GENETICAL SOCIETY OF GREAT BRITAIN

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STUDIES ON HETEROGENEOUS CLONES (HETEROCLONES) IN *STREPTOMYCES COELICOLOR*

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Some of the recombinant colonies which develop on selective media after plating spores from a mixed culture of two complementary auxotrophic strains of *Streptomyces coelicolor* turn out to contain a mixture of parental and recombinant phenotypes. The hypothesis is put forward that these heterogenous colonies (heteroclones) develop from plating-units containing a highly unstable heterozygous nucleus which undergoes segregation during the development of the colony. Some of the parental markers appear amongst the segregants with reduced frequencies, or are even lacking altogether. With this limitation, the genotypes and frequencies of the segregants are predictable on the basis of the linkage relations of the markers deduced from standard selective analysis. The observed deficiency of certain markers is interpreted as being due to hemizygosity of some loci in the heterozygous plating-units. Pre-zygotic elimination has not been critically shown ; it is evident, however, that extensive post-zygotic elimination occurs.

Genetic analysis of the heteroclones has confirmed the location of several markers in two linkage groups, and permitted the mapping of new mutations. The heteroclones provide a convenient complementation test, and allow the study of the fate of the zygotic nucleus in this highly organised bacterium.

COMPLEMENTATION BETWEEN ADENINE REQUIRING MUTANTS IN YEAST

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Mutants at the ad_1 and ad_2 loci of *Saccharomyces cerevisiæ* require adenine for growth and produce an intracellular red pigment. Of 173 mutants isolated, 63 were found to be at the ad_1 and 110 at the ad_2 locus. Tests for interallelic complementation have been made on a sample of 25 ad_2 mutants. Complementation is indicated by growth of the diploid on minimal medium and suppression of pigmentation. On the basis of complementation patterns 16 groups of mutants may be distinguished and a linear map of the locus can be constructed showing seven sub-units. No indication of complementation between ad_1 mutants has been found.

Back mutation studies on the ad_2 mutants show that there is considerable variation in reversion rates even between mutants in the complementation group. Preliminary tests indicate that these reversions are not due to suppressor mutations at different loci.

PURIFICATION OF GENETICALLY DETERMINED GLUTAMIC DEHYDROGENASE VARIETIES

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At the *am* locus of *Neurospora crassa* a number of alleles are known which affect the enzyme glutamic dehydrogenase. Of the primary mutant alleles, am^1 is associated with a protein very similar to the normal enzyme except that it appears to have no activity, while am^2 and am^3 are associated with varieties of the enzyme with very poor catalytic efficiency. Among revertants induced in these primary mutant strains, am^{2l} (from am^2), and am^{3a} and am^{3b} (from am^2) form somewhat more effective, but still abnormal and quite distinct, varieties of the enzyme. In addition complementation between am^1 and am^2, and between am^1 and am^3, produces still further distinct enzyme varieties.

It is now possible to isolate all these enzyme varieties in apparently pure form. Some account will be given of the properties of the purified enzyme, and the differences between the various mutant forms of it and the wild type.

THE INHERITANCE OF PSEUDOCHOLINESTERASE

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It is now known that the inheritance of low pseudocholinesterase levels is genetically determined. The work of Kalow and his collaborators has demonstrated that the low cholinesterase activity in the very great majority of cases is due to the fact that an enzyme other than normal cholinesterase is present. It is recognised by a greater resistance to inhibition by dibucaine.

Three phenotypes can be differentiated : normal homozygotes where the enzyme is inhibited to 80 per cent., heterozygotes where the inhibition is about 60 per cent. There have been reported two families in which this classification did not work and inherited low pseudocholinesterase activity seemed to be associated with a normal inhibition by dibucaine. A third such family is now reported.

Kalow suggested as a possible explanation that normal pseudocholinesterase activity might be controlled by a number of genes determining different rates. The flaw of this theory was that in that case individuals should be found with very low pseudocholinesterase activity but normal dibucaine response. One family has now been discovered which will be described, where the propositus had no pseudocholinesterase activity at all in her serum and where the two children, from a normal husband, and two siblings had abnormally low pseudocholinesterase values and normal response to dibucaine inhibition. The implications of this " silent gene " are briefly discussed.

THE MESOSOME: A NON-REPLICATING GENE-DETERMINED PARTICULATE FACTOR IN *PARAMECIUM*

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Mate-killer animals of stock 540 (var 1) *Paramecium aurelia* have previously been shown to contain two dominant genes (M_1 and M_2) either of which alone maintains growth of the cytoplasmic mu particles controlling the mate-killer phenotype. Evidence will now be given showing that the dominant genes act by controlling the production of certain other cytoplasmic factors, provisionally called " mesosomes ", one of which is sufficient to support growth of many mu particles, though about 1000 mesosomes may be present initially. On replacement of the dominant

genes by their recessive alleles, the mesosomes are gradually diluted out until by the 15th fission only about 7 per cent. of the paramecia still contain any. Following fission of a cell containing only one mesosome, the daughter lacking the mesosome rapidly destroys all its mu particles.

Treatment of living paramecia with ribonuclease results in elimination of all mesosomes, and thus indirectly of all mu particles.

THE NATURE OF *t*-ALLELES IN THE MOUSE

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" Mutation " of *t*-alleles in the mouse is the result of crossing-over in an abnormal chromosome region (Lyon and Phillips, 1959). A study of the " mutants " should therefore reveal whether the different properties of *t*-alleles can be ascribed to linearly arranged factors or depend on the whole length of abnormal chromosome. The properties of the allele t^6 include : modification of expression of dominant alleles at the *T*-locus, homozygous lethality, male sterility, abnormal male segregation ratio, and crossover suppression. Mutants derived from t^6 show that the lethal factor is not at the *T*-locus but is close to the nearby locus of *tf*, while the *T*-modifying factor shows no recombination with *T*. The allele t^{h7}, derived from t^6, decreases the expression of *T* rather than enhancing it. This suggests that it carries a duplication of the *T*-modifying factor, and hence that this factor has a gene activity which is reduced, but greater than half normal.

From this one may surmise that the fundamental chromosome abnormality in *t*-alleles is a loss of specific pairing combined with reduced gene activity. This would lead to crossover suppression, unequal crossing-over, and hence to duplications, deficiencies and the other observed properties.

RECOMBINATION AND RESPONSE TO SELECTION

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The work to be described involves location of the genes concerned with the responses to selection for high sternopleural chaeta number in the three lines dp 1, dp 2 and dp 6 (Thoday and Boam, *Genetical Research*, *2*, 161), which showed such remarkable parallelism of response. The techniques of chromosome analysis used will be described.

It has been shown that dp 1 is distinguished in the main by two high chaeta number genes between *h* and *eyg* on chromosome III, and present evidence suggests the same will prove true of dp 2 and dp 6.

These lines started from a population with 19 chaetæ per fly. dp 1 rose to 22 chaetæ before dp 2 and dp 6 were derived from it, and was then stable against natural selection and against 15 generations of back selection. The evidence suggests that at the start most dp 1 chromosomes were LL at the two loci, but that at least one was LH and one HL. The 22 chaeta level would be stable because the LL chromosome had been eliminated. Further response in each line would depend upon the formation of HH by recombination.

THE CHROMOSOMES IN CONGENITAL ANOMALIES OF INFANCY

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The chromosome constitution is reported in three newborn infants with congenital anomalies. One infant is clinically similar to cases reported as trisomic for

a chromosome of the 13-15 group. A second infant had congenital heart disease, horseshoe kidney, flexion deformity of the wrists and a peculiar facies. A third, an apparent male, had hypospadias, acute adrenal failure and was chromatin positive in both blood and buccal skin.

The relationship of these infants to others reported in the literature with congenital abnormality and unusual chromosomal findings, is discussed.

AN EXTRA CHROMOSOME WITH SATELLITE ON THE LONG ARM IN A FEMALE PATIENT

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It is almost universally agreed that all the acrocentric chromosomes in the human karyotype can be satellited. The satellites are seldom, however, observed in their maximum number, but, when present, have always been on the short arm of the chromosome. The karyotype of the present case, a female aged 13 with mental retardation and epilepsy, had an additional small acrocentric chromosome with the satellite on the long arm. Although, in the absence of meiotic observations, it is not possible to identify this chromosome with certainty, the simple explanation would suggest a pericentric inversion of No. 22 (Denver classification).

The parents and normal sister were investigated cytologically but no similar chromosome was found.