

it opens up the possibility that the localized crossover, in distinction to the Poisson distributed crossovers, may be effected by the mechanism proposed by SAX⁸, suggesting that the localized crossover may occur *after* a chiasma has been initiated resulting from strand tension at the crossing point of the chiasma. On this hypothesis visible chiasmata are assumed to represent crossing points which originated by crossingover (à la DARLINGTON⁹) and were not resolved by crossingover (à la SAX). Delayed crossingover occurring at these points might be expected to be unequal while crossovers occurring just as the chiasma begins to move might be equal. Thus the fundamentals of both DARLINGTON's and SAX's hypotheses may obtain simultaneously, the former explaining the Poisson distributed exchanges and the latter explaining the localized exchanges which resolve entanglements (resulting from Poisson distributed exchanges) that tend to accumulate in specific regions.

The localized crossover, spanning a definite but restricted region, accounts for the phenomenon interpreted as chromosomal interference. If we define two regions by the genes *A*, *B* and *C* (Fig. 1a) then, when the localized exchange occurs between *A* and *B*, it does not occur between *B* and *C* and *vice versa*. Under these circumstances the low frequency of concomitant recombination for the two regions results not from mutually interfering *pairs* of exchanges (as was originally assumed by MULLER and STURTEVANT), but rather as a logical necessity which follows from the spatial placement of a single event. It is important to point out that if crossingover occurs at the time and place proposed by DARLINGTON there is no physical reason why one exchange should inhibit the occurrence of another as was assumed in the original hypothesis of chromosomal interference. This present view does not encounter this difficulty. Thus, the discovery of the localized crossover (by tetrad analysis) provides a hypothesis explaining low coincidence in terms consistent with the occurrence of the negative chromosomal interference encountered in tetrad analysis and with the fact that localized crossovers cannot be detected by the classical (single strand) method in which the hypothesis of chromosomal interference was formulated originally. The present hypothesis makes it possible to generalize the concept of chromosomal behavior from *Ascomycetes* to *Drosophila* since it is not inconsistent with the high frequency of 2-strand double exchanges nor the negative chromosomal interference found in *Neurospora* which would have predicted high coincidence values in *Drosophila*.

E. E. SHULT and C. C. LINDEGREN

Biological Research Laboratory, Southern Illinois University, Carbondale (Ill.), May 14, 1957.

Zusammenfassung

Die bei *Drosophila* beobachtete Herabsetzung der Häufigkeit von Doppelaustausch innerhalb von benachbarten Chromosomenabschnitten lässt sich nicht nur durch die klassische Annahme einer gegenseitigen Interferenz zwischen benachbarten crossing-overs erklären, sondern kann auch durch das Vorkommen von lokalisierten crossing-overs innerhalb von bestimmten, engbegrenzten Chromosomenregionen bedingt sein.

sierten crossing-overs innerhalb von bestimmten, engbegrenzten Chromosomenregionen bedingt sein.

A Mendelian Gene for Albinism in Natural Cave Fish

As previously reported (ŞADOĞLU¹), the *Characimide* *Astyanax*, a normal eyed and dark pigmented fish which lives in overground waters, can be crossed with the pigmentless or \pm feebly pigmented blind cave fish *Anoptichthys* which is accepted to be its descendant or close relative. The F_1 hybrids are fertile. *Anoptichthys* is found in different populations which were described as different species and which are genotypically different from one another. In the F_2 generations of both Sabinos and Chica cave fish with normal river fish, recombination of the genes which are responsible for pigmentation and those for eye structure, is observed on a polygenic basis. The number of degenerative genes found in the Sabinos population which lives in the inner part of the cave system, are greater in number than those observed in the Chica population which is located nearer the overground river, Rio Tampaon. The Pachon population found at the innermost cave has even more degenerative genes than the Sabinos fish. The Pachon cave fish used as a grandparent to produce a F_2 generation was captured in nature some years ago. It carried, besides many other genes for pigment reduction, a gene which in homozygous condition prevents pigment formation even in the eyes. The F_2 generation, which results from the cross between Pachon and the overground river fish resulted in 26% (278) albinotic individuals having unpigmented, red eyes of the most different degrees of development from practically none to \pm normal. The body of these albino fish is completely pigmentless. The remaining 74% (787) of the F_2 individuals have dark eye pigment even when their eyes are of the most rudimentary type. The body colour of these non-albinotic individuals shows a series of gradations between the body colour of normally pigmented individuals and that typical for cave fish, the pigmentation of which has been much reduced. Thus, in these 74%, the number of polymeric genes present in each of the F_2 fishes controls the degree of melanophore formation. On the other hand, in the albinotic individuals the formation of pigment does not take place, because of the presence of an epistatic inhibitory gene pair. It is very interesting to note that such a great mutational step causing the extinction of all the melanine which up to now is known only in domesticated fish (*Salmo irideus*, *Xiphophorus helleri*, *Mollienisia 'sphenops'*, *Dermogenys pusillus et al.*), could be found in cave fishes under absolutely natural conditions.

P. ŞADOĞLU

Zoological Institute, University of Istanbul, June 6, 1957.

Zusammenfassung

Es konnte gezeigt werden, dass beim Höhlenfisch *Anoptichthys* – neben zahlreichen anderen degenerativen Genen – ein Gen für Albinismus vorkommt.

¹ P. ŞADOĞLU, *Copia* 1956, 113.

⁷ E. E. SHULT and C. C. LINDEGREN, *Genetica* 28, 165 (1956).

⁸ K. SAX, *J. Arnold Arboretum* 11, 193 (1930).

⁹ C. D. DARLINGTON, *J. Genetics* 31, 185 (1935).