THE OPERATIONS OF A LETHAL FACTOR IN *APOTETTIX EURYCEPHALUS* (GROUSE LOCUSTS)¹

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This paper deals with (1) the discovery in *Apotettix eurycephalus* Hancock of an autosomal lethal factor linked with those for the dominant, elementary color patterns, (2) the approximate, uniform stage of embryonic development attainable by the homozygous lethals, and (3) the apparently increased viability and probably vigor of individuals heterozygous for the lethal over sibs homozygous for its normal allele.

Specimens of *A. eurycephalus* have been brought in from the regions of Tampico, Mexico, Houston and San Antonio, Texas, annually, or biennially, over a period of twenty-two years, and bred in the greenhouse laboratory. The accumulated genetic and cytological evidence seems to justify the assumption that the closely linked factors for the thirteen dominant, elementary color patterns are all located along one, the smallest of the six pairs of autosomes (NABOURS 1925, 1929; NABOURS and ROBERTSON 1933). It is to be shown in this paper that the factor for lethal has a definite place also on the first pair of chromosomes (see chart 1).

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THE DISCOVERY OF THE LETHAL

In the summer of 1929, two sisters, one with the dominant color pattern combination $YmfZ\Theta TG/O$ and the other $YmfZ\Theta TG/RK$, reproducing parthenogenetically in separate cages, gave 7 O/O females and 8 RK/RK females, respectively, and none of the ordinarily expected combinations $YmfZ\Theta TG/YmfZ\Theta TG$. Following these results, the other specimens of this stock were conserved, so that, in 1930 and subsequently, many matings were made which further definitely indicated the presence of a factor for lethal in a particular position on the chromosome bearing the dominant color pattern combination $YmfZ\Theta TG$.

The chromosome bearing the lethal, since it was marked by the genes for the color patterns, could be traced back through the pedigrees to a female carrying the factors $Ymf(le?)/(le?)Z\Theta$ in which there was a crossing over to produce the $YmfleZ\Theta$ (T and G , to complete $YmfleZ\Theta TG$, were added subsequently by crossing over). Each of these homologs, $Ymf(le?)$

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and (*le?*) $Z\Theta$, was further traced back to specimens collected at West End Lake, San Antonio, Texas, in October, 1926. The $Z\Theta$ had been paired homozygously several times in the laboratory without disclosing a lethal, but the Ymf had not been so tested. Therefore the lethal factor was probably brought in from nature linked with that for the Ymf color pattern. However, there had been remote opportunity for linking the lethal from other sources with Ymf by crossing over; or it might have originated by mutation at some time in the laboratory.

In the females carrying the lethal, crossovers have occurred between the locus, or possibly the extremely closely linked loci, of the color pattern genes B , H , M , Y and Ymf , on the left, and the locus, or closely linked loci, of O , R , Z , Θ and W , approximately 6.9 points to the right. In the 119 of these crossovers that were tested 70, or 59 percent occurred between the lethal and the locus, or loci of O , R , Z , Θ , W ; and 49, or 41 percent, between the lethal and the B , H , M , Y , Ymf locus, or loci. These crossings over therefore show not only that the lethal is on the same chromosome pair with the genes for the elementary, dominant color pattern, but that it is markedly nearer the latter group (see sample matings and chromosome map, chart 1).

THE EMBRYONIC STAGE ATTAINABLE BY THE HOMOZYGOUS LETHALS

Sufficient numbers of heterozygous lethals were bred to ascertain that approximately one-fourth of the offspring, as determined by the color pattern combination expectations, never appeared in the records which were made in the third to fifth, or even earlier, instars. However, it was not known how far the double lethal embryos developed, if development was ever initiated. Accordingly the batches of eggs were removed from the ground soon after the newly hatched offspring were observed to have emerged. It was quite easy to distinguish the shells of the hatched, from the unhatched eggs, and the former closely approximated the numbers of the newly hatched offspring in each case. From 3766 such eggs, 2652 offspring, and nearly the same number of shells, were counted, and there were 1090, or 28.95 percent, unhatched. The small discrepancy of 24 is attributed to errors in counting egg shells and offspring. From the group of non-lethal bearing, control matings, under similar breeding conditions, there were 1899 offspring, and approximately the same number of shells, and 102 or 5.07 percent, of unhatched eggs among 2012 eggs counted. Such unhatched eggs did not usually contain embryos, development probably never having begun. These data show then that the homozygous lethal is responsible for the failure to hatch of at least 23.88 percent of the eggs from the heterozygous lethal bearing parents.

Nearly all of the unhatched eggs of the heterozygous lethal parents

removed from the ground, after the viable sibs had emerged, showed advanced embryonic development. The pigmented eyes could be noted through the shells. After preparation, many of the embryos were removed from the shells, some of them preserved whole, and others sectioned. No irregularities in development, nor deformities, could be detected from a careful study of the whole embryos, and the stained sections of these homozygous lethal embryos, as compared with controls killed four or five days before time for hatching. In both controls and double lethals at this stage blastokinesis was complete, but the appendages were immature. The mesenteron contained a considerable mass of yolk and was thin-walled and the mesoderm around it was loosely arranged. The dorsal organ or "heart," the Malpighian tubules and the trachea were plainly visible. Very little muscular development was evident. The caeca of the anterior end of the mesenteron were still solid and only slightly differentiated.

It remains to be ascertained whether the embryos which never hatch develop normally until four or five days before time for emerging and then die suddenly, or the whole or part of the embryonic progress is slower so that they are incompletely developed and die at about the regular time for hatching. There is then the alternative that the double lethal embryos may develop more rapidly at certain stages and then slow down till they are found to be actually behind the normals at the time for hatching. It is not thought that the death of the double lethals occurs long before the time for emergence of the viable sibs, because the tissues are still in good condition, though they remain so for only a few hours thereafter. These questions are reasonably within the sphere of experimentation, and it is hoped that answers will be forthcoming.

THE STIMULATING EFFECT OF THE HETEROZYGOUS LETHAL

The presence of the lethal on only the one chromosome of the pair does not make any, at least striking, difference in the appearance of the individual. There is no sex-limited effect, as the sex ratio is normal. However, the tabulation of 9657 offspring of lethal-bearing parents, marked by the genes for the dominant color patterns, recorded in the third to fifth instars, revealed 6560 heterozygous lethals, and 3097 homozygous non-lethals. The former were therefore 122 individuals, or 3.91 probable errors, more viable, or above the expected ratio of 2:1. In 7607 offspring from parents one only of which carried the lethal, 3969 contained it, while 3638 did not. This comprised a difference in favor of the viability of the heterozygous lethals of 165.5 individuals, or 5.63 probable errors.

The question of control now arises, and we have concluded that there is possibly none available further than to check the heterozygous lethal with its homozygous normal allele, which is probably sufficient. However,

the following appended table indicates a similar comparison of the factors for the heterozygous *T*, *G*, and *K* color patterns with their respective homozygous alleles.

TABLE 1

The comparative viability of heterozygous lethals, homozygous non-lethal sibs, and controls.

TYPE OF MATING	NUMBER OF HETEROZYGOTES	NUMBER OF HOMOZYGOTES (+ / +)	DEVIATION FROM EXPECTED RATIO	$\frac{D}{PE}$
$\frac{le}{+} \times \frac{le}{+}$	6560	3097	122.0	3.91
$\frac{le}{+} \times \frac{+}{+}$	3969	3638	165.5	5.63
$\frac{T}{+} \times \frac{+}{+}$	6001	5985	8.0	.22
$\frac{K}{+} \times \frac{+}{+}$	4914	4781	66.5	2.003
$\frac{G}{+} \times \frac{+}{+}$	4382	4329	26.5	.84

Another set of data, including those of *T*, *G* and *K*, of table 1, was compiled at random from the record books. From 58,475 offspring of heterozygotes with dominant color patterns, or closely linked groups of dominants, mated with the normal recessives, or alleles, there were recorded 29,345 heterozygotes to 29,130 homozygotes, a difference of only 107.5 or 1.32 probable errors.

If these data are available as a control, of which we are in some doubt, then the increased viability of the heterozygous lethals over the sibs homozygous for the allele, is even more outstanding. Regardless of the controls in question, it seems that the lethal in the single dose significantly improves the viability, and that in the double dose it causes death at the stage 4-5 days before time for hatching.

A principle of rather wide implication and importance may have come to light through this discovery which may not be new, although we have been unable to find an account of a previous observation of exactly the same kind. It is conceivable that the effect of the lethal, however it operates, is to aid viability and vigor when there is not so much (only half) of it, and discretely to limit life to the stage 4-5 days before hatching when there is too much (a homozygous dose) of it. It may not be unreasonable to suggest that factors of this kind are sometimes responsible for hitherto unexplained variations in viability, vigor, and longevity in foetal as well as postnatal stages of domestic animals and man. Certain races noted for their marked vigor and viability, yet low in prolificity, may be thus bionomically influenced.

SUMMARY

1. An autosomal lethal factor in *A. eurycephalus* has been found to be located between the locus, or loci of those for the dominant elementary color patterns, *B*, *H*, *M*, *Y*, and *Ymf* and the locus, or loci, for those of *O*, *R*, *Z*, Θ and *W*, and somewhat nearer the former.

2. The embryos carrying the homozygous lethal develop apparently normally till within 4–5 days of hatching, and then invariably die.

3. The individuals heterozygous for the lethal survive better than their sibs homozygous for its normal allele. The effect of a double dose of the lethal is to cause death at a uniform embryonic stage, and the single dose stimulates in some way to greater viability and probably vigor.

4. It is suggested that such lethal factors may be quite numerous, some of them causing death, either pre- or postnatal, when they are in homozygous quantity, but giving increased size, vigor, and general viability when they are in heterozygous amounts, and that they are probably also present and thus operating in domestic animals and man.

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