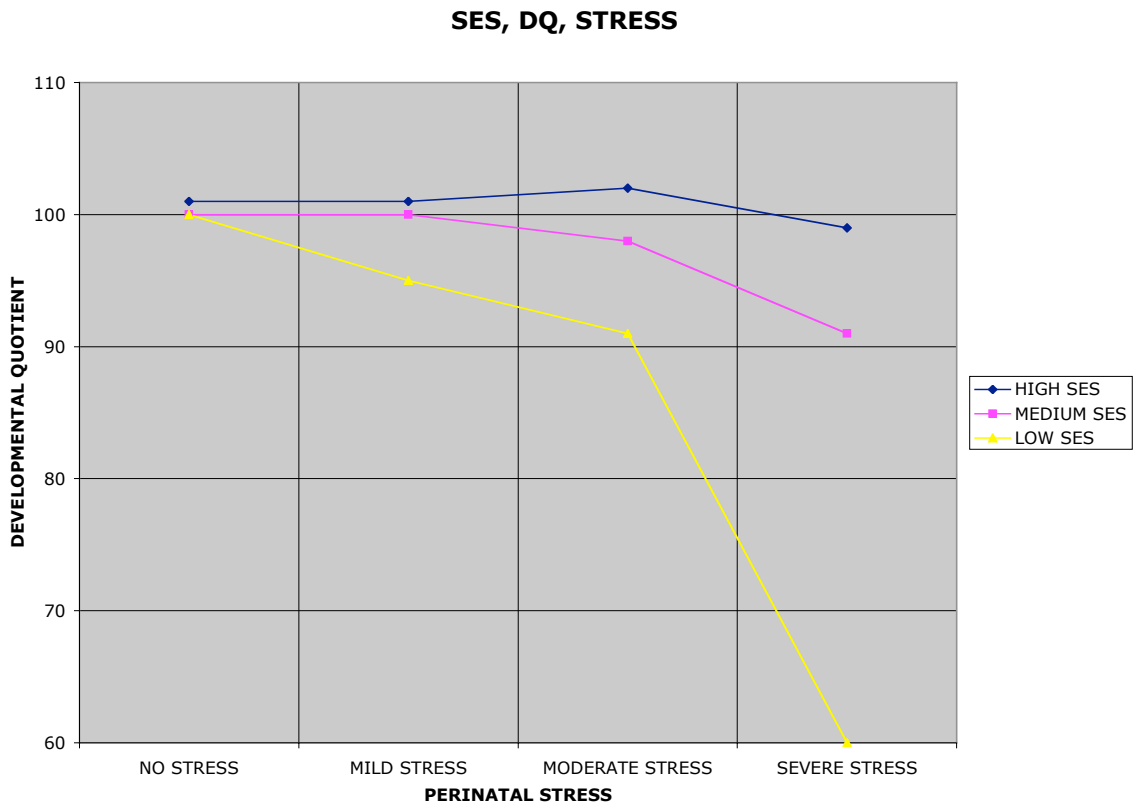
What counts as language, and what gets processed in the "language area" (left temporal lobe) is also extremely variable. The left temporal lobe of deaf people processes perception of movement, picture identification, and the recognition of faces, which are localized in the right hemisphere of hearing people. The reason is that visuospatial perception is part of sign language and is therefore processed as language in the left hemisphere "language centers" of sign users. Clearly, what cortical centers do depends upon the social form of language – what language requires of the cortex – rather than upon genetic programs (Sacks, 1989, pp. 101-106; Sacks, 1990; Neville, 1991).[5]

In other words, the physiological substratum of psychological phenomena is organized from the top down rather than from the bottom up. Psychological phenomena definitely exit in neuronal structures; they do not float freely as spirits in the head. However, the substratum of cortical neurons is organized by cultural experience, not by endogenous chemicals in their own right. Repudiating genetic determinism of psychology thus does not lead to a Cartesian dualism where mental phenomena are separated from the body (Ratner, 1991, chap. 5).

Culture does not simply accentuate or reduce biological influences on psychology -- as interactionist models propose. Rather, cultural factors and processes supercede biological determinism of psychological phenomena. Cultural factors organize the form and content of psychological phenomena (cf. Ratner & Hui, 2003) – as well as organizing the structure and function of biological phenomena such as the cortex.

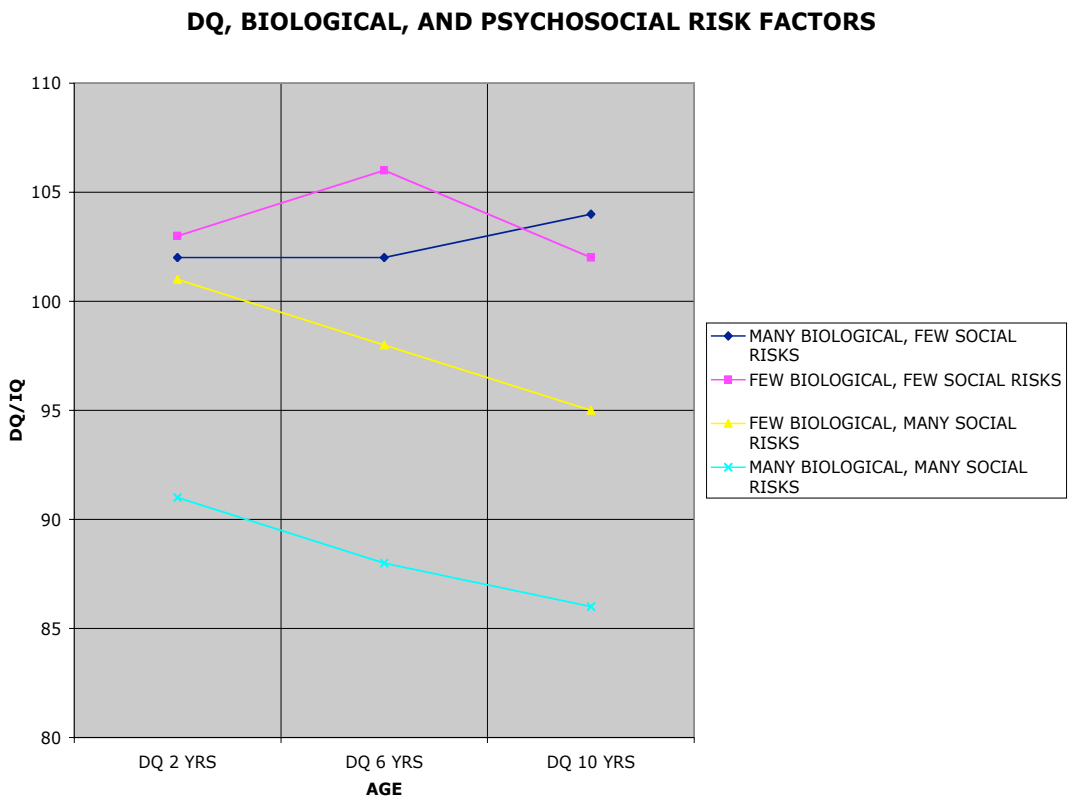
The way that culture supercedes biology to generate behavior is demonstrated in the relation of biological risk factors and psycho-physiological development. In general, the impact of biological risk factors

is a function of social experience. Positive experiences can override biological defects. The latter only impede psycho-physiological development when they are reinforced by deleterious experience. Werner (1989) found that the effect of perinatal stress on developmental quotient was modulated by socioeconomic status, as the following figure depicts.



Gollnitz, et al. (1990) similarly found that a composite developmental quotient and intelligence quotient (DQ/IQ) was more a function of social experience than of biological risks. The composite DQ/IQ combined scores on the WISC IQ test, Raven's matrices, measures of motoricity, play, speech, and social behavior. The following figure from Gollnitz, et al. depicts the DQ/IQ score at different ages as a function of biological and

social risk factors. It demonstrates that children who have many biological risk factors but few social risk factors have high DQ/IQ scores, just as high as children with few biological and social risk factors. On the other hand, children with many social risk factors and few biological risk factors manifest developmental impairment by 10 years of age (cf. Ratner, 2002, pp. 73-74).



This research should make us doubt claims that psychological phenomena are controlled by biological factors such as genes,

neurotransmitters, neuroanatomy, hormones, sense organs, body physique, and infantile reflexes.[6]

Notes

1. Even if we accepted a definition of schizophrenia as general disorientation, the Times' report of genetic transmission is suspect. It provides no quantitative data about the correlation between genetic defects and "schizophrenic" symptoms. More egregious is the fact that the entire article is based upon research by a private corporation, Decode Genetics, rather than by independent researchers. The company is in partnership with the drug company Roche to develop drugs to counteract the aberrant gene's effects. It is by now well known that medical research by corporations is inadmissible because is it corrupted by the drive for profit. Drug companies routinely publish studies which favor their own (expensive) drugs but which are contradicted by independent researchers. The New York Times has exposed this scandalous situation often. It is inappropriate for the Times to then publish the uncorroborated claims of Decode Genetics and Roche about genetic causes of schizophrenia when these companies have a financial incentive for promoting a bio-medical cause of schizophrenia that can be treated by the companies' expensive medication.

2. An additional example which illustrates this point is the manner in which Viagra enhances men's sexual functioning. Viagra works by relaxing smooth muscles in blood vessels throughout the body. This enhances blood

flow throughout the body (which is why Viagra was originally used to lower hypertension). Relaxed blood vessels and inflow of blood are necessary for erections. Viagra can help this physical aspect of sex by promoting vasodilation in general. But Viagra is not specifically a "sex drug." It does not selectively target sexual behavior; nor does it affect sexual desire. If a man feels no desire, Viagra will not enhance his sexual behavior. Viagra only affects sexual behavior to the extent that the latter depends upon a general physiological process -- vasodilation.

Serotonin affects depression in analogous fashion. It has broad affects on the body and psychological competencies. It does not specifically target the physiology or psychology of depression. The individual must generate appropriate thoughts, feelings, and motives from her normalized physiology and psychological competencies in order to escape depression.

3. One may believe that as long as SSRIs relieve depression, it doesn't matter how they work or whether serotonin causes depression directly or indirectly as I have contended. However, science demands an understanding of the real nature of things and the processes by which they come to be. It makes all the difference if serotonin causes depression in the same way that a fungus causes your foot to itch, or whether serotonin generates a set of physical states which have no direct connection with depression and which are only associated with depression occasionally and because of how people incorporate awareness of them in interpreting external events. This is invaluable for understanding the nature of human psychology and its relation to biology. It has vital practical importance for knowing how to effectively enhance psychological functioning.

4. The chemical imbalances that occur during depression usually disappear when one completes psychotherapy for depression, without taking any medications to correct the imbalance. This suggests that the imbalance is the body's physical response to psychological depression. Further evidence that serotonin level results from emotions is that it drops when people fall in love and when they develop obsessive-compulsive disorders (Marazziti, 1999).

5. Cortical localization of psychological functions also differs in different cultural groups. Tsunoda (1973, 1979) reports the fascinating fact that in Japanese people, human sounds such as humming, laughter, cries, sighs, and snores, along with animal sounds and traditional Japanese instrumental music, are processed in the verbal-dominant hemisphere. However, Westerners process all of these in the non-verbal hemisphere. In the Westerner, the dominant hemisphere deals with logic, calculation, and language, while the non-dominant hemisphere deals with pathos and natural sounds, and Japanese music. On the other hand, in the Japanese, the dominant hemisphere deals with logic, pathos, nature, and Japanese music. Importantly, Americans brought up in Japan evidence the Japanese pattern of cortical allocation. Conversely, Japanese individuals brought up speaking a Western language as their mother tongue develop the Western pattern of brain localization. These facts indicate a social rather than biological cause of the cortical localization of psychological functions.

6. Cf. Ross & Pam (1995), and Ratner (1991, pp. 282-303) who debunk research in biological psychiatry which purports biological bases and

treatments for mental illnesses; Davidson, Jackson, & Kalin (2000), and Levenson (2003), who demonstrate that emotions manifest a highly variable relation to biological processes and are not tightly controlled by the latter.

Bronfenbrenner (1975) demonstrates that conclusions about the genetic control of intelligence from studies of identical twins reared apart are faulty. The correlation of IQ scores among these twins actually reflects experience more than genotype. Only those separated MZ twins whose environments and experiences are similar have highly correlated IQs. Separated twins whose environments and experiences are dissimilar have low IQ correlations. Separated MZ twins raised in communities of similar size and economic base (e.g., mining or agriculture) had IQ correlations of .86, while those residing in dissimilar localities had IQ correlations of .26 (ibid., p. 121). Separated twins who had the same number of years of schooling had a small average IQ difference of only 1.45, whereas twins who had an educational difference of 14 years had IQ differences of 24 points (ibid., p. 118).

Joseph (1998, 2001a, 2001b) makes this same point in debunking twin studies on schizophrenia, personality, and crime which conclude that these are genetically transmitted.

Lancaster (2003, chap. 18) refutes research which claims that homosexuality is genetically caused.

Gottlieb (2003) summarizes research on animal growth and behavior which confirms Werner's and Gollnitz's findings that environmental influences dramatically modify genotypical inclinations.

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