We must know. We will know.

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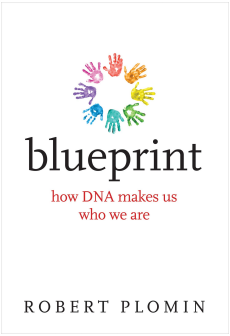
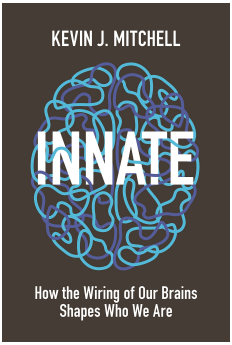
Abstract

Two new books on behaviour genetics and heredity argue that the effects from 'non-shared environment' are due to random noise, a combination of measurement error and chance developmental variation.[1]





- Robert Plomin *Blueprint* (MIT Press, 2018). ISBN: 9780262039161. 280 pp.
- Kevin J. Mitchell *Innate* (Princeton University Press, 2018). ISBN: 9780691184999. 304 pp.

[1] `http://galton.org/reviews/Plomin-Mitchell/plomin-mitchell.html`.

The nature-nurture question, though often advertised as delusional or uninteresting, even as the product of some or other nefarious plot, has been steadily investigated ever since Galton first framed it in 1865.[2] Along the way there have been some interruptions of service and prolonged bursts of inactivity. With appropriate resources, much more progress could have been made, and what we know today is the result of scrimping together funding and accumulating results over many years. Since this is an empirical question, contingent on what happens to be the case, as opposed to something that could be settled on theoretical grounds, resources are essential. These two books present an up to date summary of the evidence so far and preview exciting prospects for the future. Following David Hilbert, 'we must know—we will know!'

The key question is which *differences* in conditions lead to *differences* in outcomes. It is not about which factors are required to get any outcomes at all. Trivially, any organism needs an environment in which to develop. Many of the misconceptions about behaviour genetics come from subtly confusing these two ideas. Although Galton's research programme has drawn a lot of criticism over the years, it has always been the only real game in town for answering this question. Caviling and carping do not not constitute an alternative research programme.[3] It is tempting to say that the results are now in, but this errs on the side of caution. There has never really been an alternative explanation of the empirical patterns established in *Hereditary Genius* (1869) worth taking seriously.[4] What followed was a meticulous elaboration of all aspects of the ideas established by Galton: pedigree analysis, adoption and cross-fostering studies, comparison of siblings, identical and fraternal twins, reared together and reared apart, and more sophisticated designs.[5]

As Robert Plomin reiterates in *Blueprint* (2018) the results of these gross-level comparisons, which do not directly analyze DNA, are overwhelming and unambiguous. Heritability is substantial for every human behavioural trait, including intelligence and every dimension of person-

[2]Galton 1865a; Galton 1865b.

[3]Kamin 1974; Rose, Lewontin, and Kamin 1984; Devlin et al. 1997.

[4]Galton 1869a; Galton 1869b; Galton 1875.

[5]Burks 1928; Newman, Freeman, and Holzinger 1937; Shields 1962; Juel-Nielsen 1965; Bouchard et al. 1990; Neale and Cardon 1992; Plomin et al. 2005.

ality. This heritability *increases* with age, peaking in late adulthood. Earlier studies biased these estimates to the lower end by concentrating on children and adolescents, who have always been easiest to study.[6] A classic case of looking for the car keys under the street lamp. One should estimate at least 50% heritability for all behavioural traits, some higher, some lower. There is little difference here between physical and behavioural traits. In 2015 a comprehensive meta-analysis confirmed these findings, summarizing 17,804 traits from 2,748 publications including 14,558,903 partly dependent twin-pairs.[7]

The variation that remains is harder to explain. Identical twins do not turn out identically, though they are very similar. We now know that this is not caused by the environment shared by people when they are raised, that is by differences between families.[8] Indeed, whereas genes make people both similar and different, to the extent that they have the same or different genes, the 'environment' appears to only make them different, and not to make them the same at all. This varies over time: in adolescence shared environment shows some effect but it declines to nothingness with age. To the extent that relatives are similar, that is almost entirely due to their shared genes. Thus siblings are only moderately alike despite sharing a family environment. Of course, this only holds within the range of variation actually experienced by people in the real world. Which is what we can ethically study, as opposed to some imaginary world in which, say, all newborns are brain-damaged by hammer-blows to the head, or lobotomized. That is why we must examine the way the world really happens to be be, rather than the way it might be in the bizarre fantasies of a madman.

'Shared environment' is subject to misinterpretation. Emotionally, it invokes feelings about caring, stable home life, books to read, education, nutritious food, scarcity of gunshot wounds, and so on. It turns out that to the extent that differences actually exist on these dimensions, they do not have enough effect to explain differences in outcomes, though it is not hard to imagine extreme conditions where they might.

[6] A confusion that was taken as a feature in Devlin et al. 1997, who were obviously determined to find the lowest possible heritability estimates and still hit 0.4.

[7] Polderman et al. 2015.

[8] Rowe 1994.

In the same way, access to food does not explain why many Americans are fat and some are thin. We need food, as we need basic conditions for growth, but there are sharply diminishing returns. Massive doses of vitamin C do not, contrary to Linus Pauling, do much once you have a minimal dose, and those not pressed into the Royal navy get enough fairly easily, even unconsciously. Nor is it the case that this common sense of environment is non-genetic. Firstly it contains copies of an individual's genes, carried by parents and relatives, and all the other genes. Secondly it is to a significant extent actively selected and constructed by those genes. This was obvious to the cyto-geneticist Cyril Darlington in the 1950s when he wrote *The Facts of Life* (1953), later republished as *Genetics and Man* (1964).[9] To Darlington, nature not only outweighed nurture, it completely dominated it since it helped to construct it. Though Lionel Penrose and other ardent 'environmentalists' sneered at the idea, it has been validated and now *measured* by behaviour genetic studies. Plomin refers to this phenomenon as the 'nature of nurture'. Nurture is itself under genetic control. Genes seek out and help to make conducive niches for themselves.

It became undeniable in the 1980s, as results rolled in from Thomas Bouchard's standard-setting Minnesota Study of Twins Raised Apart, that 'shared-environment' had negligible lasting effect. (One should note that this study was *not* supported by the National Science Foundation.[10]) Efforts turned to understanding the rest of the variance, attributed vaguely to 'non-shared environment'. The search was plagued from the beginning by the ambiguity just noted: the word 'environment' is not well-defined. It encompasses absolutely anything, including random noise and measurement error. And indeed nothing turned up. Plomin reports that all the obvious suspects, such as unique peer groups and differential treatment within the family,[11] show no measurable lasting effect after 30 years of searching. Mitchell argues in *Innate* (2018) that we might have expected this from the beginning, since if these sorts of influences exist at all, they surely exist to some degree at least *between* families as well. A persuasive argument, though not with-

[9]Darlington 1953; Darlington 1964.
[10]Bouchard et al. 1990; Segal 2012.
[11]Harris 1998.

out objections: a niche *might* compose and amplify a very large series of small influences to something substantial, in a way that simple differences between families might not. But here one needs evidence rather than speculation, and the glass is not even partly-full, it is bone-dry. Plomin states that he recently gave up looking for these mysterious non-shared effects once he realized that the differences between twins which these effects represent are not consistent over time for many traits: the twins flip their scores when remeasured. Thus the effect is at least in part just subtle measurement error. Heritability estimates should be corrected upward to account for unreliability of the instrument, a position that many advocated from the beginning.

Both Plomin and Mitchell argue that developmental randomness may supply the rest of the missing variation, but whereas Plomin notes this in passing, Mitchell devotes most of his book to elaborating the argument from a neurological point of view. Since genes are instructions and not, contrary to Plomin's title, blueprints, they suffer from problems of fidelity in execution. To paraphrase Mitchell, you cannot make the same barbecued brisket twice. Even if identical twins have instructions which do their best to build a particular kind of human, random conditions and errors during their execution, and the subsequent steps needed to build not only proteins but cells and eventually the behaviour-endowing organ that is the brain, are unavoidable. These disturbances compose to produce different results. Because we have no realistic prospect of modeling them, it makes sense to think of these events as random, even if they might be the product of a deity with an odd sense of humour, and not just an inordinate fondness for beetles. For the purpose of the nature-nurture discussion, Mitchell prefers to chalk them up to nature. Certainly they have nothing to do with the 'environment' in any common sense of the word, because all such senses try to get at something systematic.

Now that large samples of actual DNA from individuals have been collected and sequenced, a feat unthinkable in Galton's day, more direct analysis of the influence of genes has become possible, opening up innumerable natural experiments. The number of subjects grows all the time, and the sheer amount of DNA collected from each individual grows too, as the cost of 'chips' for such analysis falls. We are no

longer limited to twin and adoption studies. Though these still make up most of what we currently know, we will know much more in the future. As Plomin explains, earlier attempts to directly identify genes with effects on traits, including his own, were frustrated by the small samples of DNA available, and the misconception that genes with large effects should be found. They were not, so after several failed attempts Plomin very nearly retired to a sailboat. One has visions of a *sea-org* for jaded behaviour geneticists.

It turns out that the individual mutations in genes which persist in the pool and are not selected out almost always have very small effects. Genome-wide analysis only picks up these effects in very large samples. At least 300,000 individuals are required. Added together, small effects can make up anything from similarly small to very large composite effects. It is no accident that intelligence appears to be normally distributed (strictly speaking, binomially). It very nearly is, with some skewing by the small number of harmful genes of major effect. Which is why IQ scores have long been normally distributed by design. This is just the venerable Central Limit Theorem in action. Nobody who absorbed Arthur Jensen's *Bias in Mental Testing* (1980) would have supposed that genes of large effect were implicated.[12]

Individuals can be scored by the extent to which they possess a set of additive mutations known from genome-wide analysis to be minutely favourable or unfavourable for a trait. Such scores are known as *polygenic scores*, or less exactly as *polygenic risk scores*. Though even the best of these scores now explain just less than 14% of variance on an outcome like educational attainment (a study led by Bouchard's successor at Minnesota, James Lee) that percentage will steadily grow with database sizes and whole-genome sequencing rather than sampling, and is far stronger than alternative predictors like socioeconomic status.[13] One intriguing result of these polygenic scores has been their implication that psychological disorders like schizophrenia and major depression are related in unexpected ways, because many of the same 'generalist genes' are implicated. Disorders usually described until now in

[12] Jensen 1980.

[13] Lee et al. 2018.

categorical terms must be reconceptualized as spectra, with polygenic scores predicting where on a spectrum people may lie. The cumulative effect of small predispositions appears to move some individuals over the edge into what is obviously a persistently disturbed mental state. This emphatically validates Galton's work in *Inquiries into Human Faculty* (1883), which investigated the prevalence and heritability of the tendency to see visions among otherwise sane people.[14] Which is to say people who would not be classified as schizophrenic, but were part of the way there.

One might say that, though there once was a 'debate' about nature versus nurture, it is now all over bar the shouting. Informed proponents of zero-heritability have scarpered. Which does not mean that the carnival barkers of *X-studies* departments have left the stage, just that technical criticism has vanished. The critics retain only sound and fury. The field itself has long moved on to calculating better and better polygenic scores.

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[14] Galton 1879; Galton 1883.

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