(2) Average number of labeled segments/100 µm of stretch length. While in control, this number decreases, in Puromycin treated fibres the number increases (though fluctuating) with increase in the length of pulse time.

(3) Average size of the unlabeled gap and number of gaps/100 µm of stretch length. In control, the gap size remains more or less constant, whereas with Puromycin, the gap size, though fluctuating at the beginning, decreases gradually and then remains constant with increasing duration of pulse time. In control, the number of unlabeled gaps per 100 µm stretch decreases, while with Puromycin, the number tends to increase with increasing time.

(4) The rate of fork movement. In control, the rate is at first high, then drops down sharply and then gradually decreases with increasing pulse time. In Puromycin treated preparations, the rate though 2 to 10 folds less follows the same curvilinear regression as found in the control set.

It appears from the results that Puromycin induces a stage of replication found in early embryogenesis by activating the number of initiation sites, inducing clustering of replicons and reduced replicon size.

Furthermore, the results suggest that there may be two classes of replicon families as suggested by Hori (1979) and others. Puromycin inhibits the rate of fork movement in both types of replicon families.

References: Edenberg, H.J. & J.A.Huberman 1975, Ann.Res.Genet. 9:245-284; Hori, T. 1979, Jap.J.Genet. 55(1):41-54; Hori, T. & K.G. Lark 1974, J.Mol.Biol. 88:221-232; Van't Hof, J., A.Kuniyuki & C.A.Bjerknes 1978, Chromosoma (Berl.) 68:269-285.

Basden, E.B. Leyden Park, Bonnyrigg, Midlothian, Scotland. The Species as a block to mutations.

Mutants (phenotypes) of D.melanogaster and of a few other species of Drosophila have been described in detail. The number discovered since 1907 in melanogaster alone is many, many thousands and of every category.

There are no lists, however, of mutants that might be expected but are not found. One type will be discussed here, and for this purpose the species of Drosophila are grouped into two distinct divisions, viz: (1) The clear-wings. These are species whose wing-blades (including veins) are clear, hyaline, and quite unmarked. Clear wings include affinis, ananassae, funebris, hydei, melanogaster, pseudoobscura, subobscura, etc. (2) The marked-wings. Species whose wings bear a naturally pigmented spot or spots, or cloud, or pattern. Included here are hawaiian picture-wings, immigrans, robusta, quinaria-group, virilis-group, etc.

As far as is known there are no mutants (visible mutations) of any clear-wing species that have pigmented wing marks. Converesely, there are no mutants of marked-wing species that have unmarked wings. Excluded from clear-wing mutants are suffused general yellowing or darkening (as in yellow, black, dusky, ebony, sooty of melanogaster, and the shadowy smudge along the costa of subobscura at certain seasons), melanotic tumors, blood blisters, and developmental disturbances (e.g., black spotted wings (DIS 58:203), dumpy-oblique lethal vortex, and speck).

Wild-type marked-wings have one or more regular precise wing areas that are naturally and discretely pigmented in fully hardened flies. If the marks are multiple, any mutation would have to be assessed on the disappearance of all rather than on some of that particular type.

Thus it appears that at the species level there is a block to the apparently simple shift to or from pigmentation in the wings. In other words, the species is a block to some mutations. However, in a few species the male and female wings differ, one sex being clear-wing, the other marked-wing. Examples are D.tristis of the western palearctic and some species of the melanogaster-group. Evidently many clear-wing species do contain plenty of pigment in their bodies but it does not occur in discrete, localised spots in their wings. Yet two closely related species may belong to the opposite divisions. So where have all these intraspecific mutations gone?

Anyone has my consent to quote this note. I am grateful for information from M.Ashburner, H.Gloor, Oswald Hess, Claude W. Hinton, Costas B.Krimbas, Dan L.Lindsley, K.G. LUning, Dwight D. Miller, Toyohi Okada, D.Sperlich, Lynn H. Throckmorton, L.Craymer, and A-M.Jönsson(née Perje).