incompetent non-chromosomal *petite* mutants (for review see Roodyn & Wilkie, 1968). In general, cell division is a necessary prerequisite for the mutation, although ethidium bromide acts as a potent mutagen on non-dividing cells (Slonimski *et al.*, 1968). We have shown that cells of *S. cerevisiae* N.C.Y.C. 239 after growth in glycerol liquid medium (Luha *et al.*, 1971) undergo mutation to *petite* in the absence of cell division on incubation in 2% glycerol; 100% mutation can be approached.

Optimal conditions for the mutation have been investigated. The physiological state of the cells inoculated into the starvation medium is important insofar as the mutation rate of cells harvested during exponential growth is higher than that of cells harvested during approach to stationary phase. Replacement of the starvation medium glycerol with either glucose or ethanol leads to cell death.

Rifampicin (100 µg/ml), an inhibitor of bacterial RNA polymerase, prevents starvation-induced mutation. Sodium nalidixate (50 µg/ml), an inhibitor of DNA replication, and cycloheximide (0.25–0.5 µg/ml) increase the initial mutation rate. The starved cells are also extremely sensitive to mutagenesis by low concentrations of ethidium bromide (0.05 µg/ml). The ethidium bromide-induced mutation is not affected by the presence of rifampicin, but can be partially alleviated with sodium nalidixate.

Starvation-induced *petite* mutation is shown to be preceded by a fall in Q_{O_2} (µl of O_2 taken up/h per mg dry wt.), which suggests that a general degeneration of the mitochondria may be occurring. However, although rifampicin does prevent the mutation it does not affect the loss of Q_{O_2} . Analytical ultracentrifugation on a CsCl gradient of DNA extracted by the technique of Williamson *et al.*, (1971) shows that mitochondrial DNA detectable by this method has been lost after 3h in the presence of ethidium bromide and after 24h in the absence of ethidium bromide.

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A Model for the Transfer Ribonucleic Acid-Messenger Ribonucleic Acid Complex of Protein Biosynthesis

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Peptide-bond biosynthesis takes place in a complex containing aminoacyl-tRNA, peptidyl-tRNA and an mRNA sequence. A structure for tRNA and for this complex can be deduced from the tRNA base-pairing pattern with the addition of two simple postulates. First, the correlations between the structures of the amino acids specified by the genetic code and the corresponding codon-anticodon sites (Thomas, 1970) suggest that aminoacyl group and anticodon lie together in a tRNA molecule. Secondly, combining this first postulate with the observation that a polynucleotide helix in some cases apparently occupies only half of the space within the helix periphery suggests that tRNA may have a tetra-helix structure.

These postulates lead to a single structure for tRNA (disregarding variations in detail). The model demonstrated shows that two such tRNA molecules and an mRNA single helix fit closely together and that a simple rotatory translocation places the peptidyl group in contact with the incoming aminoacyl group (the peptidyl-tRNA must disconnect from mRNA). The model provides a simple account, using polynucleotide components only, of the movement of tRNA and mRNA through the complex on a primitive site. It says nothing about the protein components involved. The present very simple model may require modifications to accommodate some chemical results, e.g. the photo-linkage of thiouridine and cytosine (Yaniv *et al.*, 1969).

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