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Commentary: An explosion without a bang

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Consider a conflict between the following two accounts of the Kennedy assassination: (i) the Oswald theory (that Kennedy was killed by a guy named Oswald, who was born in New Orleans to those particular parents), and (ii) the conspiracy theory (that the whole thing was planned and carried out by the CIA and other government agencies). Now imagine counterfactually that, despite all the plausible evidence accumulated over the years and pointing to Lee Harvey Oswald as the culprit, it is eventually proved that he was not in Dallas at all on that fateful day and that the assassin was in fact his elder brother, Robert Oswald, Jr. Notice that the Oswald theory, the way it was described above, would strictly speaking still be true even under the new circumstances (because it only claimed that 'Kennedy was killed by a guy named Oswald, who was born in New Orleans to those particular parents'). Nevertheless, is it not quite clear that we would all think that those who had defended the Oswald theory in the past were badly off the mark and that they surely have some serious re-examining to do?

Something similar has actually happened in the nature–nurture controversy and yet, somewhat surprisingly, not much re-examining has been done. Please bear with me a little until I develop the analogy. Recall first that the term 'environment' in the heredity–environment debate has usually been construed very broadly to refer to *all* phenotype-affecting factors *except genes*. Despite this broad definition, however, historically the environmentalist accounts of differences in, say, intelligence or personality traits have typically focused on a much narrower range of variables, such as parental attitudes and parental characteristics, socio-economic status (SES), school quality, family situation, neighbourhood features etc.

The main reason for this narrow focus was that it always just seemed so obvious that these very variables (all in some way connected with the family one was raised in) must be among the most important influences on psychological traits. This seemed obvious even before doing any systematic research. For who would have doubted that, for example, having more intellectual stimulation at home or attending better schools must raise the children's cognitive abilities at least to a certain degree? Likewise, it seemed to make perfect sense to presume that parental violence towards children would tend to increase the probability of the children themselves later engaging in antisocial behaviour. The research appeared to confirm these intuitions because it did turn out that the children's intelligence in adulthood and their later antisocial tendencies, respectively, were indeed positively correlated with those antecedent conditions. True, complaints were heard occasionally that correlation is not causation and that the impact of these family-related environmental factors had not been conclusively established, but these grumblings were mostly dismissed as methodological quibbles that could not undermine the happy consensus between common sense and social science that these factors are trait relevant.

And then the unsettling empirical data started coming in. Specifically, if these family-related environmental factors really shaped psychological traits, one would have expected that genetically unrelated children who are adopted in the same family should be much more similar to one another than those who are raised in different families. But for some strange reason, the reality refused to cooperate. For many traits, the correlation between (biologically unrelated) adoptees in the same family turned out to be zero or very close to zero, particularly in adulthood. This outcome took everyone by surprise. Everyone would have initially assumed that living in the same home must make the children somewhat more similar. Everyone, that is, except those rigid, 100% genetic determinists—who, let us be frank, never existed anyway!

However astonishing this empirical discovery was (as it definitely was), it did not make a splash. Or, to mix the metaphors, it was like an explosion without a bang. The lack of reaction to such an amazing result is itself amazing. It is not just that this truly remarkable finding was not widely reported in newspapers, magazines or popular science publications. The event was also largely ignored in many relevant parts of psychology. No re-examination there, no questioning of the fundamental presuppositions, no paradigm shift.

It was, to return to our opening fantasy, as if people still kept blaming Lee Harvey Oswald despite the new evidence that exonerated him, or if they started blaming his brother but with no expression of surprise on their face and with no change in the old approach that had been leading them astray for years. Reverting to our real-world case, some just continued with the family-based explanation of psychological differences, completely unconcerned with the apparent empirical disconfirmation of their preferred aetiology, while others simply switched to defending the alternative non-family environmentalism pretending that there was no discontinuity with their previously held view of psychological development. In many circles, there was no willingness to recognize that a totally unexpected and potentially path-breaking discovery has been made: that, contrary to common wisdom, it was not the notorious suspect (family-related environmental factors) who was responsible for the environmentally caused psychological differences but that, rather, it was all the work of the mysterious and until then totally neglected, sinister 'brother' (the impact of environments *not* shared in the same family).

And it is not as if the news about the tectonic shift in the environmental causation was not communicated well. On the contrary, the most important messenger by far was Plomin and Daniels's target article about this, an article that was published in a widely read interdisciplinary journal[1] and followed by two dozens of expert commentaries as well as the authors' response. Later the behaviour geneticist David Rowe wrote an excellent and highly readable book about the limits of family influence.[2] Another source that, exceptionally, caught a lot of public attention is Judith Harris's book, *The Nurture Assumption*,[3] which also spread the news about the surprisingly low impact of family environments on children's psychology.

This claim about the low environmental impact should be first qualified and then potentially expanded. First, speaking about family-related environmental variables having no effect on intelligence or personality differences, this actually applies only to differences in adulthood and also only to the normal range of environmental variation. In cases of severe environmental deprivation there *are* negative psychological effects. But there is no symmetry here: the exceptionally stimulating and high-end positive environments do not bring a measurable advantage. Second, even in connection with environments that are not family associated, there is a possibility that these variables will not have any systematic influence of psychology at all, and that the environmental impact will consequently reduce to the hodgepodge of accidental, idiosyncratic, unpredictable and largely uncontrollable effects. If that happens to be the case it would be impossible to arrive at a coherent and theoretically satisfying account of psychological development. This possibility is what Plomin and Daniels called 'a gloomy prospect', a scenario that was later pursued in more detail in Turkheimer and Waldron.[4]

Plomin and Daniels tried to refocus the attention of researchers from shared to non-shared environments. They were aware that this would amount to 'a dramatic reconceptualization of psychological

environments', but their call had a strong empirical support and could not be dismissed easily. I cannot try to explain here why their message did not sink in nor why it often seemed that there was no awareness of the message at all. I will just give several illustrations of this remarkable phenomenon.

In a 1994 issue of a leading academic journal, *Child Development*, several distinguished scholars published articles in which they argued that it has been shown empirically that poverty has a negative effect on children's intellectual and personality development. This claim was later criticized[5] on the grounds that the causal conclusion about the impact of poverty (a family-shared environmental factor) was fallaciously derived without controlling for the possibility that genetic factors might be at least partly responsible for the effect. Rowe and Rodgers made a very straightforward and cogent methodological point that if the level of poverty (variable A) is correlated with the level of different psychological traits (variable P), we cannot legitimately infer that A is causing P if we also know (as it seems we do) that A is correlated with genetic characteristics (variable B). There are actually research designs that can help us disentangle the causal influences of A and B, and the objection was simply that the causal attribution was made prematurely and without using the existing safeguards. Although Plomin and Daniels's article pointing to the aetiological impotence of family-shared environments obviously presented an additional reason to tread very slowly here and not to jump to causal implications based on statistical data, the friendly methodological warning was not heeded and the critics' perfectly valid point was lost in the ensuing discussion that went off a tangent and largely ended in irrelevancies.

In an article about child abuse in a recent encyclopaedia about child development, we can read that 'children suffering multiple types of abuse or neglect tend to have a poorer outcome than children who suffer only one type or incident of abuse or neglect' and that other studies document similar 'long-term *effects*' of child abuse and neglect.[6] Again, the variation in children's 'outcomes' may well be due to the amount of abuse or neglect they suffered but for all we know it may also be the result of some genetic characteristics that the children share with their more or less abusive parents. We simply cannot choose between the two rival hypotheses so long as the only thing we can rely on is the mere correlation of the effect P with *both* A and B. The somehow 'intuitive' and 'natural' assumption that abuse is the causally operative factor cannot be favoured *a priori*. If anything, after Plomin and Daniels this assumption should be actually treated with increased suspicion.

Several years ago National Institute of Justice (which is the research, development and evaluation agency of the US Department of Justice) has issued a report on the so-called 'cycle of violence' hypothesis. It claimed that being abused or neglected as a child increases the probability of later arrest as a juvenile by 59% and for a violent crime by 30%.[7] The basis for this claim was a longitudinal study of two groups followed from childhood to young adulthood, one of the groups with recorded cases of child abuse and the other one without it, and with the two groups matched according to sex, age, race and approximate SES. It apparently did not occur to the authors of that study that despite the match the two groups could still differ on the presence/absence of the genetic predisposition for violence, which would be transmitted from parents to children and which would create the statistical association between early abuse and later violence, *without the former causing the latter at all*. I am not saying of course that this is true, but merely that this possibility could not be excluded and that for this reason the evidence as presented was actually worthless for trying to figure out what was going on. Many other scholars defending the cycle of violence theory are similarly methodologically cavalier in attributing causal force to a family variable,[8] again despite the fallaciousness and the *prima facie* implausibility of such claims.

The accumulated evidence putting into doubt the power of family-shared influences is sometimes resisted in a strange way. For example, in his recent book about intelligence the prominent psychologist Richard Nisbett responds to that evidence with the following derisive comment: 'By now, if you have children, you could be wondering why you spent good money to move to a more expensive neighborhood with better schools, or for that matter why you squander money on books and orthodontia, waste time driving them to violin lessons and museums, and drain off emotional energy holding your temper so as to set a good example'.[9] Notice first Nisbett's sophistical introduction of 'orthodontia' in the debate about *intelligence* differences between children. (Needless to say, no one ever claimed that the straightness of children's teeth was not affected by family-shared factors.) Second, parents can surely do something to improve some of their children's skills (say, by arranging violin lessons) but it is doubtful that they can thereby affect the relevant psychological traits (say, musicality). And third, contrary to what Nisbett suggests, parents do have an excellent reason to continue driving their kids to museums, buying them books, not losing temper with them, etc., 'even if they believe that all this will not shape their children's psychology'. The parents might think that the additional effort is worthwhile simply because in this way they would make their children's lives *more pleasant*.

Sometimes the lack of evidence of family-shared influences is just interpreted as an indication that these factors operate in a very sophisticated way. So Jerome Kagan says: 'Eleanor Maccoby, a colleague and a distinguished developmental psychologist, wrote that

the contribution of parental practices to children's personality cannot be viewed in isolation. Each parental behavior or parental personality trait is part of a complex system that in some respects is unique to each parent-child relationship'.[10]

But if each parental behaviour is unique, it would not make sense to talk about effects of different *types* of parental behaviour. Furthermore, how could the impact of parental behaviour be then studied at all? Actually its elusiveness starts to look a lot like the famous historical case of the luminiferous ether and the alleged empirical impossibility of measuring the Earth's speed relative to it. In that controversy, the scientists in the end dismissed the fairly counterintuitive idea that there was a 'conspiracy of nature' that made the Earth's 'true' speed undiscoverable. Einstein famously said that 'God is sophisticated but not malicious', meaning that nature does not use elaborate tricks to hide the existing effects from us. If ever more involved and complicated scenarios are necessary to explain why X did not manifest itself despite our many and various attempts to detect its presence, the time will come at some point to conclude, reasonably, that X actually does not exist. Ditto for at least some hypothesized family-shared influences on psychological differences.

When wondering about why the behaviour geneticists' message about the absence of family-shared effects often did not get through, different avenues of explanation should be explored. A possible contributing factor might be that the message was politically unpalatable both to the political left (e.g. because the non-existence of shared environmental effects would undermine any social reforms aiming to equalize people's abilities) and to the political right (because it would undermine the traditionalist's belief in the importance of family).

But occasionally we have also to blame the messenger. For in some cases the message was so toned down and emasculated that the full flavour and the exciting nature of the new discovery were not communicated at all. For instance, in a recent introduction to behaviour genetics we can read: 'However, many studies suggest that shared home environment does not do very much to make siblings resemble each other in terms of personality and actions. Each child turns into a distinct character who behaves in individual fashion, despite parents' efforts to raise all their children impartially and despite similarities in genotypes of the siblings'.[11]

First, the phrase 'many studies' is quite vague. It leaves the reader guessing whether there were perhaps many other studies with the opposite results. Putting 'most studies' instead of 'many studies' would be much more helpful and informative. Second, the statement that 'shared home environment *does not do very much* to make siblings resemble each other' leaves open the interpretation that home environment does make siblings similar to a certain extent but perhaps not very much (what is 'very much' actually?). But this interpretation would overstate the impact of home environment, because for key psychological characteristics (such as intelligence and personality traits) those sharing home environment show zero or near-zero correlation in adulthood. And third, the claim that 'each child turns into a distinct character who behaves in individual fashion' trivializes the momentous nature of the new piece of knowledge. It is *not* as if we have recently discovered that the children raised in the same home still have distinct characters and their own individualities. We knew that all along and this truism is not worth mentioning in a psychology textbook. Yet, the really important and surprising news—that the key differences between children's individualities cannot be traced to their home environments—happens either not to be reported at all or is treated as a wild and empirically implausible speculation.

In conclusion, what can be said about Plomin and Daniels's article? It is a classic paper that relatively early systematized and publicized the behaviour genetics results about the unexpectedly low influence of family-shared environments on psychological differences. It also gave rise to a number of new studies that tried to make sense of these findings. And yet in many quarters, where its relevance should have been specially appreciated, the paper was often ignored or misinterpreted, with its most important message sometimes being diluted to the point of triviality. So in contrast to other classic papers whose main points have usually been completely absorbed into the body of scientific knowledge and which are today read mainly for their historical importance, this article has a dubious distinction that even after more than 20 years its impact is still far from over.

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Commentary: Reading Plomin and Daniels in the post-genomic age

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If there were a holy grail of research questions for human behavioural scientists it would probably be the following: To what extent are our life outcomes due to nature and to what extent to nurture? We have watched the proverbial pendulum swing back and forth over the decades with respect to this question. Today, where it rests is unclear: on the one hand, scientists are being inundated with enormous amounts of genetic data that would seem to augur for an intense focus on the hereditary roots of behaviour. On the other hand, genome-wide association studies have failed to (additively) account for prior broad sense heritability estimates, sending human geneticists off onto a mystery novel plot in search of the 'missing heritability'. Explanations have ranged from the salience of epigenetic processes to the importance of higher order allele interactions. And, of course, it could be that those broad sense heritability estimates were wrong in the first place.

Where did we get our estimates of the heritability of social traits such as personality dimensions (e.g. the big five), IQ, delinquency and even earning power? They came from behavioural geneticists such as David Rowe and Richard Plomin. In this paper—'Why are Children in the Same Family so Different from One Another?'[1]—and elsewhere, Plomin and his co-author, Denise Daniels, argued for two basic methodologies to assess the degree to which population variation in social outcomes was explained by genetics (additively), by shared environment and by unshared environment: kinship studies (specifically monozygotic and dizygotic twin comparisons) and adoption studies. In the first of these methods, Plomin and Daniels argue that by comparing social outcomes among genetically identical twins (i.e. monozygotic twins who share 100 percent of their nuclear genes) with those from fraternal twins (i.e. dizygotic twins who share, on average, 50% of their genes, just like singleton siblings), we can properly estimate the genetic, shared environmental and non-shared environmental components of traits.

In the most naïve approach, genetic heritability is calculated as two times the difference between the intra-class correlations of identical and fraternal twins. However, as they acknowledge in their paper, much more complex structural models have been offered to account for various complications such as the fact that—as a result of assortative mating at the parental level—fraternal twins may share >50% of their genes. Likewise, they point out that the 'equal environments' assumption has been relaxed in many models (i.e. the notion that the environmental similarity for fraternals is the same as it is for identicals). That is, for the naïve calculation mentioned above, it is necessary to assume that the covariance between environment and genetics is zero. Put in another way, the simple estimation of heritability requires the rather heroic assumption that identical twins experience the same degree of similarity in environment as do (same sex) fraternal twins.