Table 1. Relationship between cell death in the eye-antennal discs and adult abnormalities in the head of tumorous head adults.

Abnormalities probably due to cell death	Number of abnormalities scored
Antennal and arista missing	20
Eye reduced	326
Eye missing	16
Head to palpus	15
Palpus missing	85
Rostralhaut to palpus	16
Palpus malformed	51
Total abnormalities attributable to cell death	529

Total number of all types of abnormalities scored in the population	1367
Percent of abnormalities probably caused by cell death (a)	38
Expressivity for total population (i.e., number abnormalities per half head) (b)	1.45
Calculated percent of discs that should show cell death (a/b) if our assumptions are correct	26
Actual percent of discs showing cell death	27

Bownes, M. and M. Seiler. University of Essex, Colchester, England and University of Freiburg, Germany. UV irradiation of Drosophila embryos.

Using UV irradiation it is possible to cause polarity reversals in the eggs of some insects. In Smittia double-abdomen embryos, where the head and thorax are replaced by a second abdomen in mirror image symmetry to the original position of the abdomen, can be induced with 100% efficiency (Kalthoff, 1971). In Drosophila, however, double abdomens could not be induced by UV and the only reorganized embryos were ones containing 8 abdominal segments occupying the whole egg with no head or thorax and no loose tissue at the anterior (Bownes and Kalthoff, 1974). The maternal effect mutation bicaudal (bic) of Drosophila leads to the production of similar embryos (Bull, 1966; Nusslein-Volhard, 1977). It is possible that since bicaudal mothers can produce this defect in the organization of pattern in the embryo that eggs laid by these mothers would also be sensitive to UV irradiation-induced polarity reversals. Eggs were collected from both bic/vgB mothers which normally produce some bicaudal eggs and bic/bic mothers which rarely produce bicaudal eggs when homozygous, but often produce them when hemizygous. Eggs were UV irradiated at the anterior pole at the nuclear multiplication stage using 285nm wavelength. As can be seen in the table the proportions of abnormal embryos are not altered very much by the different genetic backgrounds, although the number of undifferentiated and abnormal embryos is significantly increased in all cases by the UV. Furthermore, no bicaudal embryos and no increase in the number of abdomen-only embryos were observed. Thus the genetic instability in these flies which causes them to produce double abdomens does not make them more responsive to UV irradiation. One possible explanation for this is that the bicaudal mutation alters the initial establishment of positional information whereas UV ir-

Survival of UV-irradiated eggs laid by females with different genotypes

Genotype	Experiment	Total	% Hatched	% Undifferentiated	% Abnormal
OrR	control	264	76	17	7
	UV irradiated	632	12	34	54
OrK	control	226	75	22	3
	UV irradiated	259	31	35	35
bic/bic	control	1128	81	14	5
	UV irradiated	1680	29	30	41
bic/vgB	control	264	29	60	11
	UV irradiated	536	1	77	22

radiation alters its interpretation. Thus the bicaudal embryos resulting from mutation and the UV-induced double abdomens found in other species of insects may well be produced by entirely different pathways at the molecular level.

References: Bownes, M. and K. Kalthoff 1974, J. Embryol. Exp. Morph. 31: 329-345; Bull, A.L. 1966, J. Exp. Zool. 161: 221-242; Kalthoff, K. 1971, Wilhelm Roux Archiv 168: 63-84; Nusslein-Volhard 1977, Wilhelm Roux Archiv 183: 249-268.

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Bregliano, J.C., A. Bucheton, J.M. Lavige, A. Pélisson and G. Picard. University of Clermont-Ferrand II, France. Hybrid dysgenesis in Drosophila melanogaster: the I-R system.

Since 1970 we have been working on a non-mendelian female sterility in Drosophila melanogaster. On the basis of the fertility of F_1 females, two main classes of strains, inducer and reactive, can be distinguished. Crosses between reactive females and inducer males give rise to daughters (SF females) showing a more or less important reduction of fertility, while reciprocal crosses produce only normally fertile daughters (RSF females). SF sterility is characterized by several specific physiological features (Picard et al., 1977). A survey of more than 200 strains indicates that all wild populations are inducer, whatever may be their geographical origin, while both inducer and reactive are found among long established laboratory stocks.

We have demonstrated that SF sterility results from an interaction between a chromosomal factor (I factor) responsible for the inducer condition and a genetic state, called reactivity, responsible for the reactive condition. Both conditions show a great range of variations. According to the amount of reduction of fertility of SF females, reactive and inducer strains can be arranged from "strong" to "weak": the stronger the parental strains, the higher the reduction of SF female fertility (Bucheton et al., 1976). All wild populations tested so far are strong inducer.

Reactivity corresponds to a cytoplasmic state controlled by a polygenic system, with a long delayed effect (Bucheton and Picard, 1978). I factor is chromosomal and may be linked to any chromosome of inducer strains. Two kinds of chromosomes, inducer (i^+) and non-inducer (i^o) have been found in inducer strains according to their ability or not to carry I factor, respectively. Through heterozygous males bearing both i^+ and reactive originating (r) chromosomes, I factor is transmitted following a strict mendelian pattern. In contrast, in heterozygous females, even in those carrying only one i^+ chromosome, every r chromosome may acquire irreversibly I factor, often with a high frequency, by a process called chromosomal contamination (Picard, 1976, 1978). Several evidences indicate that I factor might be a transposable element (Pelisson, 1978). Chromosomal contamination occurs only in females in which the I-R interaction exists but not in inducer females. Indeed, although i^o chromosomes can contaminated in SF females as well as r chromosomes, some inducer strains maintain a stable i^+/i^o polymorphism. The lack of chromosomal contamination in i^+/i^o inducer females allows the mapping of I factor. The first results support the idea that there are only a few sites on each chromosome but the data do not permit a decision as to whether or not these locations are the same on all homologous chromosomes of various strains (Pelisson and Picard, 1979).

We recently showed (Picard et al., 1978) that the I-R interaction leads not only to SF sterility and chromosomal contamination but also to high levels of several dysgenic traits in the female germ line (X non-disjunction, lethal and visible mutations). Some of the mutations observed are very unstable, suggesting they might result from insertions. It is of course tempting to hypothesize that they are insertions of I factor. The I-R interaction does not seem to have any effect on the male germ line.

The I-R interaction clearly enters in the field of hybrid dysgenesis. It is now firmly established (Kidwell, 1979) that there are at least two causally independent systems displaying many common features: the I-R and the P-M systems. The latter produces dysgenic traits in both female and male germ line, especially male recombination. These observations make it necessary to take a critical look at many studies done for 40 years on Drosophila melanogaster, mainly on mutator effects. In most cases, the experimental schemes do not exclude the possibility that the high mutability observed results from strain interactions rather than from widespread mutator genes acting in natural populations. Moreover, we claim that it is no longer possible for Drosophila geneticists to neglect the I-R and P-M classifications of the stocks they use.