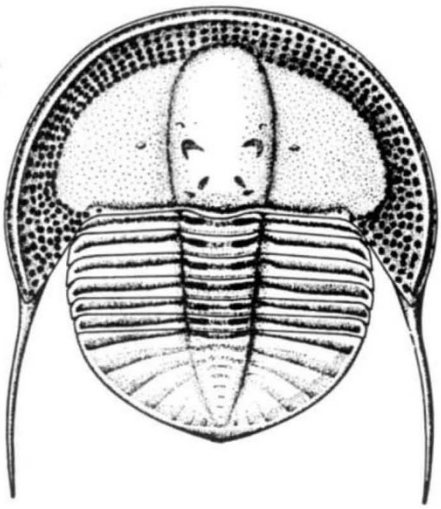
TRILOBITES

An Australian Trinucleid



A new genus and species of trilobite—an extinct marine arthropod which flourished in the early Palaeozoic—has been recovered from Upper Ordovician siltstones near Parkes in New South Wales. According to K. S. W. Campbell and G. J. Durham of the Australian National University (*Palaeontology*, **13**, 572; 1970), it is the first member of the family Trinucleidae to be described from Australia. They have given it the name of *Parkesolithus gradyi* and this figure shows their reconstruction which illustrates the trilobite's semicircular cephalon with its unornamented spines and the six-segmented thorax.

RODENTICIDES

The Search Continues

from a Correspondent

IN Britain, at least, some far reaching changes in the control of rodent pests are on the way, as a result of the development of resistance to anticoagulant rodenticides. Some of these changes were discussed at a symposium on rodenticides held by the Pesticides Group of the Society of Chemical Industry in London on February 15.

In the first contribution, Mr R. Scott (Royal Borough of Kensington and Chelsea) suggested that, besides resistance, the casual manner in which anticoagulant rodenticides were used was an important element in the complex of factors contributing to current increases in infestation by the urban house mouse. This theme was continued by Mr J. H. Greaves (MAFF, Pest Infestation Control Laboratory) (PICL), who pointed out that the apparently inexorable spread of populations of resistant rats which now occupy some thousands of square miles of agricultural land in Britain has arisen largely from the continued use of anticoagulants in the affected areas, a practice which has been unavoidable in the absence of alternative rodenticides that are equally safe and convenient to use.

Dr A. D. Martin (PICL) suggested several physiological mechanisms in rodents which may be open to attack by particular classes of compounds. Among his favourites were new types of anticoagulants and antimetabolites of nicotinamide, but the list included drugs with immunosuppressive, hypoglycaemic and anticorticosteroid activities and many others. Two intriguing suggestions were that the target species might be presensitized or that rodenticides could be potentiated by the respective use of inducers or inhibitors of metabolizing enzymes. The problems, however, of developing basic ideas of this sort were vividly illustrated by further contributions which showed that relatively little progress in specific lines of research had so far been made. This research has included attempts to upset thermoregulation in the mouse (Mr R. A. Davis, PICL) and to remove the limitations of alphachloralose as a rodenticide (Mr J. O. Bull, Rentokil Laboratories) through the use of a combination of compounds.

Dr P. B. Cornwell (Rentokil Laboratories) went on to describe an extensive series of experiments aimed directly at securing improvements in existing rodenticides through the application of microencapsulation techniques. He concluded, however, that though the principle might be valid, basic improvements in the characteristics of release of the microcapsules were needed to yield results of practical value.

The door was held open to a possible, if unpredictable, quick success by Mr F. P. Rowe (PICL), who described a screening project in which compounds submitted by industry are tested for rodenticidal activity on a confidential basis. Doubts were expressed about the immediate commercial prospects for new rodenticides, for resistance is not yet sufficiently widespread to create a major

Picking Up Chick Genes

WHEN an inert nucleus from a chicken erythrocyte is introduced by cell fusion into the cytoplasm of a different cell type derived from another species, it resumes synthesis of nucleic acid and chick specific proteins. Finding out how the chick genes are reactivated is, in itself, an exciting enough prospect, but in next Wednesday's *Nature New Biology*, Harris and his colleagues at Oxford take the technique a stage further by introducing genetic material from chick erythrocytes into nuclei of mouse cells.

The chick nuclei were introduced into the cytoplasm of mouse A9 cells by the now conventional means of inducing cell fusion with ultraviolet inactivated Sendai virus. The mouse cell strain lacked the enzyme inosinic acid pyrophosphorylase, but the chick cells were derived from birds which did not have this disadvantage. The hybrid cells were then grown up in a medium in which cells lacking this enzyme activity die.

Only cells in which the chick erythrocyte nucleus has been reactivated can therefore survive in this medium. But within three or four days of the cell fusion, the heterokaryons enter a mitosis during which the erythrocyte nuclei disintegrate. Any cells which survive after this time must therefore have a functioning chick gene for inosinic acid pyrophosphorylase, presumably located on a chromosome inside the mouse nucleus. The corresponding enzymes synthesized by mouse and by chick can be distinguished by their electrophoretic properties, so Harris and his colleagues were able to show that the survival of cells in the selective medium did indeed result from the synthesis of the chick-coded enzyme.

But looking at the chromosome constitution of the surviving cells showed that no chick chromosomes were present. This implies that during the disintegration of the chick erythrocyte nucleus, some of its genetic material has been incorporated into the mouse chromosomes. That there must be very little such chick material was shown by testing the cells for chick specific antigens on their surface; this is a very sensitive test indeed, and the genes which determine these antigens are widely dispersed on the chromosomes of the chick cell. No such antigens could be found on the surface of these cells, which implies that the extent of chick material picked up by the mouse nucleus must be very small indeed.

What is the state of the chick genetic material in the mouse nucleus? Is it integrated into the mouse chromosome or is there some other form of attachment? The rate at which the gene coding for inosinic acid pyrophosphorylase is lost from cells can be estimated by transferring cells into a normal medium in which the enzyme is no longer needed for survival, and then transferring to a medium which selects against cells which produce the enzyme. This gives an estimate of the number of cells that have lost the enzyme activity during growth in the normal medium. Normal mouse cells lose the enzyme at a frequency of less than 1 in 10^6, but in the hybrid cells the frequency was 20 per cent.

This means that the chick genetic material has been only loosely integrated into the mouse cells. Harris and his colleagues have not yet shown conclusively that the chick genetic material is in the nucleus of the mouse cell, but it seems unlikely that it has remained in the cytoplasm.