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GENE GENIE

A review of *A Troublesome Inheritance: Genes, Race and Human History* by Nicholas Wade. New York, NY: Penguin (2014), 288 pages.
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The most controversial aspect of this book is the title. By attempting to tie folk concepts of human differences to commonplaces of genetics research, Wade has conjured up a storm of protest. Indeed, some of the responses to the book have been absurdly over-the-top in terms of scaremongering and threats. So much so, that Scooby Doo and his friends might wonder if the tales of scary monsters were a deliberate attempt to keep meddling kids away from something really interesting down at the disused old fair ground.

In respect of this we will merely note that scientific support for folk concepts is no more likely to be forthcoming in genetics than it is in any other field. What interests the readers of this journal are the more interesting questions of what Wade's book contributes to our understanding of human behavioral sciences.

In the first half of the book Wade tries to summarize and synthesize the results of the last ten years of analyzing the human genome. In the second half, he attempts to give grounds for believing something like Fukuyama's (1989) somewhat Whiggish view of history. We will leave detailed commentary on this latter aspect of his book to others. However, some behavioral parts of this thesis appear premature to us for reasons we will go into below.

For a variety of reasons, some of which are interesting and profound, and some merely historical accident, the study of human behavior has been bifurcated. The biology of humans has been commonly understood to be fixed across the species – on the model of computer hardware. Those things that make us different from one another – simplified under the rubric of culture – has been taken to be akin to software. This has been seen as the more flexible aspect. One major (and boring) reason for this two-fold division has been that it's just methodologically – mainly mathematically--simpler to model human biological data in terms of the neutral theory. On this view, genes mainly just change due to drift without much Darwinian selec-

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tion going on. At the same time, human brains have been held to just absorb culture, like sponges – neatly explaining human differences.

There is nothing wrong with simplifications and place-holders in science. Physics, for example, is full of place-holders like "dark matter" and "dark energy" about which we know very little and we just leave on one side until we know more. However, recent advances – many of which are highlighted in Wade's book--have made both the simplifications of biological and cultural black boxes increasingly untenable. What this means is some of the simplifying assumptions of both anthropology and evolutionary psychology – indeed human behavioral science in general – are going to need revising.

One necessary revision is in terms of what it is that is under selection. The number of genes that directly code for proteins is less than 30,000 and according to some recent accounts is even lower than that (HARROW et al. 2012). This means that only a few hundred genes separate humans from chimpanzees and not many more separate humans from mice. This means, to anyone who thinks it through, that we have miscounted the units of selection. As Marvin Minsky once quipped, "Don't read any genetics research published in your lifetime".

The source of this miscounting is the roughly 98% of the genome which has been known as junk DNA. This turns out to have, in many cases, regulatory roles of turning on and off other genes in response to a huge number of external and internal triggers. The whole genetic dance is more complex than we first thought (HARROW et al. 2012). However – the basic principles remain unchanged. These regulatory genes are still under selection and many give rise to the sorts of adaptations to be discussed below. That Wade does not discuss them does undermine his later argument somewhat. However, his initial points about the reality of recent genetic selection stand.

All measured behavioral traits show some degree of heritability (TURKHEIMER 2000). Techniques like twin studies have been criticized for years, and such criticisms always carried something of the air of the kangaroo court about them. In the light of modern techniques, these criticisms seem not so much trumped-up as simply quaint and irrelevant. As COCHRAN and HARPENDING (2009) put it "SNPs don't lie".

It is still true – as evolutionary psychologists assume--that, to first approximation, humans are adapted to a Pleistocene social life. However, evolution simply did not stop at about the point that our ancestors left Africa. This realization has consequences. Similarly, we can no longer make do with the notion that all human variation can be simply lumped into the place-holder of culture without further analysis. To understand culture it turns out that we are going to have to further decompose it into its evoked and transmitted elements (BARKOW, COSMIDES and TOOBY 1992). This is not something that Wade discusses – and this again does represent something of an explanatory lacuna in his account. In fairness though the question of where culture originates has not been squarely faced by mainstream anthropology either.

Whether one reacts to these changes as exciting or threatening is going to be largely a function of one's own temperament, and the threats these changes might pose to one's ideological commitments. As such they are not scientifically interesting.

Wade's core message is threefold. Namely, that evolution has been recent, copious, and regional. We shall focus this review on what can be said for each of these claims and then pose some questions about why Wade has left out some of the most interesting and well-grounded behavioral science that bears on these questions.

Has there been recent evolution?

There is no doubt that there has. Humans are mostly the same – in that over 99% of their genes are identical. However, genes come in different forms and these forms – alleles – do vary in their proportions between individuals and between populations. It is well known that adaptations to altitude, availability of milk, or resistance to malaria have been recent adaptations. Although humans might see a strong division between minds and bodies, nature does not. Some genetic variants are strongly linked to behavioral differences in humans.

In this respect what Wade says is obviously true – indeed a commonplace of modern genetics. Selection is apparent in recent changes in allele frequencies. Wade mentions MAO-A variants and there are others that he could have mentioned too. For example, some populations show marked differences in the proportion of DRD4 7-repeat allele (KIDD, PAKSTIS and YUN 2014). Variants of this gene are associated with a range of risk-taking behaviors.

Wade's treatment of a vast subject could easily have been expanded upon especially in the area of intelligence research. An historical body of research exists noting a dysgenic trend in intelligence from as early as 1857 with the work of Benedict Augustin Morel (LYNN 2011). Ever since then, scholarly papers have appeared attesting to a dysgenic trend. Wade does refer to the increase in intelligence scores when people move from poorer to richer countries. He does not, however, refer to this as the Flynn effect. The only thing is, the Flynn effect does not occur on 'genetic *g*' as it is primarily an increase in phenotypic intelligence (LYNN and HARVEY 2008). Broadly, the construct *g* evidences a negative correlation of about 0.4 with the Flynn effect which admittedly leaves a fair amount of variance unaccounted for in this association. Nonetheless, the argument is bolstered by the fact that Flynn effects are not associated with Jensen effects which suggest significant correlations between high *g*-loaded items and *g*. In essence, the Flynn effect is 'hollow' regarding *g* and is caused mainly by environmental effects (TE NIJENHUIS and VAN DER VLIER 2013; WOODLEY and MEISENBERG 2013).

Genetic *g* is operationalized as scores on *g*-loaded test items and the dysgenic trend occurs primarily on *g* and so *g* loadings and heritabilities are likely strongly

associated (MEISENBERG 2010; MEISENBERG and KAUL 2010; WOODLEY and MEISENBERG 2013). Moreover, the case for genetic *g* is made all the stronger due to recent evidence suggesting that modern-day populations are slowing down on simple reaction time, a measure which is itself highly correlated to genetic *g* (also referred here to chronometric *g,* ARMSTRONG and WOODLEY 2014) and not as observable on non-*g*-loaded test items (WOODLEY, TE NIJENHUIS and MURPHY 2013a, b, c, d; 2014). This dysgenic trend has been in existence in Western populations since around the 1850s (WOODLEY 2012a). Evidence for a genetic basis for intelligence if there ever was one!

Indeed, not only has a Flynn effect been noted since the 1920s and indeed first documented by Runquist in 1936 (LYNN 2011, 2013) but an anti-Flynn effect (negative Flynn effect) is now being observed evidencing a decadal decline of between 0.26 and 4.3 IQ points in various European countries (DUTTON and LYNN 2013; TEASDALE and OWEN 2008) but the link between the anti-Flynn effect and *g* is less clear at this stage. Certainly aspects such as different teaching styles or differing societal pressures are possible contenders for the reversal of the Flynn effect seen in recent years (SHAYER and GINSBURG 2009). Regarding the wealth of nations and IQs, Wade concludes that LYNN and VANHANEN's (2002) correlations are simply that; associations with no clarity as to the direction of cause and effect. Does higher intelligence result in greater wealth or is it wealth which results in higher intelligence? There is more clarity now, in that, however feasible an idea it may be that wealth creates the necessary optimal environments to increase IQ, these increases are evidenced on the non-genetic component of intelligence and is recognised as the Flynn effect. Wealth may well bring about an upward movement of IQ but this move is constrained within genetic parameters (WOODLEY 2012b).

Wade could further bolster his argument for the genetic basis of intelligence if he included research attesting to dysgenic trends in intelligence. The mechanism for the concurrent upward movement of non-*g* loaded tests (Flynn effect) and the downward movement of genetic *g* (dysgenesis) is explained by teasing apart the differential effect of *g*-loaded items of intelligence test batteries. This is speculatively referred to as the co-occurrence model (WOODLEY 2012a; b). The countervailing Flynn effect shows increases in intelligence test scores over time but this is starting to reverse in some countries. The reversal is partly due to the upper limits placed on environmental influences by intelligence as well as the dysgenic effect on intelligence. To dismiss, as Wade does, the importance of the genetic basis of intelligence is an error. Simply stating that the genetics behind intelligence is polygenic is a bit of a cop out. Admittedly, numerous genes with small effects underlie the genetic influences on intelligence (DAVIES et al. 2011). With a little bit of effort into reading the dysgenic intelligence literature research, the argument for the further study of the genetic basis of intelligence would have been made stronger. Also, Wade refers to the link between an individual and the greater society within which she is placed by stating '… intelligence is a quality of individuals, not of societies'. The Breeder's equation in fact states the opposite and has been known since Fisher's

1918 paper on the topic. The Breeder's equation (as it is now referred to) simply states that the response to selection is equivalent to the product of narrow-sense heritability and the selection differential. It is the selection differential which states that there is a difference between the population mean and the mean of the parental population (in this case, referring to intelligence) (COCHRANE 2013). The difference between the parental and populations means is variance which remains unaccounted for in the next generation. In essence, a fair amount of the variation in intelligence is not accounted for by the effects of genes or families (TURKHEIMER 2000).

It is unclear why Wade does not really get involved with recent research into intelligence. It can hardly be because he is afraid to court controversy.

Has evolution been regional?

Once again – there is no doubt that it has. The adaptations noted above are all to be found in populations associated with certain geographic locales. Altitude adaptions (different ones in each case) are found in populations that live high. Lactase persistence is found in populations where a culture of cattle farming has fed back into the genome (TISHKOFF et al. 2006). There are a number of malaria adaptations that track the threat of this disease, and so on. The links to human recent behaviorally linked genes are less clear cut. However, this is no reason to ban research in this area.

Has evolution been copious?

Wade over-states the case here. It's true that the rate of genetic change may not be as uniformly glacial as we commonly supposed. However, to simply point to large scale cultural differences, and assume that they must ipso-facto rest on genetic differences is too hasty. A lot of human behavioral difference can rest on facultative adaptations to environments. Some of this has been obscured by the too-simple models we have been using to date and the frankly, overly rosy view of human origins, held by some researchers.

The main reason for thinking this is the increasing body of evidence that humans, in common with lots of other creatures, have suites of adjustments to local conditions described by life history theory (STEARNS 1976). Put simply – organisms with identical genes can show plasticity to recurring patterns of threats and opportunities that demonstrates that both of these have featured long into said organisms' phylogeny.

These adaptations are not unique to humans and the phenotypic results of such differential gene expression can be so profound. So profound, indeed, that it has made scientists initially believe that they were studying separate species. A classic example would be Coho Salmon who have two very different male morphs with very different behavioural concomitants that lock in at about twelve months of de-

velopment (SANDERCOCK 1991). Jack salmon are small and sneaky with cryptic coloration. By contrast, Hooknose salmon are large and aggressive with bright colors and physical weaponry. They are genetically identical but with totally different morphologies and behavioural strategies.

A very basic trade-off that organisms need to maximise fitness is between the allocation of resources to early maturation, sexual activity and reproduction. Organisms that are cued to a relatively easy life can afford to live slow, allocate resources to repair, and to consider future options. On the other hand, otherwise genetically identical organisms that are cued for a future risky environment will have higher fitness if they allocate resources to rapid maturation with concomitant risk-taking. Typical early cues to fast or slow life history could include infant mortality, degree of parental care, and infant hunger. Such trade-offs explain at least part of the relative height differential in Mbuti Pygmies compared to their neighbours (MIGLIANO, VINICIUS and LAHR 2007).

Mainstream evolutionary psychology has yet to fully grasp the implications of some of these developments (NETTLE 2009). For example – it is not simply the case that males have a Pleistocene-established preference for human females of particular sizes. Fat stores are useful in famine conditions but energetically inefficient in other ways. Male preferences track these trade-offs. In populations that have suffered repeated famine threats preferences shift to larger females (BROWN and KONNER 1987; KING 2013). In populations where this threat is absent the preference is for smaller females (TOVÉE et al. 1998). The mechanism can also be seen in microcosm. Males manipulated to feel poor or hungry shift their preferences towards larger females (NELSON and MORRISON 2005).

Other aspects of human development that are predicted by cues to the increased fitness of a live-fast strategy have also been found. Examples include age of first pregnancy along with characteristic future discounting in a cohort of British women (NETTLE, COAL and DICKINS 2011), trust of neighbours as measured in behavioural economics games and future discounting as measured in a raft of risk-taking behaviors predicted by cues to inequity (WILSON and DALY 1997).

The differences between trust of and thereby co-operation of neighbors in adjacent districts of Newcastle was so great that it might have been put down to basic genetic differences if it had been found in populations in different countries (NETTLE, COLLÉONY and COCKERILL 2011). However, the neighborhoods in Newcastle were geographically close and not reproductively isolated. This makes an explanation in terms of pure genetic difference unlikely.

The existence of such mechanisms argues that things like famine and social inequity have been part of human history long enough for complex adaptations to have evolved to maximise fitness in response to them. This challenges mainstream anthropological models of human forager pasts as universally egalitarian and resource rich. It also challenges mainstream evolutionary psychology models of adjustments to such a lifestyle being more or less fixed, with deviations from this representing mismatch.

Wade thinks that trust may well be a basic feature of social differences between large-scale observable human populations. He may be right. However, humans seem exquisitely tuned to the likely local results of their co-operation and trust. Its not just (the cliché goes) that far more research is needed before we can conclude that bottom-up levels of trust underlie large-scale population differences in humans. We need research that integrates all our fields rather than divides them.

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