homology meets (coming in opposite directions) consists of is a matter of some interest as it would cast some light on the nature of the pairing code. It may be a pair of chromosomes or a pair of nucleotides.

3. In 7b

\[ \begin{array}{ccccccc}
   & F & E & D & C & B & A \\
\hline
N 7 & A & B & C & D & E & F \\
\end{array} \]

Bridge + frag.

Which hypothesis is correct is difficult to determine. In case of hypotheses #1 and #3, the fragment is always the same size—it consists of the equivalent of one chromatin. On the contrary, under hypothesis #2, the size of the fragment would be variable, sometimes very small from a crossover in region 1 and sometimes larger than a one chromatin equivalent from a crossover in region 3. The observed fragments were uniformly large, so hypothesis #2 is probably not correct.

The use of other pericentric inversions is planned. If anaphase bridges are found in inversion heterozygotes where the two break points are equidistant from the centromere, then hypothesis #2 may be valid. Or if one break point is close to the centromere and there are a considerable number of anaphase bridges formed, this would tend to invalidate hypothesis #2.

Also, it is possible to isolate and examine some of the products of non-homologous crossing over if they do not cause inviability or if they do, a trisomic culture can be used.

The occurrence of non-homologous crossing over is relevant to a number of cytogenetic problems—such as whether the translocations found in the progeny of monoploids arise solely from crossing over in duplicated segments and how chromosomal aberrations are formed under natural conditions.

G. G. Doyle

2. The formation of duplications by the induction of translocations between homologous chromosomes and by the transposition of chromosome segments to non-homologous chromosomes.

Translocations between homologous chromosomes will produce chromosomes with duplicated segments in tandem (and concurrently—chromosomes with deficiencies), as shown in the diagram below.

\[
\begin{array}{ccccccccc}
   1 & 2 & 3 & 4 & 5 & 6 & 7 & 8 & 9 & 10 \\
\hline
   1 & 2 & 3 & 4 & 5 & 6 & 7 & 8 & 9 & 10 \\
\end{array}
\]

Dp \[
\begin{array}{ccccccccc}
   1 & 2 & 3 & 4 & 5 & 6 & 7 & 8 & 7 & 8 & 9 & 10 \\
\end{array}
\]

Df \[
\begin{array}{ccccccccc}
   1 & 2 & 3 & 4 & 5 & 6 & 9 & 10 \\
\end{array}
\]
Translocations between homologous chromosomes should not be uncommon. Theoretically they should occur with 1/(n-1) times the frequency of translocations between non-homologous chromosomes, where n is the haploid chromosome number. In maize this frequency should be 1/9.

Undoubtedly many such duplications have been produced in the past by X-irradiation. However, it is very difficult to detect such duplications since they would cause no pollen sterility and would in most cases have no readily observable effect on the phenotype of the plant.

There is a method by which these duplications can be isolated. Kernels or other diploid tissue, such as young ears, which are heterozygous for gene markers are irradiated with the result that duplication-chromosomes containing both a recessive gene and its dominant allele are formed. This irradiated material is crossed as the female with homozygous dominant plants and then crossed in the next generation to recessive testers. The duplication carrying plants can be identified by the fact that their progeny will be mostly of the dominant phenotype, but with a low frequency of recessives. The rationale of this method is explained below:

\[ \text{X-rayed} \]
\[ \text{N} \quad \text{O} \quad \text{A} \quad \text{N} \quad \text{O} \quad \text{A} \]
\[ \text{X} \]
\[ \text{N} \quad \text{O} \quad \text{a} \quad \text{N} \quad \text{O} \quad \text{A} \]
\[ \text{X}_1 \text{ Progeny} \]
\[ \text{If translocation between homologues did not occur} \]
\[ \frac{1}{2} \]
\[ \text{N} \quad \text{O} \quad \text{A} \quad \text{X} \quad \text{aa} \quad \text{all A/a} \]
\[ \frac{1}{2} \]
\[ \text{N} \quad \text{O} \quad \text{A} \quad \text{X} \quad \text{aa} \quad 1/2 \text{ A/a, 1/2 a/a} \]
\[ \frac{1}{2} \]
\[ \text{N} \quad \text{O} \quad \text{a} \quad \text{X} \quad \text{aa} \quad 1/2 \text{ A/a, 1/2 a/Df} \]
\[ \text{If translocation between homologues did occur} \]
\[ \text{Df} \quad (\text{Df}) \quad \text{X} \quad \text{aa} \]
If the Df chromosome does not result in the inviability of the megasponge and the ensuing haploid generation arising from it, then its presence cannot be detected when the testcross is made using it as the female as was done in the experiment to be described.

The presence of the Dp chromosome may be detected in the testcross progeny by the occurrence of a small number of recessives which are formed in the manner diagrammed above and also may arise from intrachromosomal pairing and crossing over—an event believed by Laughnan and Peterson to occur at the $A_b^1$ locus, which is a small tandem duplication.

The genetic marking system also permits the detection of another type of duplication event—the transposition of a genetically marked segment to a non-homologous chromosome. This is a three-break aberration and probably is uncommon. It is shown diagrammatically below:

It may be detected genetically by a 3:1 ratio (instead of a 1:1 ratio) in a testcross progeny. It should be noted that the transposed chromosome segment must carry the dominant gene if it is to be detected.

One hundred kernels of a hybrid between Kys and Mangelsdorf's tester ($bm_2$, $l_1g_1$, $a_1$, $su_1$, $pr$, $X$, $g_1^1$, $l$, $wx$, and $g_1$) were treated with 10,000 r and the plants crossed as the female with Kys. Eighty ears were obtained. Forty-four of the ears showed semi-sterility; in many of the ears the semi-sterility was in longitudinal sectors. A sample of 10 kernels was taken from around midsection of each of the eighty ears, from both fertile and semi-sterile sectors. Thus 800 kernels were
planted and these plants were used as the female in a cross with Mangelsdorf's tester. This resulted in 448 ears which were large enough to be classifiable. The results are given in Table 2.

Table 2

<table>
<thead>
<tr>
<th>Gene</th>
<th>No. of ears with ratios of</th>
<th>Gene</th>
<th>No. of ears with ratios of</th>
</tr>
</thead>
<tbody>
<tr>
<td>seg.</td>
<td>1:0 1:0 (aberrant)</td>
<td>seg.</td>
<td>1:0 1:1 (aberrant)</td>
</tr>
<tr>
<td>Su:Su</td>
<td>219 229 0</td>
<td>Lg:Lg</td>
<td>68 57 2 (c,d)</td>
</tr>
<tr>
<td>Y:y</td>
<td>205 242 1 (a)**</td>
<td>G1:g1</td>
<td>48 79 0</td>
</tr>
<tr>
<td>Wx:Wx</td>
<td>221 225 2 (a,b)</td>
<td>G:g</td>
<td>51 71 5 (e-i)</td>
</tr>
</tbody>
</table>

*Sandbench test on seedlings.
**Letters to designate families with aberrant ratios—will be described in text.

The genes bm2 and a1 produce mature plant characters and it is not worthwhile to check their gene segregation. The Mangelsdorf's tester used was not an a1 tester and therefore the genes a1 and pr were not tested. Of the six genes used in this study, four gave aberrant ratios. Some cases, however, are known to be spurious. All the suspects must be checked cytologically before we can say that a duplication has been produced.

The first case (a) was an ear with 34 Y Wx, 2 Y wx, 5 y Wx, and 1 y wx kernels or 36 Y:6 y and 30 Wx:3 wx. Since it is unlikely that two genes have been duplicated at the same time, it is suspected that this is a contaminant ear. The second case (b) is an ear with 67 Wx:14 wx, this perhaps is a case of transposition—the X² for a 3:1 ratio is 2.4. The ear (c) segregated 1:1 for a form of vivipary—apparently a newly induced dominant mutant—and gave 63 Lg plants and 2 apparently liguleless ones in the sandbench. The seed from this ear was separated into two classes, normal appearing kernels and kernels which had embryos that bulged outward (the embryos apparently late in going into dormancy). The first class when planted in the greenhouse gave 59 Lg:1 "Lg"; the second class gave 36 Lg:3 "Lg". The "Lg" plants were found to have normal ligules on the third or fourth and later true leaves. Therefore this is not a valid case of a duplication. The ear (d) gave 90 Lg and 1 lg. Not enough seed remained to run a test in the greenhouse. In the case of golden-1, five aberrant ratios were found out of total of 127 cases. There are (e) 69 G:1 g, (f) 87 G:1 g, (g) 34 G:1 g, (h) 94 G:1 g, and (i) 60 G:11 g. Golden is not too good a seedling marker in the background used so some or all of the cases may be spurious.

The segregation of Wx:wx can also be observed in the pollen. Tassel samples were collected from 240 plants (3 from each of the eighty families) and the pollen was stained with iodine and examined with the microscope. The results were: all Wx—105 plants, 1 Wx:1 wx—126 plants, and 8 plants which had mostly Wx pollen but with a small percentage of wx. These aberrant plants had the following ratios of Wx:wx (585:6)*, (517:58), (520:7), (514:17)*, (1584:122), (577:20)*, (540:13)*, and (510:8). There seems to be an association with semi-sterility in the pollen and the formation of wx types in these 8 plants. The asterisk following the ratio indicates that it was a semi-sterile plant.
The frequency of plants with semi-sterile pollen was 8.75%, 21 out of 240. One plant was completely sterile.

The occurrence of translocations between non-homologous chromosomes was thus very low if we accept the value of 8.75% as an estimate. Consequently, the frequency of translocations between homologous chromosomes must be very low---1/9 x 8.75% or ca. 1%. No attempt was made to select kernels preferentially from the semi-sterile ears or semi-sterile sectors of the X_1 ears. This would increase the frequency of translocations between non-homologues in the population, but it is probably true that translocations between homologues frequently do not produce semi-sterility—particularly those ones which are especially desired such as those producing duplications of the wx locus. It is known that chromosomes deficient for much of the short arm of chromosome 9 are functional through the megagametozyte.

This method of obtaining duplications needs further examination. There is probably some difficulty in duplicating genes which are close to the centromere, such as X, since a proximal break is required.

G. G. Doyle

3. Chromosome 9 mapping.

Enough 3-point testcrosses and 2-point data are finally available to order the loci provisionally. See Newsletters 33:78 and 32:100 for earlier data. Table 1 presents new 2-point testcross data, combinations with earlier samples, and information from 3-point testcrosses. Table 2 presents new 3-point data. Unquestioned orders are Wx-D_3-Pg_{12}-Ms_2-Gl_{15}-Bk_2-Bf-Bm_4 and Wx-D_3-Ar-V-Bk_2; Wx-Ms_2-Ar is indicated in some sketchy experiments. With addition of data for Wc (Burnham, Newsletter 33:74), the most logical complete map is as follows:

<table>
<thead>
<tr>
<th>Dt</th>
<th>Yg_2</th>
<th>C</th>
<th>Sh</th>
<th>Bz</th>
<th>Bp</th>
<th>Wx</th>
<th>D_3</th>
<th>Pg_{12}</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>7</td>
<td>26</td>
<td>29</td>
<td>31</td>
<td>44</td>
<td>59</td>
<td>62</td>
<td>66</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ms_2</th>
<th>Ar</th>
<th>V</th>
<th>Gl_{15}</th>
<th>Bk_2</th>
<th>Wc</th>
<th>Bf</th>
<th>Bm_4</th>
</tr>
</thead>
<tbody>
<tr>
<td>67</td>
<td>70</td>
<td>71</td>
<td>74</td>
<td>83</td>
<td>108</td>
<td>138</td>
<td>142</td>
</tr>
</tbody>
</table>

Several intervals and orders are still in doubt because of difficulties in isolating 3-point testers in these short intervals. The most uncertain placement is that of Ar and V in relation to Gl_{15}. Although Gl_{15} is easily classifiable, recombination tests with this marker have been very erratic; no definite pattern that would explain the variation has been seen.

Coincidence data suggest that the centromere may be to the right of D_3, near Pg_{12}. This would place D_3 in the short arm, with centromere placement somewhere between the limits of Anderson and Randolph (2-3 units from Wx, Genetics, 1945) and Rhoades and Dempsey (10-11 units, Newsletter 30:42, 51).