the sight, though not the sound, of a remarkable musical composition, complete with semitones, in which the second half was played by inverting the score of the first half, Roger Penrose exhibited an extraordinary series of wooden objects extending from solid representation of impossible objects to relatively simple two-dimensional sliding or slotting puzzles. He ended with a series of self-duplicating objects from the original rhombi demonstrated at the International Genetics Meeting in 1958 in Montreal to self-replicating and self-separating chains of differing units. The remarkable nature of these artefacts was made even more impressive by the manual dexterity of the demonstrator and the depth and lucidity of his commentary. Copies of this tape are available through Professor Cohen (Department of Psychology, University of Manchester).

The symposium continued the next morning, under the chairmanship of J. H. Renwick, with a historical survey and an up-to-date review of the linkage relations of the human red cell groups by R. R. Race (Lister Institute, London). This form of analysis was largely dependent on the work of the Galton Laboratory (under the serial triumvirate of Fisher, Penrose and Harris) where much of the analytical and enzymatic work was developed, and of Race's laboratory at the Lister which provided much of the data, and undertook, by hand, many of the analyses at a speed which defeated the computer. These reviews were followed by R. Ceppelini (University of Turin) who spoke on the linkage relationship of the white cell surface determinants which, unlike those known on the red cell, are defined by several determinants arranged over a segment one or two centimorgans long.

The symposium finished with the Penrose Memorial Lecture, given by Penrose's successor Professor Harry Harris who explored allelic variation in man and reviewed the work of his own laboratory, on electrophoretic evidence of functional enzymatic variants, and related to this data from the mapping of man's best known protein unit of haemoglobin, the beta chain and data on the frequency of those functionless or silent alleles which can be defined by neonatal screening for aminoacidurias.

It was appropriate that a man whose main influence was to expose his own species to the standard of professional scrutiny and objective accountancy established in the genetical study of his parasites, and of his edible and ornamental plants and animals, should be commemorated by a final session in which man was established as the leader, with his own blood, in the objective auditing of claims of selection and chance or, as Kimura might have it, of drift and drive.

HORMONES

Diabetes Control

from a Correspondent

THE role of insulin in the control of plasma glucose concentrations is well known. More recently, the protein hormone glucagon has also been implicated. It has been found that diabetic subjects with high plasma glucose (hyperglycaemia) not only have lower levels of effective insulin activity but also have increased plasma glucagon compared with normal subjects. This may occur because the hyperglycaemic suppression of glucagon release requires insulin. Diabetes is thus a bihormonal disease. Both hormones seem to regulate plasma glucose by their action on the liver, where they are known to have opposite effects on the synthesis and degradation of stored glycogen.

One of the mechanisms by which these hormones exert their opposing actions in liver cells is by stimulation or inhibition of membrane-bound adenyl cyclase, the enzyme which regulates the intracellular concentration of cyclic AMP (Illiano and Cuatrecasas, *Science, N.Y.*, **175**, 906; 1972). Thus it would seem that a decrease in plasma glucagon in a diabetic subject might offset the effect of the lower insulin level. Because glucagon is a protein hormone, one method of depressing its effect would be to produce antibodies against it.

This possibility has now been tested by Epand and Douglas working with immunised rabbits (*Biochem. biophys. Acta*, **320**, 741; 1973). Although bovine, porcine, rat, human and rabbit glucagons are known to be chemically identical, rabbits are still able to produce anibodies to bovine glucagon when the latter is mixed with a suitable adjuvant before injection. Epand and Douglas reasoned that as glucagon is known to stimulate the release of insulin, rabbits whose effective glucagon concentration is decreased by the presence of specific antibodies should have a correspondingly decreased concentration of insulin. Because of the antagonistic effects of the two hormones the resulting concentration of blood glucose should be similar to normal.

To test their theory Epand and Douglas compared the plasma glucose, insulin, and glucagon antibody levels of rabbits immunised with glucagon injections for 4 weeks, with control animals. All the experimental animals produced antibodies to glucagon, but none to insulin. The glucose concentrations in plasma samples of experimental and control animals were virtually the same. The insulin levels of the immunised group of animals, however, were less than half those of the non-immunised rabbits. These results suggest strongly that the presence of glucagon antibodies in immunised rabbits renders them able to maintain normal blood sugar concentrations with a reduced level of plasma insulin. It is thus likely that the presence of glucagon antibodies lowers the concentration of active glucagon in the plasma which, in turn, decreases the secretion of insulin. The combined reduction of the concentrations of the two antagonistic hormones has little effect on blood glucose.

Epand and Douglas also show that immune rabbits can use administered glucose as quickly as the control rabbits in the glucose tolerance test, but without as great an increase in plasma insulin. The production of glucagon antibodies in a human subject has been reported (Stahl *et al., Horm. Metab. Res.*, **4**, 224; 1972). These results therefore suggest a potential use for glucagon antigenicity in diabetic therapy. Less insulin would be required to maintain normal blood glucose levels of a diabetic patient who has been immunised with glucagon antibodies.

PEST CONTROL

Attack on Rodents

from a Correspondent

RODENT pests are most effectively controlled with poisons. Such rodenticidal agents are hazardous, their biological actions are by no means specific for the target species, and their use presents a danger to both man and domestic animals. New approaches to the problem of rodent control, by chemosterilisation and genetic means, are discussed by Marsh and Howard in a recent issue of the *Bulletin of the World Health Organisation* (**48**, 309; 1973). Chemosterilisation has reached the experimental stage of development, whereas the genetic approach is still highly speculative.

The eventual application of both lines of attack will mean, except in instances where an immediate and complete disappearance of pests is required, a gradual and a biologically far sounder depletion of rodent populations than is possible with present methods of control. This steady and more natural dilution of a rodent population density may, depending on the situation, be advantageous, of no consequence or disadvantageous. In instances of rodent-borne diseases a steady decline in numbers would be advantageous; there would be no sudden appearance of disease vectors from poisoned rodents as would occur in the case of a sudden drop in numbers resulting from the use of rodenticides. In areas such as rangeland, crop-producing areas, sewers and warehouses, where complete eradication of pests is not necessary, chemosterilisation and genetic control measures will be entirely satisfactory. And in cases where use of the rapidly effective rodenticides is necessary, chemosterilisa-