to their shape, the chance of spore impaction being greatest on leaves of minimal cross-section (Gregory[1]).

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Direct Effect of Preferential Segregation on the Origin of Polysomy

INVESTIGATIONS of reverse linkage[1,2] in *Saccharomyces* (excesses of non-parental ditype tetrads yielding a frequency of recombination greater than 50 per cent) have revealed that the centromeres of the adenine–inositol chromosome and the proline–lysine chromosome demonstrate affinities for each other at the first meiotic division[3]. (Reverse linkage is not detectable in organisms in which a long heterochromatic region intervenes between the centromere and the euchromatin, permitting random recombination between the centromere and the euchromatic genes.) The centromeres of both chromosomes are of two types, designated 'up' (U) and 'down' (D). In reverse linkage for genes closely linked to these centromeres (genes controlling the synthesis of inositol and proline, respectively) the mating is $U_1D_2 \times D_1U_2$ (Fig. 1). Preferential segregation has been shown to occur in nearly every meiosis of this mating by the close agreement between the distances between inositol and proline obtained by (*a*) one mapping function assuming 100 per cent non-parental preferential segregation (which yielded the distance 36·25) and (*b*) another mapping function which utilized only the frequency of tetratype tetrads and thus was independent of assumptions involving preferential segregation (which yielded the distance 37·50). Thus in nearly all resultant tetrads the gametes contain U_1U_2 or D_1D_2 centromere combinations. Unless recombination occurs between the gene and its corresponding centromere, only non-parental ditype tetrads are obtained. The mechanism controlling the preferential segregation of these non-homologous centromeres is inferred to comprise their synapsis just prior to meiosis I and their passing together to the poles in that condition at the first division. Marker-pairs which are in reverse linkage in one family may appear in direct linkage in a related family. This is inferred to result from the new mating U_1U_2 by D_1D_2.

From these findings it would follow that the mating U_1D_2 by D_1D_2 should produce a high frequency of unequal partitioning of chromosomes at the first division—U_1 passing to one pole and $D_1D_2D_2$ to the other. After the second division two D_2D_2 gametes would be produced disomic, or attached, for the proline–lysine chromosome and two non-viable spores deficient for the proline–lysine chromosome (if they were formed at all). Any hybrid resulting from a mating of one of these disomic gametes with a normal haploid would produce a clone trisomic for the proline–lysine chromosome. It is suggested that polysomy in *Saccharomyces*[4] as well as in all other organisms is the direct effect of this mechanism. It is important to point out that this mechanism differs fundamentally from that involved in the induction of polyploidy since it does not entail an extra division of chromosomes but merely a preferential segregation of chromosomes already present due to an imbalance of the types of centromeres.

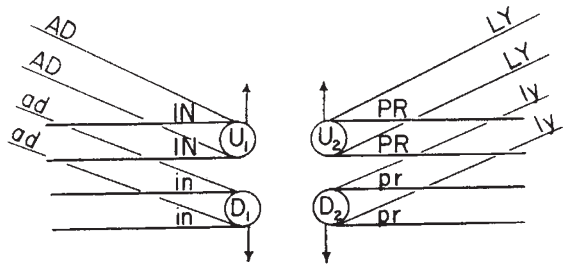

Fig. 1. Diagram showing how reverse linkage occurs in a hybrid U_1D_2 (*IN pr*) D_1U_2 (*in PR*) through the affinities of the U_1U_2 and the D_1D_2 centromeres which orient them on the spindles and consequently direct them to opposite poles. Unless cross-overs occur between the centromere and *PR/pr* or the centromere and *IN/in* only non-parental ditype tetrads are produced

In inbred *Saccharomyces* stocks fertile hybrids are not found frequently and many test matings must be made before one is obtained in which a high frequency of viable four-spored asci occurs. Genetical analysis in this laboratory is confined to those families in which high frequencies of four-spored viable asci occur, thus obviating difficulties which arise due to polysomy, since this phenomenon might obscure the basic question of genic stability upon which our attention has been concentrated. The operation of this mechanism in the formation of trisomics in organisms used for single strand genetical analysis would not generally be detected because chromosomal elimination in the reduction division at the formation of the egg excludes the detection of chromosomal deficiencies resulting from unequal partitioning of chromosomes at the first division. The low viability of eggs from certain females may be due to this type of imbalance. A genome containing a few long chromosomes as in *Drosophila* might be expected to exhibit a low frequency of trisomics among the longer chromosomes even if preferential segregation were a common event, because disomic gametes would produce inviable zygotes due to the extreme genic imbalance and thus eliminate at their origin all trisomic zygotes. In *Nicotiana*, with a larger total number of chromosomes, trisomics might be expected to survive.

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Structure of Turnip Yellow Mosaic Virus

IN this communication we report some of the results of the early stages of an X-ray diffraction study of crystals of turnip yellow mosaic virus[1,2]. The two most important conclusions from the interpretation of the X-ray diagrams concern: (*a*) the packing of the virus particles in the crystal; and (*b*) the arrangement of protein sub-units in the individual virus particle.

Crystals of this virus were first studied by Bernal and Carlisle[3,4], who found a cubic unit cell of side