FURTHER DATA ON GENETIC MODIFICATION OF RUMPLESSNESS IN THE FOWL

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Introduction

We have recently described (1934) the inheritance of a mutant gene (Rumpless **Rp**) in the fowl which is responsible for changes in the structure of the caudal and synsacral regions. In our original material and in the experience of other investigators this gene behaved as a dominant. Fowls heterozygous for the gene lacked the free caudal vertebrae, one or two synsacral vertebrae, the fleshy rump, the tail feathers and the uropygial gland. This condition, which was likewise characteristic of the few known homozygotes examined, we referred to as complete rumplessness. The gene was shown to segregate normally, and both hetero- and homozygous embryos were of approximately normal viability.

After crossing with certain normal fowls a new class of heterozygote was observed in which all of the affected characters except the absence of synsacral vertebrae were modified toward the normal, although in varying degree. Such "intermediate rumpless" fowls showed some development of the rump, had tail feathers, caudal vertebrae (often fused), and some of them had uropygial glands, occasionally functional. The factors responsible for these modifications toward normal proved to be hereditary, and after selection and inbreeding among members of the modified stock, ratios approaching $\frac{1}{4}$ complete rumpless: $\frac{1}{2}$ intermediate rumpless: $\frac{1}{4}$ normal were obtained from *inter se* matings of intermediates; while from matings of intermediates by normals from the same stock there resulted chiefly normals and intermediates, with very few complete rumpless. This suggested that factors tending to produce normal development of the tail had been introduced from the normal unrelated stocks, and that in the presence of such factors the rumpless gene acted as a recessive. We supposed that such genes would be retained in normal stocks because of the protection which they conferred against abnormal development of the tail brought about by environmental vicissitudes of the sort exemplified by our "accidental" (non-genetic) rumplessness (Dunn & Landauer, 1925) and by Danforth's (1932) demonstration that rumplessness may be induced in normal fowls by subjection of early

embryos to fluctuating temperatures. The effects of such genes on the dominance of rumplessness could thus be viewed as incidental to their normal physiological activity, and their selective value (and hence their retention or accumulation in normal stocks) as due not to the risks of recurrent mutation as Fisher had supposed but rather to the insurance which they provided against abnormal development from all causes.

New Evidence

The evidence which we had obtained was incomplete in several respects, and we have endeavoured to remedy these defects by new experiments. These have been only partially successful, but since we shall not be able to continue our work on this problem we shall record the final data which permit decisions on three points.

(1) *Homozygosity of "modifying genes"*

We supposed that the genes modifying rumplessness toward normal were recessive and multiple and were transmitted both by intermediates and by normals of selected intermediate ancestry. "When complete homozygosity (of modifying genes) is reached, crosses of intermediates by normals of similar genetic constitution should segregate only for the intermediate and normal conditions and no completely rumpless fowls should be produced, i.e. complete rumplessness should have become recessive" (1934, p. 226). This test was made in 1935. An intermediate male was mated with normal sibs from the selected stock. The result was clear: the progeny consisted of 148 intermediates and 132 normals (adult descriptions). No complete rumpless progeny appeared. It is probable then that a sufficient concentration of homozygous "modifiers" had been reached to induce some normal development of the tail in every rumpless heterozygote. The same result might arise if a single dominant "modifier" were present in homozygous form in the intermediate male, but this is extremely unlikely: (1) because *inter se* matings of fowls which from their ancestry should be heterozygous for such a dominant (e.g. our mating 1933–1, Table IV, 1934) have not given the ratios of 3/4 intermediate to 1/4 complete rumpless expected on this assumption; (2) because of the gradual increase in the proportion of intermediates among all rumpless heterozygotes from 22 per cent in 1926 to 100 per cent in 1935. It is probable then that the modifiers are multiple and recessive and that many of them have become homozygous in the stock following selection

(2) *Homozygosity of completely rumpless fowls*

The second question left open was whether all complete rumpless fowls from *inter se* matings of selected intermediates are homozygous for the rumpless gene. This was required by our assumption that modification toward normal occurred only or chiefly in rumpless heterozygotes.

The test is not entirely decisive because of the increasing difficulty of classification as the intermediate class continues to approach more nearly to the normal. Eleven completely rumpless females (from *inter se* matings of intermediates) were tested by mating with a normal male from the same stock. There resulted seventy-nine intermediates and eighteen progeny classified as normal at 2–3 months of age. All except one of these "normals" had been recorded as intermediate or questionable at hatching. Three "normals" were dissected when adult. All had normal tail vertebrae, but each lacked one synsacrocaudal vertebra, as do the intermediates. This evidence, together with the observations at hatching, make it quite likely that the normals in this case were rumpless heterozygotes showing extreme modifications towards normal. Of the individual rumpless birds tested seven produced only intermediate progeny; four gave only intermediates and birds of the questionable "normal" or intermediate type (one gave twenty-three intermediates and two intermediate (?) and was certainly **RpRp**; one gave twenty-three intermediates and eight (?) and was probably **RpRp**; one gave seven intermediates, one intermediate (?) and one normal (?) and was probably **RpRp**; one gave four intermediates and one intermediate (?), an insufficient test). Although not decisive, the test shows that most completely rumpless fowls from matings of intermediates are probably homozygous **RpRp.** Complete rumplessness, which at the beginning of the experiments was characteristic of heterozygotes, is probably now expressed only in the homozygote.

(3) *Does modification occur only in heterozygotes?*

A related question was whether modification toward normal was confined to the heterozygotes. Our previous evidence suggested that this was so, since we obtained about 1/4 rumpless, 1/2 intermediate and 1/4 normal from *inter se* matings of intermediates. Two further generations of selection and inbreeding among the intermediates, however, have thrown doubt on the earlier assumption. The results of matings among intermediates during 1934 and 1935 are shown in Table I, compared with the results previously published.

The proportion of fowls with the rumpless gene which show the intermediate condition has increased steadily with increasing concentration of "modifiers". If the "modifiers" affect only the heterozygotes the limiting value of this proportion should be $66\frac{2}{3}$ per cent; this is definitely exceeded in the 1934 and 1935 results. The proportion of fowls inheriting the rumpless gene itself (homozygotes and heterozygotes together) has not changed significantly from the 75 per cent expected, showing that the viability of homo- and heterozygotes relative to normal has remained constant and thus cannot account for the changed proportion of intermediates. It is thus probable that some of the homozygotes have also been modified toward normal. The descriptions of most of the rumpless fowls during the last two years were based on external appearance and palpation only. Several fowls included among the complete rumpless in Table I were dissected and found to contain anky-

TABLE I

Results of matings between rumpless intermediates (from adult descriptions only)

Year	Rumpless	Rumpless intermediate	Normal	Total	% of intermediates among all rumpless	% with rumpless gene
1927–32	200	147	135	482	42·4	72·0
1933	62	152	74	288	71·0	74·3
1934	38	136	50	244	78·1	77·7
1935	17	124	54	195	88·0	72·3

losed and reduced caudal vertebrae. We believe that many of our apparently rumpless fowls have now some characters of the intermediate condition (remains of caudal vertebrae, etc.), and that complete rumplessness has practically disappeared from our stock, even in fowls homozygous for the rumpless gene. Coincident with this change has been a further modification of heterozygotes toward normal until it is now impossible to distinguish many heterozygotes from normals except by dissection and examination of the synsacro-caudal vertebrae.

The "modifiers" thus act both on hetero- and homozygotes. In similar concentrations they modify heterozygotes relatively more than homozygotes toward normal. In extreme cases they may even extinguish all external character differences between heterozygote and normal. Genes with such effects need not be regarded as specific modifiers of rumplessness or of relative dominance. They are apparently constituents of the normal gene constitution, tending to promote the normal development of an important part which is especially subject to variation

from different causes. No apparent effect of these genes on the normal type has been observed. They would be retained in the normal type because of their obviously favourable effect on development. Our previous evidence (1934) had shown that one of their effects was to render recessive such harmful mutations as rumplessness. It is now shown that their effects go beyond this, and tend to extinguish also the homozygous effect of the rumpless mutation. This may be most simply conceived as a by-product of their activity in producing the normal characters.

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