



## Genetics, Epigenetics, and Flexibility: A Reply to Crozier

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GENETICS, EPIGENETICS, AND FLEXIBILITY:  
A REPLY TO CROZIER

Crozier (1992) criticizes me for “two major errors” (p. 219). One is to have distinguished between “naturalists” and “geneticists” since, as he points out, the two approaches can be pursued by the same person. This does not seem worth a quibble, except to insist on the point behind the distinction: Hamilton’s rule *has* been used in two different ways, one natural historical (as a decision rule tested by field studies) and the other genetic (as a predictor of allele-frequency change). Crozier confirms this point when he says (p. 220) that “Hamilton’s rule is a predictor of allele-frequency change and not a ‘model of gene expression’ *except in terms of studying the results of such change*” (italics mine). Studying the results of such [evolutionary] change—phenotypes, and the conditions of their expression—could serve as a definition of what naturalists (who may also be geneticists) do. I did not refer to either approach as “wise” or “foolish” (Crozier 1992, p. 219).

The other criticism has to do with my portrayal of genetic models. It is certainly correct and important to point out that not all genetic models of altruism (traits costly in terms of the performer’s fitness) specify or assume obligatory expression of alleles for altruism. Crozier cites many models postulating alleles with conditional expression or incomplete penetrance, and, although this is not mentioned by Crozier, I also discussed them (West-Eberhard 1987) in order to show why they are not necessary to explain the evolution of a population capable of conditional worker behavior. I regret the misleading statement (1988, p. 128) where I mentioned “a gene for worker altruism (that is, one whose bearers are obligate helpers)” as if that were the only kind treated by genetic models. But I wish to emphasize that this does not affect my argument that *all* models depicting genes for altruism, including those with conditional expression, may be irrelevant to the evolutionary establishment of conditional worker behavior in a population. If the epigenetic hypothesis for the evolution of the worker phenotype is correct, a gene for altruism need not be involved. Instead, helping behavior and sterility can occur as a side effect of selection for “selfish” traits like aggressiveness and group living whose mean benefits more than compensate their mean costs (including the cost of a certain frequency of sterility). Kin selection would further reward helping behavior in groups of kin but is not necessary to explain the spread of the underlying genes.

I worry that Crozier’s lengthy defense of quantitative theories in general will give the mistaken impression that I disparaged them in general. The epigenetic hypothesis (West-Eberhard 1987) challenges only one kind of genetic model (that

treating alleles for altruism) in only one context (the origin of social insect workers), and the challenge applies whether the model is verbal or quantitative.

Crozier's final paragraph, regarding "source laws" and "consequence laws" (from Sober 1984), points the way toward understanding how comparative study and phenotype-transition hypotheses relate to genetic models of evolution. As Crozier says, genetic models describe results of selection (consequence laws) for a particular kind of trait. Phenotype transition models like the one I proposed attempt to reconstruct the phenotypic changes that might have led (under selection in certain contexts) to the evolution of a novel trait. Such hypotheses, and the "source laws" they may illuminate for natural selection, make it possible to identify the kinds of genetic models (consequence laws) appropriate to describe a particular change. Accordingly, the "epigenetic" transition hypothesis suggests that ordinary models of selection without genes for altruism are sufficient to explain the evolution of worker behavior beginning with an ancestral workerless population. That is, a genetic model is inappropriate when it proves inconsistent with the source law best supported by comparative study (which is *necessarily* qualitative). (Neither phenotype-transition nor game-theoretic models should be seen as crude or facile versions of quantitative genetic models; each deals more gracefully than the others with a different aspect of evolution.)

What about the subsequent elaboration of the worker phenotype? A "flat" view of the phenotype (disregarding mechanisms of development) might, again, suggest that models for conditionally expressed alleles would apply. Analysis of the phenotype including its development, however, indicates that several classes of traits are involved: those regulating expression (e.g., sensitivity to external cues and response thresholds), "specific modifiers" (*sensu* Turner 1976) of the worker phenotype (traits controlled by the regulatory apparatus and expressed in workers, but not queens), and "nonspecific modifiers" (expressed in both castes). Only one of these (the specific modifiers) might require a model of a conditionally expressed allele for altruism. Altruism alleles affecting regulation need not be conditionally expressed, and a *general* model for an evolutionary increase in worker altruism would have to encompass both conditionally and unconditionally expressed alleles.

By now it should be clear that my use of "epigenetic" does not mean "nongenetic." I have never argued that "evolution may proceed . . . without genetic innovation" (Crozier 1992, p. 221), nor have I departed from a genetic definition of evolution, made explicit (West-Eberhard 1988, p. 126) to avoid misinterpretations of this kind. All of the evolved traits I discussed—for example, cyclic reproductive behavior, aggressiveness, and group life, and their pleiotropic effects—presumably reflect the genetic makeup of the individuals performing them. Indeed, it seems likely that even caste determination (by which some individuals end up as helpers and others as egg-laying queens) depends to some degree on genotype (e.g., heritable differences in aggressiveness, hormone titers, etc.), especially in relatively simple societies lacking extensive manipulation of the brood (which can overwhelm heritable variation). In this respect I would criticize models, cited approvingly by Crozier, in which "queenness strikes at random" (p. 219) with respect to genetic makeup. By ignoring epigenesis—the

regulatory architecture of flexible traits—such models disregard the kinds of genetic variation that permit adaptive adjustment of individual decisions and caste ratios. For evidence of genotypic influence on determination of conditional alternatives, see West-Eberhard (1989) and Hazel et al. (1990).

I hope that readers shocked by Crozier's citation (p. 219) of my statement that "eusociality can originate 'without genetic innovation'" (freely interpreted by him to possibly mean *evolution* without genetic change) will examine the original text (West-Eberhard 1988, p. 127). It discusses environmental change as one conceivable way (in a list including evolved, genetic changes) that conditional phenotypes can be triggered for the first time in individuals genetically predisposed to express them due to selection in other contexts. This is just old-fashioned preadaptation and genotype-environment interaction, seen at the dawn of an evolutionary transition. Until we can discuss the environment as building block and cue (not just an agent of selection) without being suspected of advocating some weird form of "nongenetic evolution," we will not progress far toward understanding the evolution of plastic traits.

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