A PLEA FOR PSEUDO-PROCESSES

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A PLEA FOR PSEUDO-PROCESSES*

BY

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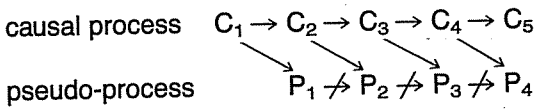
I

IS ALL EXPLANATION causal explanation? Puzzles abut barometer readings "explaining" storms and shadow lengths "explaining" flagpole heights make it attractive to think so. Wesley Salmon [1984] has endorsed this causal thesis. Not content to take the concept of cause as primitive, he has tried to provide a noncircular account of the difference between causal processes and what he calls "pseudo-processes." My interest here is not in the adequacy of his theory (on which see Sober 1986), but in the phenomenon he seeks to explicate. One way to test the causal thesis is to assess the explanatory import of pseudo-processes.

Consider two of Salmon's examples. A beacon on the floor of the Astrodome produces a circle of light on the ceiling. As the beacon is rotated, the circle of light traverses the ceiling. The moving circle on the ceiling is a pseudo-process. A car moves along a road and casts a shadow on the shoulder. The moving shadow is a pseudo-process.

Salmon's two examples have this in common: the stages of a pseudo-process are not related to each other as cause to effect, but are each effects of causes found elsewhere. The structure is as follows (arrows represent causal connections):



If all explanation is causal explanation, then earlier stages in a pseudo-process cannot explain later ones. A science intent on uncovering causes will discard pseudo-processes as mere shadows of the explanatory mechanisms to be found elsewhere.

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The ceiling image path and the car's shadow are obviously pseudo-processes. But sometimes it is a difficult and far reaching scientific discovery to show a pseudo-process for what it is. An example of the first importance was August Weismann's [1889] formulation of his principle of the continuity of the germ plasm. A parent's phenotype does not *cause* the phenotype found in the offspring. Rather, each is the result of the "germ plasm" (which we now call the genome) found in each, where parental genotypes cause those found in offspring. Phenotypes that run in families are pseudo-processes, contrary to Lamarckian doctrines about the inheritance of acquired characters.[1]

If earlier stages of a pseudo-process cannot explain later ones, then Weismann's discovery implies that my eye color is not explained by my parents'. I will not now take a stand on this isolated example; the stakes are really much larger, concerning as they do the status of an entire science.

Before Mendelism was rediscovered around the turn of the century, Francis Galton laid the foundations for the theory of quantitative inheritance. A fundamental achievement of this science is its characterization of a concept of inheritance[2]—h^2, called "heritability"—that applies to phenotypes and that can be measured in utter ignorance of their genetic basis (if any).

When the Modern Synthesis in evolutionary theory integrated Mendelism and Darwinism during the 1930s, it assimilated Weismannism as well. One therefore might expect the science of quantitative inheritance to have withered; if parental phenotypes do not cause offspring phenotypes, what explanatory use could there be for the concept of heritability? But quite the opposite happened; the science that Galton founded has developed into a robust and important part of contemporary theory.

The need for such a science is not far to seek. The genetic bases of many characteristics (one might even say—practically *all* traits) are unknown; yet, an evolutionary treatment of such characters requires a measure of inheritance. I now will say a little about how heritability is understood and about one role it plays in the theory of natural selection.[3] This will show, not just that heritability has heuristic or predictive utility, but that it can be explanatory.

Heritability measures the correlation between parental and offspring phenotype. Let's take height as an example. We represent the difference between the average height in a population and the average height of various parental pairs (the "midparent height") along the x-axis. These may have positive or negative values, depending on whether the midparent is taller or shorter than average. Along the y-axis, we represent the difference between the population average and the heights of various offspring. Each data point describes the height of a child and the average height of his or her parents. If taller than average parents tend to have taller than average children (and short parents tend to produce short children), the data points will form a football-shaped cluster, tilted Southwest to Northeast. We then draw a best-

fitting regression line through those points; its slope is the trait's heritability (h^2).

Let X_o be the average offspring height from parents with midparent value X_P; let X be the average height in the population. Then the graphical relationship just described takes the form.

$$(X_o - X) = h^2(X_P - X).$$

Again, heritability describes the degree to which offspring tend to resemble their parents. A maximum value of 1 means that the average height of offspring from a given parental pair will be the same as the midparent height. A minimum value of 0 means that a parental deviation from the mean is not expected to be reflected in the offspring's height; when $h^2 = 0$, the offspring average will be the population mean, regardless of the offspring's parents' heights.[4]

How does the concept of heritability figure in discussions of natural selection? For simplicity, let us imagine that selection works only on survivorship, not on fertility. That is, organisms have different probabilities of surviving from the zygote to the adult stage; the survivors then randomly mate and the various parental pairs have the same number of offspring. These zygotes then make their way to the adult stage under the same selection regime, randomly pair and reproduce, the process cycling through anew.

Just to simplify further, suppose that at a certain stage in the passage from fertilized egg to adult, *truncation selection* occurs; individuals taller than some fixed value are allowed to reproduce whereas ones shorter than that threshold are not. The difference between the average height among those permitted to reproduce ($X_{w,t}$) and the population average (X_t) is the *strength of selection*.

What will happen in the next generation, after the selected individuals of the first generation reproduce? How much of an increase in height will one observe between this generation and the previous one (censusing in both cases before selection occurs)? This difference, between X_{t+1} and X_t, is called the *response to selection*. It is a function of the heritability of the trait and the strength of selection:

$$(X_{t+1} - X_t) = h^2(X_{w,t} - X_t).$$

response to selection = (heritability) (strength of selection).

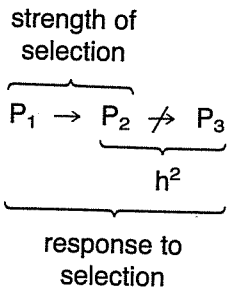
Clearly, a large response to selection demands strong selection pressure and a high level of heritability. In our example, the individuals who reproduce must be much taller than average and they must themselves tend to produce tall children.[5]

It is important to recognize that no mention of genes has occurred in this discussion. It is widely supposed that height is a trait influenced by many genes (as well as by the environment), but this plays no role in the model. Substitute any character you please in this story, and the prediction equation remains the same; if length of surname were used as the selected character, and if offspring tend to resemble their parents in this respect (as they surely do), the equation would apply. There need be no gene for surname length in this case nor for height in the other.

Plant and animal breeders use models like the one just sketched to predict and explain the changes they produce by artificial selection. And evolutionists discussing *natural* selection (i.e., in the wild, not in the laboratory or on the farm) also use this sort of approach, when they investigate the fitness consequences of a phenotypic trait whose genetic basis is unknown. A response to selection, whether large or small, may be explained by the strength of selection and the heritability.

Heritability is a measure defined on a pseudo-process; it looks at parent-offspring lineages and describes the connection of phenotypes at one stage with phenotypes at the next. Weismannism asserts that parental phenotypes do not cause offspring phenotypes; rather, each traces back to a common cause—the parental genotypes. I suppose this means the distribution of phenotypes among offspring is not caused by the distribution of phenotypes found in their parents.

The strength of selection describes the difference between the phenotypic mean in the parental generation before selection (P_1) and the phenotypic mean in the parental generation after selection (P_2). Heritability describes the difference between the parental phenotypic mean (P_2) and the phenotypic mean in the offspring generation (P_3). To invoke the first two of these in the explanation of the third is to invoke a pseudo-process.[6] This is illustrated below (braces drawing together the parameters that the relevant concept connects):



If all explanation must be causal explanation, then the introduction of a pseudo-process into the chain of explanation must break it. But nothing of the sort occurs here.

It might be suggested that the explanation just sketched is causal after all, since the strength of selection measures the degree to which differential mortality *causes* the mean height to increase in the passage from zygote to adult. I do not deny that selection is a cause; but the focus of my argument is on the second link in the explanatory chain, not the first. An explanation of P_3 that traces it back to P_2 and thence to P_1 is causal precisely when P_1 causes P_2 and P_2 causes P_3. But in the example, individuals' heights cause some to survive to become parents, but parent's heights do not cause the heights of their children.

The causal thesis may be salvaged by weakening it to the point of triviality. If all that is required is that the *explanans* provide "information about the cause," then facts about heritability certainly fill the bill. Heritability is now judged explanatory because it tells us that the causal mechanism of inheritance—whatever it was—must have been such that the relevant phenotypes had the degree of heritability they did. But this formulation is of no avail, if the theory of explanation is to show why barometer readings fail to explain the weather.[7]

A fall back position for the causal thesis might be that causal explanation is an explanatory *ideal;* this would allow that the theory of quantitative inheritance may be explanatory, while insisting that it is less so than an overtly genetic explanation. But even this more modest causal thesis is unsustainable. Not only are pseudo-processes useful when their underlying causes are unknown; in addition, pseudo-processes can play explanatory roles that causal processes cannot.

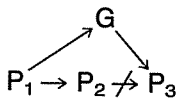
To see why, we must shift to a new *explanandum*. Rather than explaining a single trait's response to selection, let us imagine that we confront a set of such traits spread through very different species. These may differ in their genetic mechanisms; indeed, some may have no genetic basis at all. But these traits may yet have in common the fact that they exhibited the same response to selection. The explanatory task is to say what, if anything, these populations have in common that accounts for their similar behaviors.

Though no common element may be available at the level of genetic mechanism, it yet may be true that the traits had identical heritabilities and were subjected to selection pressures of identical intensities. It is a familiar idea that *supervenient properties* allow one to formulate explanatory generalizations that would be invisible from the point of view of finer-grained characterizations. The present point about explanation follows from the fact that a pseudo-process like parent/offspring chains of phenotypic resemblance itself supervenes on one or another causal mechanism of inheritance.

Successive readings on a barometer comprise a pseudo-process; so too do the successive phenotypes in an ancestor/descendent chain.[8] Few would say that a barometer reading at one time helps explain its reading later on; but, if I am right, the theory of quantitative inheritance allows phenotypic

distributions among parents to help explain those found among offspring. How to explain this difference?

The following answer elaborates an idea suggested to me by Ellery Eells. Heritability describes a pseudo-process, but once Weismannian facts about the genome are interpolated, we can see a difference between parent/offspring correlation and the correlation of today's barometer reading with tomorrow's. If G is the distribution of genotypes in the parental generation after selection, we may flesh out the causal story as follows:



An *indirect* causal connection can be found between P_1 and P_3. The phenotypic mean before selection (P_1) is a causal contributor to the *genotypic* array after selection *(G)*. Selection for being tall effects a change in gene frequencies, because phenotype and genotype are correlated. What is more, these parental genotypes *(G)* causally influence the array of phenotypes found among the offspring (P_3). Mendelism describes how parental genotypes *(G)* produce zygotic genotypes, and then laws of development show how these yield the array of offspring phenotypes (P_3).[9]

Nothing comparable can be said about a series of correlated daily barometer readings. Not only does today's barometer reading not cause tomorrow's. In addition, there is no further causal factor that intervenes between today's reading and the one found the day after tomorrow that links these two as cause to effect.

What general lesson does this suggest about the explanatory value of pseudo-processes? Heritability remains a pseudo-process. It is just that the pseudo-process from P_1 to P_3 by way of P_2 has as its material basis the causal process from P_1 to G to P_3. Perhaps we should conclude that pseudo-processes are explanatory only if the events they link are also connected by causal processes. This may be on the right track, but it should be cold comfort to the thesis that all explanation is causal explanation.

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NOTES

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[1]A more detailed explanation of the difference between Weismannian and Lamarckian theories of inheritance is given in Sober [1984], Chapter 4.

[2]I say "a" concept, since this is not the only way to make sense of the nature/nurture distinction. See the discussion of the "norm of reaction" concept in Sober [1984], Section 4.1.

[3]My exposition will be drawn from Roughgarden [1979], Chapter 9. A more detailed discussion can be found in Falconer [1960].

[4]It is no a priori matter that h^2 must fall between 0 and 1. If tall parents had children who were taller still, and short parents had children who were shorter still, the slope would be greater than 1. And if tall parents tended to have short children, and short parents tended to have tall children, the slope would be negative. In these cases, the concept of heritability is not employed.

[5]So as to avoid the charge that some or all of these concepts merely describe without explaining, one might think of each as estimated from samples drawn from the population. I discuss this problem with respect to the concept of fitness in Sober [1984], Chapter 2.

[6]I also would suggest that the heritability of a trait can explain the resemblance of parent and offspring, but this may be less persuasive, in that it raises the spectre of dormative virtue explanation, on which see Sober [1984], Section 2.2.

[7]In Sober [1983], I argue that a nontrivial reading of the causal thesis makes it vulnerable to a style of explanation I call "equilibrium explanation."

[8]To make these fully parallel, it may be useful to think of the lineage as made up of uniparental organisms; otherwise, the "chain" is really a "net."

[9]The various laws of transformation pertaining to different stages in the life cycle are discussed in Sober [1984], Chapter 1.

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