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A response to the commentaries by Craddock and Fotaki

Ronald J. Angel

University of Texas at Austin, United States

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Commentaries

These are excellent commentaries by Fotaki (2011) and Craddock (2011) on my article "Agency versus Structure: Genetics, Group Membership, and a New Twist on an Old Debate" (Angel, 2011). The commentaries are informed, constructive, and civil, and this debate format is a useful method of fostering a dialog related to an important, or rather several important issues. I and the commentators are basically in complete agreement. They have identified some injudiciousness in certain of my statements which require clarification. In addition, Fotaki takes the discussion into a new domain that I would have very much liked to have pursued had I had the space (Fotaki, 2011). She restates the issues I addressed in a succinct and articulate manner and draws upon important social theorists to develop an argument for a continued focus on structure and social factors in the explanation of racial and ethnically-based health vulnerabilities that speak directly to the intent of the original article.

I, the commentators, and probably most everyone else, are in agreement that factors that we label biological or social are inevitably and inextricably intertwined. Although the language of "agency" and "structure" may have no place in polite dinnertime conversation, the rhetorical objective is to reiterate the point that the distinction is far from straightforward, either theoretically or empirically. As with "biological" or "social", the terms are merely convenient ways of referring to different analytical foci. Clearly what we refer to as "social" is created and reproduced by biological entities acting in accordance with genetically evolved capacities. As Craddock (2011) notes, the choice of language with which one describes the world reflects one's disciplinary background and practical objectives. There can be little doubt that the old debate over "nature" versus "nurture" belongs in textbooks on the history of science. Fotaki, though, restates my point that it is unlikely that specific genetic variations will explain complex behaviors and social outcomes. As she states, some behaviors and even complex abnormalities, including certain psychoses, may well be shown to be related to a limited set of such variations. Distinguishing among various outcomes and their relation to explanatory factors at very different levels of analysis is crucial. As I discuss below, problems with the combination of explanatory and outcome factors from very different levels of analysis lies at the core of my argument.

Craddock also objects to my assertion that one would not develop group-specific screening procedures. My statement is too abbreviated and requires greater elaboration which again was not possible in the original article. Individuals of Mexican or Native American origin, as well as African–Americans as Craddock (2011) notes, are as groups at elevated risk of Type II diabetes. Yet all three groups are characterized by substantial genetic variation. There has been and is now a great deal of intermarriage and even intergroup sex. Racial and ethnic categorizations represent political labels or census categories rather than meaningful genetic classifications. Clearly the identification of meaningful risk profiles is vitally important and those defined by age, one's own medical history, one's family's medical history, one's clinical profile, etc. allow one to target diagnostic as well as treatment efforts. My reference was to classifications based on supposed racial and ethnic classifications, and perhaps other social labels. One would not, I imagine, propose a specific diabetic test for Mexicans, Native Americans, or African–Americans. One would use the standard assessments

E-mail address: rangel@austin.utexas.edu.

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regardless of the race or ethnicity of the patient. What a racial identity or classification per se might add to a specific social and clinical risk profile is unclear. Craddock makes the point that race-specific results of any class of drugs have not been documented. He also notes that an excess focus on race-specific biological risk factors could have negative social consequences.

Craddock also corrects my statement about the decoding of the entire human genome. My reference was a rhetorical flourish that is clearly an overstatement. Much work is necessary before we adequately understand human genetics and develop effective treatments for specific diseases. My point was that much progress has been made and that much more will be made in the years to come. Craddock's observation that social policy will have to be aware of genetics, even if it is not driven by genetic findings, goes directly to the point.

Fotaki raises issues that are central to the intent of the original piece. Within the word limit there was simply no room to raise the issue of health disparities and their causes or introduce the important theorists she mentions. This commentary serves perhaps as a completion by proxy and I appreciate this important addition to the discussion. My article was a revision of a keynote speech presented at a conference on bio-biological factors related to health differentials among Hispanics and other groups. My research has focused on racial and ethnic differentials in illness and access to health care for decades and I am keenly aware of the institutional factors that create racial and ethnic health vulnerabilities. These are inextricably related to a history of colonization, exclusion, and institutionalized inequality. The observation that genomics could be used to explain away the social roots of health inequalities, or more broadly any undesirable social outcomes, goes right to the point of my article. An excessive focus on biological factors of any sort to explain differential social outcomes to the exclusion of structural and institutional sources of vulnerability represents the real danger.

Fotaki's observation that the unintended implication of the article is that genetics alone hold the key to improvements in public health was definitely unintended and completely contrary to my intent if that is how it can be read. This commentator's development of a theoretical structure based on Foucault, Bourdieu, and Butler is right on target. I am in complete agreement both with the theorists she chooses and the proposed explanatory structure. Such a theoretical model should inform all research focused on racial, ethnic, and gender-based differentials in health and other outcomes.

Clearly our ultimate objective should be to improve the health of individuals and populations. Yet there are theoretical as well as practical issues to which the article relates. Basic problems related to epistemology and measurement lie at the core of comparative research. The intent of my article was to problematize the combination of variables from different levels of analysis, an issue which relates directly to attempts to employ biological predictors to explain higher-order behavioral or social outcomes. The danger of misattribution of social causes to individual characteristics and traits is very real. As the philosophical and systems theory principle of emergence holds, higher-order processes cannot logically be explained solely by lower-order processes. After several decades of teaching research methods to social scientists and engaging in many projects myself I am keenly aware of the complexity of most social scientific constructs and of the imprecision inherent in not only measuring them, but also in providing conceptually precise definitions.

I am also very interested in translation, which introduces an additional dimension of complexity into the measurement and comparison of complex outcomes in groups that speak different languages. My reference to Caspi (2002) was intended to highlight the very different nature of biological predictors and complex behavioral or social outcomes. The definition and measurement of antisocial behavior is very different conceptually and operationally than measurements of specific hormone levels or polymorphisms. Delinquent and troubled boys live in a complex world that is structured by far more than their genes.

The emerging focus on biology is timely and necessary and attempts to include biological factors in the explanation of complex behavioral and social outcomes inevitable and understandable. We have probably reached the point of seriously diminishing returns in the use of self-reported behaviors and risk factors to predict self-reported symptoms and behaviors. Today social scientific researchers are under tremendous pressure to collect information on biomarkers of all sorts. In the social sciences it is not as yet clear how we will make the best, or even the most legitimate, use of this information. Social group membership does not define any specific genetic, health risk, or behavioral profile. Individual genetic differences in every characteristic clearly exist and are important in the understanding of disease susceptibility. At the aggregate level, though, the fact of genetic variation is logically very different. Individuals may have genetically determined differences in specific intellectual capabilities but attributing group difference in performance on such measures as IQ tests to genes represents pseudo-science. Understanding how genetic information can be combined with information from history, political economics, cultural studies, and other disciplines to help explain complex social outcomes requires ongoing examination and debate.

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