Curtis, Czernik and Tilley have now found (*Radiat. Res.*, **34**, 315; 1968) that carcinogenic changes induced by radiation can also persist in a latent form for at least 9 months. The carcinogenic alteration induced by radiation was manifested only after injection of carbon tetrachloride. During the period from 1 to 9 months there was, however, little evidence of any intracellular recovery from the carcinogenic change. In this respect this particular form of sub-lethal injury seems to behave differently from the damage leading to chromosome aberrations. The carbon tetrachloride could also be regarded as acting as the promoter of carcinogenesis, after initiation by ionizing radiation. The mechanism of promotion in this case is presumably the wave of mitosis induced by the carbon tetrachloride. This is in line with the commonly accepted view that malignancy depends on a stimulus for mitosis in addition to the original change, which may be interpreted as a mutation.

The reason why cell division is a necessary condition for the expression of malignancy may be connected with the suggestion, put forward most recently by Mayneord (*Brit. J. Radiol.*, **41**, 241; 1968), that normal cells can act as mitotic inhibitors. A single cell bearing a malignant mutation would then be prevented from dividing by the influence of surrounding normal cells. Uncontrolled mitosis would begin only when a barrier to this mitotic inhibition was formed by a group of potentially malignant cells. In other words, a single mutant cell cannot express its malignancy until it forms a clone, either through the natural divisions occurring in the organ concerned or through mitosis induced by a promoter.

Peptidyl Transferase

from our Cell Biology Correspondent

In 1964 Traut and Munro suggested, on the basis of experiments on the reaction of puromycin with *E. coli* ribosomes charged with polyphenylalanine, that the enzyme responsible for catalysing peptide bond formation during protein synthesis was part of the 50*S* ribosomal subunit, presumably one of the ribosomal proteins. In the puromycin reaction, the puromycin molecule acts as an analogue of an amino-acyl-*t*RNA, which has the amino group but not the carboxy-*t*RNA group. As a result of nucleophilic attack of the puromycin amino group on the carboxy linkage of the peptidyl-*t*RNA, the puromycin attaches to the peptide chain, which is liberated, free of *t*RNA, from the ribosome. In the current issue of the *Journal of Molecular Biology* (**35**, 333; 1968), Maden, Traut and Munro report further characterization of the puromycin release reaction which strongly supports their original conclusion.

Using an improved assay to distinguish free polyphenylalanine from the substrates of the reaction, they find that, in the absence of added GTP and supernatant factors, puromycin effects the release of polyphenylalanine from washed 70*S* ribosomes and from purified polyphenylalanine-*t*RNA-50*S* ribosomal subunit complexes. The general characteristics of the release reactions with either 70*S* or 50*S* ribosomes are the same; both reactions, for example, are blocked with chloramphenicol, and apparently the minimal requirements for release are simply the presence of monovalent or divalent cations and buffer. Under ideal conditions 70 to 80 per cent of the polyphenylalanine is released.

These results, and the observations that sulphydryl groups which inactivate supernatant factors and specific inhibitors of the GTP reaction in protein synthesis have no effect on the puromycin release reaction, mean that neither GTP nor supernatant factors can be involved in peptide bond formation. And these latest results, together with those Munro reported last year, which show that the 50*S* subunits catalyse the reaction between puromycin and formylmethionine-hexanucleotide (CAACCA-Met-F), eliminate the possibility that the puromycin release reaction is catalysed by a supernatant enzyme which is specifically bound to and protected by the 50*S* ribosome. The only conclusion is that peptidyl transferase is an integral part of the 50*S* ribosome. All the available evidence supports the contention that the puromycin release reaction is brought about by the same mechanism as authentic peptide bond formation. Briefly, the release reaction occurs only with the L form of puromycin and not with the D form; it depends on native ribosomes and on monovalent or divalent cations, and is inhibited by inhibitors of protein synthesis.

If GTP and supernatant factors are not required at the peptide bond formation step in protein synthesis, they are presumably required for the preceding step, the shift of the peptidyl-*t*RNA from the amino to the peptidyl site in the ribosome, which must be effected by a translocase system. This has yet to be proved, but as Maden *et al.* note, well defined systems are available to study this reaction.

The Pursuit of the Receptor

from our Molecular Biology Correspondent

For some time now the noses of molecular biologists, twitching in the wind, have been turning in the direction of nerve functions in general, and the nature of the receptors in particular. Even though no successful attempts at the isolation of a receptor substance from the nerve cell membranes have so far been reported, the methods of protein chemistry have already been applied with considerable effect to study receptors *in situ*. It is, in fact, already allosteric; or at any rate Changeux and Podleski (*Proc. US Nat. Acad. Sci.*, **59**, 944; 1968) have shown that the response of the excitable membrane, in terms of the potential developed, varies in sigmoidal manner with the concentration of the activator carbamylcholine. In the presence of different activators the sigmoidal character may be lost, and the response becomes hyperbolic. This is interpreted in terms of an allosteric receptor protein in which the two conformations correspond to the polarized and depolarized states of the membrane, acetylcholine and other activators binding preferentially to the one form and inhibitors to the other. Be this as it may, the cooperative character of the response seems clear, and the evidence is consistent with the involvement of a single protein species in the interaction with acetylcholine.

This protein is very clearly a quarry worth pursuing, and important progress in this direction has now been made by Karlin and Winnik (*Proc. US Nat. Acad. Sci.*, **60**, 668; 1968), who have brought off a successful attempt at affinity labelling. This is a well-tried