of the many variables. One way to do this is to isolate temperature sensitive mutants of the different proteins involved in cyclic nucleotide metabolism and the preliminary results of such an approach are described by Willingham, Carchman and Pastan (*Proc. natn. Acad. Sci., U.S.A.*, **70**, 2906; 1973). In order to select for mutants of cyclic AMP metabolism Willingham *et al.* made use of the fact that high levels of this nucleotide enhance the adhesiveness of mouse 3T3 cells to plastic. Cells with low amounts of cyclic AMP would therefore be expected to round up and lose contact with the substratum, and this behaviour was observed in one clone of mutagenised cells, after shifting them from 39° to 23° C. The change occurred within 5 min and disappeared after a few hours at the new temperature, when the cells spread out and reattached to the dish. This behaviour correlated very neatly with changes in cyclic AMP inside the cells; 2 min after the temperature shift cyclic AMP was at a minimum, but it soon increased and by 2 h reached concentrations up to five times greater than in control cells before declining. Immediately after the temperature change the mutant cells released cyclic AMP into the medium and this, at least in part, accounts for the drop in intracellular cyclic AMP.

Inter alia Willingham *et al.* also observed granular deposits on the dishes in which the mutant cells had been grown for several days. Electron microscope pictures revealed that cell processes were attached to these deposits rather than directly to the plastic but, apart from showing that they contain some protein, the authors were not able to account for this strange phenomenon. The site of the *ts* mutation in these cells has not yet been identified, though adenylate cyclase and phosphodiesterase appear normal. Somewhat surprisingly the transient change in adhesiveness was seen when cells were shifted either up or down in temperature, and there seemed to be no critical temperature at which the cells responded, suggesting that the mutation may not be as straightforward as one would like.

Animal cells with inheritable differences in cyclic AMP metabolism have also been studied by Gilman and Minna, in two papers in a recent issue of the *Journal of Biological Chemistry* (**248**, 6610; 1973). Rather than isolating mutants in the manner of Willingham *et al.*, they surveyed a variety of already existing rat and mouse cell lines for their ability to respond to catecholamines and prostaglandins with an increase in intracellular cyclic AMP. Cells were classified as either β^+ or β^- (responders and non-responders to β-adrenergic stimulators) or P^+ or P^- (responders and non-responders to prostaglandin E_1). Hybrids made between β^+ and β^- cells were all β^-, at a time when almost all the parental chromosomes were still present, whereas similar hybrids between P^+ and P^- cells were all P^+. It is tempting to assume that the phenotypes β^+ and P^+ are associated with the presence of receptor proteins in the plasma membrane which are capable of activating adenylate cyclase, but it remains to be seen why the regulation of expression of the two phenotypes is so different.

MEDICAL GENETICS

Penrose Honoured

from a Correspondent

A SYMPOSIUM to commemorate Lionel Penrose, who died suddenly last year during a retirement which had curtailed neither the breadth of his curiosity nor the practical application of his experience to the problems of mental defectives and their relatives, was held in London on November 16 and 17 during the 173rd meeting of the Genetical Society.

The first session was chaired by the President of the Royal College of Physicians, C. A. Clark, who pointed out that Penrose was the first medically qualified holder of the Galton Chair. The session was then introduced by B. Childs (Johns Hopkins University, Baltimore) who exhibited the direct and elegant approaches he is using in the study of patients who have specific difficulties in reading, and was even able to supplement the message of his slides by exploiting the technical difficulties due to the screen being smaller than the slide image. J. H. Edwards (Birmingham Maternity Hospital) then spoke of the difficulties implicit in the disjunctive use of monogenic and polygenic in observational genetics and the alternative approach of estimating the frequency and strength of the most powerful allele present.

E. T. O. Slater (Institute of Psychiatry, London) then spoke on the hereditary dementias of adult life, with special reference to Huntington's chorea, a condition in which the heterozygote cannot yet be identified in the interval between fertility and morbidity, and sporadic cases cannot be diagnosed with precision. In the final paper of the session, C. A. B. Smith (Galton Laboratory, University College, London) explored yet another of Penrose's interests with yet another parameter defining gametic correlations, this time with the remarkable property that the series of related constants are further related by simple but unusual numerical operations on their subscripts. The final paradox seemed to imply that, so far as gametic correlations are concerned, where, as well as with whom, our ancestors lived may have contributed appreciably to conventional measures of inbreeding.

The next session, which appropriately dominated the meeting, was a video tape presentation by J. Cohen and L. J. Lawler (University of Manchester) and by R. Penrose (University of Oxford), one of Lionel Penrose's three sons. After an introduction of Penrose's works in pencil, woodcut and oil, and

Interaction of tRNA with 5S Ribosomal RNA

SEVERAL years ago it was suggested that the 5S RNA of the 50S ribosomal subunit is important for transfer RNA-ribosome binding. In particular, it was recognised that the sequence UpGpAp-ApCp of 5S RNA could interact with the TψC loop of tRNA. Evidence has since been accumulating to support this hypothesis; first, it has been shown that the fragment TpψpCpGp specifically binds to a 5S RNA-protein complex; second, non-enzymatic binding of aminoacyl tRNA is inhibited by the same fragment; and, third, two adenines of 5S RNA, which are essential for biological activity and the binding of TpψpCpGp to the 5S RNA-protein complex, are on the surface of the ribosome.

To decide the possible biological significance of these data, Richter, Erdmann and Sprinzl studied the effects of the tetranucleotide (TψCG) on tRNA dependent reactions, and they report their results in *Nature New Biology* next Wednesday (December 5). They found that when the oligonucleotide binds to the 50S ribosomal subunit it inhibits elongation factor (EF)-Tu-directed aminoacyl-tRNA binding, and EF-Tu-linked GTP hydrolysis and ribosome-dependent synthesis of 'magic spot' I. The anticodon loop and the CGA end of tRNA molecules have already been shown to function in protein synthesis. These results show that the TψC loop is almost certainly also involved.

As further evidence the authors note that in a recently proposed three-dimensional model of yeast $tRNA^{phe}$, the TpψpCp is on the surface allowing it, theoretically, to interact with ribosomes, and also that equilibrium dialysis experiments have shown that the TψC loop is not base-paired in solution. Although convinced that the oligonucleotide TpψpCp of tRNA binds to ribosomes, the authors are not completely certain that the binding site is on the 5S RNA and intend to investigate this matter further.

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