Nascent Doubts May Presage Conceptual Clarity: Reply to Surbey

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Wahlsten, D. Nascent doubts may presage conceptual clarity. Canadian Psychology, 1994, 35, 265-267 (reply to commentary by M.K. Surbey).

Article:

It is difficult for anyone to acknowledge that an idea he or she has been leaching may be invalid. I faced this situation several years ago and, after a period of vacillation, concluded that heritability analysis, the dominant paradigm in behavioural genetics, was not merely weak at its foundations but actively misleading to the nonspecialist; hence, it ought to be relegated to the history of science along with phlogiston, penis envy and cold fusion. During a postdoctoral fellowship year with McClearn and DeFries at the Institute for Behavioral Genetics at the University of Colorado in 1968-69, I eagerly absorbed the lessons of Falconer and my mentors, who were foremost authorities on heritability analysis. These lessons were acquired easily because mathematically they were so similar to the classic rendition of the analysis of variance presented by Hays and Winer, the stans6cal gurus of psychology graduate students in that period.

Two events blunted the pleasure of the exercise. The article by Jensen (1969) extolled heritability and pointed to its practical application with the question: "Is there a danger that current welfare policies, unaided by eugenic foresight, could lead to the genetic enslavement of a substantial segment of our population?" (p. 95) What seemed innocent enough when used on the farm took on sinister overtones in political debate. Then came the study by Henderson (1970) showing how the estimate of the heritability coefficient was dramatically altered by rearing conditions in a manner that could not be explained by an additive model.

In a review of the genetics of animal learning (Wahlsten, 1972), I cited numerous heritability estimates but expressed nascent doubts. Further evidence of heredity-environment interaction accumulated in the literature and, as a speaker invited by Joseph Royce to participate in the 1978 Banff conference on Theoretical Advances in Behavioral Genetics, I found the earlier doubts giving way to forthright opposition (Wahlsten, 1979). Since then, the many direct demonstrations of control of gene action at the molecular level by environmental conditions have amply confirmed the earlier conclusions.

Evidently the author of' this commentary now has doubts ;about heritability analysis arid, while vehemently defending the practice, sees no future for it. This leads to a lack of coherence. It appears the commentator has turned up the heat too far, the sauce has separated, and we are presented with an unappetizing mix of technical misunderstandings seasoned with hyperbole.

Serious Complications

"The Intelligence of Heritability" does not erect a straw man by highlighting the additive model. Additivity of genetic and environ-mental components *is the conceptual foundation* of heritability analysis. The eminent theorist Sewall Wright, whose professional life spanned the entire history of quantitative genetics from 1917, began his exposition of the topic with this cautionary note: "The determination of the genetic component is simple enough if it can be assumed that the effects of genes ;are wholly additive in relation to each other and to environmental variations. A first rough approximation may often be obtained on this hypothesis, but there may be serious complications ..." (Wright, 1969a, p. 418) The foundation is not, as the commentator asserts, that $h^2 = V_G/V_P$; that formula is a conclusion from an algebraic derivation that requires P = G + E or some more elaborate

equivalent. There is a world of difference between a premise and a conclusion. Without additivity for an individual, heritability in a population is utter nonsense.

It is not true that the model of development in my Figure 4 and population heritability are unrelated because they pertain to different levels. Only if there are two developmentally separate causes adding to yield the phenotypic value of an individual is the phenotypic variance in a population equal to the sum of variances of the two components. I reject the model of an individual that is actually used to justify heritability calculations. It is this additive model which proponents of heritability must somehow defend. Of course, some authors pay lip service to heredity-environment interaction. As my essay states: "Many sources introduce the model with a G×E interaction term attached ..." Once they excise this dangling appendix, which almost always seems to happen in research with humans, they can-not thwart my criticism by claiming they did display an interaction term on page such-and-such and then proceed with a purely additive model. I am quite certain that most students and many of my colleagues for that matter, first at the University of Waterloo and now at the University of Alberta, do not understand the false assumptions inherent in heritability calculations. Are folks at Mount Allison University so much more sophisticated statistically? I am skeptical of this.

Several comments made about ANOVA make me doubly skeptical. The author lectures me to the effect that including a $V_{G\times E}$, term makes the model "both additive and multiplicative." Two things which are multiplicative cannot be additive at the same time. Furthermore, the expression G×E does not mean the two things arc multiplied; it simply means *any* departure from strict additivity, whatever its mathematical form. The ANOVA model is quite general and makes no claim about the kind of interaction that may be present in the data. The ANOVA devised by Fisher is not a model of development; it is a method for analysing a factorial experiment. It is a very good method if the research design is of that type and the sample size is adequate to evaluate non additivity. Heritability analysis also seeks to *separate variance into components* but it goes further and makes untenable assumptions about the biological relations between heredity and environment. It is possible and even wise to reject heritability while retaining ANOVA for one's research. Fisher himself (1951, p. 217) cautioned about "the so-called coefficient of heritability, which I regard as one of those unfortunate short-cuts which have emerged from biometry for lack of a more thorough analysis of the data." Heritability is based on fundamentally false assumptions of additivity but ANOVA is not. ANOVA does not drop the interaction term unceremoniously the way advocates of heritability usually do, and interaction is not in the least lethal for ANOVA. Indeed, when replicated genotypes of experimental animals are available to be reared in different controlled environments, a sophisticated ANOVA can illuminate gene-environment interaction in the context of a genetic analysis (e.g., Gebhardt and Stearns, 1992). Interaction is lethal for the simple-minded kind of heritability analysis that often appears in psychology texts. A multitude of facts demonstrates that heredity and environment are interdependent causes. Those who pre-tend this is not so and point to nonsignificant interaction terms to justify obsolete views take unfair advantage of the weakness of ANOVA to detect interaction and thereby mislead their audience.

RESPONSE TO SELECTIVE BREEDING

There are several other claims in this commentary that I cannot support. (a) We are told that heritability was "never designed" to help "understand the origins of individual differences." Surely the author could not be serious about this. (b) We are asked to believe that heritability "has broader implications concerning the amenability of traits to ... environmental influence ..." Here we observe what Lewontin (1991) refers to as `The vulgar error that confuses heritability and fixity ..." The truth is that many bona fide, totally heritable single genes render the individual *more sensitive* to environmental variation, e.g., the phenylalanine hydroxylase mutation in humans and the diabetic gene in mice. (c) The commentator expresses confidence in the ability of a heritability coefficient to predict the response to selective breeding. Here again, Sewall Wright was more circumspect, pointing out "The impossibility of long-range prediction on the basis of any practically obtainable knowledge of the genetic situation in the foundation stock ..." (Wright, 1969b, p. 563) and noting: "Realized heritability is usually less than the heritability calculated from the foundation stock." (p. 562) (d) There is also the claim that it is possible for "the genotype to mold its own phenotypic expression by seeking out or creating new environments." On the contrary, a genotype by itself can do nothing. Only an intact, whole organism can

interact with its environment, and the organism is not the isomorphic embodiment of its genotype. (c) Is *"Homo sapiens* ... the only species capable of significantly altering its own environment"? The very oxygen we breathe is an atmosphere created by species capable of photosynthesis (Kasting, 1993). Microscopic organisms and_ industrious worms form and till the soil, prey animals adapt to the presence of predators, and almost everything from caddis fly larvae to prairie dogs build shelter. It has been argued persuasively that behavioural change can precede and shape evolutionary change by modifying the physical or social environment in a wide diversity of species (Bateson, 1988; Gottlieb, 1991). (I) Genes are not the only things transmitted from parent to offspring. In a very real sense, even nonhuman animals possess culture or learned traditions and even phenotypes can be passed from generation to generation (Bonner, 1980; Cavalli-Sforza and Feldman, 1978).

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