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Short report

Agency versus structure: Genetics, group membership, and a new twist on an old debate

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ABSTRACT

The decoding of the human genome and advances in genetic medicine promise great advances in the prevention and treatment of disease. These powerful methodologies, though, raise serious intellectual, ethical, and practical questions when they are employed in explanations of complex higher-order behavioral and social outcomes. There can be little doubt that all human behavior reflects complex gene/environment interactions, but isolating the unique contributions of genes and environment in the explanation of overdetermined behavioral and social outcomes may not in principle be possible. When dealing with groups that differ significantly in histories of discrimination and exclusion biological explanations must be employed with caution even as they promise great strides in dealing with specific diseases.

New methodologies in genetic research, such as genome-wide association studies, that can search through millions of individual genes to identify those potentially associated with specific pathologies promise great advances in disease prevention and treatment (Cichon et al., 2009; Psychiatric GWAS Consortium Coordinating Committee, 2009). Like all powerful technologies, though, these new techniques and approaches are not without risks, depending on their use. In the case of genetic and biological explanations of behavioral outcomes those include attributing to biological factors variance that is in fact due to social factors. Debates concerning the role of biology in the determination of human aptitudes and social traits are often rancorous given the racist and unscientific nature of much early work and the nefarious purposes to which biological explanations of group differences have been put. For example, during the 19th century Italian physician Cesare Lombroso proposed a theory that criminality is inherited and that criminals could be identified by specific "atavistic" physical characteristics (Lombroso, 2006). Lombroso also observed that criminals often have tattoos, a physical characteristic that today would place a huge number of individuals under suspicion.

In another example from the 19th Century sociologist Richard L. Dugdale published a study of the "Jukes", a pseudonym he used to refer to a New York hill family among whose members he documented generations of criminals, paupers, prostitutes and other social misfits descended from one female, again supposedly proving the heritability of complex behavioral predispositions (Dugdale, 1877). Even well into the 20th Century members of the eugenics movement who feared that the higher fertility of the inferior classes would debase the gene pool advocated forced sterilization and other procedures to limit their number (Kevles, 1987). Racist Nazi atrocities associated with the Holocaust and the racism that Black Americans have experienced, and racist practices in other countries assure that many observes will find biological explanations of group differences, especially when they might be construed as even implying group inferiority or superiority, to be morally reprehensible (Fraser, 1995).

Beyond the potential racist overtones, though, approaches such as those associated with sociobiology or evolutionary psychology are criticized on methodological grounds (Gould, 1981; Hagen, 2005; Lerner, 2006). For example, although twin studies clearly demonstrate the heritability of even complex traits, they have been criticized for the supposed inability of analysis of variance approaches to distinguish environmental from genetic effects (Lweontin, 2006; Richardson & Norgate, 2006; Rutter, Pickles, Murray, & Eaves, 2001; Wahlstein, 1990). To be sure, behavioral genetics has progressed well beyond the simplistic theories of Lombroso, Dugdale, and the early eugenics movement. Yet, race-based explanations of supposed group differences in complex outcomes continue to be published (e.g., Rushton, 2000), and there are still issues to be debated related to the various domains in which biology in general can be most fruitfully applied. Medical genetics will no doubt greatly advance our understanding of the

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complex interaction among genes and environment in disease expression. The understanding of complex cultural, political, and social behaviors, on the other hand, will in all likelihood benefit little from advances in genetic and genomic research.

In this discussion I identify what I see to be the domains in which an understanding of biology, and especially behavioral genetics, offers great promise in improving health levels, as well as areas in which an understanding of genetics will be far less useful, and potentially even harmful, from the perspective of a social scientist. First, though, let me be clear that reductionism is not confined to biological or genetic explanations of human behavior. It is a danger that potentially affects all intellectual approaches that privilege their own disciplinary perspective. Economic determinism is one clear example. In psychology, classical behaviorism and operant conditioning assumed an almost complete organismic plasticity and treated the social and physical environments as the major, if not the only determinants of personality and behavior (Watson, 1930).

From a theoretical perspective it is useful to relate the discussion of genetic and biological explanations of complex behaviors and social outcomes to the concepts of agency and structure. In social theory "agency" refers to the capacity of individuals to act as agents on their own behalf, either individually or collectively (Ritzer, 2008; Ritzer & Gindoff, 1994). Structure, on the other hand, refers to all those factors that limit humans' ability to act as autonomous agents. These include social class, education, religion, gender, ethnicity, customs, norms, geography, weather, and much more, including basic biological and genetic factors. The real intellectual challenge then is to ask whether it is possible to isolate the impact of various factors, especially those at very different levels of analysis, on complex behavioral and social outcomes. The question takes on particular salience when one's ability to control one's life is constrained by social class factors related to historical disadvantages associated with race and ethnicity.

Certain modern social theorists reject the notion that agency and structure are conceptually or practically distinct (Berger & Luckman, 1966; Bourdieu, 1977; Giddens, 1986). From this perspective agency cannot be conceived of except in relation to structure. Genetics provides evidence that this is the case with gene/environment interactions: The effects of agency and structure, or genes and environment, cannot be isolated. Behavioral genetics suggests that all behaviors reflect complex interactions of the genome and the environment in potentially hugely complex ways.

Environment/organism interactions have interested health researchers for decades, but more recently the mapping of the human genome has given rise to the possibility of a new depth of understanding into how individual genetic factors interact with environmental factors to affect physical and mental health (Asimit, Yoo, Waggott, Sun, & Bull, 2009; Bertram et al., 2008; Cichon et al., 2009; Craddock, O'Donovan, & Owen, 2008; Mahon et al., 2009; Psychiatric GWAS Consortium Coordinating Committee 2009; Treutlein et al., 2009). Such an understanding represents a major undertaking because of the inherent complexity of the systems involved (Burmeister, McInnis, & Zöllner, 2008; Richardson & Norgate, 2006; Rutter et al., 2001). Although twin studies have shown that almost all human characteristics are heritable, finding a single, or even a limited number of polymorphisms or genes that account for individual differences is usually impossible since in the determination of complex outcomes, including most diseases, multiple genes interact in complex ways (Burmeister et al., 2008; Lerner, 2006). Defining and delineating the environment in which organisms live is also a challenge. Although characterizing the environment of fruit flies might be simple, for humans the environment includes the social, cultural, political, and economic systems that immediately affect individuals' and groups' opportunity structures and the external forces that affect their behavior and life chances.

The potential utility of a genetic approach or genetic explanations clearly depends on the question of interest. Medical genetics will rapidly further our understanding of physical and mental illness. It is likely that we will see rapid progress in the understanding of how aspects of the environment, both physical and social, influence the expression of specific disease predispositions. But other questions related to the impact of race and ethnicity as social constructions, or questions related to the impact of group membership or characteristics, will not necessarily benefit from a discourse based on the language of genetics or biology more generally. Racial and ethnic identities or labels are social, psychological, and even political constructions rather than categories that reflect a specific genetic profile. Understanding reasons for the seriously high rate of high school dropout among Mexican-origin students, or the very low levels of wealth among African Americans, requires an appreciation of specific structural disadvantages.

It is clear that the health disparities related to race and ethnicity must be addressed from multiple levels of analysis. Approaches that focus solely on the individual or biological levels run the risk of failing to understand the impact of macro structural factors that determine group-specific educational and occupational opportunities and disadvantages, as well as the living and work conditions that expose individuals and communities to occupational and environmental pathogens. On the other hand, approaches that ignore biology fail to take advantage of opportunities to refine individual risk profiles.

Both theoretically and practically then it is imperative that we deal with the very real problems involved in the combination of different levels of analysis. The problems in explaining higher-order structures and outcomes on the basis of lower-order phenomena (the macro/micro problem) or the dangers inherent in employing collective characteristics in explanations of individual outcomes (the ecological fallacy) are today the same as those that have bedeviled theorists and researchers historically. We know, for example, that Mexican-Americans are, as a census category, at elevated risk of diabetes and its complications, even as they enjoy a mortality advantage over non-Hispanics (Markides & Coreil, 1986). Yet, like all human populations the Mexican-American population is highly differentiated genetically, and the ethnic label itself represents more of an administrative or political category than a meaningful genetic or medical classification. The racial and ethnic group labels that are commonly used in social, behavioral, and medical research reflect significant health-related socioeconomic differences; they are political categorizations that by definition and construction reflect group characteristics of social significance. Their relation to biological traits or predispositions is only approximate.

The promising contribution of genetics is its potential for more accurately identifying those Mexican-origin individuals at highest risk of specific diseases and their complications. But this objective applies to members of other racial and ethnic groups as well. It is doubtful that one would evolve group-specific screening procedures. Poor populations in general require constant screening for, and monitoring of, hypertension and diabetes, as well as other diseases associated with poverty. Refined risk profiles provide useful individual-level information that has little connection to ethnicity per se. Again, the racial and ethnic labels that are routinely used in research reflect politically significant macro-level collective characteristics that are of limited use in predicting individual-level (micro) outcomes. The demonstration that Mexican-origin individuals are, as a group at elevated risk of diabetes is a useful finding, but the explanation of that elevated group

risk requires the examination of a wide range of historically- based structural disadvantages reflected in disparities in education, income, diet, and more.

A classic paper that appeared in Science magazine in 2002 illustrates the potential difficulties in employing genetic factors to socially defined outcomes (Caspi et al., 2002). Caspi et al. report the findings of a longitudinal study of a large sample of boys in Dunedin, New Zealand who they have followed from birth to adulthood. The researchers call upon two very different bodies of research to build their case for a genetic/environmental interaction in predicting complex behavioral outcomes. They begin with several studies that have shown that boys whose parents treat them in erratic, coercive, and punitive ways are at elevated risk of conduct disorder, antisocial personality symptoms, and violent crime in adulthood. But of course not all boys who are mistreated grow up to engage in antisocial behavior. Other animal studies and at least one human study suggest that a functional polymorphism in the gene encoding the enzyme monoamine oxidase A (MAOA), which metabolizes neurotransmitters, is associated with different levels of aggression. Low levels of MAOA are associated with high levels of aggression. The authors found that in their data maltreated boys with a genotype conferring high levels of MAOA expression were less likely to develop antisocial problems than boys with low levels of the enzyme.

A particularly serious problem arises, though, in the definition and operationalization of the behavioral outcome. Although the authors employ a rigorous procedure for defining antisocial behavior the definition is inevitably based on social norms and institutional factors like the probability of being arrested for a violent crime. Antisocial behavior is not the same sort of variable as levels of MAOA or the presence of a specific gene. When groups are the unit of analysis explanations that are based on specific polymorphisms are not necessarily useful, and might even be dangerous. The fact that the Caspi et al. results have not been consistently replicated underscores the need for caution. Attributing the high arrest and incarceration rates among black males to an interaction of specific biomarkers and environment is not straightforward. There are simply too many uncontrolled variables associated with such overdetermined processes, and these clearly include many social and situational factors.

Caspi and others cited earlier have further elaborated genetic environmental interactions in the determination of other complex behavioral outcomes, including personality types in adulthood, IQ, depression, psychosis, post-partum mood symptoms, and personality (Caspi et al., 2005; Hicks, South, DiRago, Iacono, & McGue, 2009; Legrand, Keyes, McGue, Iacono, & Krueger, 2008; Mahon et al., 2009; Turkheimer et al., 2003). In the end it is important not to lose sight of the importance of considering all levels of analysis in assessing individual and group risk factors. Collective educational deficits or labor force disadvantages, though, are unlikely to be explained even partially by genetics. William Shockley and others assume that important behavioral and social characteristics of groups can be attributed to biologically-based predispositions, even though group membership does not imply genetic invariance. The potential risk is poor science and retrograde social policy. While refining individual risk profiles for specific diseases by including biomarkers may be a useful objective, identifying risk profiles for social outcomes is far more problematic. One might legitimately ask whether it even makes sense to look for the genetic markers of such outcomes.

The misuse of genetic profiles is a real danger. One can imagine a world in which an applicant for a government job is denied a security clearance on the grounds that he or she has the genetic profile of a potential security leak. In a more likely scenario, if given the opportunity insurance companies might deny individuals coverage on the basis of membership in a group with an unprofitably high risk profile. Since the possibility exists it is almost inevitable that Genome-wide association studies (GWAS) comparisons of criminals and non-criminals will be carried out. How that information will be interpreted or used is as yet unclear. Very likely it will be deemed to be useless. The possibility for real harm clearly exists though.

Currently the genetic approach is new and novel. We have decoded the entire human genome, but the promising next steps are not entirely clear. In ten years the novelty will have worn off and what today is new and novel will be part of normal science. Through experimentation and trial and error those areas in which genetics proves useful will emerge and thrive. Those where it is less useful will not. The challenge for us is to remain ever vigilant in order to benefit from new and potentially promising approaches while avoiding pseudoscience and approaches that are more rhetoric or theoretical sleight of hand than real contributions. When dealing with socially vulnerable groups that caveat cannot be expressed strongly enough.

Conclusion

Twin studies make it clear that there is very little about human biology and even fairly high level behavior that does not have a substantial genetic component. That fact, though, offers little guidance for the use of aggregate characteristics in predictions of complex individual outcomes. If someone identifies him or herself as a member of a social group with high rates of diabetes a physician would no doubt test glucose levels and look for other markers of the disease. But the doctor should do that anyway regardless of the patient's group membership. Although a better understanding of genetics holds out hope for more effective prevention and treatment of disease, it remains unclear that knowledge of group membership can be combined with biological and social factors to help understand the unique health risks of specific individuals. Individual risk must be assessed on an individual-level.

Much conceptual as well as practical work remains to be done. Through it all our fundamental concern with the autonomy and dignity of human subjects and the social and political consequences of our efforts must remain at the forefront. How genetics can inform social policy remains unclear. The Caspi articles provide intriguing examples of genome/environment interactions in the determination of complex behavioral and social outcomes, but knowing that certain children are at elevated risk of developing antisocial behavioral traits if they are abused provides little useful policy-relevant information. Child abuse should be socially condemned, outlawed, and punished regardless of the genetic vulnerability of the victim. A recent review of research on the potential differential vulnerability of children who grow up in poverty claims to provide policy-relevant information. The authors review a large body of research that suggests that certain children are more seriously harmed by growing up in poverty than others, a finding which they argue calls for more egalitarian social policies (Lundborg & Stenberg, 2009). While the authors' call for more egalitarian social policies is laudable, it is not necessary to document differential vulnerabilities to poverty to advocate policies aimed at its elimination. Certainly one would never advocate that certain resilient children who can be identified through genetic testing should be abandoned to poverty. Perhaps the demonstration of gene/environment interactions involving complex social outcomes is of theoretical interest, but it may be of little practical importance. The determination of which questions and what domains of human suffering can be better addressed though an understanding of genetics and biology is on ongoing question.

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