responsible for the lower gene frequency in population D. The selection pressure against genes on the X-chromosome is much higher in ♂♂ than in ♀♀, since these genes are in a hemizygous state and always expressed in ♂♂, if there are no modifiers concealing them. In a natural population of D. melanogaster from Banyuls (Bösiger, 1962) the frequency of mutants is twice as high in ♀♀ than in ♂♂. The greater fragility of ♂♂ could add to the lower mutant gene frequency. The effect of modifier genes, introduced by the crossing of the Samothrace ♂♂ with the foreign Küssnacht ♀♀, may have prevented in some cases the phenotypical expression of present mutants.

4. The X^2 test shows, that the observed distribution of frequencies of mutant genes fits well with a Poisson distribution for the populations B and C, at the 1% level only for population A and not at all for population D.

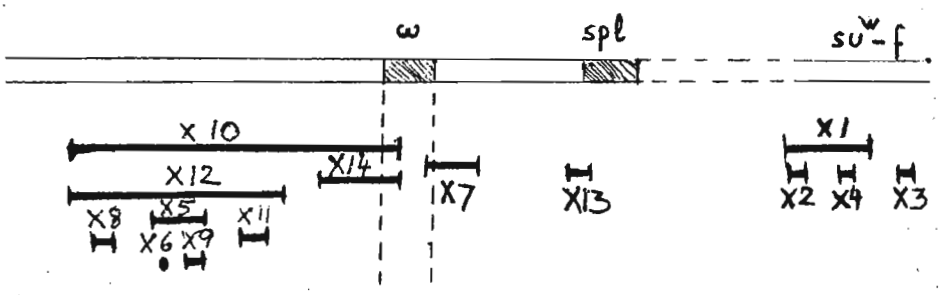
5. A preliminary morphological comparison of the mutant phenotypes found by Gordon, Spurway and Street (1939) in England, by Buzzati-Traverso (1941) in Italy and by Prevosti (1952) in Spain with those found in Greece shows that the great majority of the phenotypes are common in the four countries. The list and description of the mutants found in Greece will be given later.

Bösiger, E., 1962, Bull. Biol. France et Belgique, 96:3-122. Buzzati-Traverso, A., 1941, Scient. Genetica, II:1-34. Gordon, C., Spurway, H. and Street, P., 1939, J. Genetics, 38:37-90. Prevosti, A., 1952, Genetica Iberica, 4:95-128.

Lifschytz, Eliezer. Hebrew University, Jerusalem, Israel. Induced X-chromosome lethals covered by $Y \cdot w^+$.

7100 y ac sc chromosomes were irradiated with a 2000 r dose of X-rays. 363 chromosomes carried lethal mutations. 14 of these lethals (3.9%) were covered by a $Y \cdot w^+$ chromosome produced by Brosseau et al, (1961).

The lethals covered by the $Y \cdot w^+$ chromosome were crossed among themselves in all possible combinations in order to determine their allelism. Lethals that were suspected to be "point mutations" after the allelism test were checked further for crossing-over disturbance in the y-pn-w region. One group of lethals showed free recombination with the markers and proved to be proximal to f. They are also covered by $Y \cdot ma\text{-}1^+$ and by $Y \cdot B^S$. These lethals were thus located in the most proximal region of the X-chromosome, and are probably covered by su^W-f. All the lethals except one showed disturbances in the crossing over frequency in their immediate vicinity. The following "complementation map" is consistent with the data collected so far:



Since we selected for lethals covered by the $Y \cdot w^+$ compound, deficiencies extending beyond the region of the X-chromosome included in this compound were automatically excluded. This may explain the fact that only 3.9% of the lethals were found in a section comprising 10% of the cytological length of the X chromosome.

Most of these lethals do not disturb crossing-over between distant markers, such as y-sn, and would have been classified as "point mutations" by routine genetic procedures. These results suggest that many X-ray induced lethals that pass for "point mutations" are actually small aberrations.

Note that lethals X10, X14 and X7 are allelic to w, but X7 complements the lethal effect of both X10 and X14. This is in line with Lefevre's (1965) findings, and suggests that a deficiency for the w-locus is non-lethal.

We started to accumulate chemically induced lethals covered by $Y \cdot w^+$ in order to compare the "complementation map" of lethals induced by the various mutagenic agents.