## Individual differences or different individuals? That is the question

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I am unhappy about the approach used by Plomin & Daniels (P&D) to explain why single children in the same family are so different from one another. They use the traditional approach of behavioral genetics (BG). That approach fails to account adequately for particular differences in psychological make-up (Nyborg 1983; 1986). A better approach would be to use a person-specific analysis. Let me briefly illustrate how P&D happen to sit between two chairs and suggest a person-specific, double-chair solution to their problem.

P&D get the wrong answer to the right question for the following reasons. BG originated in quantitative and population genetics and abounds with valuable information from family, twin, and adoption studies about how much of the total phenotypic variance of a given trait can be ascribed to genetic and to environmental variance, respectively. BG methods are, however, completely unable to cope with individualized data. BG methods provide a heritability estimate, but that measure is neither informative - nor even a first step - with respect to pinning down causal agents in a particular child's ontogenesis. The dilemma of whether to calculate population averages or to study particular persons, having individual development in mind, is most certainly not new to psychology. From time to time it appears in the literature as the nomethetic-idiographic controversy or as the problem of clinical case studies versus experimental psychology. No one has yet succeeded in finding a solution to the painful problem of dealing simultaneously with two methodologically incompatible psychologies, and P&D try to have it both ways with their nomothetic approach.

Another problem with applying the classical BG approach in the present context is that people differ in susceptibility to various environmental influences, but the BG approach chosen by P&D averages out such differences. Some people go through life rather unaffected by environmental pressures. Other people have sensitive periods, and still others are abnormally sensitive all the time. P&D clearly miss this point of individuality by generalizing that nonshared environment is the most important source of environmental variance for personality, psychopathology, and IQ.

A third problem with P&D's BG approach is that it cannot cope with covariant development or "positive manifold" expression of clusters of cognitive and personality traits in individuals. Usually, cognition and personality are considered separately in BC analyses. But even if considered together, their relationship is dealt with statistically rather than in individualized, biological terms, or the connection is left unexplained by a reference to unknown genes interacting in unspecified ways. The lack of person-specificity in P&D's approach may be acceptable to some as a first step as long as they are interested in anonymous, average trends in a population. But even so, it can go wrong. P&D note, for example, that estimates of "gender differences account for only a small (1%-5%) portion of the variance of sibling differences in development. conclusion that gender is unimportant for psychological differentiation is clearly challenged by simple everyday observations of profound gender-related differences in prepubertal play pattern and temperament, of an astonishingly large and persistent bias in the educational and occupational choice repertoire, of the tremendous gender-related differences in the social power structure, and so forth. Knowing the gender is by far the best basis for predicting membership in engineering or helping professions. It is, by the way, saddening to see that psychological tests do not clearly reflect the easily observable gender bias

A final problem with the approach used by P&D is that of the "fallacy of negative proof" (Fischer 1970). Their logic is as follows. Shared environmental factors fail to account for similarities between siblings (low correlations for fraternal twins, etc.). Therefore, differences between siblings are caused by nonshared environmental factors. Nobody has illustrated this fallacy better than Lewis Carroll (1869): "I see nobody on the road," said Alice. "I only wish I had such eyes," the King remarked in a fretful tone. "Able to see Nobody! And at that distance too!"

In order to obtain the right answer to P&D's important question, we have to perform a person-specific analysis. Such an analysis starts with the single individual, not with an average, and is the only way to safely establish a genuinely general trend (Nybord 1974). We also need a more flexible and dynamic kind of analysis than BG allows in order to keep up with the complexity of the question. I suggest that we start by acknowledging our tremendous biochemical individuality, and then try to probe deep down under the skin of single individuals rather than satisfying ourselves with an outside look. To find the singular we must study the dynamics of how their genes and their environment influence their person – specific internal milieu and condition their unique differentiation. If we find enough people with similar dynamic characteristics we might have stumbled upon something general.

Let me give an example of how such an individualized analysis can run. Experiments show that exposure of an organism to gonadal steroids has profound effects on its body, its brain (McEwen 1983; Toran-Allerand 1984), and on the phenotypic expression of cognitive and personality traits (Nyborg & Nielsen 1981; Nyborg 1983; 1986). What happens is that the steroids selectively modulate sets of genes by influencing their protein production and thus "masculinize" or "feminize" the brain. Fetal secretion of, and differential receptor sensitivity to, gonadal hormones are partly under genetic control by the fetus itself, partly influenced by placental and maternal secretions, the latter again being under environmental influence in an intricate dynamic interplay that changes radically over time. Add to this that the outcome of the steroidal impact also depends on "sensitive" periods in neural development that are probably controlled by fetal genes other than those controlling the steroid output in addition to being partly under environmental control. The early, predominantly organizational effects of steroid hormones on the fetus later combine at puberty with activational effects of surges in plasma steroid values. All these effects contribute to individual variations and to person-specific covariation of bodily, cognitive, and personality traits (Nyborg 1984). The mechanisms *are* intricate, but it should be possible to study them at different levels of complexity.

The sex hormonal history of a given person has been found to be a better predictor variable for later psychological differentiation than is the karyotype, the genital type, or any particular environmental agent (Nyborg 1983; 1984; 1986). If this finding can be confirmed in future studies, it seems likely that the power of person-specific analyses of biochemical individuality to explain why people become different surpasses that of the statistically anonymizing, dichotomous gene-environment analyses.

Perhaps the time has come for a change of paradigm in the developmental study of cognition, personality, and psychopathology. Averaging individuals will no longer do. The last ten years of research in psychoneuroendocrinology suggest that different individuals and not individual differences are what matter most when we ask questions about how a particular child developed differently from another child. Moreover, similarities across different individuals, not average differences between them, are the stuff that general trends are made of.

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