themselves practising. There are other cases, however, where no such feedback is apparently necessary: chickens deafened soon after hatching give all the normal species calls (Konishi, *Z. Tierpsychol.*, **20**, 349; 1963).

Fentress has now given a particularly clear demonstration of a complicated sequence of movements developing in the absence of obvious feedback (*Science*, **179**, 704; 1973). The grooming movements of mice are characteristic of the strain to which the mouse belongs and have several definable components which follow each other with a fair degree of regularity. The forepaws sweep over the snout and various parts of the face and contact the tongue in a complex pattern. Mice which have had one or both forepaws amputated at birth so that they have only stumps also "groom": they sit in the hunched posture characteristic of a normal grooming mouse; their tongues, shoulders and stumps move just as if they had functional paws. They show the same increase in amount of grooming that normal mice do during their third week after birth, and the normal coupling between shoulders and tongue movements occurs even though the tongue has no paws to lick. Most striking of all, the shoulder traces out large amplitude sweeps over the eye and the eye closes at just the right time even though there is no paw there to touch it. The sequence of movements is also similar. Normal mice go from licking to single rapid strokes along the snout and the mice with amputated paws also show this transition.

Although there may, of course, be subtle as yet undetected differences between normal and pawless mice, it is clear that what might be thought an essential stimulus, namely, the touch of the paws on the face, is not necessary either for the development or for the execution of a complex series of movements involving several parts of the body.

TISSUE CULTURE

Fun with Cytochalasin B

from our Cell Biology Correspondent

ADDING a dash of cytochalasin B to cultures of mammalian cells and watching what happens seems to have become a diverting pastime for many cell biologists, and there is no denying that this interesting drug causes cultivated cells to undergo remarkable changes. Perhaps the most dramatic and ultimately useful property of cytochalasin B is that it can, when used in appropriate conditions, cause the enucleation of most cells in a population. Goldman, Pollack and Hopkins (*Proc. US Nat. Acad. Sci.*, **70**, 750 ; 1973), for example, by ingeniously harnessing centrifugal force with cytochalasin B, have obtained populations of enucleated BSC-1 cells and BHK-21 cells attached to coverslips and their data clearly indicate that these enucleate cells are viable. They can, for instance, be removed by exposure to trypsin solutions from the coverslips and then replated whereupon they resume the typical morphology of cultivated fibroblasts (BHK-21 enucleates) or epithelial cells (BSC-1 enucleates).

Obviously the enucleates must contain whatever information, in other words, molecules are necessary for attachment, spreading and form. The enucleates are also capable of pinocytosis and locomotion ; they are susceptible to contact inhibition of locomotion, and Pollack and Goldman have apparently shown that they support virus replication. Presumably, therefore, there is in the cytoplasm of these cells sufficient amounts of the macromolecules involved in these various processes to allow them to continue for several hours after the loss of the nucleus. And one does not need much imagination to devise interesting experiments that exploit that fact. What, for example, happens when enucleate cells are infected with transforming viruses or fuse enucleate cells of one sort or another to nucleated cells using inactivated Sendai virus to mediate fusion? Such experiments should be fun to do and the results may even be rewarding.

Defendi and Stoker (*Nature New Biology*, **242**, 24 ; 1973) have recently drawn attention to another useful property of cytochalasin B, namely its ability to induce general polyploidy in populations of BHK-21 cells and presumably cells of other lines. Regardless of whether or not the target of the drug is a cells microfilament system, and that is a vexed question, in appropriate conditions it causes the nuclei of BHK-21 cells to divide but prevents cytokinesis, the division of the cytoplasm. As a result binucleates accumulate and when the drug is removed many of these binucleates undergo a further round of DNA replication before mitotis and cytokinesis so that many cells emerge tetraploid. As Defendi and Stoker point out, although many drugs induce polyploidy by interfering with the mitotic spindle cytochalasin B induces polyploidy without drastically reducing viability. It ought, therefore, to be possible to use the drug to induce polyploidy in experiments designed to test how the chromosomal constitution of a cell affects the expression of phenotypic characters. In particular it should be possible to exploit cytochalasin B to test the idea, championed by groups led by Harris, by Pollack and by Sachs, that a cell's chromosome balance regulates its tumorigenicity.

In short cell biologists now have at hand a drug that can be used to yield viable enucleates or viable polyploids. The bags from such a happy hunting ground should be considerable.

BACTERIA

Colicide

from our Molecular Biology Correspondent

THE colicins are proteins, secreted by strains of *Escherichia coli*, which have the property of rapidly and specifically killing various related bacteria. This phenomenon has been examined at a number of structural and biochemical levels: in the best documented case, that of colicin E3, the site and nature of the lethal act have now been established, and a certain amount is known about how the intruder insinuates itself into the cell. The target of colicin E3 is the 16S RNA of the smaller ribosomal subunit, which it severs near the 3′ end, with consequent annihilation of biosynthetic function. There are some odd aspects of this reaction, in particular that the colicin will not attack isolated RNA, or even the lone 30S subunit. This can in principle be explained in terms of ribosomal conformation, which, as a variety of independent lines of evidence suggest, undergoes some sizable change when the subunits dissociate ; it has also been suggested, however, that the colicin is not in itself a nuclease, but rather a regulator of a dormant nuclease already present on the ribosome. Some evidence in favour of the first explanation now comes from Dahlberg *et al.* (*Biochemistry*, **12**, 948 ; 1973).

They find that the cleavage of the 16S RNA by the colicin is subject to inhibition by some antibiotics which are known to operate directly on the ribosome. Thus streptomycin, which binds to the ribosome, deranges the assembly of the polypeptide chain, with apparently an accompanying structural disturbance, will prevent cleavage of the RNA by the colicin in streptomycin-sensitive, but not in resistant cells. Two other antibiotics, which are not thought to have the same ribosomal binding site as streptomycin, function similarly, both *in vivo* and *in vitro*. This argues strongly in favour of stringent conformational requirements that specify the circumstances in which the colicin E3 will interact with the 30S subunit. Dahlberg *et al.* report that the selfsame scission of the 16S RNA occurs also when *Bacillus stearothermophilus* ribosomes are exposed to colicin E3, although the sequence at the 3′ end is quite different from that in *E. coli*. This too then apparently reflects a structure-dependent specificity. Two antibiotics, erythromycin and also kasugamycin, which is known to interact with the 16S RNA at the 3′ end, that is to say, in the very