

SHORT NOTES

Effects of disruptive selection with negative assortative mating

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Prout (1962) has called attention to what he calls a difference between my results in a disruptive selection experiment involving negative assortative mating, and those of others. Since his conclusion from my results differs from mine I feel it necessary to draw attention to the *similarity* between my results and those of the other workers referred to.

Prout states (p. 379) that his 'finding is to be contrasted with Thoday's (1959) results where the increased variance in his D⁻ line (. . .) was accompanied by an increase in additive genetic variance. In fact, reference to Table 10 will reveal that this result of Thoday's stands alone as the only case where the genetic variance was detectably increased by this mode of selection.'

By contrast I stated in my summary (item 2): 'D⁻ selection resulted in an increase of chaeta-number, some deterioration of developmental stability (homeostasis) as measured by sternopleural asymmetry, but little if any change of variance that could not be attributed to the correlation of variance and mean.'

It seems that Prout has attached weight that I clearly did not attach to the crude heritability estimates I gave. Reference to my Fig. 2 will show that only one of the D⁻ tests gave a response in the first generation of directional selection greater than the *mean* response of Dronfield. This is why, though I concluded that there was a real difference between my D⁻ and S lines, I was quite specifically cautious when comparing D⁻ and the Dronfield population from which it came saying, 'The D⁻ line *seems* (italics new) to respond more than the Dronfield stock to one generation of selection, but thereafter its response decreases so that after three generations of directional selection it has diverged no more and perhaps less than the stock'.

My experiment in fact produced results which seem to me essentially the same as those of Falconer & Robertson (1956) with which Prout contrasts them. The D⁻ and S results were in agreement with Robertson's (1956) theoretical demonstration that D⁻ selection should maintain gene frequencies and stabilizing selection should lead to fixation. I have since shown more clearly that D⁻ selection can maintain gene frequencies but have never claimed that it has increased genetic variance, though I believe that in appropriate circumstances it could do so.

Prout's discussion (p. 381) of my stabilizing line also seems questionable to me, where he argues that the loss of fitness in my line, if due to homozygosity, would have to be due to homozygosity at those loci under selection, i.e. those controlling sternopleural chaeta-number, and consequently implies that I have made 'the extreme assumption that homozygosity at any locus causes a decrement in fitness'. Since I am one of those who has argued *against* this assumption (e.g. Thoday, 1955) I may perhaps point out that, in a small population, rendering several sterno-pleural chaeta-number loci homozygous will render some of the linked loci homozygous, and that there is no need to suppose that deleterious homozygosity would have to be due to homozygosity of chaeta-number genes.

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