An ovary of an adult female Drosophila melanogaster consists of a parallel cluster of ovarioles, each of which is differentiated into an anterior germarium and a posterior vitellarium (see Koch, Smith, and King 1967, their Figure 1). The vitellarium is composed of a series of interconnected egg chambers which lie in single file. Each chamber is in a more advanced developmental stage than the one anterior to it, and each contains an oocyte and 15 nurse cells surrounded by a monolayer of follicle cells (ibid., Figure 2). The egg and its fifteen nurse cells are fourth generation descendants of a single germlarial cell called a cystoblast. The cells formed by the mitotic activity of a cystoblast are called cystocytes, and it is within region 1 of the gerarium that such mitoses occur (ibid., Figure 2). The cystocytes generated from a single germlarial cystoblast form a branching chain of cells. The 16 cells are connected by 15 canals, each surrounded by a ring rich in protein (ibid., Figure 6). The cystocytes can be characterized by the number of ring canals each contains. Two cystocytes (designated 1e and 2e) are interconnected and each possesses four ring canals; two cells (3e and 4e) contain three canals each, four cells (5-8e) contain two ring canals each, and eight cells (9-16e) have but one canal each (ibid., Figure 7). Cells 1e and 2e undergo a different type of nuclear differentiation from cells 3-16e. Since only these two cells enter meiotic prophase, they have been named pro-oocytes, while the remaining fourteen are called pro-nurse cells. The production of fourth generation cystocytes and their differentiation into pro-oocytes and pro-nurse cells begins during the pupal stage, and continues throughout adult life (King, Aggarwal and Aggarwal 1968). In each 16-cell cluster, however, one of the two pro-oocytes eventually switches to the nurse cell developmental pathway (Brown and King 1964).

Pro-oocytes may be readily differentiated from pro-nurse cells at the electron microscopic level by their nuclear morphology (Koch, Smith and King 1967). Ribbon-like synaptonemal complexes are seen only in pro-oocyte nuclei (King, Aggarwal and Aggarwal, their Figure 13). In the nucleus of the pro-oocyte

1 Three spellings of this adjective are found in the literature (synaptinemal, synaptenemal, and synaptonemal). The first spelling was used by Moss, who coined the term synaptinemal complex. However, we suggest that the last spelling is the etymologically correct one (G. synaptos, fastened together + nema, thread). Throughout this paper the terms leptonema, zygonema, pachynema, and diplonema are used as nouns; whereas leptotene, zygotene, pachytene, and diplotene are used as adjectives.
destined to become a nurse cell all synaptonemal complexes degenerate during stage 1. Throughout this paper the staging of Drosophila oogenesis developed by King, Rubinson, and Smith (1956) and King (1964) will be employed.

In *Drosophila melanogaster* the nuclei of pro-oocytes and of young oocytes are unsuitable for cytological study at the light microscopic level because of their small size (see Figure 3). However, there are species with larger chromosomes in which a correlation is possible between light and electron microscopic observations of cells in meiotic prophase. It is known, for example, in *Aedes aegypti* primary oocytes (Roth 1966) and in *Lilium longiflorum* microsporocytes (Roth and Ito 1967; Moens 1968) that synaptonemal complexes are first seen during zygonema, reach their maximum lengths during pachynema, and degenerate during diplonema. Wettstein and SoteLO (1967) have shown from reconstructions of serial sections that in the pachytene spermatocytes of *Gryllus argentinus* an uninterrupted synaptonemal complex extends the length of each pair of homologues. Furthermore, the morphology of the synaptonemal complex is the same in each of the different bivalents.

In *Drosophila melanogaster* leptonema must immediately follow the postmitotic DNA replication in cystocytes 1e and 2e, since synaptonemal complexes appear in the nuclei of pro-oocytes shortly after their formation by the division of third generation cystocytes 1d and 2d. Synaptonemal complexes increase first in number and then in average length. The period during which the synaptonemal complexes appear and grow must be zygonema. This stage thus occurs during the time the pro-oocytes move posteriorly through the germarium and during stages 1 and 2. Pachynema is completed in oocytes residing in the anterior portion of the vitellarium, since the combined length of all synaptonemal complexes reaches a maximum between stages 3 and 4. The complexes then degenerate and by stage 7 none are visible (see Koch, Smith and King 1967, their Figures 3, 4, and 12).

From studies on other species we know that synaptonemal complexes are rich in protein (Moses 1964), that they can dissociate from post-pachytene homologues, and that sometimes these dissociated complexes stay intact long enough to pair laterally with one another to form "polycomplexes" (Roth 1966, his Figure 12). The syntheses of such structurally complex ribbons must be under genetic control, and, if they are essential to meiotic recombination, there should exist mutations which suppress crossing over because the synthesis of synaptonemal complexes is retarded or abolished. A mutation in *Drosophila melanogaster* which is a case in point is the recessive crossover suppressor in chromosome 3 of G Owen.

This spontaneous mutation (symbolized *c(3)G*) was discovered in 1917 by Marie and John G Owen. Subsequently J. G Owen (1933) reported that the mutant gene was located on the third chromosome at about locus 55. More recent data (Lewis 1948) place the genetic locus at 58 and the cytological locus between 89A1 and 89B3. G Owen showed that in females homozygous for *c(3)G* crossing over in the entire chromosomal complement is reduced to a small fraction of normal. Externally the mutation produces no visible effect in either the homozy-
gous or the heterozygous condition. In 1964 G. F. MEYER reported (without giving any details) that he could not find any synaptonemal complexes in oocyte nuclei from homozygous \(c(3)G\) females. It is the purpose of this paper to give a detailed account of the cytology of such females.

**MATERIALS AND METHODS**

The mutant females used in this study were of genotype \(st\) \(c(3)G\) \(+\) \(ca\). LINDSLEY and GRELL (1968) can be consulted for descriptions of the other mutants employed. The flies were grown upon DAVID's medium (DAVID 1962) at a temperature of 25°C and a photoperiod of 12 hours of light and 12 hours of darkness. Heterozygous females \((st\) \(c(3)G\ ca/TMI, \(Me\ ri\ sbd\)) and females from an Oregon R, wild-type strain were used as controls unless noted otherwise. Females were provided with wild-type mates in all cases.

First a study of egg production and percent of eggs hatching was made using the procedure of KING (1955). Next ovaries were dissected from etherized females immersed in Drosophila Ringer's solution (see BUTTERWORTH, BODENSTEIN, and KING 1965, pg. 142 for the recipe). Subsequently Feulgen-stained whole mounts were made of some of the ovaries according to the procedure of KING, BURNETT, and STALEY (1957, p. 242). These ovarian preparations were used to determine the distribution of egg chambers in various developmental stages. In other cases ovaries were transferred to a fixative and after dehydration were imbedded in plastic. The techniques of cytological processing are given in KOCH and KING (1966). A Leitz-Fernandez-Moran microtome equipped with glass knives was used to cut 1 µ longitudinal sections of germaria or young egg chambers which were mounted on glass slides for light microscopy. An aqueous 0.025% solution of azure B (at pH 9) was used as a general cytoplasmic and nuclear stain. This dye binds to all nucleic acids, proteins, and lipids bearing negatively charged groups at pH 9 (KING 1960). Outlines of sectioned germaria were drawn at magnifications of 1270X on tracing paper using a Wild-Heerbrugg M20 microscope equipped with a drawing tube. Stacked tracings were used to analyze the distribution of cystocytes in each germarium.

Electron microscopic observations were made of sections each approximately 60 µ thick. Germaria and young chambers were longitudinally sectioned using an LKB Ultrotome. Sections were picked up in groups of four on electron microscope grids. A Hitachi 11A electron microscope was used to take overlapping photographs of images magnified 5000X of areas from one section on each grid. Tracings were made from prints enlarged 2.5 times. A given cell could be followed through successive tracings and its connections to other cells determined. In this way cells 1e and 2e could be distinguished from cells 3–16e, for example.

**RESULTS AND CONCLUSIONS**

Individual records were kept of the daily egg production for ten females homozygous for \(c(3)G\). Over the period between the 3rd and 8th day of adult life each fly laid an average of 55.1 ± 3.3 eggs per day. The average percentage of eggs which hatched was 30.7 ± 2.6. The average number of ovarioles per female was 31.9 ± 1.9 for 17 \(c(3)G/c(3)G\) females. The values for the average number of developing chambers per vitellarium did not differ significantly between \(c(3)G/c(3)G\) and \(c(3)G/+\) females ranging in age between 0.5 and 10 days (see Table 1). The distributions of oocytes between the various developmental stages were also determined in ovaries from females of differing ages (Figure 1). No significant differences were seen in the distributions between \(c(3)G/c(3)G\) and \(c(3)G/+\) females. On the other hand a significant difference does exist between the
TABLE 1

Number of oocytes per vitellarium as a function of age and genotype

<table>
<thead>
<tr>
<th>age (days)</th>
<th>c(3)G/c(3)G</th>
<th></th>
<th>c(3)G/+</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Average chambers per vitellarium</td>
<td>N</td>
<td>Average chambers per vitellarium</td>
</tr>
<tr>
<td>0.5</td>
<td>10</td>
<td>1.9</td>
<td>7</td>
<td>2.0</td>
</tr>
<tr>
<td>3.5</td>
<td>10</td>
<td>7.0</td>
<td>10</td>
<td>6.1</td>
</tr>
<tr>
<td>7.0</td>
<td>10</td>
<td>5.9</td>
<td>6</td>
<td>5.3</td>
</tr>
<tr>
<td>10.0</td>
<td>5</td>
<td>5.7</td>
<td>10</td>
<td>5.1</td>
</tr>
</tbody>
</table>

$N =$ the number of ovaries observed.

The number of 16 cell cysts in the germaria of $c(3)G$ and in wild type germaria (see Table 2 and Figure 2). Wild-type germaria contain twice as many clusters of fourth generation cystocytes on the average as do mutant germaria.

The rate at which egg chambers develop in a functioning ovariole of Drosophila

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**FIGURE 1.**—The stage distribution of the egg chambers in Drosophila ovaries as a function of age and genotype. See Table 1 for the numbers of ovaries observed in each series.
TABLE 2
Average number of a single cells and 2, 4, 8, and 16 cell clusters per germarium
for females of different genotypes

<table>
<thead>
<tr>
<th>Genotype</th>
<th>N (avg)</th>
<th>1</th>
<th>2</th>
<th>4</th>
<th>8</th>
<th>16</th>
</tr>
</thead>
<tbody>
<tr>
<td>c(3)G/c(3)G</td>
<td>1.4</td>
<td>1.7</td>
<td>0.9</td>
<td>1.1</td>
<td>4.0</td>
<td></td>
</tr>
<tr>
<td>+/+</td>
<td>7</td>
<td>1.9</td>
<td>1.9</td>
<td>1.0</td>
<td>0.9</td>
<td>7.6</td>
</tr>
</tbody>
</table>

N = the number of germaria analysed.

The cytology of oocyte nucleus was studied at the light microscope level in both Feulgen-stained whole mounts and in azure B-stained sections. The results of this study are presented in Figure 3. In wild type the chromosomal filaments begin to condense during stage 3 and are incorporated into the compact karyosome by stage 4. In c(3)G this process is speeded up, since a well-defined karyosome is first seen in stage 3. Thus the stage in c(3)G equivalent to pachynema appears to be shortened. In both + and c(3)G the karyosome persists throughout subsequent stages. The karyosome becomes still more compact during stages 11 through 13. Late in stage 13 the nuclear envelope breaks down, and the karyosome is liberated into the ooplasm. This DNA-containing structure which lacks a nuclear envelope is called a karyosphere (KING, RUBINSON, and SMITH 1956). The post-pachytene tetrads arise from the karyosphere, and the first meiotic metaphase occurs after the egg leaves the ovariole. The data presented in Figure 4 are derived from electron micrographs taken of every fourth section from serially sectioned nuclei from pro-oocytes and oocytes residing in c(3)G ovarioles. The comparative data for wild-type oocytes are from the literature (KOCH, SMITH, and KING 1967, their Figure 12). It is obvious that synaptonemal complexes do not occur in c(3)G oocytes belonging to the same developmental stages where in wild type the formation of the ribbons begins, reaches a maximum, and ceases. Electron micrographs taken at identical magnifications of sections at similar levels through nuclei of stage 3 oocytes are presented for wild type and c(3)G in Figure 5.

In the wild type Drosophila oocyte each synaptonemal complex appears as a tripartite ribbon about 0.1 μ wide consisting of parallel, dense, lateral elements surrounding a medial complex. The medial complex contains a system of transverse rods oriented perpendicularly to the lateral elements and separated from...
them by a clear area, the central "space." It is reasonable to assume that the transverse rods form a strutwork which holds the lateral, chromosome-containing elements in their parallel configuration. If this is the case, then the clear areas to either side of the stacked rods must contain fibrils that bridge the gap, but which are not generally resolved in electron micrographs. See Roth (1966, his Diagram 2) for a reasonable, three-dimensional conceptualization of the structure. The finding that the formation of synaptonemal complexes is never initiated in the mutant suggests that c(3)G+ is required for the synthesis of the subunits of which the strutwork is composed.

**DISCUSSION**

Whitehouse (1967) has put forward a model in which the chromosomes of eucaryotes are considered to be cycloids. Each loop of a cycloid corresponds to a set of copies of a gene forming a chromomere. At meiosis the copies of the gene are detached as a result of intrachromatid crossing over between the first and last members of the series. Only the master copy remaining in the chromatid is allowed to cross over with its allele in a non-sister chromatid. The detached duplicate copies are subsequently restored to the chromatid by crossing over between one of their number and the master copy. Whitehouse assigns to the synaptonemal complex as one of its functions the holding in place of the detached chromomeres until the time for their reattachment comes around. This hypothesis would predict that in the absence of synaptonemal complexes all detached loops of duplicated genes would be released from the chromatid, and only a few would ever reattach at the appropriate loci. Since such denuded chromatids are presumably incapable of functioning normally, one would expect c(3)G females to be rendered sterile. Since they are capable of producing viable euploid as well as aneuploid offspring, Whitehouse's model seems untenable.

The minimum time interval during which synaptonemal complexes are present in a Drosophila oocyte is 4 days; whereas the maximum time spent by a cystocyte in mitotic prophase is 0.5 days (Koch, Smith and King 1967). In c(3)G homozygotes the time spent in meiotic prophase is halved. Therefore the prolonged time spent in meiotic prophase is required at least in part for the formation and functioning of synaptonemal complexes.

The stubbloid deficiency of Drosophila melanogaster includes the locus of c(3)G. Hinton (1966) has shown that crossing over is abolished in females of
<table>
<thead>
<tr>
<th></th>
<th>+ c(3)G</th>
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<th>+ and c(3)G</th>
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<tr>
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<tr>
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<tr>
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<tr>
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<tr>
<td>S₁₄</td>
<td><img src="image43.png" alt="Image" /></td>
<td><img src="image44.png" alt="Image" /></td>
<td><img src="image45.png" alt="Image" /></td>
</tr>
</tbody>
</table>

10 \( \mu \)
Figure 4.—The combined lengths of all synaptonemal complexes as a function of nuclear volume. The points shown as $S_1$-$S_8$ represent data from serially sectioned nuclei from oocytes in egg chambers belonging to stages 1–8 from Chicago wild-type or c(3)G females. These points each represent one nucleus. The numbers above the points give the number of sections analyzed per nucleus. The numbers in parentheses give the number of sections passing through the karyosome. Each point labeled by a P represents the average value calculated from data derived from sectioned pro-oocytes. The number above the P gives the number of nuclei studied. In the case of c(3)G 14 sections were photographed for one nucleus; 15 for the other. Point P (symbolized by an open circle) is the average value calculated from eight serially sectioned pro-oocyte nuclei from a germarium from a fly homozygous for fused and c(3)G+.

genotype c(3)G/Df(3)sbd$^{103}$ as well as in c(3)G/c(3)G. Crossing over is reduced to about one half of its normal value in females of genotype Df(3)sbd$^{103}$/+. In c(3)G/+ females, on the other hand, crossing over is slightly increased.

Thus c(3)G behaves more like an amorphic than a hypomorphic allele. In the homozygote not even the earliest stages in the formation of synaptonemal complexes can be detected. Furthermore whether alone or in double dose, the pheno-

Figure 3.—Drawings of the light microscopic cytology of the nuclei from gerarial pro-oocytes (PO) and oocytes from egg chambers in consecutive stages ($S_1$-$S_{14}$) from wild-type and c(3)G females. The nucleolus which is drawn as a solid sphere breaks into fragments which disappear by stage 9.
type is nearly the same in terms of crossing over. On the other hand, \( c(3)G^+ \) appears to produce different phenotypes when in single dose, in double dose, and when in combination with \( c(3)G \). Another way of describing HINTON’s results is that \( c(3)G^+ \) can function normally when \( c(3)G \) is present in the same nucleus, but not when its homologue is deficient for the segment missing in \( Df(3)sbd^{105} \). Two alternative explanations suggest themselves. The first is that \( c(3)G^+ \) cannot function normally unless it can pair with another \( c(3)G \) gene, but that either \( c(3)G \) or \( c(3)G^+ \) will do. The second explanation is that the formation of synaptonemal complexes requires the combined action of two closely linked genes, \( S \) and \( t \). Only one dose of \( S \), but two doses of \( t \) are required for proper function. Under this model, wild type = \( S \ t \), \( c(3)G = s^e \ t \), and \( Df(3)sbd^{105} = s^o \ t^o \) (is deficient for both). Females of genotype \(+/c(3)G \ (S \ t/s^e \ t)\) would behave normally since one \( S \) and two \( t \) genes occur per diploid nucleus. Females of genotype \(+/Df(3)sbd^{105} \ (S \ t/s^o \ t^o)\) show decreased crossing over because the requirement of two \( t \) genes is not met.

Our preliminary electron microscopic studies of oocyte nuclei from \( Df(3)sbd^{105} \) /+ females demonstrate that while synaptonemal complexes are present in germarial pro-oocytes (Figure 6), they are not present in oocyte nuclei in chambers in the vitellarium which correspond to stages 2 and 3. Thus the development of synaptonemal complexes is precociously terminated in \( Df(3)sbd^{105} /+ \) females. It follows that the hypothetical \( t \) gene may determine the interval during which the subunits of the synaptonemal complexes are synthesized, and the length of the interval could depend upon the dosage of \( t \).

Since crossing over occurs at about half the normal rate in \( Df(3)sbd^{105} /+ \) females, it is clear that some crossing over is occurring before the synaptonemal complexes have reached their maximum lengths, and it also follows that some crossing over is also occurring during stages 2 and 3 in the wild type-nucleus. The premeiotic replication of DNA occurs within the nuclei of oocytes before they enter the vitellarium (CHANDLEY 1966). Thus meiotic crossing over is occurring long after the massive DNA replication which gives the oocyte its 4C DNA content. Such an argument renders untenable the hypothesis advanced by PRITCHARD (1960) which explains recombination in eucaryotes as due to copying errors occurring at the time of DNA replication.

Meiotic crossing over does not take place in the male of \( Drosophila melanogaster \) (MORGAN 1912), and synaptonemal complexes do not occur in the prophase nuclei of primary spermatocytes (MEYER 1960). According to LE CLEC (1946) in females homozygous for \( c(3)G \) pairing of homologous chromosomes is seen in oogonial metaphases and larval salivary gland nuclei, and somatic crossing over occurs. Somatic pairing and crossing over take place between the homologous autosomes of male \( D. melanogaster \) (STERN 1936). Although somatically
Figure 6.—An electron micrograph of a sectioned nucleus of a pro-oocyte from the germarium of a $Df(3)sbd^{105}/+$ female. The arrow points to a longitudinally sectioned segment of a synaptonemal complex. Dense parallel lateral elements lie to either side of a striated medial complex. m, cytoplasmic mitochondrion; r, rim of ring canal.

Paired salivary gland chromosomes have been investigated at the electron microscope level, there are no reports of synaptonemal complexes associated with them. From the above data we conclude that somatic pairing and somatic crossing over can occur in Drosophila in the absence of synaptonemal complexes.
Somatic crossing over may be widespread in its occurrence in plants and animals, but it takes place at a frequency hundreds or thousands of times lower than is the case for germinal crossing over. The fact that somatic crossing over is relatively common in Drosophila is no doubt due to the occurrence of somatic pairing in these and related insects. However, in most eucaryotes pairing of homologues does not occur in the nuclei of somatic cells. Germinal crossing over generally occurs in both sexes (although often at different rates). Electron microscopists studying a wide range of eucaryotic species (including fungi, flowering plants, annelids, molluscs, crustaceans, spiders, insects, fish, amphibians, birds, lower mammals, and man) have seen synaptonemal complexes inside the nuclei of germ cells undergoing synaptic stages of meiotic prophase (Moses 1964). In those species where gametogenesis in both sexes has been examined with the electron microscope, synaptonemal complexes have been observed in both sexes in cases where crossing over occurs in both sexes. These facts taken together with our data make it seem likely that the formation of synaptonemal complexes is essential for meiotic crossing over in most eucaryotes.

In addition to showing that meiotic crossing over was almost completely abolished in female Drosophila melanogaster homozygous for c(3)G, GOWEN (1933) reported that among the offspring of such homozygotes were matroclinous females and patroclinous males, haplo- and triplo-IV males and females, metamales and metafemales, intersexes, and triploids. He concluded that the c(3)G females were producing eggs, some of which were diploid for the X, for the 4th, for all the autosomes, or for all four chromosomes, and others which lacked the X or the 4th chromosome. GOWEN suggested that nondisjunction during the first meiotic division was the phenomenon producing the exceptional offspring because disomic eggs formed by c(3)G females always contained both grandparental chromosomes. However, although the rate of nondisjunction in primary oocytes was very high, it was not elevated to the point where the chromosomes were being distributed completely at random. Nondisjunction was not increased in c(3)G/+ females. Zygotes containing one or both major autosomes in single or triple dose would be inviable, and such lethal embryos presumably account for the majority of the eggs which fail to hatch (70% in our experiments). If we conclude that the absence of synaptonemal complexes is the cause of the abolition of meiotic crossing over, it seems reasonable to use the same argument to explain the elevated rate of primary nondisjunction.

HINTON (1966) has shown that nondisjunction is increased above the control rate by almost 300 times in Df(3)sbd105/c(3)G females; whereas the rate of nondisjunction in Df(3)sbd105/+ females is only about 40 times that of controls. We know that the formation of synaptonemal complexes is prematurely terminated in Df(3)sbd105/+ oocytes. It follows that, if even abnormally short synaptonemal complexes are formed, the rate of nondisjunction is reduced below the value seen when none are formed.

Can one erect a theory to explain how synaptonemal complexes function during zygonema and pachynema to promote normal disjunction of bivalents subsequently? One obvious explanation would be that bivalents must be held
together by chiasmata which fail to terminalize in order to segregate properly at first meiotic anaphase (Darlington, 1931). Synaptonemal complexes in promoting crossing over would insure that each bivalent contains such a chiasma. While such a hypothesis may describe the true situation in many species, it is untenable at least for female Drosophila melanogaster because there is clear genetic evidence (1) that the fourth chromosomes segregate normally although they cross over very rarely (Grell 1964), and (2) that in the absence of a pairing partner nonhomologous chromosomes may pair during meiosis and segregate normally without having crossed over (Grell 1967).

There is supporting cytological evidence from other species that pairing can occur between nonhomologous chromosomal segments during meiotic prophase. Using the light microscope McClintock (1933) was able to detect such non-homologous pairing during the pachytene stage in Zea mays. Such non-specific pairing occurs over small non-homologous regions when synopsis is interrupted by structural heterozygosity. Non-homologous pairing also takes place in some monoploid individuals when homologous partner chromosomes are absent. At the electron microscope level we have reports that synaptonemal complexes form at sites of such non-homologous associations. For example, in Lilium longiflorum a short complex can form in the region where a univalent folds back and synapses with itself (Moens 1968). Typical synaptonemal complexes are also seen in the microsporocyte nuclei from hybrids between Lycopersicon esculentum and Solanum lycopersicoides (Menzel and Price 1966). In such hybrids synopsis between nonhomologous chromosomes is a common event. Thus it is clear that the subunits of the synaptonemal complex are nonspecific in the sense that they can, if required, form a strutwork binding together nonhomologous chromosomal segments.

The formation when no homologous partner is available of synaptonemal complexes between nonhomologues and their subsequent correct distribution to daughter nuclei demonstrates that while synaptonemal complexes are non-specific in terms of the chromosomes they can zip together, they are essential for proper disjunction. The intimate, precise pairing of homologues so characteristic of larval Drosophila salivary gland chromosomes is presumably due to an attraction between DNA segments having identical or nearly identical base sequences. Such homologous pairing forces may also exist between leptotene chromosomes and may thus provide the necessary mechanism that will align homologues prior to the formation between them of synaptonemal complexes. Previous homologous pairing may function to insure that complexes normally zip only homologues together.

We therefore postulate that the parallel alignment of chromosomal segments characteristic of meiotic synapsis is due to the assembly of an intervening molecular strutwork, the synaptonemal complex of electron microscopists. Meiotic crossing over cannot occur unless synaptonemal complexes are made. However, such crossing over only accompanies the formation of synaptonemal complexes between homologues. If two chromosomes are held together by synaptonemal complexes, they will pass to opposite poles during first meiotic anaphase, whether
or not they are conjoined by one or more chiasmata. Thus meiotic crossing over requires both homology and synaptonemal complexes; whereas disjunction requires only complexes. A major function of a synaptonemal complex is to insure proper meiotic disjunction of the component chromosomes in a bivalent. The poleward movement of chromosomes depends upon the interaction of chromosomal centromeres with traction fibers. Perhaps during the time that the synaptonemal complex holds the members of each prophase bivalent in parallel the adjacent centromeres are positioned in some fashion which makes it impossible for them to move to the same pole at the subsequent anaphase.

An equally attractive hypothesis is that in some species the synaptonemal complexes which perform this function are retained between achiasmatic bivalents or are lost and then synthesized at a second time, nearer to first meiotic metaphase. Such complexes may occur only in the centromeric regions and thus be more difficult for the electron microscopist to detect. In Drosophila females the plus allele of \( c(3)G \) could be required for the synthesis of the subunits utilized in constructing synaptonemal complexes at both times. The plus allele of a gene such as \( \text{claret-nondisjunctional} \) might function only during the second synthesis, since crossing over is normal in homozygous females, while meiotic nondisjunction is greatly (Lindsley and Grell 1968).

The high frequency in man of serious disease associated with chromosomal abnormalities arising from nondisjunction has only been appreciated recently. There are publications (see Sarles et al. 1968) citing family groups in which relatives show nondisjunction involving different chromosomes. Such reports suggest that genes like \( c(3)G \) may exist in human populations.

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**SUMMARY**

Cytological observations were made of oogenesis in *Drosophila melanogaster* females homozygous for the recessive crossover suppressor in chromosome 3 of Gown. Since the formation of synaptonemal complexes is never initiated in the oocyte nucleus, we suggest that the \( c(3)G^+ \) gene is required for the synthesis of structural subunits common to all these organelles. Less time is spent by \( c(3)G \) oocytes in stages which in wild type correspond to zygonema and pachynema. We conclude that the prolonged time normally spent in meiotic prophase is required in part for the formation and functioning of synaptonemal complexes. Since meiotic crossing over is abolished in \( c(3)G \) females, synaptonemal complexes are required for meiotic crossing over. Such crossing over occurs during the time synaptonemal complexes are forming. Females homozygous for \( c(3)G \) are semi-sterile due to the production through meiotic nondisjunction of large numbers of aneuploid zygotes. We suggest that a major function of each synap-
tonemal complex is to insure proper meiotic disjunction of the component chromosomes in a bivalent, whether or not crossing over has occurred between them.

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