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Boomsma, D.I.

published in

Behavioral and Brain Sciences
1987

DOI (link to publisher)

[10.1017/S0140525X00055989](https://doi.org/10.1017/S0140525X00055989)

document version

Publisher's PDF, also known as Version of record

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citation for published version (APA)

Boomsma, D. I. (1987). Absence or underestimation of shared environment? *Behavioral and Brain Sciences*, 10(1), 19-20. <https://doi.org/10.1017/S0140525X00055989>

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way interaction with any other gene that happens to be heterozygous in either parent, our chance of exactly matching in effective genotype is halved. The differences are genetic, but not represented as such in the additive model, making them part of "that which (etc.)"; and they are at least not fully shared. The utility of refining the fractionation of "environmental" variance is therefore limited by the extent to which it might well not be environmental at all.

Given the same parental genotypes and the same potential distribution of the elements thereof, the a priori probability of one sibling having a given genotype will be the same as that for another of the same sex, regardless of the transmission mechanism. For all but the simplest modes of transmission, that number is small and its square very small.

Very early in our genetics course for medical students we attempt to focus their attention on the mechanisms by which genetic diversity among humans is generated and distributed, and to convey in our lectures some appreciation of its potential range, by some variation of the following:

Human gametogenesis selects one from each of 23 pairs of chromosomes in each parent. Any given child represents one of 2^{46} (about 70 trillion) possible choices of a normal set of chromosomes from those of the parents, assuming each chromosome comes to the child exactly as it passed from a grandparent to one of the parents. They rarely do that. Individual genes mutate, and members of chromosome pairs exchange parts with each other before being sorted into future gametes. This recombination averages in excess of one exchange per chromosome arm, the longer arms having more.

Recombination of genes between chromosome homologues increases potential genetic variation. Each point on the "average chromosome" at which recombination might take place doubles the base of the exponentiation. Given even one point on each chromosome arm (two per chromosome) at which recombination might occur, the number of available structures for each chromosome increases from two to eight, and potential genotypes from 2^{46} to 8^{46} . This is more than the total of human gametes ever fertilized, and still a drastic underestimate. The children of unrelated parents have more differences from which to choose.

It seems at least as reasonable to consider the observed range of sibling differences anomalously narrow as to believe it oddly broad. Perhaps many of the possible combinations are phenotypically equivalent. The extent of euploid prenatal mortality, plus postnatal lethals, indicates that perhaps as many as half are forbidden. The potential variety, even between siblings, remains astronomical. I am not satisfied that current linear genetic models account for anywhere near all of it.

Absence or underestimation of shared environment?

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How different children from the same family can be is well illustrated by the famous Mitford family. The six sisters from this upper-class English family were brought up in the twenties and thirties under very similar conditions: Not allowed to go to school or to have friends outside the family, they were all educated at home. Two sisters became convinced fascists. Unity lived in Germany as a member of Hitler's inner circle until the war broke out and Diana married the head of the British Union of Fascists. Of the other sisters, two did not seem to have any interest in politics, Nancy flirted a little with socialism, and Jessica became a devoted member of the American Communist Party. In her own account of these events Jessica Mitford (1977, p. 24) asks: "What propelled us in these different directions?" Her answer is "the Zeitgeist of the thirties." And in "Unity

Mitford: A Quest," David Pryce-Jones (1976, p. 4) writes: "Out of this childhood . . . the one evolved as a fascist and the other as a communist: one experience, but two outcomes."

Plomin & Daniels's (P&D's) target article is an important contribution towards understanding such differences among children of the same family. They have performed a useful service in demonstrating that behavior-genetic methods can be used, not only to estimate genetic variance, but also to partition environmental variance into a part shared by family members and a part unique to each individual. Their discussion of research on psychological characteristics can be supplemented with other examples: The importance of nonshared environmental factors is also evident in physiological measures such as lipid and lipoprotein levels (e.g., Namboodiri, Kaplan, Heuch, Elston, Green, Rao, Laskarzewski, Glueck, & Rifkind 1985) and blood pressure (e.g., Iselius, Morton & Rao 1983).

When various measures seem to show such a consistent picture of large unique influences and absence of shared family environment then either the phenomenon is real or there is a chance that our methods somehow underestimate the importance of shared environmental factors. One crucial assumption in behavior-genetics research is that all participating groups represent a random sample of all genotypes and environments present in the population. If nonparticipation is spread along the entire range of family environments, no problem arises. If certain groups are less likely to participate, however, then the effects of shared environment will be underestimated. In general, because children are adopted by parents of above average IQ and socioeconomic status, there is a fair chance that adoption studies are biased against detecting the influence of shared family environment. Alternatively, the absence of shared environmental effects in the American population may be caused by the large degree of uniformity of the environment produced by the schools and other public agencies (Woodworth 1941, in Scarr & Carter-Saltzman 1983, p. 219-20). In his discussion of a French adoption study by Schiff, Duyme, Dumaret, Stewart, Tomkiewicz, and Feingold (1978), Willerman (1979) also suggested that the standard deviation of environments sampled in American adoption studies may be too narrow to provide a test of social class environmental effects. Schiff et al. compared adopted children reared in high socioeconomic environments with their own full or half siblings who were brought up by their true mother in a lower social class. Adopted children had higher IQ (110.6 versus 94.7) and less failure in school (13% versus 55%). This contrast is close to that in the general population between children of upper-middle-class parents and unskilled workers. Schiff et al. emphasized that the adopted children and their own siblings are biologically equivalent so that the contrast between them is mainly of environmental origin.

Even if nonshared environmental factors are important in explaining sibling differences, nonshared environment is not the only part of the answer to the question posed in the title of P&D's article. Only for differences between identical twins are nonshared environmental factors the sole explanation. Jinks and Fulker (1970) showed how genetic variance may also be partitioned into genetic effects between and within families. If the behavior we study is influenced by genetic factors then the other part of the answer is genetic factors that are not shared by family members.

Finally, P&D's question of whether most nonshared variance is systematic or specific may be answered by multivariate behavior-genetics methods such as those developed by Martin and Eaves (1977) and Fulker (1979). Martin and Eaves found, in their analysis of monozygotic (MZ) and dizygotic (DZ) twins on five of Thurstone's Primary Mental Abilities, that most nonshared environmental variance was specific. That is, the environmental factors not shared by family members were also specific to different mental abilities. In contrast, the influence of shared environmental (and genetic) factors was more systematic in that they had a general influence on all abilities. Fulker (1979)

provides a multivariate analysis of specific cognitive skills. According to his analysis of Loehlin and Nicols's large twin study of the National Merit Scholarship Qualifying Test, social environment appears to be very important in cognitive development. His analysis also suggests that shared family environmental effects are of a similar nature, whatever cognitive skills are involved, and that nonshared family influences exert little general effect. In the same article Fulker also carried out a multivariate genetic analysis of Taubman's twin data on schooling, occupation, and earnings. In this example, the general nature of shared family environment was even clearer. Moreover, this example showed that genetic and shared environmental factors that influence schooling subsequently influence adult occupational status and income. Nonshared environmental factors had little later influence. However, large independent genetic and nonshared environmental effects played the major role in explaining later differences in occupation and income.

Evolutionary hypotheses and behavioral genetic methods: Hopes for a union of two disparate disciplines

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Plomin & Daniels (P&D) present one of the most startling and important findings to emerge from behavioral-genetic research in the past decade – the pervasiveness of nonshared environmental influences on personality, psychopathology, and cognition. The finding is startling because nearly all current theories of environmental influence emphasize, implicitly or explicitly, environmental factors shared by children within the same family, such as socioeconomic status or common parental child-rearing attitudes, and these factors appear now to be of little causal import. The finding is important because it remains one of the few clues we currently have about how environments influence dispositions.

Because of its recency, little theoretical guidance yet exists to facilitate the empirical search for systematic sources of nonshared environmental influences. Recent work in evolutionary biology, however, points directly to specific hypotheses that can be tested with behavioral-genetic methods. This commentary will address a few of these. The comments are meant to suggest the possibility of a fruitful union between two biologically oriented disciplines that have developed in apparent isolation from one another: *behavioral genetics*, which has developed powerful methods that can be applied to nearly any domain and are therefore largely content- and theory-free, and *evolutionary biology*, which has proven a fertile source of hypotheses but, at least with humans, has not developed powerful methods to test these hypotheses.

The following list provides some evolutionarily based hypotheses about systematic sources of nonshared environmental influences that could be tested with behavioral-genetic methods:

Hypothesis 1: *Parents will expend more effort and confer greater resources on those children that are best able to translate parental investment into reproduction.* Based on Trivers's (1972) theory of differential parental investment, strategies that ensure distribution of parental investment to offspring that can best convert such favoritism into gene copies will be selected (see also Alexander 1979). [See also Vining: "Social Versus Reproductive Success" *BBS* 9(1) 1986.]

Hypothesis 2: *Because optimal reproductive strategies typically differ for human males and females, parents will socialize their male and female offspring differently.* Specifically, males will be socialized to be more aggressive, hasty, and wanton in sexual conduct. Females will be socialized to be more circum-

spect, cautious, and coy in sexual conduct. Furthermore, males will be socialized to embody characteristics that females value highly in mate selection (e.g., ambition, industry, good financial status); females will be socialized to embody characteristics that males highly value in mate selection (e.g., frugality, physical attractiveness) (Buss 1985; in press; Buss & Barnes 1986; Symons 1979; Trivers 1972). [See also multiple book review of Symons's *Evolution of human sexuality*, *BBS* 3(2) 1980.]

Hypothesis 3: *Parental favoritism toward male or female children will interact with the socioeconomic status of the parents.* Specifically, males will be more favored by their parents in higher socioeconomic groups, whereas females will be more favored by their parents in lower socioeconomic groups (Alexander 1974; Dickmann 1979; Trivers & Willard 1973).

Hypothesis 4: *Characteristics that covary with paternity confidence (e.g., physical or behavioral similarity to the father; effectiveness of mate-guarding tactics) will partly drive differential parental investment (cf. Daly & Wilson 1982).* The more similar a child is to the father, the greater will be the father's investment in that child. The evolutionary rationales stemming from theories of paternity confidence (Alexander 1979; Hartung 1985) and of genetic similarity (Rushton 1984) yield this prediction.

Hypothesis 5: *Birth-order will sometimes determine parental favoritism.* Older children are sometimes better able to translate parental investment into gene copies and so will be favored under certain conditions (Alexander 1979). Major exceptions will occur with the last-born as parental investment need not be saved for additional or future children (Alexander 1979).

These hypotheses, drawn from the existing literature, represent a small sampling of differential environments implied by evolutionary concept. They remain to be operationalized. Their potential effects on differential behavioral development (e.g., self-esteem, ambition, industry, coyness, aggression, impulsivity, dominance) also remain to be examined. Additional evolutionary hypotheses will undoubtedly be generated, and many will prove to be misguided or false upon empirical scrutiny. But evolutionary theory does provide a rich theoretical perspective that can guide the search for systematic sources of nonshared experiences and their behavioral consequences. Evolutionary hypotheses can be tested with behavioral genetic methods. Drawing on each other's strengths in this way provides a step toward unifying these powerful scientific disciplines. [See also multiple book review of Kitcher's *Vaulting Ambition*, *BBS* 9(4) 1986.]

Genes and environmental factors in the determination of behavioral characters

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It is well established that the expression of all phenotypic characters, quantitative as well as Mendelian, depends to varying degrees on both genetic and environmental influences. In the last few years our information about the nature and functioning of individual genes has become much more concrete. Environmental influences can be controlled in certain types of experiments, such as studies of the growth of microorganisms and plants, and even studies of some developmental processes in animals such as the fly *Drosophila*. In some more general characters, however, the environmental and genetic factors involved in the expression of particular phenotypes are ill defined and difficult to analyze. These characters include fitness, life cycle characters, and behavioral traits. In the case of behavior, this is partly because the definition of environment is frequently negative. The twin design and the sibling adoption