Zusammenfassung

Das Auftreten der sogenannten «AP»- oder «PL»-Kristallisationsreaktion in gelagerten Samen (Sperma) von drei Spezies (Kaninchen, Bulle und Mensch) wird beschrieben und mikrophotographisch demonstriert. Es wird versucht, eine Interpretation und Korrelation zwischen einigen der früheren wichtigsten Laboratoriums- und klinischen Beobachtungen zu geben.

Morphogenesis of Melanotic Tumours (pseudotumours) and its Genetical control, in three Wild Stocks of *D. melanogaster*

Melanotic masses are already visible during the 3rd larval stage, in the wild stocks tu-A_2, tu-B_3 and melanotic e 144. Their percentages in the adult stage are: A_2, 74·0% ± 3·296; B_3, 100%; melanotic e 144, 68·5% ± 2·659. The phenotype is almost the same in all stocks, and is fully viable.

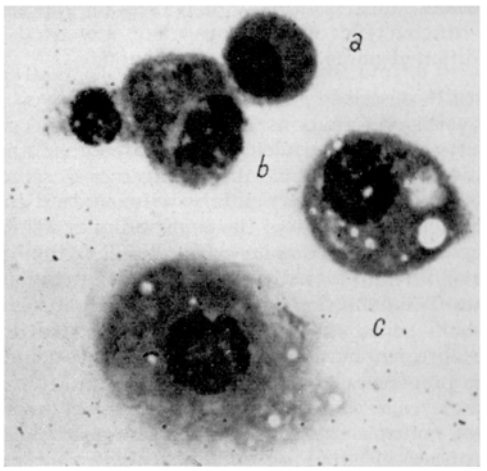

Fig. 1.—Haemolymph cells: *a* small basophilic cell; *b* medium-sized basophilic cells; *c* large cell (× 1500).

Morphogenesis progresses along the following steps, which are basically the same in all three stocks, with some minor differences:

(1) Release of the cells from the lymph gland, either as migration from the gland (tu-A_2, melanotic e 144) or as detachment of gland lobes (tu-B_3);

(2) Differentiation of a number of typical cells (large cells, see Figure 1) outside the gland, showing frequencies from 3% up to 10% or more;

(3) Formation of clumps of large cells, which are very thin and, lately, are embedded in melanine.

In two tumorless stocks $\left(\text{Varese and } \frac{+}{+}\ \frac{\text{CyL}}{\text{Pm}}\ \frac{\text{H}}{\text{SbMe}'}\right)$ the percentage of the large cells is lower than 3%, and (especially in Varese) the gland remains intact. A genetical analysis (replacing whole chromosomes by means of marked tumourless stocks) was brought about, which gave these results: the three morphogenetic steps mentioned above are under control of different parts of the genome. The three major chromosomes control (although with different intensity) both the differentiation of the large cells, and the behaviour of the lymph gland (no localization within the chromosome has been attempted); melanization is only controlled by the second chromosome (a short portion close to the left end in A_2, and another close to the right end in B_3).

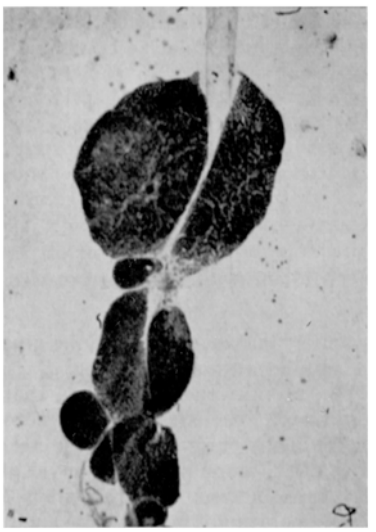

Fig. 2.—Entire lymph gland of Varese stock (× 100).

At least in melanotic e 144 it has been proved that the genes acting on melanization are different from those localized on the same chromosome, but acting on the other morphogenetic steps. Several loci with similar effect (polygenes) are located in the second chromosome (tu-B_3), to control melanine production.

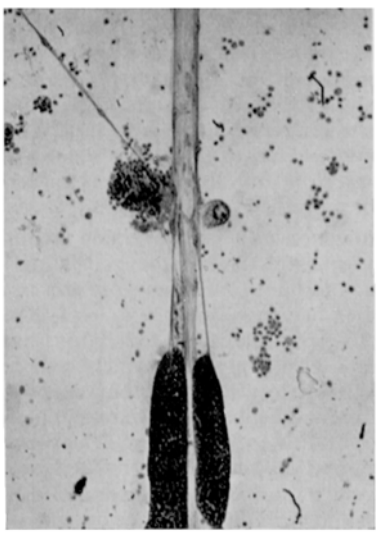

Fig. 3.—Entire lymph gland of A_2 stock (× 100).

To conclude, in the stocks studied so far, the melanotic masses are the end result of a complicated developmental procedure, corresponding to a similarly complicated system of multichromosomal factors.

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Riassunto

Gli autori descrivono la genesi dei tumori melanotici benigni (pseudotumori) in tre ceppi vitali di *D. melanogaster*, riconoscendovi tre tappe (disintegrazione o rottura della ghiandola della linfa, differenziamento fuori della ghiandola di cellule particolari e comparsa di melanina entro masse di tali cellule). In pari tempo constatano che queste tappe sono controllate da tre sistemi complessi di fattori, distribuiti nei tre cromosomi maggiori.

Priority in Gene-Conversion

When a scientist makes a discovery he gives it a name which often implies an understanding of the phenomenon, e.g., the atomic theory implies that atoms are unsplittable ultimate particles and the name has been retained in spite of the paradox. When several simultaneous discoveries of the same phenomenon occur, confusion reigns until the problems of priority are solved. If the phenomena are operationally indistinguishable the name applied by the first discoverer, no matter how paradoxical, has precedence. The subsequent 'discoverers' get credit for confirmation and if confirmation is precise and extensive, while the original discovery was based on scanty data, confirmation may bring more credit than discovery but discovery alone confers the privilege of naming the phenomenon. Scientific etiquette requires that every worker compare any supposed first discovery with similar phenomena previously reported to prove that the supposedly new phenomenon is operationally different. Deliberate failure to observe protocol very properly lays one open to the charge of plagiarism since it may involve an attempt to appropriate scientific property by improper means. Interpretations of others must either be refuted formally (point by point) or accepted with adequate reference if one is to justify his own position as a qualified worker. To ignore the interpretation of another worker in full knowledge is either deliberate plagiarism or a studied insult.

The phenomenon of gene-conversion comprises the interaction by genes in the heterozygous state resulting in change from dominant to recessive or *vice versa*. The term was coined by WINKLER[1] in 1930 to explain and describe nonreciprocal recombinations found by tetrad analyses of mosses and basidiomycetes which could not be explained by crossing-over. Recombination and crossing-over are different phenomena. BRIDGES[2] pointed out ... 'recombination of *linked* characters is a special use of the term (recombination) instead of its general use, which (does) not specify the method of recombination ... Whenever one uses the term crossing-over one refers now to the mechanism behind the recombination of the characters or of the genes for the characters... Crossing-over is something which occurs to the chromosomes at a particular point along their length.' Crossing-over is exchange between homologous chromosomes; recombination is the appearance of two nonallelic genes in combinations reciprocal of those present in the two original parents. Recombination is most frequently effected by the reassortment of chromosomes which generally insures random recombination of genes on different chromosomes.

RENNER[3–5] beginning in 1937 and RENNER and SENSENHAUER[6] confirmed WINKLER in a series of spectacular papers which showed that gene-conversion in the heterozygous state may occur in the soma although WINKLER considered it to be more common at meiosis. RENNER considered his work a confirmation of WINKLER's hypothesis. LINDEGREN[7] proposed gene-conversion as an explanation of phenomena of recombination not explicable by crossing-over in yeast which were shown by MUNDKUR[8] to be independent of other possible recombinatory mechanisms. LINDEGREN[9–13] *et al.* published an extensive series of papers corroborating the occurrence of recombinations not explicable by crossing-over or other conventional mechanisms. RENNER's papers were pointed out to LINDEGREN in 1956 during a discussion with R. GOLDSCHMIDT and LINDEGREN[14] hastened to credit RENNER with previous confirmation of the phenomenon. EMERSON[15], who is himself a specialist on *Oenothera*, attacked the concept of gene-conversion without quoting RENNER's confirmatory work on *Oenothera*. The purpose of the present communication is to point out that a phenomenon operationally indistinguishable from WINKLER's gene-conversion has recently been 'discovered' by many others, all of whom fail to quote any of RENNER's papers. Each of the 'discoverers' of gene-conversion invented a new and a different name for the phenomenon.

WINGE[16] described 'interallelic crossing over'. It is clear by this designation that WINGE implies crossing-over between different alleles of the same gene and that an equivalent term would be intragenic crossing-over. This is, however, an unfortunate paradox since it conflicts with the definitions both of the gene and of crossing-over. Crossing-over is defined as an exchange occurring between the *loci* of different (nonallelic) genes on homologous chromosomes. This definition has the peculiar advantage of defining both genes and crossing-over. The occurrence of reciprocal recombinations of phenotypes between characteristics previously considered to be the multiple effects of a single gene comprises the event which defines the supposed multiple effects of a single gene as the different effects of two different genes. Ambiguities due to nonreciprocal recombination can only be resolved by tetrad analysis. If tetrad analysis is unavailable no definitive solution is possible. Cross-overs can only occur between genes that are nonallelic since genes are indivisible by crossing-over by definition just as the atom is indivisible in intermolecular exchange. The parallel is even more precise, for genes, like atoms, may exist in different forms; changes in their finer structure may differentiate one gene (or one atom) from another but do not change a given

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