LETTERS TO THE EDITOR

Phenodeviants and Genetic Homeostasis

October 6, 1960

To the Editor
Dear Sir:

Since Heuts in his Socratic dialogue on evolution (Archives de Philosophie, vol. 23, 1960) recalled Woodger's observation, that among biologists the term *mystical* merely refers to something one disagrees with, I have ceased objecting to being so labelled. But I still object to irresponsible distortion and attribution to me of hypotheses diametrically opposed to the ones I have offered for examination in my book, "Genetic Homeostasis." Granted, that by injudicious usage of such terms as "heterozygosity *per se*," and "obligate level of heterozygosity," I have provided infinite opportunities for quoting me out of context as espousing one or another view. When, however, explicit statements are to be found which leave no doubt as to what is meant, it seems to be a highly dubious procedure to interpret the terms, inadequate as they may be, *ad libitum*.

Thus a favorite target has been figure 7 on page 67 of the book, which is sometimes referred to as evidence of my failure to accept the idea that gene action is specific. Yet the discussion accompanying the figure distinguishes between specific gene action (or function) and interchangeable gene effects on the phenotypic level to which the diagram refers. Similarly, I have been represented as holding the view that balanced polymorphism is the situation to be found at all loci, in all populations of all species, at all times. On the contrary, I have repeatedly pointed out that no population can afford to maintain too many heterotic loci or blocks simultaneously. Furthermore, a concrete model of temporal succession of balanced polymorphisms is outlined in detail on p. 113 of the book.

The present remarks are provoked by the statement of Morton, appearing on p. 358 of the current volume of this journal: ". . . Lerner's mystical thesis that phenodeviants cannot in principle be referred to any specifiable set of loci, but represent the effect of too high a level of homozygosity *per se* (an hypothesis that would require that *all* types of phenodeviants be increased in affected individuals and their sibs). . . ." There seem to me to be three possible ways in which this assertion could have been made in the presence of reiterated explicit state ments on my part (e.g. on page 69 of the book) that the hypothesis advanced (whether it is right, partly right, or wrong) is that overly homozygous individuals are developmentally labile, but that the direction in which this lability will express itself, i.e. the particular type of phenodeviant which will show a rise in incidence under inbreeeding, is determined by the specific alleles present. Of the three possibilities, I am unwilling to accept deliberate misrepresentation nor failure of comprehension of what I wrote (even though, as noted above, I do plead guilty to providing far too many openings for being quoted out of total context). The remaining one is simply that Morton has not read the book he cites, or at least has not read it sufficiently carefully. This, I am sure, is not his loss, nor a matter of great significance in general. Yet, were it not for his failure to follow the common custom of verifying before publication what the thesis he discusses actually was, he would not, I assume, have constructed and then demolished a man of straw, under the curious illusion that this operation has something to do with proving or disproving the hypothesis I proposed.

I. Michael Lerner
Department of Genetics
University of California
Berkeley 4, California