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On Schönemann on Guttman on Jensen, via Lewontin

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On the whole, I find much more to agree than to disagree with in the commentaries of Roskam and Ellis and Gustafsson on Guttman's target article. Jensen speaks clearly in his own defense. Therefore, given limited space, I will focus my further comment chiefly on Schönemann's contribution to the present debate.

By way of preliminary, consider a thought experiment proposed by Lewontin (1970) over twenty years ago, in an early critique of Jensen (1969). Suppose two handfuls are taken from a sack containing a genetically diverse variety of corn, and each grown under carefully controlled and standardized conditions, except that one batch is lacking in certain nutrients that are supplied to the other. After several weeks, the plants are measured. There is variability of growth within each batch, due to the genetic variability of the corn. Given that the growing conditions are closely controlled, nearly all the variation in the height of the plants within a batch will be due to differences in their genes. Thus, within populations, heritabilities will be very high. Nevertheless, the difference between the two groups is due entirely to an environmental factor — differential nutrition. Lewontin didn't go so far as to have the one set of pots painted white and the other set black, but you get the idea. The point of the example, in any case, is that the causes of between-group differences may in principle be quite different from the causes of within-group variation.

Now, to our immediate concerns. Ask yourself, what will be the distribution of the plant heights, in Lewontin's (1970) experiment, when both batches of corn are taken together? Will it be normal? No. Within each group, the distribution may well be normal, if many genes affect growth, each having a small effect, but the combined population of heights will be bimodal, with each subpopulation normally distributed around its own mean. We can extend the example to envision several measures taken on each plant — height, maximum breadth, average leaf length, and so on, thus changing univariate to multivariate normality for each group around its centroid, but the overall distribution remains bimodal: the plants that were well-nourished and the ones that weren't.

What is the basic assumption with which Schönemann begins his mathematical derivation? That the pooled population distribution is multivariate

normal. That is, Schönemann has ruled out by assumption precisely the situation described above, the situation that Lewontin (1970) and others have judged to be of major interest — the possibility that between-group differences might be due to quite different causes from those responsible for within-group differences.

What Schönemann proceeds to do next, if I understand him, is to sort cases into two groups *based solely on their observed scores*, and then make the argument that the structure of the measurements within and between the groups will be the same. In terms of the original Lewontin (1970) thought experiment, what we seem to be doing here is raising genetically diverse corn under environmentally diverse conditions, thus yielding multivariate normality of measurements, and then putting white labels on all the large plants and black labels on all the small ones. This does not strike me as a very felicitous modeling of racial differences in the United States. For one thing, it implies that on average the plants with black labels will be genetically inferior to those with white labels. (They will have an average environmental disadvantage as well). For another, it suggests that racial membership is an outcome of an individual's development, rather than a function of his ancestry.

I don't suppose for a moment that Schönemann holds such beliefs about US race differences; quite the contrary, I would suspect. I'm simply making the point that mathematical arguments following from assumptions not solidly grounded in the facts of the situation in which one intends to apply them can be hazardous — or worse. This was my objection to Guttman's target article as well.

However, let's push Lewontin's (1970) original experiment a bit further. Suppose we take multiple measures on each plant, and factor analyze them, obtaining a g — in this case, a general growth factor. This factor emerges, let us suppose, because of dynamic biological interrelationships within plants, such that the plants that grow higher also tend to spread wider, have longer leaves, and so on. But it is at least conceivable that these characteristics will hang together whether the determinants of luxuriant growth are favorable genes or a favorable environment. If this is the case, the equivalent of "Spearman's hypothesis" will be borne out in Lewontin's experiment. That is, the plants in the well-nourished group will be taller, bushier, have longer leaves, and so on, because of an environmental difference between the groups, whereas the plants within each group vary in this manner wholly because of genetic differences that lead to differences in metabolic efficiency, or the like.

Thus "Spearman's hypothesis" could, in principle, hold in cases where the between- and within-group differences were due to entirely different causes, provided that those causes represent alternative ways of influencing a dynamically coherent system, the system that accounts for the pattern of relationships

among the measurements. This is not, I believe, the way most people have thought of Spearman's hypothesis.

I remind you, however, that this is a thought experiment. Saying that it *could* be this way does not mean that it *is* this way. Jensen's reply to Lewontin's (1970) original thought experiment could equally well be applied to this elaboration of it. Jensen (1970, 1973) argued that although Lewontin's example was possible in principle, it was unlikely that one would have such a sharp segregation of between- and within-group factors in the real world. One can readily find an average difference between US blacks and whites on some environmental factor presumed to affect intellectual performance, such as amount or quality of education. But it will almost certainly be the case that individuals within each of the two groups will also vary a great deal on such a factor. If the factor were really of overwhelming importance, why does the within-group heritability remain so high?

Artificial examples are easy, real life is harder, and although thought experiments (and mathematical derivations) have their merits, if one wants to know what the actual causes are of racial differences in intellectual performance, there is no substitute for direct research on the matter. Jensen, to his credit, has done a fair amount. There is plenty left to do. Some years ago, Lindzey, Spuhler, and I, in a book on race differences (Loehlin, Lindzey, & Spuhler, 1975), outlined a number of research possibilities. Most of them remain viable today.

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