Response to such stresses has been measured as days to 50% mortality at 25±0.5°C and 65-70% relative humidity in 3 x 1 inch glass vials with polyurethane foam stoppers for three stress treatments: (a) empty vials; (b) agar + sucrose - 7 ml of 0.15% w/v agar and 0.4% w/v sucrose medium; (c) paper + sucrose - a 20 x 13 cm piece of absorbent paper (Kleenex tissue) pressed into the bottom of the vial and 2.5 ml saturated sucrose solution added and absorbed by the paper. Additional treatments imposed were: sex - males only, females only, males and females in 1:1 ratio; density - 10, 20 or 30 flies per vial.

This 3 x 3 x 3 factorial was set up with 2 replicates. The flies used were progeny of a sample taken from a stock population cage which derived from 96 females captured at Yarrawonga, N.S.W. (locality 5 of Barker and Mulley 1976). These progeny emerged during a 12 hour period, and were aged for 3 days in well-yeasted vials before allocation to treatment vials. Mortality in each vial was recorded daily.

Analysis of variance of days to 50% mortality (i.e., from 3 days of age) showed a significant effect only for stress treatment ($P<0.001$). The means, which were significantly different from each other, and the maximum number of days survived, were: (a) empty vials - 3.8 days, 8 days; (b) agar + sucrose - 14.9 days, 23 days; (c) paper + sucrose - 18.6 days, 25 days. The significantly longer average survival in the paper + sucrose treatment, as compared with agar + sucrose, was presumably due to an initial higher humidity.

For comparison, in other experiments using medium containing dead yeast, but for a different strain of D. buzzatii, mean age at 50% mortality was 34.7 days, and maximum survival was to 90 days of age.

Clearly, D. buzzatii shows high tolerance to these environmental stress treatments (see also Parsons and McDonald 1978), which would be adaptive in their natural habitat, and the results do not preclude the possibility of survival for many days in a non-cactus environment, such as during migration from one cactus patch to another. It is hoped that current field studies will determine whether such migration does occur.

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References: Barker, J.S.F. and J.C. Mulley 1976, Evolution 30:213-233; Parsons, P.A. and J. McDonald 1978, Experientia 34:1445-1446.

Hardy, R.W. University of California, San Diego. Crystal aggregates in the primary spermatocytes of XO males in D. melanogaster.

In another note in this issue we describe a small region in the middle of the Y chromosome which when missing results in formation of the crystal aggregates in the primary spermatocyte described by Meyer et al. (1961) in XO males. These aggregates are seen in live preparations with phase contrast optics. In the present note we locate a gene on the X chromosome which determines the shape of the aggregates.

Crystal aggregates occur in either of two forms, needle-shaped or star-shaped (Meyer et al., 1961). Aggregates are found in both nucleus and cytoplasm but the latter are larger and more easily seen. They persist through the meiotic divisions and can also be found in developing spermatids. Their exact molecular composition is not known, but they seem to contain both protein and lipid (Cox et al., 1976).

Meyer et al. (1961) reported that in XO males whose X chromosome is FM4, the needle-shaped crystal aggregates normally found are replaced by star-shaped ones. Additionally, Cox et al. (1976) report the occurrence of star-shaped aggregates in spermatocytes of both FM4/0 and FM6/0 males and further suggest that the change in morphology may be due to a specific inversion (In(1)3C;4E-F which is superimposed on $In(1)sc^8$ + In(1)d1-49 in both of these chromosomes).

We have found star-shaped crystal aggregates in two stocks with X chromosomes of normal sequence. We designate the locus responsible for modifying the shape of the crystal aggregates as stellate (ste). The allele responsible for the needle-shaped aggregates is designated ste^+ since this allele seems to be the one more commonly found in laboratory stocks. The allele responsible for the star shape is designated as ste. It is not known which, if either, allele is dominant.

In one instance the stellate phenotype was found in XO males carrying an X chromosome marked with y w^a, and in the second it was found segregating in our Canton-S wild type stock. Females heterozygous for ste from both sources and sc ec cv ct^6 v g^2 f were crossed to XY/O males, and recombinant sons were dissected and the phenotype of the crystals in the spermatocytes noted. The crystals were star-shaped when the g-f region came from the X chromosome being tested and needle-shaped when it came from the marked X chromosome. Furthermore the recombinants between garnet and forked gave the results tabulated below.

Origin of ste-bearing X chromosome	Recombinants				Total males
	g ste f^+	g^+ ste^+ f	g ste^+ f^+	g^+ ste f	
Canton-S	3	2	10	7	258
y w^a	0	2	5	12	---

8.5% recombination was observed between g and f compared to a standard map distance of 12.1 units. The distribution of ste among the recombinants between g and f indicates that the stellate locus is 17% (7/41) of the distance from g to f which places it at 46.5 on the standard map of the X chromosome.

References: Meyer, G.F., O. Hess and W. Beerman 1961, Chromosoma 12:676-716; Cox, G.N., J.D. White and B.I. Kiefer 1976, Genetics 83:S17.

Hardy, R.W. and J.A. Kennison. University of California at San Diego, La Jolla, California. Identification of a small Y chromosome region responsible for meiocyte and spermatid abnormalities typically observed in XO males.

A most striking effect of deletion of the Y chromosome from the primary spermatocyte is replacement of the lampbrush loop structures with crystals of a proteinaceous nature (Meyer et al. 1961). In addition, recent studies of XO males demonstrated abnormal meiotic organelle and chromosome distribution probably as a consequence of aberrant meiotic spindle formation (Lifschytz and Hareven 1977; Lifschytz and Meyer 1977). Furthermore, as with crystal formation (Meyer et al. 1961), the distribution of meiocyte cellular components is more nearly like wild type in spermatocytes having the long arm of the Y chromosome (Y^L) present (Lifschytz and Hareven 1977).

We have investigated these phenotypes in males carrying deficiencies for small regions of the Y chromosome, that is to say, regions which are thought to contain only a single fertility factor. A deficiency for such a small region is generated by combining specific segregants from different male-fertile XY translocations. In particular, segmental aneuploidy for one small region, a region approximately in the middle of Y^L, results in spermatocyte and spermatid abnormalities closely resembling those seen in the light microscope for XO and XY^S males (Lifschytz and Hareven 1977; Lifschytz and Meyer 1977) and in the electron microscope for XO males (Kiefer 1973). Sixteen primary spermatocytes are formed which contain crystals characteristic of XO males. Additionally, mitochondria and chromosomes are distributed abnormally during meiosis resulting in the formation of abnormal nebenkerne and micronuclei in the sperm-