with inflammatory disease, the origin of the principal fibril protein is so far unknown, being derived by proteolysis of a larger protein precursor. Work with this "non-immunoglobulin" protein, or protein A, was reported by Dr Franklin's group who have successfully produced an antiserum to protein A, and have found an antigenically related component to the protein in the sera of patients. Dr J. Natvig (Institute of Immunology and Rheumatology, Oslo) and his colleagues reported similar findings.

The final afternoon was devoted to a WHO session on immunopathology of parasitic disorders. Professor S. Cohen (Guy's Hospital Medical School) discussed the *in vitro* IgG antibody which acts in malarial infections at the merozoite stage by blocking the attachment to the erythrocyte and therefore the parasitic cycle. Why then does immunity take so long to develop? There seem to be three reasons—reinfection with serologically different strains, reinfection from foci in hepatic cells, and, third, the antibody induces antigenic variation in the parasite.

The principal chronic disease syndromes in schistosome infections can be attributed to fibrotic lesions associated with the granulomatous response of the host to the schistosome eggs trapped in the tissues. Dr K. Warren (University Hospitals, Ohio) has extracted from the eggs soluble antigens which induce and elicit granulomatous inflammation. Injection of the eggs intravenously into mice produced lung granuloma; particles the same size as the eggs induced no response, but these particles plus adsorbed antigen resulted in granuloma. Supernatant from cultured granulomas and soluble antigens contains a macrophage inhibition factor. The pattern of schistosome infection therefore seems to be that the egg, sequestered in the host tissue by accident, secretes soluble antigen through the shell micropores; sensitized lymphocytes secrete lymphokines and macrophage inhibition factor, with the formation, finally, of granulomas which coalesce to give the pathological condition.

An important new development now being tested by Dr Warren is anti-eosinophil serum. Earlier attempts to prepare this have failed because of lack of large numbers of eosinophils, but Dr D. Colley (VA Hospital, Tennessee) has now developed a technique for producing them in quantity and Dr Warren has been able to prepare an antiserum. Dr Colley described his method in relation to his work with schistosomiasis. Using the same murine model as Dr Warren, he found that injection of proteose peptone into 8-week-old mice infected with *S. mansoni* resulted in a peritoneal exudate of about 50 to 80% eosinophils, which could be further purified to about 90%. Dr Colley found that eosinophilia, a prominent feature of early granuloma formation, followed injection of either eggs or soluble egg antigen, the principal episode being closely correlated with the development of the immune response. Infected mice depleted in T lymphocytes develop antibody to the egg antigen but not the cell mediated response, nor the main peak of eosinophilia. Dr Colley also showed that isolated eosinophils from the peritoneal exudate migrate on exposure to the egg antigen; the cells cluster round and on to the eggs, but on emergence of the miricidium from the egg they are to be found on this and not on the empty eggshell.

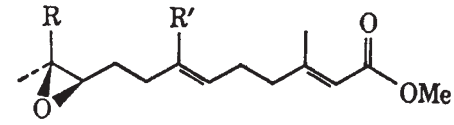
A controversial ending to the meeting was provided by Dr Allison who conveyed the feelings of an informal group who had met to discuss the nomenclature of certain killer cells. For a long time it has been known that T lymphocytes can kill target cells but recently there has been attention on another system—lymphoid cells with receptors for the Fc component of antibody on the target cell which initiates a cytotoxic reaction. Specificity is ensured by the presence of antibody on the target cell. The nomenclature of these cells has become confused and the group suggested the name "K" cell for these killer cells (Greek κλαστος; *klastos*, broken in pieces). Dr Raff warned, however, that as it is possible that cells of both lymphocyte series and the monocyte macrophages system can kill, it seems premature and unwise to group them together under one name. This point will obviously continue to be discussed by immunologists and it is the intention of the originators of this idea that it should be circulated among all the workers in this field.

JUVENILE HORMONE

New Compound Isolated

from our Insect Physiology Correspondent

SCHMIALEK discovered some twelve years ago that the material with juvenile hormone activity present in the excreta of the mealworm *Tenebrio* is a mixture of farnesol and its oxidation product farnesal. Whether these substances are derived from the food of the larva or are metabolized by the insect has never been clarified. But the observation at once suggested that the natural hormone of insects is likely to be a related terpenoid. Farnesol has only a very weak juvenile hormone activity. Schmialek then found, working jointly with Hoffmann–La Roche, that the more lipid-soluble derivative farnesyl methyl ether is far more active. In some insects it has about one quarter of the activity of the natural hormone; in others it is far less active. A year or two later Bowers and colleagues in the US Department of Agriculture found that a compound of more widespread activity could be produced by taking the methyl ester of farnesenic acid and introducing an epoxy ring at C10, 11 ($C_{16}H_{26}O_3$).



This compound provided a very helpful guide for Röller and his colleagues in the purification and analysis of the natural juvenile hormone of the cecropia silkmoth, the principal component of which proved to have the same structure with the exception that two of the three methyl groups which form side chains in the sesquiterpenes, namely those at R and R′, are replaced by ethyl groups ($C_{15}H_{30}O_3$). Shortly afterwards Meyer *et al.* demonstrated the presence in the cecropia extract of a smaller amount of a less active compound in which R=ethyl and R′=methyl ($C_{17}H_{28}O_3$).

Three years ago Röller and Dahm succeeded in showing that the corpora allata of the silkmoth maintained in organ culture will secrete both these juvenile hormones. In the current number of the *Proceedings of the National Academy of Sciences, USA* (**70**, 1509; 1973), Judy *et al.* report a number of refinements of the organ culture procedure; and they have applied these methods to isolate the juvenile hormones secreted by the corpora allata in the adult female of the sphingid moth *Manduca sexta*. They point out that by these means the amount of hormone obtainable from each animal is increased some hundred times; by adding labelled methionine to the artificial culture medium almost quantitative labelling of the side chains of the hormone is effected; and the extraction and purification of the hormone are much simplified.

From 179 cultures containing 738 pairs of glands they were able to isolate 14 μg of pure hormone. Using highly economical procedures, many of them novel and ingenious, they were able to characterize exhaustively the chemistry of the hormone. It contains two components: 5.3 μg of the $C_{16}H_{26}O_3$ compound, known hitherto only as a synthetic "mimic" of the juvenile hormone, and 8.7 μg of the $C_{17}H_{28}O_3$ compound as isolated by Meyer from cecropia. The principal compound isolated by Röller ($C_{18}H_{30}O_3$) is completely absent in *Manduca*. Although the C_{16} and C_{17} compounds are present in approximately equal amounts, the true terpene C_{16} compound when tested on *Galleria* is about 100 times less active than the C_{17} terpenoid—although Judy *et al.* suspect that in *Manduca* itself the difference is much less.