up in concentrations of 20 per cent in lanolin were made to the ears of the animals once daily for periods up to 6 weeks. Biopsies were taken at weekly intervals and the material fixed in phosphate buffered 2 per cent osmic acid and embedded in 'Araldite'. Sections stained with uranyl acetate in methanol and aqueous lead citrate were examined in a Siemen's Elmiskop I electron microscope.

Micro-invasion by basal cell pseudopods was observed in material from 2 weeks' treatment onwards and there was an associated destruction of the superficial connective tissue with fragmentation of the collagen (Figs. 2 and 3). No such changes were observed in control material from animals either untreated or treated with lanolin alone.

It is of considerable interest that hydroxyanisoles as a group can induce micro-invasion, in view of the demonstrated association between micro-invasion and neoplastic change in epithelial cells.

The result with BHA is particularly significant because butylated hydroxyanisole is used extensively as an anti-oxidant in foodstuffs, although it must be pointed out that the concentrations used in these experiments greatly exceed the concentrations of anti-oxidant present in foods. It does not necessarily follow that the effect of BHA in chronic low dosage would be the same as it is in the high dosage used in these experiments. Nevertheless the data on the possible carcinogenicity of BHA are insufficient to exonerate it[4].

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P. A. RILEY

Department of Chemical Pathology,
University College Hospital Medical School,
London, WC1.

P. SEAL

Electron Microscopy Unit,
Imperial Cancer Research Fund,
Lincoln's Inn Fields,
London, WC2.

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Primary Structure Homology within the 23S Ribosomal RNA

OUR understanding of the evolution of the cell will depend largely on our gaining an understanding of the evolution of the cell's translation apparatus, and in this context I wish to discuss the recent finding that the RNA sequences (seen in a T1 ribonuclease digest) surrounding the methylated bases in the *Escherichia coli* 23*S* ribosomal RNA (*r*RNA) are all repeated twice in the molecule[1]. This has been interpreted to mean either that the 23*S* *r*RNA molecule is actually a dimer of two identical halves, or that the 23*S* cistron has evolved through a gene duplication and joining mechanism[1]. The dimer interpretation seems to be ruled out by the fact that there are many more kinds of sequences in the 23*S* *r*RNA than there are in the 16*S* *r*RNA, and so, reasonably, the former should be larger than the latter, if most sequences were singly represented in each[2]. In view of the evolutionary significance of the second possibility, however, I wish to point out that there is at least one other reasonable interpretation of these data (which does not invoke gene duplication) and so they cannot be taken as evidence for an origin of the 23*S* *r*RNA cistron by a duplication and joining involving a smaller cistron.

It seems reasonable to assume that the ribosome contains two sites for *t*RNA binding[3] which are nearly identical in composition, and that the methylated bases reside in (and are essential to the function of) these sites. If these assumptions are valid then the twice repeated segments in 23*S* *r*RNA could have evolved as a result of identical functional constraints operating in their evolution—in other words, this would be a case of "convergent evolution" not "divergence" from a common ancestor.

An additional point that tends to reinforce this alternative interpretation is that the introduction of odd bases into RNA requires information, undoubtedly in the form of RNA sequence in the vicinity of the base to be altered, which could put additional pressure for constancy on those sequences surrounding such bases.

I should point out, however, that certain other evidence actually does suggest an origin of the 23*S* *r*RNA cistron by a gene duplication mechanism. I refer to the reports that in *E. coli* there exists primary structural homology between the 16*S* and 23*S* *r*RNAs, seen in the complete competition between 16*S* and 23*S* *r*RNAs for hybridization with *E. coli* DNA[4,5]. If true, this may mean that 23*S* *r*RNA comprises two halves, each homologous to the entire 16*S* *r*RNA molecule, and so each half homologous with the other. The study, showing the methylated base-containing segments in the 23*S* *r*RNA to be two-fold redundant[1], revealed no similarity between those segments in the 16*S* *r*RNA molecule and their counterparts in the 23*S* molecule, however, so it is still reasonable that these particular redundancies came about through a "convergent" evolution.

CARL R. WOESE

Department of Microbiology,
University of Illinois,
Urbana, Illinois.

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Effect of Actinomycin *D* on the Assimilation of a Cytoplasmic Determinant in Amoebae

CERTAIN cellular properties of the large free living amoebae, *Amoeba proteus* and *Amoeba discoides*, such as sensitivity to streptomycin and nuclear diameter, have been shown to be influenced by self-replicating cytoplasmic hereditary determinants[1]. *A. proteus* is more sensitive to streptomycin and has a greater nuclear diameter than *A. discoides*[2,3]. Micro-injection of small quantities of cytoplasm of *A. discoides* into recipient *A. proteus* results in the transfer and expression of the determinant studied—for example, lowered sensitivity to streptomycin—for a small fraction of the clones derived from cells surviving the operation[1].

The factors which influence the assimilation and presumably replication of non-homologous cytoplasm are unknown. The small percentage of altered clones, comparable with the small percentage of transformants obtained in bacterial studies[4], could be a consequence, for example, either of a small chance of sampling the presumed randomly distributed determinant, or of the reaction of the recipient cell to "foreign" cytoplasm.