also followed by puffing but not by pupariation. The result is not due to supplementation of endogenous titres by injection because no ecdysteroids are detectable using the new antisera (Horn *et al., J. Insect Physiol.*, **22**, 901; 1976) before or immediately after the 'gate' period. Hormone titre only increases when pupariation commences where the level is 0.11–0.15 nmol g^{-1}. Similarly Whitehead found that stage III tsetse fly larvae are devoid of hormone during sclerotisation of the polypneustic lobes *in utero* as well as during and after birth. The radioimmunoassay first detected ecdysteroids during pupariation and the titre rose eventually to 0.8–0.9 nmol g^{-1} after pupal–adult apolysis.

A more practical observation was that 10^{-5} M ecdysterone and inokosterone added to blood fed through a membrane to *Glossina morsitans* females after ovulation caused 55–80% of the eggs and larvae to be aborted. The systemic action may be at the level of the Malpighian tubules (which were activated *in vitro* to secrete urine (Gee, Koolman and Whitehead)) and the uterine glands which nourish the larva, rather than directly on the offspring.

P. Berreur and coworkers (CNRS, Gif sur Yvette, France) demonstrated a simple device for positioning blowfly larvae before ablation of the ring gland complex containing the gland responsible for ecdysone synthesis. Twelve days after the operation the permanent larvae which result contained 0.08 nmol g^{-1} of ecdysteroids whereas the titre at pupariation was normally ten times this level. Furthermore, wing disks of the ablated larvae were unable to incorporate ^{3}H-uridine normally after injection into the larva. □

Parasite invasion

from V. R. Southgate

The fifteenth meeting of the British Society of Parasitology, entitled Parasite Invasion was held at the Zoological Society of London on October 29, 1976.

PARASITES must, at some stage in their life-cycle, move from one host to another, then find appropriate sites within the host to which they become morphologically, physiologically and biochemically adapted. To do this often requires penetration. Penetration mechanisms of parasites are relatively poorly understood, although recent advances have revealed glimpses of the levels of complexity involved. Therefore it seemed appropriate to review current knowledge and ideas on the mechanisms of parasite invasion and the accompanying physiological changes which take place. It is of fundamental importance to understand these diverse processes which parasites, in order to be successful, have evolved in association with their hosts.

Parasites attach to host cells/tissues in many ways, and the degree of specificity controlling attachments and invasion varies. For example, P. L. Long (Houghton Poultry Research Station) and C. A. Speer (University of Montana) reported that sporozoites of coccidia will enter a wide variety of cell types *in vitro*: however, *in vivo* studies demonstrate that site specificity is strong although factors influencing this are unknown. In contrast, L. H. Bannister (Department of Biology and Anatomy, Guy's Hospital) reported that the merozoite of *Plasmodium* will adhere to several cell types, but penetration only occurs when the apical surface of the merozoite contacts a red blood cell membrane. Thus, invasion depends upon the recognition of specific receptors, and interest has been aroused in the possible relationships of the receptors of *P. vivax* and *P. knowlesi* with blood group antigens of the Duffy class (Miller and Carter, *Expl Parasitol.*, **40**, 132; 1976). The signal for invasion remains elusive. Different groups of viruses have assorted methods for cell attachment and entry: for example, D. A. J. Tyrrell (Clinical Research Centre, Harrow) indicated that influenza viruses attach to cells by 'interlocking' viral glycopeptides to host cell neuraminic acid residues. If attached to cells which are unsuitable for penetration, these viruses are able to detach themselves by secreting neuraminidase. Helminths perceive certain stimuli through complex sensory receptors which apparently initiate behavioural patterns such as release of cytolytic secretion. The schistosome cercariae which invade man respond to rise in temperature and fatty acids of the host skin (Schiff *et al., J. Parasitol.*, **58**, 476; 1976).

Thus, evidence is increasing to suggest a molecular basis for attachment and signals for invasion, which in turn is helping to explain specificity.

What are the actual mechanics of invasion? Our knowledge of the processes involved are limited, especially in the protozoa. It seems likely that well-defined cell organelles are important. Cinemicrographic studies suggest invasion is an active process on the part of the sporozoite of coccidia, and the conoid (a truncated hollow cone of 6–8 spirally arranged fibrils) is capable of protrusion and contraction. The merozoite of *Plasmodium* somehow induces the host cell membrane to invaginate: the invagination 'sucks' the merozoite inwards. Possibly, substances secreted from the rhoptries and micronemes (merozoite organelles) are absorbed into the host cell membrane, thereby altering its biophysical properties. B. E. Matthews (University of Wales, Bangor) demonstrated that helminths pass through tissue barriers using one or a combination of three methods: mechanical invasion; the use of cytolytic secretions; or normal processes, such as feeding.

Why are there alterations in metabolic pathways after invasion? J. Barrett (University of Wales, Aberystwyth) considered two factors to be of major importance: the metabolism of infective helminth larvae is geared to survival whereas that of adults is directed towards reproduction. Also, the physical environment is different, for example, in homeotherms, the temperature and partial pressure of CO_2 rises, and the partial pressure of O_2 falls. Basically, the metabolism of infective larvae is aerobic whilst that of adult parasites is anaerobic—the switch from one to another is controlled genetically and by physical factors. P. I. Trigg (National Institute of Medical Research, Mill Hill) and W. Gutteridge (University of Kent) argued that difficulties associated with *in vitro* culture and separation of protozoan parasites from their host cells for biochemical analysis are the two main reasons for the incomplete picture of physiological and biochemical changes during life-cycles. These problems are not so acute with the extracellular trypanosomes. Consequently, the morphological and metabolic differences between the vertebrate blood and insect midgut forms are comparatively well-documented. Interpretation of the physiological and biochemical changes in coccidia and *Plasmodium* have been based largely on ultrastructural and cytochemical observations, but the recent advances in the continuous cultivation of *P. falciparum* by Trager and Jensen (*Science*, **193**, 673; 1976) and Haynes *et al.* (*Nature*, **263**, 767; 1976) may help to rectify this inbalance.

Advances in techniques in the future will enable a more precise identification of specific receptors at the molecular level on both parasite and host to be attempted.

Parasitologists will also be trying to uncover the nature of signals which initiate behavioural patterns. Advances in *in vitro* culture of protozoan parasites and use of natural barriers *in vitro* for infective larvae will make collection of material for biochemical analyses easier, but problems of clean separation of intracellular protozoa from host cells will have to be overcome. □