artefacts indicate that the cemetery was incorporated into small plots and built on until about AD 250. About 100 Roman coins, minted between about AD 40 and AD 250, have been recovered but, surprisingly enough, the excavators have found very few coins of the period 250 to 400—usually by far the commonest on Roman sites in Britain. Dr Stead infers from this that Roman occupation of the site stopped as abruptly about 250 as the Belgic use of the cemetery stopped in the 40s. The explanation seems to be that about 250 the Romans built the town walls around Verulamium and moved all outlying settlements within the walls. Nobody knows why.

ANTIBIOTICS

How Streptomycin Works

from our Cell Biology Correspondent

IT is well known that streptomycin interacts with ribosomes and somehow blocks protein synthesis and this may be the reason why it kills cells. As long ago as 1961 (*Nature*, **192**, 633), Spotts and Stanier proposed that in sensitive bacteria, streptomycin inhibits the association of 70*S* ribosomes with messenger RNA. Inevitably this proposal—made long before it was realized that the 70*S* ribosome dissociates into its two subunits at the completion of each round of protein synthesis and then re-forms at the initiation of the next round—has turned out to be an over-simplification. According to recent experiments reported by Luzzato, Apirion and Schlessinger in the current *Proc. US Nat. Acad. Sci.* (**60**, 873; 1968), streptomycin prevents the successful initiation of protein synthesis by inducing aberrant initiation complexes. In other words, the *m*RNA, 30*S* and 50*S* ribosomes associate, but peptide bond formation is blocked.

Luzzato *et al.* added streptomycin to cultures of the so-called fragile mutant of *E. coli* which, because of the ease with which it is lysed, is a useful strain for analysing ribosome distributions. They then measured the proportion of ribosomal subunits, 70*S* ribosomes and larger polysomes in the cells. After the addition of the antibiotic there was a striking decrease in the proportions of large polysomes and free 30*S* and 50*S* subunits and an accumulation of 70*S* ribosomes. Pulse labelling experiments with this system suggest that each 70*S*-*m*RNA complex that forms after the addition of the streptomycin is blocked so that protein synthesis does not commence, while identical complexes formed before the addition of the streptomycin continue to synthesize protein. Streptomycin therefore seems to block chain initiation but not chain elongation.

In vitro experiments bear out this conclusion. Streptomycin at concentrations as low as one molecule per molecule of *m*RNA completely blocks protein synthesis in cell-free systems from streptomycin sensitive strains of bacteria programmed with *R*17 RNA, under ionic conditions where initiation is dependent on formyl-methionine incorporation. On the other hand, protein synthesis can continue in a cell-free system if the initiation step is complete when the streptomycin is added, or if initiation is forced by high concentrations of Mg^{++} ions. Furthermore, as expected, a cell-free system from a streptomycin resistant strain is not affected by streptomycin. The exact nature of the initiation complex blocked by streptomycin is not yet known, but *t*RNA is required for the formation of the 70*S* ribosome-*m*RNA complex.

These experiments certainly show that the blockage of chain initiation is sufficient to explain the bactericidal effect of streptomycin although they do not, of course, prove that this is in fact the primary event in its bactericidal action. On the other hand, the results do indicate that the misreading of the genetic code which streptomycin can induce plays no part in its bactericidal action because, at the concentrations at which the antibiotic kills cells, protein synthesis is blocked and streptomycin can have no effect on translation.

MICROBIOLOGY

Polyurethane Biodeterioration

from our Microbiology Correspondent

INCREASING commercial use of polyurethanes has been a noticeable trend in the plastics industry in recent years. The properties of these synthetic polymers have stimulated their application in many ways, as adhesives, hard-finish paints, fabric treatments and foams. One less attractive feature of polyurethane plastics is their susceptibility to biodeterioration, caused chiefly by fungi. In particular, it has been suggested that the polyester type of polyurethane is readily attacked by fungi, whereas the polyether types may be largely resistant. This view of differential susceptibility of polyurethanes has been based on reports of commercially formulated polyurethane products and, because the precise specifications of such formulations are rarely disclosed, it has not been possible to decide whether the observed fungal attack has been on the plastic itself or on some other component in the product.

A critical examination of this question was made by Richard Darby and Arthur Kaplan at the US Army Natick Laboratories and their conclusions are now reported in *Applied Microbiology* (**16**, 900; 1968). The rationale of the approach was to observe the fate of a large series of specially synthesized polyurethanes in the presence of selected test fungi in the hope of finding correlations between polymer configuration and fungal susceptibility. In commercial terms, the results of such a study could underwrite the design of new, fungus-resisting polyurethanes. A hundred polyurethanes were synthesized from four diisocyanates and 25 diols; the latter included alkane diol esters of adipic acid (to produce polyesters) and polyethylene glycols, polypropylene glycols, alkane diols and bisphenols (to produce polyethers).

The polymerizing catalyst in these syntheses did not affect fungal growth of the test species, which included *Aspergillus*, *Penicillium*, *Pullularia*, *Trichoderma* and *Chaetomium*. Darby and Kaplan found that the polyether polyurethanes were indeed poor substrates for fungal growth. Most of the polyethers tested were highly resistant to attack and only low molecular weight linear alkane diols or high molecular weight polypropylene glycols were even moderately degraded. The resistance of these compounds may reside in the length of the carbon chains between urethane linkages, and in the extent of methylene group substitution.