COMPLEMENTATION AT THE YELLOW LOCUS IN DROSOPHILA MELANOGASTER

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AS a general rule allelic or pseudoallelic mutants when compounded produce either an intermediate phenotype or one equivalent to the lesser departure from wild type. Cases have been infrequently described where the compound of presumed alleles manifests a complementary phenotype, i.e. one more normal than that of either allele. By and large allelic complementation in Drosophila has been sporadic and has not seriously challenged the unitary concept of the gene. One noteworthy exception has been the complementary phenotypic effects associated with the various scute bristle (*sc*) mutants, effects which formed the basis of the step-allelomorph theory of genic subdivision. Although invalidated, step-allelomorphism has its current counterpart in the complementation alleles and complementation maps so thoroughly developed in Neurospora by GILES, by FINCHAM and by CATCHESIDE and their several collaborators.

EXPERIMENTAL RESULTS

Two general phenotypic classes of sex-linked, recessive yellow body color (y) mutants have been described in *Drosophila melanogaster*. One, typified by the original y mutant, manifests a yellow body and yellow bristle phenotype. The second, of which the mutant y^2 is typical, manifests a yellow body but black bristle coloration. STONE (1935) described a unique example of complementation at this locus. He observed that the mutant y^{35a}, a y-type X-ray induced in *In(1)99b*, produced when compounded to y the usual y phenotype but when compounded to y^2 resulted in a complementary, wild-type phenotype. The heterozygote y/y^2 is phenotypically equivalent to homozygous y^2. Unfortunately y^{35a} was lost before a more detailed study could be pursued.

A re-examination and extension of complementation among y mutants was made possible by the chance finding that the spontaneous mutant y^{Si} which occurred in the $In(1)sc^{S1}$ chromosome had complementation properties identical to y^{35a}. The complex X-chromosome $y^{Si}In(1)sc^{Si}$ $In(1)$dl-49 B v (B=Bar eye, v= vermilion eye color) obtained from PROFESSOR H. J. MULLER for use as a balancer chromosome was found, as listed in Table 1, to produce a near wild-type phenotype when compounded with y^2 but not to the other y mutants to which it was tested. Independently, FRYE (1960) also uncovered the complementary interaction of y^{Si} and y^2.

A second y type mutant of rather unusual origin, y^{59b}, was found to complement with y^2 and with none of the other y mutants to which it was tested as

TABLE 1

Phenotype of y *mutant compounds*

y mutant	y	y^2	y^{59b}	y^{3p}	y^{31d}	y^{50k}	y^{51g}	y^{60b}	y^{60j}	y^{Si}
y	y	y^2	y	y^2	y	y	y^2	y	y	y
y^2		y^2	y^+	y^2	y^2	y^2	y^2	y^2	y^2	y^+
y^{59b}			y	y^2	y^2	y	y^2	y	y	y
y^{3p}				y^2	y^2	y^2	y^2	y^2	y^2	y^2
y^{31d}					y^2	y^2	y^2	y^2	y^2	y^2
y^{50k}						y	y^2	y	y	y
y^{51g}							y^2	y^2	y^2	y^2
y^{60b}								y	y	y
y^{60j}									y	y
y^{Si}										y

tabulated in Table 1. The origin of y^{59b} briefly described previously (GREEN 1960) merits repetition here. Males of the genotype $y^2 su\text{-}w^a z$ ($su\text{-}w^a$ = suppressor of white-apricot, z= zeste eye color) irradiated with 5,000r X-rays were crossed to females homozygous for the complex X chromosome inversions sc^8 and dl-49 and the recessive markers y, w^a, v and f (forked bristles). Among approximately 150,000 F_1 females scored, one wild-type female was found. On progeny testing this female produced three types of males in approximately the following frequencies: 50 percent $y\ w^a v\ f$, 25 percent $y^{59b} su\text{-}w^a\ z$ and 25 percent $y^+\ su\text{-}w^a\ z$. Thus two events followed irradiation: the reversal of y^2 to y^+ and the mutation of y^2 to y^{59b}. Subsequent genetic tests established the presence of both $su\text{-}w^a$ and z in both the mutated X chromosomes. Insofar as can be determined by genetic tests, y^{59b} is devoid of any structural change. Crossing over between y^{59b} and the tightly linked $su\text{-}w^a$, about 0.2 percent to the right, occurs freely.

Of particular interest is the finding that the complementation potential of y^2 is confounded by the mutant *sc*. While compounds of y^2, derived from a variety of stocks, and y^{Si} or y^{59b} were without exception complementary, those of coupled $y^2 sc$ and y^{Si} or y^{59b} were noncomplementary and were indistinguishable from homozygous y^2. As a test of the specific influence of *sc* upon y^2, thirteen independent phenotypically complete and one partial X-ray induced reversals of *sc* induced in $y^2 sc$ X chromosomes (GREEN 1961) were employed. Where each y^2 complete *sc* reversal was compounded with y^{Si} or y^{59b}, the complementary phenotype was restored. Conversely, the compound of y^2 partial *sc* reversal with y^{Si} or y^{59b} was clearly y^2 in phenotype. The influence of *sc* upon y^2 was similarly demonstrated in triploid females. Females of the genotype $y^{59b}/y^{59b}/y^2$ although lighter in color than wild type were nonetheless distinctly darker than females $y^{59b}/y^{59b}/y^2 sc$.

Since in Neurospora there is a high correlation between complementation and recombinational (spatial) separation of mutants (CASE and GILES 1958, 1960), it is important to know whether pseudoallelism exists at the yellow locus. Two cases of apparent recombination between noncomplementary y mutants have been found. In one, a cross involving attached-X females of the genotype $y\ sc$

$w^a ec/y^2 su\text{-}w^a$ $w^{a4} spl$ (*ec*= echinous eye, w^{a4}= white-apricot[4], *spl*= split bristles) and heterozygous for the autosomal inversions *Cy* (Curly wing) and *Ubx^{130}* (Ultrabithorax[130]), a single *$y^+ sc\ w^a ec$* attached-X female occurred. A progeny test established the genotype of this female as *$y^+ sc\ w^a ec/y\ sc\ w^a ec$*. Since *y* has in extensive tests not been observed to back mutate (GREEN 1961), the most satisfactory explanation for the origin of the y^+ female requires two assumptions. First, that *y* lies to the left of y^2, and second, that a nonreciprocal single crossover occurred between *y* and y^2. This would lead to the y^+ female of the required genotype. A second y^+ female was found among the progeny of the cross females *$y\ sp^2\text{-}w\ spl/y^{51g}\ w^h$; $Cy/+$; $Ubx^{130}/+$* × males *$y\ w\ spl\ sn^3$* (y^{51g} = a spontaneous y^2-like mutant kindly supplied by DR. J. SCHULTZ, w^h = honey-white, $sp^2\text{-}w$ = spotted[2]-white, a *w* pseudoallele). The exceptional female proved on progeny testing to have the genotype *$y^+\ sp^2\text{-}w\ spl/y\ w\ spl\ sn^3$*. Assuming y^{51g} has its locus, as does y^2, to the right of *y*, a single crossover between *y* and y^{51g} satisfactorily accounts for the *$y^+\ sp^2\text{-}w\ spl$* chromosome. These results imply the existence of pseudoallelism at the *y* locus.

DISCUSSION

Although the data do not unequivocally establish pseudoallelism at the *y* locus, they are sufficiently suggestive to warrant the conclusion that complementation need not invariably occur between recombinationally distinct mutants. This conclusion is in keeping with more extensive analyses of complementation in Neurospora (CASE and GILES 1958, 1960). On the other hand complementation between y^2 and y^{59b} is explicable as complementation between spatially inseparable mutants. Complementation between such mutants is not unique, having been described among certain lozenge mutants (GREEN and GREEN 1956). The derivation of y^{59b} from y^2 concommitant with a reversion to y^+ is most readily explained as simultaneous mutational events at the y^2 locus. Those detailed arguments submitted by MULLER, CARLSON and SHALET (1961) to rationalize the occurrence of mutations in nondividing chromosomes apply with equal rigor to the concurrent induction of y^{59b} and y^+ from y^2. Attempts to explain the occurrence of y^{59b} as a mutation at a site adjacent to y^2 or as a minute loss proved to be consistently unsatisfactory because they fail to account for the simultaneous occurrence of the reversal event. Other explanations require the occurrence of too many coincidental events to be seriously considered. Because y^{Si} mutated in the sc^{Si} chromosome whose left break point is proximal to the *y* locus, a description of its precise spatial relations is precluded.

It is not immediately obvious why *sc* should so strikingly modify the complementation function of y^2 especially since superficially the phenotypes of these mutants appear to be entirely unrelated. Of some interest in this connection is the observation that both y^2 and *sc* together with a number of other seemingly unrelated mutants are partially suppressed by the autosomal suppressor of Hairy wing (*su-Hw*) (BRIDGES 1932; LEWIS 1949). Suppression by *su-Hw* implies that y^2 and *sc* influence a common or related developmental pathway and therefore it is not too difficult to conceive of one modifying the other.

SUMMARY

Complementation at the yellow locus originally described by STONE (1935) is redescribed and extended. Evidence is submitted showing that complementation occurs between allelic mutants. The influence of the mutant scute upon the complementation reaction is demonstrated.

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