viscosity of which can be accounted for in terms of components A and B, two transitions in viscosity were observed as KCl concentration was increased. Increasing the salt concentration to 50mM-KCl resulted in a 50% decrease in viscosity, attributed to the polyelectrolyte charge-shielding effect. The second transition, indicated by a 25% decrease in viscosity between 0.5M- and 1.5M-KCl, was examined further. Although there was no variation in the molecular weight, nor any interconversion of mucoprotein components A and B, there was a marked change in the hydrodynamic properties of the mucoprotein. The s^0 values of mucoprotein components A and B decrease from $s_{25,w}^0$ 18.7 and 4.9S to 16.1 and 3.9S respectively when the concentration of KCl was raised from 0.2M to 2.5M. Values of intrinsic viscosity, of K_s (Creeth & Knight, 1965) and of the frictional ratios show that the mucoproteins A and B contracted from expanded hydrated spheres in 0.2M-KCl to asymmetric compact equivalent hydrodynamic ellipsoids in 2.5M-KCl. This transition is only partially reversible, and indicates the occurrence of definite structural changes within the mucoprotein.

The viscosity of component A was highly shear-dependent in 0.2M-KCl, this shear-dependence being almost absent in 2.5M-KCl. Further, at zero shear in 0.2M-KCl the intrinsic viscosity of component A showed an anomalously high increase in viscosity with concentration, reflecting the ability of the mucus to form a gel under physiological conditions. The absence of this anomalous increase in viscosity in 2.5M-KCl indicates that the expanded hydrated structure, as well as the highly polymerized form of the mucoprotein (component A), is necessary for gel formation. The concentration-dependence of shear suggests that gel formation does not involve strongly co-operative interactions (Rees, 1969).

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Mechanisms of Colicinogeny

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Nine colicin factors were examined in *Escherichia coli* K 12 with respect to the effect on colicin synthesis of the hosts *recA* (recombination-deficiency) allele and the relative amount of each colicin that is free or cell-bound. The results can be summarized as

follows: group 1 (colicin factors E1a-16, E1-K30, E2-P9 and K-235), *recA*⁺/*recA* ratio of bound colicin ≥ 16 and bound/free colicin ratio in *recA*⁺ host ≤ 1 ; group 2 (colicin factors Ia-CT4, Ib-P9, V-K30 and V-K94), *recA*⁺/*recA* ratio of bound colicin ≤ 2 and bound/free colicin ratio in *recA*⁺ host > 8 ; group 3 (colicin factor B-K98), *recA*⁺/*recA* ratio of bound colicin ≥ 16 and bound/free colicin in *recA*⁺ host > 8 .

Colicin synthesis may result from de-repression of the colicin operon in group 1, from conversion (i.e. constitutive synthesis) in group 2, and from conversion accompanied by a high rate of vegetative plasmid replication in group 3. A hypothetical group 4, with *recA*⁺/*recA* ratio of bound colicin ≤ 2 and bound/free colicin ratio in *recA*⁺ host, may be impossible.

Relationships between the Uptake of Uracil and Amino Acids by *Candida utilis*

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Candida utilis was grown at 30°C in a minimal medium with ammonia as the nitrogen source. When leucine was added to such a culture, there was no change in either the rate of protein synthesis, as measured by the incorporation of radioactive sulphate, or growth rate. However, when the medium also contained radioactive uracil, the rate of incorporation of this latter into RNA increased by about 50%. Chemical analyses showed that the addition of leucine had no effect on the protein and RNA contents of the cells. Instead, the contribution of exogenous uracil to the pyrimidine nucleotide pools increased from about 40 to 60% of the total, so that the RNA made after the amino acid was added was of greater specific radioactivity.

These findings suggested that leucine increases the rate of uptake of uracil by *C. utilis*. This was confirmed directly. The maximum velocity of uracil uptake was almost doubled in yeast grown with leucine present, whereas the Michaelis constant was unaltered. Experiments with cycloheximide showed that this apparent increase in the number of sites available for uracil uptake depended on protein synthesis. The kinetics of the appearance of the increased uptake of uracil, and also the decay of this activity when leucine was removed from cultures, support the interpretation that leucine acts through an inductive process.

These actions of leucine are restricted neither to this amino acid nor to the incorporation of uracil. Growth in the presence of leucine also stimulated the uptake of uridine, cytosine and adenine as well