Robert Plomin: *Genetics and Experience: The Interplay Between Nature and Nurture* (Thousand Oaks, CA & London: Sage, 1994. Pp. xvi + 189. ISBN 0-8039-5420-4 (Hb) US$ 38.95 UK£ 31.95; ISBN 0-8039-5421-2 (Pb) US$ 17,95 UK£ 12.95)

by **ARTHUR R. JENSEN**, School of Education, University of California, Berkeley, CA 94720, U.S.A.

This well-written and highly readable little book explicates the most recent status of fact and theory in what was once called the nature-nurture controversy. This picture, which has predominated in the behavioral genetics literature for at least several years now, is here well-organized and presented succinctly by one of the leading figures in the recent developments of this field. Robert Plomin, now at the Institute of Psychiatry in London, is perhaps the most prolific researcher in contemporary behavioral genetics and one of the most innovative. He writes with clarity and authority on the topics of this book, which have been the focus of his own pioneering research.

First, it must be understood that the quantitative-genetic analysis of behavioral traits concerns individual differences in a trait, not the development of the trait in a given individual. This has been a source of confusion in the popular media's presentation of the nature-nurture debate. From the beginning, among those who work in this field, it has been axiomatic that both heredity and environment are equally and totally important in the individual's development, as no organism can even exist without both nature and nurture, or heredity and environment. The legitimate scientific question at issue has always concerned the causal factors in individual differences in observable or measurable phenotypic traits and how these differences are affected by genetic and environmental influences. Phenotypic individual differences in a population are measured as the variance, or the mean squared deviations of individuals from the group mean. Quantitative genetics provides the methodology for partitioning the total phenotypic variance of a given trait into its genetic and environmental components, including their covariance (GE cov) and their interaction (G X E), or nonadditive contributions to the total variance.

Representing this problem in terms of "nature versus nurture" or as "nature or nurture" is of course sheer nonsense, and has always been so regarded, from Galton's day to the present. This is just one more of the many misrepresentations of the issue in the popular culture.

What, in fact, has evolved in this field, from Galton to the present, is a shift in causal formulations that might be characterized as moving from the conception of "nature and nurture" to that of "nurture via nature." It is this transition in theory and research on nature-nurture that is the subject of this book. Its title, Genetics and Experience, is most appropriate, because it emphasizes the individual's actively experiencing and selecting what the environment offers rather than being passively subject to environmental influences. Here the individual's environment is no longer viewed merely as a hand of cards dealt to the person, which the person then plays as best as possible given the person's particular genotypic propensities. Rather, the environment is viewed more like a big cafeteria from which

individuals make diverse selections that are compatible with their differing genotypes. Plomin contrasts these two views as instructionist (in which environment limits and shapes the individual's development) and selectionist (in which the individual selects from the environment only that which is most compatible with his or her genotypic propensities). "The essence of the selectionist argument is that what looks like instruction from the environment may be selection from built-in options" (p. 18). In other words, the organism is not a passive recipient of its environment, but seeks out and shapes the environment to suit its genotypic nature. It is not that just any environmental factors may be powerfully influential for a given individual, it's that different individuals have selected specific aspects of the environmental cafeteria that are most influential for them. Hence environmental factors that influence behavioral development largely operate to make children in the same family different from one another.

Plomin points out one of the important discoveries of the last decade, namely that "Many measures of the environment widely used in the behavioral sciences as indices of the environment show a genetic contribution" (p. 4). Moreover, some of the measures widely used in the behavioral sciences as indices of the environment are found to have some degree of heritability, that is, they reflect individuals' genotypes as much as they reflect the individual's objective environmental circumstances. A parent's treatment of a child, for example, is influenced not just by the parent's characteristics, but by the child's own genotypic tendencies. These ideas, originally, suggested in 1953 by the eminent Oxford geneticist, Cyril D. Darlington, were more recently formalized by Plomin and others in terms of genotype-environment covariance (GE cov), which comprises much of what is usually considered simply as environmental variance but actually involves the genes as well. GE cov refers to the fact that genotypes and environments have correlated effects; environmental influences are not entirely random with respect to genotypic tendencies; both may work in the same direction. Theoretically, and in some cases empirically, GE cov is analyzable into three components, passive, reactive, and active. The passive aspect is that part of the GE cov that is not directly caused by the individual's own volition or behavior; it is most prominent in infancy and early childhood, when the individual has relatively little control over the physical and social environment. It results from whatever genetic similarity exists between the parents and the offspring, since they share half their genes. For example, musical parents may provide a more musical environment, quite independent of their child's own behavior; but their child is also more likely to have inherited genes for greater musical sensitivity than is possessed by the average child and is therefore is more overtly responsive to musical sounds. Then there is the reactive component of the GE cov, whereby others in the child's environment react to its genotypic propensities. To continue the musical example, the parents, or others, notice the child's unusual responsiveness to music and therefore provide more of what attracts the child's interest. Music lessons are provided, teachers are enthusiastic about the pupil's progress, and the child's proclivity is further cultivated. By early adolescence the individual's autonomy and social environment have greatly broadened and the third component of the GE cov, the active aspect, now comes into full play. With or without encouragement, the individual actively seeks musical experiences, practices an instrument spontaneously, associates with musically talented peers, joins an orchestra, goes to concerts, buys recordings, and reads about music and musicians. Doing all these things to create a highly musical environment appears to "come naturally" to the individual and may even occur despite parents' efforts to discourage such consuming

interest. (Beethoven's father tried to impose a musical environment on all three of his sons; he succeeded only with one.) This is indeed the abstract biography of every musician.

The same principles can be observed in the development of individuals in every walk of life. Genotypic characteristics influence experience and thus are differentially amplified by the opportunities for experience afforded by the environmental cafeteria. Plomin explains the various analytic models available to behavioral geneticists for detecting the components of variance attributable to the passive, reactive, and active components of the GE covariance from studies of twins, siblings, and adoptions, and gives many examples of such studies.

Some of the empirically established findings in recent years have resulted in a rather surprising revision of our knowledge of the behavior-genetic analysis of certain well-researched traits, particularly individual differences in intelligence, or psychometric g, the general factor common to all types of cognitive performance that meet some objective standard of accuracy or proficiency. The IQ is a fair approximation to g, but also reflects other factors besides g, such as verbal, spatial, numerical, and memory. The g factor per se, which is identified by the factor analysis of a battery of diverse cognitive tests, is slightly more heritable than is IQ as measured by standard test batteries, such as the Wechsler Intelligence Scales. The total environmental variance (including GE cov) is typically analyzed into shared environment (environmental influences shared by children reared in the same family and differing between families) and nonshared environment (i.e., environmental influences that differ among children in the same family and are specific to each child). Both shared and nonshared environmental influences contribute to the total phenotypic variance, in addition to genetic factors. Whereas previously is was believed that, in intellectual development, shared environmental effects (or environmental differences between families) contributed the larger part of the environmental variance, more recent age-trend studies of genetically unrelated individuals reared together show that this is true only during early childhood. Between early childhood and maturity, the shared component of environmental variance diminishes from about 40% of the total phenotypic variance to virtually zero. Over the same period, the nonshared environmental (or individual differences within families) effects on IQ remain relatively constant at about 20% of the phenotypic variance. What increases markedly from infancy to adulthood is the genetic variance, or heritability. In other words, through individuals' genetically influenced selective experience of the environmental cafeteria, genotypic tendencies become increasingly expressed in phenotypic behavior. The most recent evidence from large studies of monozygotic twins reared apart is that the broad heritability of IQ reaches about 70% in early maturity and about 80% by late maturity. Similar trends are seen in the domains of personality and temperament, though to a somewhat lesser degree than for mental ability.

Plomin also points out that there are genetic factors that show up in some measures of the environment and that influence experience per se independently of any particular psychological traits, unless one can speak of some trait of general experience-seeking or sensitivity to the environment. Some individuals apparently are genetically more exposed and open to the effects of experience than are others, independently of other measurable psychological traits, such as the well-known ability and personality factors.

The main themes explicated by Plomin in terms of their methodologies and current empirical evidence can be summarized briefly in terms of seven hypotheses, only the last of which has not yet accrued empirical support but is currently under investigation: (1) Genetic differences among individuals contribute to measures of the environment. (2) The genetic contribution to measures of the environment is greater for measures of active experience. (3) The genetic contribution to measures of the environment is due in part to psychological traits. (4) Genetic differences among individuals contribute to differences in experience independent of psychological traits. (5) Genetic factors contribute to links between environmental measures and developmental outcomes. (6) Processes underlying genetic contributions to experience change during development. (7) Specific genes that affect experience will be identified. Plomin's book may leave some readers with the impression that all of the nongenetic variance in human traits is attributable to specifically identifiable causes and may therefore even be intentionally manipulable eventually. However, there appears to be a base level of unaccounted for nongenetic variance which is attributable to wholly random microenvironmental events. This microenvironmental variance was first recognized by R.A. Fisher as "random somatic effects of the environment." It was emphasized later by C.D. Darlington in terms of "biological noise" and unequal division of the cytoplasm in the earliest stages of the zygote's development and to which he attributed a large part of the observed physical and behavioral differences between monozygotic twins reared together.

Recent analyses indeed show that a large part of the variance within MZ twin pairs fits a model of random microenvironmental variation (Jensen, 1996). In any single case, it would be difficult, if not impossible, to identify (or to control) the specific microenvironmental influences responsible for the difference in a given characteristic between any set of MZ twins. The sample random nongenetic factors must also affect singletons in the same way. While the specific sources of random environmental effects are not identifiable in the individual case, there are many likely candidates, virtually all of a biological nature, such as mother-child incompatibilities due to immunoreactive factors during fetal development (e.g., the Rh factor), mother's health, age, parity, drugs, X-rays, childhood diseases, nutrition, and many other factors, each contributing a small random nongenetic effect to the phenotypic variance. General advances in obstetrics, immunology, health care, and nutrition are probably reducing this source of variance to some degree in the populations of industrialized countries.

Reference

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