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Review of *A Troublesome Inheritance* by Nicholas Wade

Laura R. Stein[1]

A Troublesome Inheritance: Genes, Race and Human History, by Nicholas Wade. New York: Penguin Press, 2014. x + 278 pp. 978-1-5942-0446-3 (hardcover). US $27.95.

In his book *A Troublesome Inheritance*, Nicholas Wade, a former science writer for the *New York Times*, suggests that studying racial differences should not be avoided but embraced. A key point of Wade's argument is that racial and ethnic differences in behavior can be boiled down to genetic differences and that these genetic differences in behavior result in differences in cultures and societies, some of which are less well "adapted" for the present day than others. Wade uses the fact that little is known about the translation of genes to behavior to make spurious claims under the guise of sounding scientific. Phrases such as "there is reasonable evidence that trust has a genetic basis" (184) is just general enough to wiggle out of having to actually *provide* that "reasonable evidence." Close examination, however, shows that such evidence is scant, should be approached with caution, or does not exist. Yet phrases such as this pepper the book and are used to then tell just-so stories about human societal development, without reasonable evidence.

The genetics of behavior have proven notoriously difficult to pinpoint. Wade posits that, because selection has shaped genetic underpinnings of morphology and physiology, it follows that selection also shapes genes related to behavior. This is not a controversial assumption in itself; indeed, the field of behavioral ecology focuses on trying to understand the selective pressures that, over time, have shaped behavior. In animal models, where one can manipulate the environment, breeding, and individual genes, it should be easier to determine causal genes linked to behaviors than in humans. Yet even under these conditions, we still know very little (and, it should be noted, Wade acknowledges this multiple times throughout the book, himself highlighting the speculative nature of his argument). Not only do we know very little about the genetic basis of behaviors; we know even *less* about how genes may influence societies.

One of the major behaviors the book focuses on is aggression. Wade describes a study where rats were assayed for aggressive behaviors, and the extreme phenotypes (i.e., the most and least aggressive individuals) were selected to breed. The researchers found that aggressive behaviors (and it should be noted that behavior was measured as aggression toward humans, not to other rats) were linked to two loci, altogether including 1,083 genes (Albert et al. 2009). These loci were also found to be associated with the size of the adrenal gland, responsible for producing "stress" hormones often associated with aggression. So, even though specific genes underlying aggressive (or, arguably, antipredator) behaviors have not been uncovered, it can be concluded that individuals with these genotypes will always be more aggressive than their counterparts without them. However, the expression of genes is remarkably plastic; that is, whether genes will be turned "on" or "off" is dependent on environmental inputs.

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Within rats, Dr. Frances Champagne and colleagues noticed that mothers vary in the amount of care provided to their offspring. When they quantified this behavior, they found that offspring of mothers that provided less care were less social and showed more anxious behavior than offspring of mothers that provided more care (Champagne 2008). Further, they showed that moving offspring born to a low-caring mother to be raised by a high-caring mother could reverse these effects. Maternal care produced epigenetic changes (alteration of gene expression without alteration of the DNA sequence) in the development of the offspring's adrenal glands, altering behavior without altering genes. If behavior in the relatively simple social environment of mice can be substantially altered without genetic change, we cannot speculate that behaviors underlying human societies need be due to genetic shifts.

Wade presents the example of rats bred for aggression to show that there is a reasonable expectation that the behavior is genetically based. Wade then transitions into humans, making the case that we have strong evidence for a human gene involved in aggression: *MAOA*. He presents multiple studies showing that people with two *MAOA* promoters are more likely to have been involved in violent crimes and delinquency, and that these were most common in African American men (53–57). And yet, nowhere is there a mention that the effects of the two *MAOA* promoters in increasing juvenile delinquency and violence are primarily an interaction with environment: high levels of violence only appear if the subjects were maltreated or experienced high levels of stress in their home life as children (Caspi et al. 2002; Guo et al. 2008); this result also held true in a study performed entirely in Caucasian children from England and Wales (Kim-Cohen et al. 2006). In fact, one of the study authors stated in an interview that if children in the study with two promoters "have a parent who has regular meals with them, then the risk is gone" (Fox 2008). Environmental circumstances, here parental/adult care, can alter behavioral outcomes regardless of the gene sequence, as was seen in mice and rats. Differential gene expression across populations will not, therefore, necessarily result in behavioral or, to make a larger leap, societal differences. Wade acknowledges these results in one sentence at the end of the section: "Whether a propensity to violence is exercised depends on circumstances as well as genetic endowment, so that people who live in conditions of poverty and unemployment may have more inducements to violence than those who are better off" (57). Perhaps this sentence would be better placed within the discussions of the studies themselves to reflect their actual findings.

Another key to genetic underpinnings of societal success, Wade claims, is genetic differences in IQ. Intelligence (among other traits) is referred to throughout the book as "almost certainly under genetic influence" (190) and treated as such. Yet what "under genetic influence" actually means or how genes are coded into behavior are never actually explained. Genetic differences in intelligence have long been supposed to occur, as most heritability estimates place IQ within a 30–70% range (i.e., 30–70% of variation in IQ should be able to be explained by inheritance from one's parents). This is a huge range. Keep in mind, this leaves 30–70% of variation unaccounted for by heritability, suggesting a role of the environment, imprecision of measurement, or observer bias. For the moment, let's assume that IQ is a good measure of intelligence (an issue that itself is heavily debated). Similar to the above example with rats, environment cannot be discounted. Whether IQ scores are "heritable" has been shown to depend on environmental effects, such as maternal experience (Devlin et al. 1997) and socioeconomic status: a study by Turkheimer et al. (2003) found that in impoverished families, heritability of IQ was close to zero, while in affluent families, IQ appears mostly genetically based.

However, Wade ignores much of the research on IQ and environment, choosing instead to focus primarily on one study. On pages 202–209, Wade describes a study attempting to link causal "intelligence" genes (i.e., those associated with high IQ scores) to genetic diseases in Ashkenazi Jews; the idea here is that Ashkenazim experienced a trade-off between genes conferring intelligence and those linked to lethal disease. Arguably, then, there are specific genes underlying intelligence that are capable of undergoing natural selection, and in the rest of the chapter (and earlier in the book) this is taken as a given.

However, finding such genes has proven elusive. One reason that it has been difficult to identify

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specific genes associated with intelligence, or with any behavior, is that people have millions of identifiable genetic variants. By chance, if you attempt to correlate variations in behavior with a million genetic variants, some of them will be statistically significant. But are they real, or a statistical artifact? Attempts to replicate findings associating specific genes with intelligence have largely failed, suggesting many of the findings have been false positives (Chabris et al. 2012). Wade concedes that no alleles have been found with any certainty, a finding he attributes to the idea that each makes too small of a contribution to be detected with current methods (190). While genes may be associated with behavior, environment plays a substantial role in the expression of those genes. If genes are not expressed in the phenotype (i.e., if a behavior encoded by genes is not performed), natural selection will be unable to act on it either positively or negatively.

Environmental, historical, and other sociological factors are given lip service and quickly dismissed. Wade allows himself to do this by beginning the book with this warning: "Readers should be fully aware that in chapters 6 through 10 they are leaving the world of hard science and entering into a much more speculative arena at the interface of history, economics, and human evolution." In fact, he even states that "the conclusions presented in these chapters fall far short of proof" (15). If these conclusions fall far short of proof, they should not be presented in this book. A casual reader, for whom this may be their first introduction to human genetics, will not necessarily understand that these claims are not supported by scientific evidence. This is disingenuous and irresponsible. And, in a neat little trick, if you disagree with Wade's conclusions, you either are willfully refusing to acknowledge that there are some genetic differences among races, or you secretly do agree but are afraid of being labeled a racist. This is incredibly demeaning to scientists studying human genetics and discounts much of their rigorous, well-performed research.

There are many good, interesting, and worthwhile reasons to study variation in humans. Wade argues that political motivations should not stall or dictate scientific progress or areas of study. Yet the tone of this book suggests that Wade is simply "telling it like it is" to scientists who are cowed by political correctness, and this has made many scientists defensive and angry. It *is* very important that when studying race, whether through the lens of social science, cultural or biological anthropology, or evolutionary biology, we *do* take into account the abusive history of aspects of these fields. Until recently, members of scientific institutions have been overwhelmingly white and male, bringing in their own prejudices and a priori assumptions to such studies.

There are countless examples of the harmful nature of such homogeneity in science. One standout example relates to brain size. In 1906, Robert Bennett Bean, an anatomy professor at the University of Michigan, published a paper describing the smaller size of African American brains compared with European brains, in particular, the frontal cortex responsible for higher cognitive functions. Bean not only reasoned that this is proof that are African Americans intellectually inferior but also expanded this to women and the underprivileged as well. In fact, Bean had failed to find significant differences in brain size and explained in an addendum to the paper that the reason he found no difference between the brains of blacks and those of whites was because he was measuring the brains of higher classes of blacks with those of the lowest classes of whites. His preformed prejudices resulted in him altering the data to fit his conclusions. His mentor re-examined the same brains Bean used in his study and found no differences (Mall 1909).

History is rife with examples of such prejudices informing scientific thought, from inferiority of certain races, socioeconomic classes, and women (the repercussions of which can still be felt today) to the horrors of eugenics. This underscores the need for greater inclusivity in the sciences, to allow multiple perspectives and analysis. By hand waving, making unsubstantiated claims, and ignoring much of the history of human genetics studies and incredible advances in genomics, Wade's arguments on genetics and race are unconvincing and harmful and do a disservice to scientists studying human genetics and behavior.

LITERATURE CITED

Albert, F. W., O. Carlborg, I. Plyusnina et al. 2009. Genetic architecture of tameness in a rat model of animal domestication. *Genetics* 182:541–554.

Bean, R. B. 1906. Some racial peculiarities of the Negro brain.

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Am. J. Anat. 5:353–432.

Caspi, A., J. McClay, T. E. Moffitt et al. 2002. Role of genotype in the cycle of violence in maltreated children. *Science* 297:851–854.

Chabris, C. F., B, M. Hebert, D, J. Benjamin et al. 2012. Most reported genetic associations with general intelligence are probably false positives. *Psychol. Sci.* 23:1,314–1,323, doi:10.1177/0956797611435528.

Champagne, F. A. 2008. Epigenetic mechanisms and the transgenerational effects of maternal care. *Front. Neuroendocrinol.* 29:386–397.

Devlin, B., M. Daniels, and K. Roeder. 1997. The heritability of IQ. *Nature* 388:468–471.

Fox, M. 2008. Study finds genetic link to violence, delinquency. Reuters, 14 July, www.reuters.com/article/2008/07/14/us-delinquents-genes-idUSN1444872420080714.

Guo, G., X-M. Ou, M. Roettger et al. 2008. The VNTR 2 repeat in *MAOA* and delinquent behavior in adolescence and young adulthood: Associations and *MAOA* promoter activity. *Eur. J. Hum. Genet.* 16:626–634.

Kim-Cohen, J., A. Caspi, A. Taylor et al. 2006. *MAOA*, maltreatment, and gene-environment interaction predicting children's mental health: New evidence and a meta-analysis. *Mol. Psychiatr.* 11:903–913.

Mall, F. P. 1909. On several anatomical characters of the human brain, said to vary according to race and sex, with especial reference to the weight of the frontal lobe. *Dev. Dynam.* 9:1–32.

Turkheimer, E. T., A. Haley, M. Waldron et al. 2003. Socioeconomic status modifies heritability of IQ in young children. *Psychol. Sci.* 14:623–628.

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