euglobulin precipitate was removed by centrifugation before the supernatant was freeze-dried. To check the possibility of contamination with serum components, this fraction was checked against anti-(bovine serum), but no lines of identity were formed. From this cell fraction, a basic protein subfraction was isolated after passage straight through a DEAE-cellulose column at pH 7.5 in a 10mM-phosphate buffer. The eluate was bound to a CM-cellulose column at pH 4.6 in an 0.1M-acetate buffer and was removed with 0.2M-hydrochloric acid as a single peak. This basic fraction had a spectrum of antimicrobial activity similar to that possessed by the original fraction, and was used to produce an antiserum.

The antiserum gave three lines of identity against this fraction, and was used to estimate semi-quantitatively the basic protein fraction that had been released from milk cells into milk whey. A technique with a double-diffusion precipitin assay was used (Feinberg, 1959). The increase in the basic protein fraction isolated from milk was closely related to the increase in the antimicrobial activity in the milk whey from cows with an induced mild mastitis.

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Biosynthesis of Yeast Mitochondria: Effects of Rifampicin

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The *petite* mutation in *Saccharomyces cerevisiae* involves an irreversible loss of the ability of the yeast to assemble functional mitochondria. Williamson (1970) has suggested that *petite* mutants might interfere with the action of a mitochondrial DNA-dependent RNA polymerase in its synthesis of a repressor of a nuclear gene coding for an initiator of mitochondrial DNA replication. We have shown that rifampicin, an inhibitor of prokaryotic (Ezekiel & Hutchins, 1968), chloroplast

(Surzycki, 1969) and rat liver mitochondrial (Gadaleta, Greco & Saccone, 1970) RNA syntheses, considerably enhances the *petite* mutagenic effect of ethidium bromide treatment and depresses the mutagenic effects of acriflavine or nitrogen-source starvation treatments, though having no mutagenic effect alone.

Rifampicin, however, is shown to have no effect on the growth rate or respiratory activity of yeast cultured with glycerol as a carbon source, which implies that under these conditions rifampicin does not appear to interfere with transcription of mitochondrial DNA (see also Williamson, 1970). The possibility remained that conditions of glucose repression, under which the ethidium bromide and acriflavine experiments were carried out, might cause a breakdown of a mitochondrial permeability barrier to rifampicin. This possibility was eliminated by the demonstration that rifampicin did not affect the ability of the yeast to recover respiratory activity on transfer from anaerobic culture with glucose as a carbon source to aerobic culture with glycerol as a carbon source.

Wintersberger (1970) also showed that RNA polymerase isolated from yeast mitochondria is not sensitive to rifampicin. Nor is cytoplasmic RNA synthesis in yeast significantly affected by the antibiotic (Dezelee, Sentenac & Fromageot, 1970).

If the effects of rifampicin on the *petite* mutation do result from inhibition of RNA synthesis, then its action must be on a polymerase, possibly extra-mitochondrial, involved in the maintenance of functional mitochondria and representing only a small fraction of total RNA polymerase activity.

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