These results show that the effect of actinomycin-D which inhibits DNA-dependent RNA synthesis on inactivation of the SR spirochete is more predominate than chloramphenicol which inhibits protein synthesis.

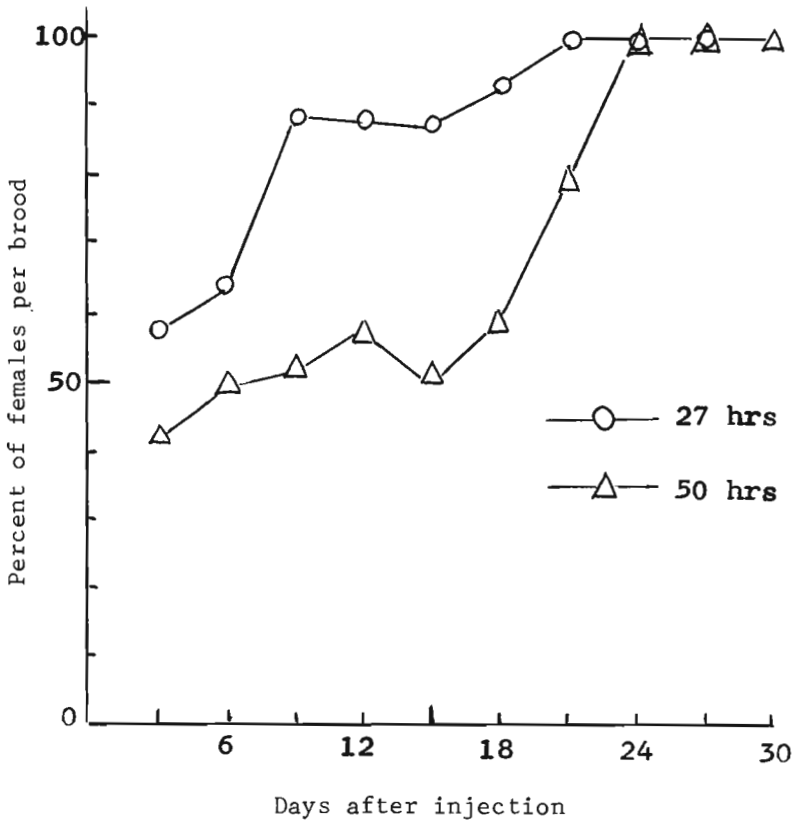

Fig. 2. Effect of actinomycin-D on SR spirochete.

Procedures of this experiment were the same as in Fig. 1.

To make clear the properties of multiplication of the SR spirochete, a more detailed examination of this sort is now underway. (Support by PHS Grant GM10238 of USA and a Grant 36001 from the Ministry of Education of Japan.)

Yoon, J.S. and W.C. Kim. Yonsei University College of Medicine, Seoul, Korea. Genetic effects of a synthetic ovarian steroid in D. melanogaster.

Effects of a synthetic ovarian steroid on genetic materials were studied in Drosophila treated with Lyndiol 2.5 (Lynostrenol 2.5 mg and Mestranol 0.075 mg/tablet). Germ cells of males ($sc^8.y.B^S/y^2\ w^i\ ct^6\ f^1$) reared on the medium containing 0.5 ml of Lyndiol (50% in Drosophila Ringer's) solution through imaginal stages were tested for genetic damage. When males treated were crossed individually to multipurpose virgin (y sc^{S1} In49 sc^8;dp bw;st p^p),

Table 1. Mutations and chromosomal abnormalities in D. melanogaster treated with Lyndiol 2.5.

	Treated		Control	
Aberrations	Total No. Studied	% With Aberration	Total No. Studied	% With Aberration
Loss of Y	8,829	0.14	6,605	0.08
Nondisjunction	18,453	0.37	12,323	0.14
Visible mutations	18,453	0.07	12,323	0.00
Lethal mutations	1,628	0.49	1,389	0.07
Translocations	1,558	0.00	1,215	0.00

increased nondisjunctions, losses of the Y chromosome, and other visible mutations were found. The rate of sex-linked recessive lethal mutation was 0.5% (8 out of 1,628 chromosomes tested) in the group treated, and no translocation was found (Table 1). The data suggest that the hormone may act as a mutagen in Drosophila.