ology. Therefore, a book on this topic is relevant at present to consolidate the existing information.

It is no fault of Raabe's that her monograph clearly illustrates the lack of definition inherent in insect neuroendocrine research. Except for proctolin and adipokinetic hormone, the insect neurohormones have generally not been purified and their chemical structures remain unknown. Hence, unlike authors of texts on vertebrate endocrinology, who are able to discuss defined hormones with specific actions, Dr Raabe has had to consider insect neurohormones largely from the standpoint of physiological responses to putative factors present in crude extracts of neurohaemal tissues.

In these circumstances the book's principal merit is in providing a summary of the large quantity of research literature available on the topic. Unfortunately, the information is presented largely in the form of a précis of research results which often leaves the reader bewildered by the array of facts. Topics reviewed range from neurosecretory cells and neurohaemal structures to evidence for the existence of specific neurohormones and their physiological effects. An addendum brings the reader abreast of significant findings that appeared during the preparation time needed for publication.

There are many useful diagrams, figures and tables that help to clarify the textual material by illustration or that summarize the diverse endocrine situations found among insect species. Some figures are overly complex with inadequate captions, however, and I found the method of reference citation, by listing authors and dates throughout the text, rather distracting.

Research libraries and researchers specializing in comparative endocrinology or insect physiology will undoubtedly find the book useful. As a first effort it will probably not be the definitive account of the subject, but it will prove valuable for bringing the serious student of insect neuroendocrinology abreast of events in this rapidly-developing field. □

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Meeting of parasites

R.J.M. Wilson

Malaria and the Red Cell: Ciba Foundation Symposium 94.
Pitman/Ciba Pharmaceutical : 1983. Pp.257. £25, $35

IN APRIL of last year, some 30 parasitologists and biochemists came together in London to exchange ideas on "malaria and the red cell". The theme of the symposium was how the malaria parasite recognizes, invades, catabolizes, alters and finally destroys the red cell. The discussions are recorded in this commendably readable volume, which is both wide-ranging in content and stimulating in that it raises as many questions as it answers.

Invasion of the red cell commences when the merozoite surface coat interacts with red cell membrane sialoglycoproteins. The glycophorins on human red cells are firmly established as specific recognition sites for *Plasmodium falciparum*, but different structures are required for other malaria parasites. Rare types of red cells, deficient in or with modified glycophorins, have been used to assess the effects on invasion of surface charge, oligosaccharide configurations and polypeptide structures proximal to the bilayer.

Apposition of the apical region of the merozoite to the red cell membrane triggers an active invasion process: a localized junction zone is formed within which fine fibrils span a 10 nm gap between parasite and red cell, an electron opaque material is extruded from the parasite and the inner leaflet of the red cell bilayer assumes an increased electron density. As recorded in the book, chief among various speculations is that a strongly cationic parasite protein is inserted into the inner leaflet of the bilayer destabilizing it and dissociating the cytoskeleton so that endocytosis of membrane ensues. These events culminate in the encapsulation of the parasite within the red cell.

The resistance of some hereditary variants of red cells to invasion, as well as changes in susceptibility associated with red cell ageing, can be explained plausibly by this new information. However, it is curious that in the three most prevalent genetically determined pathological conditions of human beings, which have all been related to malaria — sickle cell anaemia, the thalassaemia syndrome and glucose-6-phosphate dehydrogenase deficiency (G-6PD(-)) — selection is probably not exerted primarily at the level of red cell invasion. Rather, invasion is rendered abortive by failure of intracellular parasites to mature.

In parasitized sickle cells, the erythrocyte membrane sustains sickling damage under low oxygen tensions and decreased intracellular *p*H. The protective mechanism in G-6PD(-) is more paradoxical since the Gd^{A-} gene seems to protect heterozygous females (cellular mosaics), but not male hemizygotes (whose cells are uniformly deficient). The sensitivity of thalassaemic and G-6PD(-) cells to oxidative stress has been seized upon as a major mechanism of resistance, since hydrogen peroxide or reactive intermediates of oxygen can decrease parasite multiplication *in vitro*. This hypothesis remains controversial but had several supporters at the symposium and will doubtless be pursued.

Elegant electron micrographs illustrated in the book show how maturing intraerythrocytic parasites modify the red cell membrane in many other ways. "Knobs", clefts and caveolae play a significant role in sequestering infected cells on the venous endothelium and may alter the transport of ions and metabolites into parasitized cells: the intraerythrocytic Ca^{2+} content, for example, increases 10–20-fold (> 90% within the parasite). The induction of new permeability pathways for amino acids and other substrates required by the parasite have an additional significance — the potential for chemotherapeutic effects at the level of the red cell membrane; antimalarials such as chloroquine are believed to form toxic complexes within the food vacuoles of the parasite itself where haemoglobin is digested.

Antigenic changes which arise from the insertion of parasite components into the red cell membrane, as well as by the exposure of previously cryptic isoantigens, are described in several sections of the book. The relevance of autoimmune recognition of red cells in immunopathology and in the induction of immunity to malaria is at present unclear. In patients with anaemia following infection with *P. falciparum*, the role of immune haemolysis seems to be small, but a gross diserythropoiesis was reported at the meeting without identification of the mechanism. At present there is no logical approach to the management of such patients.

Substantial changes also occur in the microcirculation of the spleen during malaria infection. The altered rheological properties of infected red cells contribute to splenic trapping, and cordal macrophages may kill parasites by direct contact or by soluble mediators. Exacerbation of malaria in first pregnancies might be due to parasite sequestration in a new blood reservoir, the placenta, thereby obviating protective mechanisms in the spleen. Another fascinating interaction discussed at the symposium was the modulation of the expression of parasite surface antigens by the spleen. When introduced into a splenectomized host, parasites of an antigen-positive phenotype switch to a negative phenotype due to altered expression by the entire parasite population. The parasite's ability to express alternative molecules on the surface of the red cell may be of adaptive value in the face of spleen-mediated host immunity.

Although vaccines were not widely discussed at the symposium, it is clear that efforts to elucidate the molecular basis for events which either sustain or curtail parasitization of red cells by malaria parasites have gathered considerable momentum. The growing input from the new technologies gives grounds for further optimism. □

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