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-

atids similar to those observed in XO males. Two deviations from the XO phenotype have been observed. Lampbrush loop structures which are not obvious in XO primary spermatocytes can be seen in those carrying the deficiency. Furthermore, in electron micrographs of cross sections through the primary spermatocytes both crystals and the nuclear structures shown by Meyer et al. (1961) and Tates (1971) to be present in wild type (XY) spermatocytes are seen. The second deviation from the XO phenotype is that counts of spermatid tails in cross sections of cysts close to the middle of the testis indicate a mean number of tails per bundle of 54.5, considerably higher than the 31 reported in XO males by Kiefer (1973). Additionally, many of the axonemes and mitochondrial derivatives exhibit cross sections like those seen in XO males.

Preliminary observations of small deficiencies totaling virtually all of the Y chromosome except for the proximal regions around the kinetichore suggest that only the small region noted above leads to crystals, aberrant nebenkerne and micronuclei. Males carrying some of the other small deficiencies do not have normal ultrastructure in their spermatocytes or spermatids but the extent of such aberrations is not known.

At present work is under way to further characterize the Y chromosome deficiencies both genetically and cytologically (Kennison) and to study their effects on germ line development using both light and electron microscopy (Hardy).

References: Kiefer, B.I. 1973, in: Genetic Mechanisms of Development (Ruddle, F.H., ed.) pp. 47-102, Academic Press; Lifschytz, E. and D. Hareven 1977, Developmental Biology 58:276-294; Lifschytz, E. and G.F. Meyer 1977, Chromosoma 64:371-392; Meyer, G.F., O. Hess and W. Beerman 1961, Chromosoma 12:676-716; Tates, A.D. 1971, Ph.D. Thesis, Transitorium voor Gneeskunde, The Netherlands.

Hawley, R.S. University of Washington, Seattle, Washington. Radiation-induced nondisjunction in females homozygous for In(1)sc^{8}.

In(1)sc^{8}, y w^{a} / In(1)sc^{8}, f v cv females were exposed to 3000 R, using a Co^{60} source, and then mated to Y^{S} In(1)EN.Y^{L}, v f B / O males. The progeny, resulting from eggs laid from 24-72 hours after irradiation, consisted of 1546 B females, 438 B^{+} males, 72 v f B males, 11 B^{+} females, 2 w^{a} B^{+} females, 1 y B^{+} female, and 1 v B^{+} female. The frequencies of B males (.22), which result from nullo-X ova, and B^{+} females (.005), which result from diplo-X ova, are identical to published values obtained following similar treatment of wild-type females (Hawley 1975). The recovery of 4 females homozygous for recessive markers confirms the observation of Savontaus (1975) that radiation-induced nondisjunction is not restricted to Eo tetrads.

In a second experiment, In(1)sc^{8}, y w^{a} / In(1)sc^{8}, f v cv females treated with 3000 R were mated to Y^{S} In(1)EN.Y^{L}, v f B / O; C(4)RM, ci ey^{R} females and 27 v f B males and 6 B^{+} females were selected from among the progeny. By crossing the v f B males to C(1)RM, y f / Y ; ci ey^{R} / ci ey^{R} females, 7 (24%) were shown to have resulted from eggs which were also diplo-4. Of the 6 B^{+} females, 4 (66%) were homozygous for ci ey^{R}. Following similar treatment of wild-type females, 19% of the nullo-X exceptions were also diplo-4 and 38% of the diplo-X exceptions were also nullo-4 (Hawley 1975).

These data suggest that the associations between the X and 4th chromosomes that dictate the frequency and manner of radiation-induced nondisjunction are not influenced by the location of the pericentric heterochromatin.

References: Hawley, R.S. 1975, Mut. Res. 33:391-394; Savontaus, M.-L. 1975, Hereditas 80: 195-204.

Hazelrigg, T. and T.C. Kaufman. Indiana University, Bloomington, Indiana. Newly induced mutations of doublesex.

Previous work (Duncan and Kaufman) has shown that the homoeotic gene doublesex (dsx) is located in region 84F of the polytene chromosome map. Both a recessive allele (which yields an intersexual phenotype in males and females) and a dominant allele (which transforms only females into intersexes) are known to be associated with this locus. In the present work, an EMS mutagenizing (Lewis and Bacher) screen has been performed to uncover new alleles of dsx, and also recessive lethals located in this region of the chromosome. The screen utilized a deficiency, dsx^{D+R2}, recovered as a revertant of dsx^{D}