

at P and the second, on the same chromosome strand, adjacent and proximal to the T breakpoint. When all four groups are viewed, it is obvious that the percent recombination increases when Modulator is adjacent to the test interval.

Irwin M. Greenblatt

#### 4. Discussion of the above reports.

It is clear that transposition of Modulator from the P locus results in the mutant phenotype, red pericarp. It now appears clear that these same transpositions also cause the mutant phenotype, light variegated, to occur in equal frequency with the red mutant class. This conclusion was first advanced by Greenblatt (1968) and here tested by direct count of spots within the pericarp and again by progeny counts among the backcross offspring of a homozygous medium variegated parent. In both cases, a 1:1 ratio of red to light variegated was found. The conclusion that potential red and light variegated occur in equal frequency applies to all transpositions; there are no transpositions which would produce a red type without a concurrent light variegated type. As pointed out by Greenblatt (1968), this means that transpositions occur during that restricted period in the cell cycle when the P locus is being replicated--not before this time and not after this time.

The discovery that the proximal-distal regions adjacent to the P locus on chromosome 1 receive Modulator in a most strikingly dissimilar manner is exactly the result expected if transpositions were occurring during replication of the chromosome. As outlined in Genetics (1968), the Modulator that moves from the P locus is the one that is newly replicated, i.e., it is the one which is not from the strand that will serve as the receptor site and, in terms of semi-conservative replication, it moves from the newly forming strand to the original strand. It can only move to the original strand in those regions which are themselves in the process of replicating. As reported elsewhere, at the receptor site replication may occur a second time during the single replication of the chromosome. The interpretation of the polarity differences in site locations rests on the pattern of the

chromosome's replication. Thus, the proximal location to P, found to be void of receptor sites, is that region which has already replicated at the time the P locus is replicated. The region immediately distal to P is, at the time Mp is replicated, about to replicate and serves as the most probable receptor site due to the state of replication and proximity to the source of Mp.

The formal interpretation of the pattern of receptor sites is that a short region, extending minimally 3-4 crossover units (it may be larger due to the crossover suppression expected within the immediate vicinity of the breakpoint of a heterozygous reciprocal translocation, the third marker used in these linkage tests), exists which replicates prior to the P locus, and that there is a polar progression of the replication sequence past P into the distal region for an undisclosed length. The recovery of Mp in sites proximal to P beyond the length of chromosome void of Mp is interpreted to mean that the replication sequence is also progressing proximally. Such a pattern of replication has already been described by means of cytological techniques (see Lark, *et al.*, 1971, *J. Mol. Biol.* 58:873). Such a pattern has given rise to the notation of a unit of replication known as a Replicon.

These studies of Modulator transpositions have served to build a model of chromosome replication; the pulse-radioactive labeling of chromosomes has also yielded a view of the pattern of chromosome replication; these two different approaches yield a congruent image of the pattern. Once again, genetic and cytological analysis point to the same conclusion concerning the chromosome. This time the conclusion is that the replicon can now be defined the same way cytologically and genetically.

The data in article 3 above were, to say the least, unexpected, but nonetheless gratifying. Clearly, Modulator causes an increase in crossing-over. It is very difficult to envision such a result if Modulator is within the linear length of the chromosome; if it were, the disparity in homology adjacent to the test interval would be increased and the expectation would be a decrease in crossing-over. As postulated in 1968, Modulator is thought of as being capable of synapsing with but not joined within the linear length of the chromosome and thus it serves to functionally increase synapsis in a region known to be under physical stress at the time of synapsis due to the heterozygous reciprocal translocation.

Irwin M. Greenblatt