The Internal Brand of the Scarlet W

Stephen Jay Gould

As a setting for an initial welcome to a new home, the international arrivals hall at Kennedy Airport pales before the spaciousness, the open air, and the symbol of fellowship in New York's harbor. But the plaque that greets airborne immigrants of our time shares one feature with the great lady who graced the arrival of so many seaborne ancestors, including all my grandparents in their childhood. The plaque on Kennedy's wall and the pedestal of the Statue of Liberty bear the same inscription: Emma Lazarus's poem The

New Colossus—but with one crucial difference. The airport version reads:

> *Give me your tired, your poor, Your huddled masses yearning to breathe free . . . Send these, the homeless, tempest-tost to me: I lift my lamp beside the golden door.*

One might be excused for supposing that the elision represents a large and necessary omission to fit the essence of a longer poem onto a smallish plaque. But only one line, easily accommodated, has been cut —and for a reason that can only reflect thoughtless (as opposed to merely ugly) censorship, therefore inviting a double indictment on independent charges of stupidity and cowardice. (As a member of the last public school generation trained by forced memorization of a holy historical canon, including the Gettysburg Address, the Preamble to the Constitution, Mr. Emerson on the rude bridge that arched the flood, and Ms. Lazarus on the big lady with the lamp, I caught the deletion right away and got sufficiently annoyed to write a *New York Times* Op-Ed piece a couple of years ago. Obviously, I am still seething, but at least I now have the perverse pleasure of using the story for my own benefit to introduce this essay.) I therefore restore the missing line (along with Emma Lazarus's rhyming scheme and syntax):

The wretched refuse of your teeming shore.

Evidently, the transient wind of political correctness precludes such a phrase as "wretched refuse," lest any visitor read the line too literally or personally. Did the authorities at our Port Authority ever hear about metaphor and its prominence in poetry? Did they ever consider that Lazarus might be describing the disdain of a foreign elite toward immigrants whom we would welcome, nurture, and value?

This story embodies a double irony that prompted my retelling. We hide Emma Lazarus's line today because we misread its true intention, and because contemporary culture has so confused (and often even equated) inappropriate words with ugly deeds. But the authorities of an earlier generation invoked the false and literal meaning—the identification of most immigrants as wretched refuse to accomplish a deletion of persons rather than words. That is, the supposed genetic inferiority of most refugees (an innate wretchedness that American opportunity could never overcome) became an effective rallying cry for a movement that did succeed in imposing strong restrictions on immigration beginning in the 1920s. These laws, strictly enforced despite pleas for timely exceptions, immured thousands of Europeans who sought asylum because Hitler's racial laws had marked them for death, while our quotas on immigration precluded any addition of their kind. These two stories of past exclusion and truncated present welcome surely illustrate the familiar historical dictum that significant events tend to repeat themselves—the first time as tragedy, the second as farce.

In 1925, Charles B. Davenport, one of America's foremost geneticists, wrote to his friend Madison Grant, the author of a best-selling book, *The Passing of the Great Race*, on the dilution of America's old (read northern European, not Native American) blood by recent immigration: "Our ancestors drove Baptists from Massachusetts Bay into Rhode Island, but we have no place to drive the Jews to." Davenport faced a dilemma. He sought a genetic argument for innate Jewish undesirability, but conventional stereotypes precluded the usual claim for inherent stupidity. So Davenport opted for weakness in moral character rather than intellect. In his 1911 book, *Heredity in Relation to Eugenics*—not, by the way, a political tract but his generation's leading textbook in the developing science of genetics—

he wrote:

> In earning capacity both male and female Hebrew immigrants rank high and the literacy is above the mean of all immigrants. . . . On the other hand, they show the greatest proportion of offenses against chastity and in connection with prostitution, the lowest of crimes . . . .The hordes of Jews that are now coming to us from Russia and the extreme southeast of Europe, with their intense individualism and ideals of gain at the cost of any interest, represent the opposite extreme from the early English and the more recent Scandinavian immigration, with their ideals of community life in the open country, advancement by the sweat of the brow, and the uprearing of families in the fear of God and love of country.

The rediscovery and publication of Mendel's laws in 1900 had initiated the modern study of genetics. Earlier theories of heredity had envisaged a "blending" or smooth mixture and dilution of traits by interbreeding, whereas Mendelism featured a "particulate" theory of inheritance, with traits coded by discrete and unchanging genes that need not be expressed in all offspring independent and undiluted, but that remain in the hereditary constitution, awaiting expression in some future generation.

In an understandable initial enthusiasm for this great discovery, early geneticists committed their most common error in trying to identify single genes as causes for nearly every feature of the human organism, from discrete bits of anatomy to complex facets of personality. The search for single genetic determinants seemed reasonable for simple, discrete, and discontinuous characters and contrasts (like blue versus brown eyes). But the notion that complex behaviors might also emerge from a similar root in simple heredity of single genes never made much sense, for two major reasons: (1) a continuity in expression that precludes any easy definition of traits

supposedly under analysis (I may know blue eyes when I see them, but where does a sanguine personality end and melancholia take over?); and (2) a virtual certainty that environments can substantially mold such characters, whatever their underlying genetic influence (my eyes may become blue whatever I eat, but my inherently good brain may end up residing in a stupid adult if poor nutrition starved my early growth and poverty denied me any education).

Nonetheless, most early human geneticists searched for "unit characters"—supposed traits that could be interpreted as the product of a single Mendelian factor—with abandon, even in complex, continuous, and environmentally labile features of personality or accomplishment in life. (These early analyses proceeded primarily by the tracing of pedigrees. I can envisage accurate data, and reliable results, for a family chart of eye color, but how could anyone trace the alleged gene for "optimism," "feeble inhibition," or "wanderlust"—not to mention such largely situational phenomena as "pauperism" or "communality." Was great-uncle George a jovial back-slapper or a reclusive cuss?)

Whatever the dubious validity of such overextended attempts to reduce complex human behaviors to effects of single genes, this strategy certainly served the aims and purposes of the early twentieth century's most influential social crusade with an allegedly scientific foundation: the eugenics movement, with its stated aim of "improving" America's hereditary stock by preventing procreation among the supposedly unfit (called "negative eugenics") and encouraging more breeding among those deemed superior in bloodline ("positive eugenics"). The abuses of this movement have been extensively documented in many excellent books covering such subjects as the hereditarian theory of mental testing and the passage of legislation for involuntary sterilization and restriction of immigration from nations deemed inferior in hereditary stock.

Many early geneticists played an active role in the eugenics movement, but none more zealously than the aforementioned Charles Benedict Davenport (1866-1944), who received a Ph.D. in zoology at Harvard in 1892, taught at the University of Chicago, and then became head of the Carnegie Institution's Station for Experimental Evolution at Cold Spring Harbor, New York, where he also established and directed the Eugenics Record Office, beginning in 1910. This office, with its mixed aims of supposedly scientific documentation and overt political advocacy, existed primarily to establish and compile detailed pedigrees in attempts to identify the hereditary basis of human traits. The hyper-enthusiastic Davenport secured funding from several of America's leading (and, in their own judgment, therefore eugenically blessed) families, particularly from Mrs. E.H. Harriman, the guardian angel and chief moneybags for the entire movement.

In his 1911 textbook, dedicated to Harriman "in recognition of the generous assistance she has given to research in eugenics," Davenport stressed the dependence of effective eugenics upon the new Mendelian "knowledge" that complex behavioral traits may be caused by single genes. Writing of the 5,000 immigrants who passed through Ellis Island every day, Davenport states:

> Every one of these peasants, each item of that "riff-raff" of Europe, as it is sometimes carelessly called, will, if fecund, play a role for better or worse in the future history of this nation. Formerly, when we believed that factors blend, a characteristic in the germ plasm of a single individual among thousands seemed not worth considering: it would soon be lost in the melting pot. But now we know that unit characters do not blend; that after a sore of generations the given characteristic may still appear, unaffected by repeated unions. . . . So the individual, as the bearer of a potentially immortal germ plasm with innumerable traits, becomes of the greatest interest.

That is, of our "greatest interest" to exclude by restricting immigration, lest American heredity be overwhelmed with a deluge of permanent bad genes from the wretched refuse of foreign lands.

To illustrate Davenport's characteristic style of argument, and to exemplify his easy slippage between supposed scientific documentation and overt political advocacy, we may turn to his influential 1915 monograph entitled *The Feebly Inherited* (publication number 236 of his benefactor, the Carnegie Institution of Washington), especially to part 1 on "Nomadism, or The Wandering Impulse, With Special Reference to Heredity." The preface makes no bones about either sponsorship or intent. With three of America's wealthiest and most conservative families on board, one could hardly expect disinterested neutrality toward the full range of possible results. The Carnegies had endowed the general show, while Davenport paid homage to specific patrons: "The cost of training the field-workers was met by Mrs. E. H. Harriman, founder and principal patron of the Eugenics Record Office, and Mr. John D. Rockefeller, who paid also the salaries of many of the field-workers."

Davenport's preface also boldly admits his political position and purposes. He wishes to establish "feeble inhibition" as a category of temperament leading to inferior morality. Such a formulation will provide a one-two punch for identification of the eugenically unfit—bad intellect and bad morals. The genetic basis of stupidity had already been documented in numerous studies of the feebleminded. But eugenics now needed to codify the second reason for excluding immigrants and discouraging reproductive rights of the native unfit—bad moral character (as in Davenport's fallback position, documented earlier in this essay, for restricting Jewish immigration when he could not invoke the usual charge of intellectual inferiority). Davenport writes:

> A word may be said as to the term 'feebly inhibited" used in these studies. It was selected as a fit term to stand as co-ordinate with "feeble-minded" and as the result of a conviction that the phenomena with which it deals should properly be considered apart from those of feeble-mindedness.

To allay any doubt about his motivations, Davenport then makes his political point: Feeble inhibition, leading to immorality, may be even more detrimental than feeblemindedness, leading to stupidity.

I think it helps to consider separately the hereditary basis of the intellect and the emotions. It is in this conviction that these studies are submitted for thoughtful consideration. For, after all, the chief problem in administering society is that of disordered conduct, conduct is controlled by emotions, and the quality of the emotions is strongly tinged by the hereditary constitution.

Davenport then selects nomadism as his primary example of a putatively simple Mendelian trait—the product of a single gene—based on feeble inhibition and leading almost inevitably to immoral behavior. He encounters a problem of definition at the very outset of his work, as expressed in an opening sentence that must be ranked as one of the least profound statements in the entire history of science. "A tendency to wander in some degree is a normal characteristic of man, as indeed of most animals, in sharp contrast to most plants."

How, then, shall the "bad" form of wanderlust, defined as a compulsion to flee from responsibility, be distinguished from the meritorious sense of bravery and adventure—leading to "good" wanderlust—that motivated our early (largely northern European) immigrants to colonize and subdue the frontier. In his 1911 book, Davenport had warmly praised as "the enterprising restlessness of the early settlers . . . the ambitious search for better conditions. The abandoned farms of New England point to the trait in our blood that

entices us to move on to reap a possible advantage elsewhere."

In a feeble attempt to put false labels on segments of complex continua, Davenport identified the "bad" form as "nomadism," defined as an inability to inhibit the urge we all occasionally feel to flee from our duties, but that decent folks suppress. Nomads are society's tramps, bums, hoboes, and gypsies—"those who, while capable of steady and effective work, at more or less regular periods run away from the place where their duties lie and travel considerable distances."`

Having defined his quarry (albeit in a fatally subjective way), Davenport then required two further arguments to make his favored link of a "bad" trait to a single gene that eugenics might labor to breed down and out: he needed to prove the hereditary basis and then to find the "gene" for nomadism.

His arguments for a genetic basis must be judged as astonishingly weak, even by the standards of his generation. He simply argued, based on four dubious analogies, that features akin to nomadism emerge whenever situations veer toward "raw" nature (where genetics must rule) and away from environmental refinements of modern human society. Nomadism must be genetic because analogous features appear as "the wandering instinct in great apes," among "primitive peoples," in children (then regarded as akin to primitives under the false view that ontogeny recapitulates phylogeny), and in adolescents (in whom raw instinct temporarily overwhelms social inhibition in the Sturm und Drang of growing up). The argument about "primitive" people seems particularly weak since a propensity for wandering might be regarded as well suited to a lifestyle based on hunting mobile game, rather than identified as a mark of inadequate genetic constitution (or any kind of genetic constitution at all). But Davenport, reversing the probable route of cause and effect, would not

be daunted:

> If we regard the Fuegians, Australians, Bushmen and Hottentots as the most primitive men, then we may say that primitive man is nomadic. . . . It is frequently assumed that they are nomadic because they hunt, but it is more probable that their nomadic instincts force them to hunting rather than agriculture for a livelihood.

Davenport then pursues his second claim—nomadism as the product of a single gene—by tracing pedigrees stored in his Eugenics Record Office. On the subjective criterion of impressions recorded by fieldworkers, or written descriptions of amateur informants, Davenport marked all nomads in his table with a *scarlet W* for Wanderlust, the common German term for an urge to roam). He then examined the distribution of *W*'s through families and generations to reach one of the most peculiar and improbable conclusions ever advanced in a famous study: nomadism, he argued, is caused by *a single gene*, a sex-linked recessive located on what would later be identified as the female chromosome.

Davenport reached this conclusion because he thought that nomadism ran through family pedigrees in the same manner as hemophilia, color blindness, and the other truly sex-linked recessive traits. This status can be legitimately inferred from several definite patterns of heredity. For example, fathers with the trait do not pass it to their sons (since the relevant gene resides on the X-chromosome and males pass only a Y-chromosome only to their sons). Mothers with the trait pass it to all their sons, but none of their daughters, when the father lacks the trait. (Since the feature is recessive, an afflicted mother must carry the gene on both X-chromosomes. She passes a single X to her son, who must then express the trait, for he has no other X-chromosome. But a daughter will receive one afflicted X-chromosome

from her mother and one normal X-chromosome from her father; she will therefore not express that trait because the father's normal copy of the gene is dominant.) Davenport knew these rules, so his study didn't fail on this account. Rather, his criteria for identifying nomadism as a discrete and scorable "thing" were so subjective, and so biased by his genetic assumptions, that his pedigree data turned out to be worthless.

Davenport's summary reached (and preached) a eugenic crescendo: "The wandering instinct," he stated, "is a fundamental human instinct, which is, however, typically inhibited in intelligent adults of civilized peoples." Unfortunately, people who possess the bad gene *W* (the scarlet letter of wanderlust) cannot achieve this healthy inhibition, and they become feckless nomads who run from responsibility by literal flight. The trait is genetic, racial, and undesirable. Immigrants marked by *W* should be excluded (and many immigrants must be shiftless wanderers rather than brave adventurers), while nomadic natives should be strongly encouraged, if not compelled, to desist from breeding. Davenport concludes:

> The new light brought by our studies is this: The nomadic impulse is, in all the cases, one and the same unit character. Nomads, of all kinds, have a special racial trait—are, in a proper sense, members of the nomadic race. This trait is the absence of the germinal determiner that makes for sedentariness, stability, domesticity.

Of course, no one would now defend Davenport's extreme view of single genes determining nearly every complex human behavior. Most colleagues eventually rejected Davenport's theory; he lived into the 1940s, long past the early flush of Mendelian enthusiasm and well into the modern era of understanding that complex traits usually record

the operation of many genes, each with a small and cumulative effect (not to mention a strong, and often predominant, influence from nongenetic environmental contexts of growth and expression). A single gene for anger, conviviality, contemplation, or wanderlust now seems as absurd as a claim that one assassin's bullet, and nothing else, caused World War I, or that Darwin discovered evolution all by himself, and we would still be creationists if he had never been born.

Nonetheless, in our modern age of renewed propensity for genetic explanations (a valid and genuine enthusiasm when properly pursued), Davenport's general style of error resurfaces on an almost daily basis, albeit in much more subtle form, but with all the vigor of his putative old gene—yes, he did propose one—for stubbornly persistent behavior.

We are not questioning whether genes influence behavior; of course they do. We are not arguing that genetic explanations should be resisted because they have negative political, social, or ethical connotations—a charge that must be rejected for two primary reasons. First, nature's facts stand neutral before our ethical usages. We have, to be sure, often made dubious, even tragic, decisions based on false genetic claims. But, in other contexts, valid arguments about the innate and hereditary basis of human attributes can be profoundly liberating.

Consider only the burden lifted from loving parents who raise beautiful and promising children for twenty years and then "lose" them to the growing ravages of schizophrenia—almost surely a genetically based disease of the mind, just as many congenital diseases of bodily organs also appear in the third decade of life, or even later. Generations of psychologists had subtly blamed parents for unintentionally inducing such a condition, then viewed as entirely environmental in origin. What could be more cruel than a false weight of blame added to such an ultimate tragedy? Second, we will never get

very far, either in our moral deliberations or our scientific inquiries, if we disregard genuine facts because we dislike their implications. In the most obvious case, I cannot think of a more unpleasant fact than the inevitable physical death of each human body, but a society built on the premise that King Prospero will reign in his personal flesh forever will not flourish for long.

However, if we often follow erroneous but deeply rooted habits of thinking to generate false conclusions about the role of heredity in human behavior, then these habits should be exposed and corrected — all the more vigorously if such arguments usually lead to recommendations for action that most people would also regard as ethically wrong (involuntary sterilization of the mentally retarded, for example). I believe that we face such a situation today and that the genetic fallacies underlying our misusages bear a striking similarity in style and logic to Davenport's errors, however much we have gained in subtlety of argument and factual accuracy.

Throughout the history of genetics, the most common political misuses have rested on claims for "biological determinism"—the argument that a given behavior or social situation can't be helped because people are "made that way" by their genes. Once we attribute something we don't like to genes, we tend either to make excuses or to make less effort for change. For example, many people still argue that we should deny educational benefits and social services to groups falsely judged as genetically inferior. Their poverty and misfortune lie in their own heredity, the argument goes, and therefore their condition cannot be significantly ameliorated by social intervention. Thus, history shows a consistent linkage between genetic claims in this mold and conservative political arguments for maintenance of an unjust status quo of great benefit to people currently in power.

Of course, no serious student of either genetics or politics would

now advance such an argument in Davenport's style of "one gene, one complex behavior." That is, no one today talks about the gene for stupidity, promiscuity, or lack of ambition. But a series of three subtle —and extremely common—errors leads all too often to the same eugenical style of conclusion. Somehow, we remain fascinated with the idea that complex social behaviors might be explained, at least in large part, by inherited "atoms" of behavioral propensity lying deep within individuals. We seem so much more satisfied, so much more intrigued, by the claim that a definite gene, rather than a complex and inextricable mix of heredity and social circumstances, causes a particular phenomenon. We feel that we have come so much nearer to a real or essential cause when we implicate a particle within an individual, rather than a social circumstance built of multiple components, as the reason behind a puzzling behavior. We will avidly read a front-page headline entitled "Gay Gene Found," but newspapers will not even bother to report an equally well-documented story on other components of homosexual preference with a primary social root and no correlated genetic difference.

The common source of these errors lies much deeper than any correlation to a political utility most of us do not even recognize—and would disavow if we did. I suspect that the source lies in a general view about causality that has either been beaten into us by a false philosophy about science and the natural world or may even record an unfortunate foible in our brain's evolved mode of operation. We like simple kinds of explanations that flow in one direction from small, independent, constituent atoms of being too complex and messy interactions among large bodies or organizations. To use the technical term, we prefer to be "reductionists" in our causal schemes—to explain the physical behavior of large objects as consequences of atoms in motion, or the social behavior of large animals by biological atoms called genes.

But the world rarely matches our simplistic hopes, and the admittedly powerful methods of reductionism don't always apply. Wholes can be bigger than the sums of their parts, and interactions among objects cannot always be disaggregated into rules of action for each object considered separately. The rules and randomness of a situation must often be inferred from direct study of large objects and their interactions, not by reduction to constituent "atoms" and their fundamental properties. The three common errors of genetic explanation all share the same basic fallacy of reductionist assumptions.

1. We think we have become oh so sophisticated in acknowledging that both genes and environment produce a given outcome, but we then err in assuming that we can best express this correct principle by assigning percentages and stating, for example, that behavior A is 40 percent genetic and 60 percent environmental. We must understand why such reductionist expressions have no meaning. Genetics and environment do interact to build a totality, but resultant wholes are unbreakable and irreducible to separate components. Water cannot be explained as two-thirds the separate properties of hydrogen gas mixed with one-third oxygen's independent traits—just as wanderlust cannot be analyzed as 30 percent of a gene for feeble inhibition mixed with 70 percent of social circumstances that abet an urge to hit the road.

2. We also think that we have become sophisticated in saying that many genes, not just a Davenportian unity, set the hereditary basis of complex behaviors. But we then take this correct statement and impose the reductionist error of asserting that if behavior A is influenced by ten genes and is 50 percent genetic (by the first error), then each gene must contribute roughly 5 percent to the totality of the behavior. But complex interactions are not built as the sum of independent parts considered separately. I am not one-eighth of each

of my great-great grandparents (although my genetic composition may be roughly so determined); I am a unique product of my own interacting circumstances of social setting, heredity, and all the slings and arrows of individual and outrageous natural fortune.

3. We think that we are being sophisticated in qualifying statements about "genes for" traits by admitting their only partial, and often small, contribution to an interactive totality. Thus, we think we may legitimately talk of a "gay gene" so long as we add that only 15 percent of sexual preference records its operation. We need to understand why such statements are truly meaningless and therefore worse than merely false. Many genes interact with several other factors to influence sexual preference, but no separable "gay gene" exists. Even to talk about a "gene for" 10 percent of behavior A is to commit the old Davenportian fallacy on the "little bit pregnant" analogy.

To give a concrete example of how a good and important study can be saddled with all these errors in public reporting (and also by careless statements of some participating researchers), the *New York Times* greeted 1996 with a headline on the front page of its issue for January 2: "Variant Gene Tied to a Love of New Thrills." The article reported on two studies in the January 1996 issue of *Nature Genetics*. Two independent groups of researchers, one working with 124 Ashkenazi and Sephardic Jews from Israel, the other with a largely male sample of 315 ethnically diverse Americans, found a clearly significant, if weak, association between propensity for "novelty-seeking behavior" (as ascertained from standard survey questionnaires) and possession of a variant of a gene called the D_4 dopamine receptor, located on the eleventh chromosome and acting as one of at least five receptors known to influence the brain's response to dopamine.

This gene exists in several variant forms, defined by differing lengths recording the number (anywhere from two to ten) of repeated copies of a particular DNA subunit within the gene. Individuals with a high number of repeated copies (that is, with a longer gene) tended to manifest a greater tendency for novelty-seeking behavior—perhaps because the longer form of the gene somehow acts to enhance the brain's response to dopamine.

So far, so good—and very interesting. We can scarcely doubt that heredity influences broad and basic aspects of temperament—a bit of folk wisdom that surely falls into the category of "what every parent with more than one child knows." No one should be at all offended or threatened by the obvious fact that we are not born entirely blank or entirely the same in our mixture of the broad behavioral propensities defining what we call "temperament." Certain genes evidently influence particular aspects of brain chemistry, and brain chemistry surely affects our moods and behaviors. We know that basic and powerful neurotransmitters, such as dopamine, strongly impact our moods and feelings (particularly, for dopamine, our sensations of pleasure). Differing forms of genes that affect the brain's response to dopamine may influence our behaviors, and a form that enhances the response may well incline a person toward novelty-seeking activities.

But the long form of the D_4 receptor does not therefore become the (or even a) novelty-seeking gene, and these studies do not identify this behavior as such-and-such a percent "genetic" in origin—although statements in this form dominated popular reports of these discoveries. Even the primary sources—the two original reports in *Nature Genetics* and the accompanying "News and Views" feature entitled "Mapping genes for human personality"—and the excellent Times story (representing the best of our serious press) managed, amid their generally careful and accurate accounts, to propagate all three errors detailed above.

The *Times* reporter cited the first error of assigning separable percentages by writing that "about half of novelty-seeking behavior is attributable to genes, the other half to as yet ill-defined environmental circumstances." Dr. R. P. Ebstein, principal author of one report, then stated the second error of adding up effects without considering interactions when he argued that the long form of the D_4 gene accounts for only about 10 percent of novelty-seeking behavior. If, by the first error, all of novelty seeking can be viewed as 50 percent genetic, and if D_4 accounts for 10 percent of the totality, then we can infer that about four other genes must be involved (each contributing 10 percent for the grand total of 50 percent genetic influence). Ebstein told the Times reporter: "If we assume that there are other genes out there that we haven't looked at yet, and that each gene exerts more or less the same influence as the D_4 receptor, then we would expect maybe four or five genes are involved."

But the most significant. errors, as always, fall into the third category of misproclaiming "genes for" specific behaviors—as in the Nature Genetics title previously cited: "Mapping genes for human personality." (If our professional journals so indulge, imagine what the popular press makes of "gay genes," "thrill genes," "stupidity genes," and so on.) First of all, the D_4 gene, by itself, exerts only a weak potential influence on novelty-seeking behavior. How can a gene accounting for only 10 percent of the variance in a trait be proclaimed as a "gene for" the trait? If I decide that 10 percent of my weight gain came from the calories in tofu (because I love the stuff and eat it by the ton), this item, generally regarded as nutritionally benign, does not become a "fatness food."

More importantly, genes make enzymes, and enzymes control the rates of chemical processes. Genes do not make novelty-seeking or any other complex and overt behavior. Predisposition via a long chain of complex chemical reactions, mediated through an even more intricate

series of life's circumstances, does not equal identification, or even causation. At most, the long form of D_4 induces a chemical reaction that can, among other possible effects, generate a mood leading some people to greater openness toward behavior defined by some questionnaires as "novelty seeking."

In fact, a further study, published in 1997, illustrated this error in a dramatic way by linking the same long form of D_4 to greater propensity for heroin addiction. The original Times article of 1996 had exulted in the "first known report of a link between a specific gene and a specific normal personality trait." But now the same gene—perhaps via the same route of enhanced dopamine response—correlates with a severe pathology in other personalities. So what is D_4—a "novelty seeking" gene in normal folk, or an "addiction" gene in troubled people? We need to reform both our terminology and our concepts. The long form of D_4 is a gene that produces a chemical response. This response may correlate with different overt behaviors in people with widely varying histories and genetic constitutions.

The deepest error of this third category lies in the reductionist and really rather silly notion that we can even create rigorous definitions for discrete, separable, specific traits within the complex continua of human behaviors. We have enough trouble specifying characters with clear links to particular genes in the much clearer and simpler features of human anatomy. I may be able to specify genes "for" eye color but not for leg length or fatness. How then shall I parse the continuous and necessarily subjective categories of labile personalities? Is novelty seeking really a "thing" at all? Can I even talk in any meaningful way about "genes for" such nebulous categories? Have I not fallen right back into the errors of Davenport's search for the internal scarlet letter W of wanderlust?

I finally realized what had been troubling me so much about the

literature on "genes for" behavior when I read the Times's account of C. R. Cloninger's theory of personality (Cloninger is the principal author of the *Nature Genetics* "News and Views" feature):

> Novelty seeking is one of four aspects that Dr. Cloninger and many other psychologists propose as the basic bricks of normal temperament, the other three being avoidance of harm, reward dependence and persistence. All four humors are thought to be attributable in good part to one's genetic makeup.

The line about "humors" crystallized my distress, for I realized why the canny reporter (or the scientist himself) had used this old word. Consider the theory in outline: four independent components of temperament, properly in balance in "normal" folks, but with each individual displaying subtly different proportions, thus determining our personal temperaments and building our distinct personalities. But if one humor gets out of whack by substantial over- or under-representation, then a pathology may result.

But why four, and why these four? Why not five, or six, or six hundred? Why any specific number? Why try to parse such continua into definite independent "things" at all? I do understand the mathematical theories and procedures that lead to such identifications (see my book *The Mismeasure of Man*), but I regard the entire enterprise as a major philosophical error of our time (while I view the mathematical techniques, which I use extensively in my own research, as highly valuable when properly applied). Numerical clumps are not physical realities. A four-component model of temperament may act as a useful heuristic device, but I don't believe for a moment that four homunculi labeled "novelty seeking," "avoidance of harm," "reward dependence," and "persistence" reside in my brain, either wing for dominance or cooperating.

The logic of such a theory runs in uncanny parallel—hence the clever choice of "humor" as a descriptive term for proposed modules of temperament—with the oldest and most venerable of gloriously wrong theories in the history of medicine. For more than a thousand years, from Galen to the dawn of modern medicine, prevailing concepts regarded the human personality as a balance among four humors—blood, phlegm, choler, and melancholy. *Humor*, from the Latin word for liquid (preserved in our designation of the fluids of the human eye as the aqueous and vitreous humors), referred to the four liquids that supposedly formed the chyle, or digested food in the intestine just before it entered the body for nourishment. Since the chyle arose, on one hand, from a range of choices in the food we eat and, on the other hand, from constitutional differences in how various bodies digest this food, the totality recorded both innate and external factors—an exact equivalent to the modern claim that both genes and environment influence our behavior.

The four humors of the chyle correspond to the four possible categories of a double dichotomy—that is, two axes of distinction based on warm-cold and wet-dry. The warm and wet humor is blood; cold and wet generates phlegm; warm and dry makes choler; while cold and dry forms melancholy. I regard such a logically abstract scheme as a heuristic organizing device, much like Cloninger's quadripartite theory of personality. But we make a major error if we elevate such a scheme to claims for real and distinct physical entities inside the body.

In the medical theory of humors, good health results from a proper balance among the four, while distinctive personalities emerge from different proportions within the normal range. But too much of any one humor may lead to oddness or pathology. As a fascinating linguistic remnant, we still use the names of all four humors as adjectives for types of personality: sanguine—dominance of the hot-

wet blood humor—for cheerful people; phlegmatic, for stolid folks dominated by the cold-wet humor of phlegm; choleric, for angry individuals saddled with too much hot-dry choler; and melancholic, for sad people overdosed with black bile, the cold-dry humor of melancholia. Is the modern quadripartite theory of personality really any different from this older view in basic concepts of number, balance, and the causes of both normal personality and pathology?

In conclusion, we might imagine two possible reasons for such uncanny similarity between a modern conception of four components to temperament and the old medical theory of humors. Perhaps the similarity exists because the apparatus is true, with the modern version representing a great refinement of a central fact that our ancestors could only glimpse through a glass darkly. But alternatively—and ever so much more likely in my judgment—the stunning similarities exist because the human mind is a constant thing, despite all our growth of learning and the historical changes in Western culture. We therefore remain sorely tempted by the same easy fallacies of reasoning.

I suspect that we once chose four humors, and now designate four basic constituents of temperament, because something deep in the human psyche leads us to impose simple taxonomic schemes of distinct categories upon the world's truly complex continua. After all, our forebears didn't invoke the number four only for humors. We parsed many other phenomena into schemes with four fundamental properties—the four compass points, the ages of man, the four Greek elements of air, earth, fire, and water. Could these similarities be coincidental, or does something about the workings of the human brain favor such artificial divisions? Carl Jung, for reasons that I regard as dubious, felt strongly that division by four represented something deep and archetypal in human proclivities. He argued that divisions by three lack balance and lead onward (for one triad presupposes

another for contrast), whereas divisions by four stand in optimal harmony. He wrote: "[B]etween the three and the four there exists the primary opposition of male and female, but whereas fourness is a symbol of wholeness, threeness is not."

I think Jung correctly discerned an inherent mental attraction to divisions by four. I suspect the basis for this propensity lies in our clear (and probably universal) preference for dichotomous divisions. Four represents a kind of ultimate dichotomization—a dichotomy of dichotomies: two axes (each with two fundamental properties) at right angles to each other. We may experience four as an ultimate balance because such schemes fill our mental space with two dichotomies in perfect and opposite coordination.

In any case, if this second reason explains why we invented such eerily similar theories as four bodily humors and four basic constituents of temperament, then such quadripartite divisions reflect biases of the mind's organization, not "real things" out there in the physical world. We can hardly talk about "genes for" the components of such artificial and prejudicial parsings of a much more complex reality. Interestingly, the greatest literary work ever written on the theory of humors, the early-seventeenth-century *Anatomy of Melancholy*, by the English divine and scholar Robert Burton, properly recognized the four humors as just one manifestation of a larger propensity to divide by four. This great man, who used the balm of literature to assuage his own lifelong depression, wrote of his condition: "Melancholy, cold and drie, thicke, blacke, and sowre . . . is a bridle to the other two hot humors, bloode and choler, preserving them in the blood, and nourishing the bones: These foure humors have some analogie with the foure elements, and to the foure ages in man."

I would therefore end—and how could an essayist possibly find a more appropriate culmination—with some wise words from

Montaigne, the sixteenth-century founder of the essay as a literary genre. Instead of trying to identify a propensity for wandering or for novelty seeking (perhaps a spur to wandering) in a specific, innate sequence of genetic coding, perhaps we should pay more attention to the wondrous wanderings of our mind. For until we grasp the biases and propensities of our own thinking, we will never see through the humors of our vision into the workings of nature beyond. Montaigne wrote:

> It is a thorny undertaking, and more so than it seems, to follow a movement so wandering as that of our mind, to penetrate the opaque depths of its innermost folds, to pick out and immobilize the innumerable flutterings that agitate it.

Stephen Jay Gould teaches biology, geology, and the history of science at Harvard University. He is also the Frederick P. Rose Honorary Curator in Invertebrates at the American Museum of Natural History.

Natural History ***107*** *(March): 22-25, 70-78.*