The four activities are explainable on the basis of a single thiol-dependent enzyme and a Ping Pong Bi Bi mechanism with an alternate hydrolytic step.

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Changes in Polyribosome Metabolism in *Escherichia coli* K12 after Adsorption of Colicin E3 to Sensitive Cells

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Fixation of colicin E3, a simple protein, to specific receptors in the surface of sensitive bacteria rapidly results in the complete inhibition of protein synthesis. RNA synthesis, including mRNA synthesis, continues without inhibition, but 70S ribosomes extracted from bacteria, after prolonged colicin E3 treatment, show greatly decreased activity *in vitro* deriving from some specific alteration of the 30S subunit (Konisky & Nomura, 1967).

We have now shown that inhibition of protein synthesis induced by colicin E3 is soon followed by the fairly rapid loss of polyribosomes to yield equal proportions of both 70S and 50S+30S ribosomal subunits. This effect cannot be ascribed to increased nuclease activity. Moreover, before the onset of polyribosome degradation, abnormal polyribosome complexes can already be detected, which, unlike normal polyribosomes, dissociate at low Mg^{2+} concentrations. Thus within 1 min of the complete cessation of protein synthesis at least 40% of polyribosomes are demonstrably unstable when the Mg^{2+} concentration is lowered to 1 mM.

'Pulse-chase' experiments *in vivo* indicate that the release of nascent polypeptide chains from polyribosomes is inhibited in colicin-treated cells. In contrast, other experiments indicate that protein synthesis is blocked as a result of inhibition of some initiation step. The changes in ribosome function provoked by the adsorption of colicin E3 may therefore affect a step in the protein-biosynthetic cycle, common to both polypeptide-chain extension

and initiation, as has been suggested for streptomycin (Modolell & Davis, 1969).

Colicin E3 has no effect on ribosomes *in vitro* (Konisky & Nomura, 1967) and like other colicins it does not apparently penetrate the bacterial cell surface. The colicin acts extracellularly, presumably by promoting a specific alteration of the cytoplasmic membrane. Inhibition of ribosome function is not, however, dependent on continued RNA or protein synthesis. The surface-bound colicin must therefore trigger the 'activation' (or inhibition) of some normal cell constituent, presumably membrane-bound, which then effects modification of the 30S ribosomal subunit.

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Activity of Skeletal-Muscle Ribosomes from Intact and Castrated Rats treated with Triamcinolone Acetonide

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Triamcinolone acetonide was administered at doses of 20, 5 and 0.2mg/kg by intraperitoneal injection into intact adult male rats (250g, Carworth CFHB Strain) or similar rats castrated 10 days previously. In each experiment ribosomes were isolated from five control animals (intact or castrated) and five drug-treated animals 12h after the administration of the drug. The amino acid-incorporating ability *in vitro* of the ribosome preparation from each animal was measured over a 20 min incubation period in the presence of diluted liver cell sap and energy (Bullock, White & Worthington, 1968). The method for the isolation of ribosomes was essentially as described previously (Peters, Richardson, Small & White, 1970) except that 0.1M-tris buffer was used in the homogenizing medium and treatment with amylase was omitted.

The means of the amino acid-incorporating activities of ribosomes from intact animals at triamcinolone doses of 20 and 5mg/kg were both 79% of control, and in castrated animals were 73 and 79% of control, respectively. However, at 0.2mg/kg the mean activity of ribosomes from intact animals was 93% of control, whereas that from castrated animals was 77%. When castrated animals were pretreated daily for 7 days with testosterone (2.0mg/kg per day) the mean ribosomal activity 12h after administration of triamcinolone acetonide (0.2mg/kg) was elevated to 90% of