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FM7: A "new" first chromosome balancer.

The construction of First Multiple no. 7a, genotype Ins(1)sc^8 + 15D-E;20A-B + d1-49, y^{31d} sc^8 w^a v0 B, is given under new mutants in DIS 43. The homozygous stock may be useful for M-5 type experiments.

The balancer First Multiple no. 7b, genotype Ins(1)sc^8 + 15D-E;20A-B + d1-49, y^{31d} sc^8 w^a lz^{sp} B, was obtained as a double crossover from FM7a/In(1)d149, lz^{sp} females. Homozygous FM7b females are sterile. FM7b males (because of lz^{sp}) are slow in development so that male sterile or lethal mutant stocks may be lost if they are transferred too rapidly. Stocks kept at 18° may be held an additional week between transfers to avoid this problem.

Both FM7 stocks should prove effective in suppressing crossing-over in the normal sequence w^a-B interval which FM6 does not cover in stocks containing autosomal rearrangements. FM7 differs from FM6 in that it includes the complete d1-49 inversion. Double crossovers within the d1-49 inversion are apparently nonexistent; doubles spanning the d1-49 inversion should be rare because of the additional 15D-E;20A-B inversion. Dr. J. H. Williamson kindly checked crossing-over with Fm7b/y^2 sc cv wy f sc^{v1} females heterozygous for Ubx^{130} and/or SM5; no recombinants were found among approximately 6,000 male progeny.

(FM7a is currently maintained as a homozygous stock at the Pasadena center. FM7b is currently used to balance Muller's multiple mutant chromosome, y pn w cm ct^6 sn^3 oc ras^2 v dy g^2 f os^o car sw.)

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Patterns of damage in deep orange (dor) embryos.

Recently, Hildreth & Lucchesi (Develop. Biol. 15, 536) re-examined the pattern of damage in embryos of the sex-linked female sterility mutant dor and were unable to find the "early early lethal" class described by me in 1956 (Z.i.A.V. 87, 443). On the basis of mating behavior, fertility of their stocks, and meiotic behavior in eggs from virgin dor females, they concluded that this "lethal" class in my 1956 study was probably composed almost entirely of unfertilized eggs. Because of this report, and also because Merrell (Amer. Nat. 81, 399) in his original description of the gene reported quite different developmental effects than any of the above authors, in 1967 I looked again at the pattern of damage in the dor/C1B stock I had originally studied. Eggs were collected from 20 four to five day old dor ♀♀ mated with 40 dor ♂♂ of the same age.

My results paralleled those of Hildreth & Lucchesi in that only 8% of the 118 embryos studied might be classed as "early early lethals" and were either unfertilized eggs or eggs that ceased development during early cleavage. However, there were other, and I believe significant, differences in the patterns of damage in this 1967 study. As in 1956, lethals could easily be assigned to "early" or "late" lethal classes (roughly 40 and 60% respectively of those eggs that showed embryogenesis). But in the 1967 group, development of the "late" lethals was much more advanced than had been observed in 1956, and many showed recognizable late embryonic features, comparable to Merrell's observations. The "early" lethal group also developed to a later stage (past gastrulation) than the comparable 1956 embryos. But most significantly, in none of the lethals was there the slightest evidence of the periplasmic and consequent blastoderm abnormalities observed in half of the late lethals in 1956 (documented by illustrations 7 and 8 in that paper); nor was there any evidence of the paucity of yolk or scanty cytoplasm so characteristic of the 1956 eggs. (It should be noted that failure of a zygote to develop can result either from the failure of insemination of the female or the failure of syngamy to occur even when sperm are present in the egg; the "quality" of the egg could certainly have a profound effect on the latter type of zygote failure.)

Doane (J. Exp. Zool. 145, 23) has clearly demonstrated that both environmental and genetic factors may have a profound influence on oogenesis, egg structure, and embryonic development in the ♀ sterility mutant adipose. Beatty (Proc. Roy. Soc. Edin. 63, 249) and I (Z.i.A.V. 87, 462) got marked differences in fecundity and ovarian development in the same stock of the female-sterility mutant fused reared under only slightly different conditions. Hildreth & Lucchesi (op. cit.) found that viability and fecundity of dor ♀♀ in both the FM_3