

Zeitschrift: Acta Tropica
Herausgeber: Schweizerisches Tropeninstitut (Basel)
Band: 34 (1977)
Heft: 1

Artikel: Factors affecting transmission of vector-borne blood parasites
Autor: Baker, J.R.
DOI: <https://doi.org/10.5169/seals-312243>

Nutzungsbedingungen

Die ETH-Bibliothek ist die Anbieterin der digitalisierten Zeitschriften auf E-Periodica. Sie besitzt keine Urheberrechte an den Zeitschriften und ist nicht verantwortlich für deren Inhalte. Die Rechte liegen in der Regel bei den Herausgebern beziehungsweise den externen Rechteinhabern. Das Veröffentlichen von Bildern in Print- und Online-Publikationen sowie auf Social Media-Kanälen oder Webseiten ist nur mit vorheriger Genehmigung der Rechteinhaber erlaubt. [Mehr erfahren](#)

Conditions d'utilisation

L'ETH Library est le fournisseur des revues numérisées. Elle ne détient aucun droit d'auteur sur les revues et n'est pas responsable de leur contenu. En règle générale, les droits sont détenus par les éditeurs ou les détenteurs de droits externes. La reproduction d'images dans des publications imprimées ou en ligne ainsi que sur des canaux de médias sociaux ou des sites web n'est autorisée qu'avec l'accord préalable des détenteurs des droits. [En savoir plus](#)

Terms of use

The ETH Library is the provider of the digitised journals. It does not own any copyrights to the journals and is not responsible for their content. The rights usually lie with the publishers or the external rights holders. Publishing images in print and online publications, as well as on social media channels or websites, is only permitted with the prior consent of the rights holders. [Find out more](#)

Download PDF: 13.09.2025

ETH-Bibliothek Zürich, E-Periodica, <https://www.e-periodica.ch>

Medical Research Council, Biochemical Parasitology Unit,
Molteno Institute, Downing Street, Cambridge, CB2 3EE, England

Factors affecting transmission of vector-borne blood parasites

J. R. BAKER

Introduction

It is the intention of this review to discuss adaptations which facilitate the transfer of some haematozoic («blood-stream») parasites (protozoa and helminths) from one host to another; the scope will be arbitrarily restricted by omitting tick-borne parasites (Class Piroplasma), which will be dealt with in a subsequent paper by Burgdorfer (1977), the relatively unimportant haemogregarines and – on the grounds that they are not parasites of the circulating peripheral blood – the schistosomes.

All blood parasites are faced with at least three problems in assuring the continuance of their life cycles: (i) locating, (ii) entering and (iii) leaving a host. A fourth problem, evading the host's defence mechanisms, is equally important but is beyond the scope of this article. All haematozoic helminths (Nematoda Filarioidea) and protozoa (except *Trypanosoma equiperdum*, rather a parasite of serous fluid than of blood) have a vector, the haematophagous habit of which contributes largely if not entirely to solving problems (i) and (iii) and, concerning Haemosporina and salivarian trypanosomes, problem (ii) also.

1. Transmission from invertebrate to vertebrate host

a) Morphological adaptations

Most parasites of this group (Haemosporina, *Leishmania*, salivarian *Trypanosoma*) are passively injected with the vector's saliva. Others actively penetrate the host's skin via the puncture produced by the feeding vector, or elsewhere (subgenus *Schizotrypanum*, Nematoda Filarioidea), and so reach either blood or subcutaneous serous fluid without the need for special adaptations to aid penetration of skin or mucous membrane. Many of them are, however, modified in other ways for this part of the life-cycle. In the Haemosporina, a

Correspondence: Dr. J. R. Baker, Molteno Institute, Downing Street, Cambridge, CB2 3EE, England

particular stage – the sporozoite – has evolved for this purpose, and also for the subsequent penetration of tissue cells. Most of its morphological specializations – elongate shape, motility and, perhaps above all, the anterior complex of polar rings, rhoptries and micronemes are probably directed mainly toward cell penetration in the vertebrate host (Sterling et al., 1973). Its shape however is doubtless conducive to easy passage down the insect's narrow hypopharynx, and its possession of a thick, three-layered outer pellicle may play a part in «cushioning» the organism's abrupt transfer from one environment (insect saliva) to another (vertebrate blood) presumably very different in osmolarity, acidity, composition, and – often – temperature (Duncan et al., 1960; Garnham et al., 1960; 1961; Vanderberg et al., 1961; Garnham, 1963; Terzakis et al., 1966; 1967; Desser and Wright, 1968; Desser, 1970; Terzakis, 1971; Aikawa, 1971; Klei, 1972; Sinden and Garnham, 1973; Sterling et al., 1973; Sterling and De Guisti, 1974; Wong and Desser, 1976).

Until recently there has been no evidence of the development within the phlebotomine vector of special forms of *Leishmania* destined to re-invade the vertebrate host. However, Killick-Kendrick et al. (1974) and Molyneux et al. (1975) have demonstrated the development of hemidesmosomes within the flagellar sheath attaching promastigotes of *L. mexicana amazonensis* to the cuticle of the oesophageal valve of the sandfly *Lutzomyia longipalpis*, and the differentiation in the pharynx of similarly attached forms resembling opisthomastigotes (Hoare and Wallace, 1966). These forms had a cristate mitochondrion but its volume appeared to be reduced, and they are probably pre-adapted to life in the vertebrate host; the reverse transformation (amastigote to promastigote), which occurs when *Leishmania* is removed from its vertebrate host and cultivated in vitro, involves an increase in size of the mitochondrion (Rudzinska et al., 1964; Creemers and Jadin, 1967).

The salivarian trypanosomes (subgenera *Trypanozoon*, *Nannomonas* and *Duttonella*) produce morphologically distinct metacyclic trypomastigotes in either the salivary glands (*Trypanozoon*) or proboscis (*Nannomonas* and *Duttonella*) of their vectors (*Glossina* spp.). It is generally believed that only these metacyclic forms are capable of reinfecting a vertebrate host; this belief has been rather light-heartedly questioned by Lumsden (1972), but there is no firm evidence opposing it. The metacyclic trypomastigotes of *Trypanosoma* (*Trypanozoon*) *brucei* spp. are pre-adapted both morphologically and physiologically to life in the vertebrate host. Presumably the re-acquisition of the trypomastigote form itself is part of this pre-adaptation. In addition they re-develop the external surface coat: a layer external to their limiting unit membrane composed of glycoproteins of molecular weight about 65,000, which constitute the variable antigen presented to the vertebrate host (Wright and Hales, 1970; Newton et al., 1973; Steiger, 1975; Cross, 1975). By sequentially changing these antigens, the parasite survives the onslaught of the host's immune defense mechanism (Vickerman, 1969b; 1971; 1974a, b). The coat is missing from most

developmental stages in *Glossina*, but is secreted by the metacyclic trypomastigotes in the salivary glands (Steiger, 1971; 1973). A similar phenomenon occurs with *T. (Nannomonas) congolense* (Vickerman, 1969a; 1974b; Vickerman and Evans, 1974) but – although the coat is present in haematozoic trypomastigotes of *T. (Duttonella) vivax* (Mühlpfordt, 1975), and absent from epimastigotes in the vector (Vickerman, 1973) – there is as yet no evidence that it is produced by the metacyclic stage of this species (Vickerman and Evans, 1974). Trypomastigotes and epimastigotes of *T. brucei* in the vector have a well-developed, extensive mitochondrion, with a mean volume of $12 \mu\text{m}^3$ in the former stage; it is much reduced in size and complexity in the metacyclic and haematozoic trypomastigotes, having mean volumes of $1.4 \mu\text{m}^3$ in the former and 0.3 and $3.3 \mu\text{m}^3$ in slender and stumpy haematozoic forms respectively (Böhringer and Hecker, 1975). A similar cycle of mitochondrial regression and expansion occurs in *T. vivax* and *T. congolense*, though it has been less well studied (Vickerman, 1974a); its significance will be discussed in section 1 b.

Metacyclic trypomastigotes of the subgenus *Schizotrypanum* (e.g. *T. cruzi*) are not injected with saliva but are expelled from the vector's hindgut when it defaecates during or immediately after feeding (Hoare, 1972). Presumably by active movement, they penetrate either the intact skin or the puncture wound made by the feeding vector; according to Hoare (1972, p. 355), experimental infection through intact skin «invariably succeeded». Also, if infected faeces is deposited on or carried to mucous membranes (e.g. conjunctiva), the metacyclic forms can actively penetrate the membrane. In spite of this ability, there is no morphological evidence of any special penetrative organelle in the metacyclic trypanosomes, unless indeed the fact that they «are very mobile forms» (Hoare, 1972, p. 355), «schlanker als die plumpen Blutformen» (Brack, 1968), be regarded as an adaptation to penetration.

The infective third-stage larvae of the Nematoda Filarioidea usually if not always migrate actively down the proboscis sheath of the feeding vector, and penetrate the vertebrate host's skin through the puncture wound; there seems to be no suggestion that these relatively large organisms can penetrate intact skin (Faust et al., 1970; Muller, 1975) and therefore probably no special morphological adaptation, other than motility and their characteristically elongate «fili-form» shape, is required to aid in penetration.

b) Physiological adaptations

Little or nothing is known of physiological adaptations of the haemosporine sporozoite to life in the vertebrate host. Trefiak and Dessler (1973) suggest that the lipoprotein crystalloid material, found in ookinetes and early oocysts of many Haemosporina (including *Plasmodium* spp.), and in sporozoites of some *Leucocytozoon* spp. and *Haemoproteus metchnikovi*, is a food reserve¹ which, in

¹ An interpretation questioned by Terzakis et al., 1976.

those species having very large oocysts (*Plasmodium*), is used during oocyst growth. In contrast, in *Leucocytozoon* spp. which characteristically produce small oocysts with few sporozoites, some crystalloid is retained within the sporozoites, which may enable the known extracellular survival of the latter in their vertebrate hosts for up to 11 days; *Plasmodium* sporozoites, lacking crystalloid, disappear from the host's circulation within 1 hour of injection. The oocyst of *H. metchnikovi* is intermediate in size between those of *Leucocytozoon* and *Plasmodium*. Howells et al. (1972) collated evidence that the sporozoite of *P. berghei* has cristate mitochondria which are presumably functional since succinic dehydrogenase and cytochrome oxidase were identified. However the pre-erythrocytic schizonts of the same species apparently synthesize only the latter enzyme; their mitochondria lack cristae and are presumably non-functional. Thus, unlike the situation in the salivarian trypanosomes, the functional mitochondria of the sporozoite (and the oocyst from which it was derived) are an energy source related to its own activity (cell penetration etc.) rather than a pre-adaptation to its subsequent intracellular development in the vertebrate host.

In *Leishmania* spp., the cyclical proliferation and regression of the mitochondrion (section 1a) suggests that there may be a correspondingly fluctuating functional cycle as occurs among the salivarian trypanosomes, but there is as yet no direct evidence at this stage of the life cycle. More is known of the salivarian trypanosomes, especially the subgenus *Trypanozoon* (section 1a): in the trypomastigote and epimastigote stages in the vector, the mitochondrion is well-developed and fully functional, whereas in the metacyclic and haematozoic trypomastigotes it is only partly functional, if at all; in the long slender haematozoic trypomastigotes it is functionally repressed (Vickerman, 1962; 1965; 1971; 1974a; Newton et al., 1973). Structurally, this regression begins in the epimastigote stage (Steiger, 1973). In *T. vivax* and *T. congolense* repression of mitochondrial activity in the haematozoic forms is incomplete (Vickerman, 1965; 1969; Vickerman and Evans, 1974). Thus the metacyclic trypomastigotes of *T. brucei* have developed in the invertebrate host physiological and morphological features presumably fitting them for survival when introduced into the vertebrate host, in which they immediately transform to slender haematozoic trypomastigotes. This is probably broadly true also of members of the subgenera *Nannomonas* and *Duttonella* though, as mentioned, the mitochondrial changes are less dramatic.

There is at present no evidence of any physiological adaptation of metacyclic trypomastigotes of *T. cruzi* or other stercorarian trypanosomes to prepare them for life in a vertebrate, but this may reflect merely our present ignorance of these parasites' physiology in general. It does not seem, however, that *T. cruzi* undergoes the extreme variations in respiratory physiology characteristic of *T. brucei* (Brack, 1968; Vickerman, 1974a; Brener, 1976).

There is a similar lack of evidence of physiological adaptation of the infective, third stage larvae of the Filarioidea, though the subject has been studied

little, if at all. The fact that earlier larvae are not infective (if indeed it is true; certainly the first stage larvae or microfilariae cannot continue development in the vertebrate host) suggests however that there must be some physiological preadaptation of the infective larvae to life in the vertebrate host.

Virtually nothing is known in all these groups of the factors involved in the organospecificity necessary for the parasite's onward transmission; if malarial sporozoites or *T. brucei* metacyclic forms, for example, do not enter the vector's salivary glands, they cannot reach the next vertebrate host; yet of the stimuli which induce them to undertake this essential journey, we know nothing.

2. Transmission from vertebrate to invertebrate host

a) Morphological adaptations

Since all members of this group depend upon passive ingestion by the feeding vector they have few if any morphological specializations for this phase of the life cycle. Gametocytes of Haemosporina are surrounded by what appears to be a thick, many layered pellicle (Aikawa, 1971; Aikawa and Sterling, 1974), though Sinden et al. (1976) cast some doubt on its structure, at least in *P. yoelii nigeriensis*, by describing it as a sub-plasmalemmar «discontinuous system of flattened membrane-bound sacs». Whatever the details of its structure, it is possible that this complex pellicle could be regarded as one such specialization, perhaps protecting the parasite in some way while it remains in the host's blood waiting to be ingested by a vector, or protecting it from the latter's digestive juices after ingestion. Possible changes in mitochondrial complexity within the developing gametocyte will be mentioned in section 2 b.

The microfilariae, or haematozoic first-stage larvae of filarioid nematodes, are not known to have any particular morphological feature related to transmission to their vectors, except of course their small size which enables them to pass up the latter's proboscis; although elongate (150–350 μm), none is more than 10 μm in diameter (*Wuchereria bancrofti*) – not much more than an erythrocyte – and many are considerably thinner – e.g. *Dipetalonema streptocerca*, about 3 μm (Muller, 1975). The persistent egg shell or sheath of certain species (*W. bancrofti*, *Brugia malayi*, *Loa loa*) perhaps constitutes a protective device similar to the malarial gametocytes' thick pellicle, mentioned above.

b) Physiological adaptations

The work of Hawking et al. (1968) has revealed a previously unsuspected adaptation of gametocytes of *Plasmodium* which enhances their chances of onward transmission to a vector – periodicity. With the exception of *P. falciparum*, all the species studied (*P. knowlesi*, *P. cynomolgi* and *P. cathemerium*) showed cyclical maturation of gametocytes in the vertebrate host's peripheral blood, together with a limited duration of mature infective life (5–12 h only).

The proportion of mature gametocytes reached a peak at about midnight and so corresponded well with the feeding activity of the nocturnal mosquito vectors. This synchronization resulted from the fact the gametocytes took a constant time (a few hours longer than the sexual cycle) to develop to maturity after their liberation from erythrocytic schizonts, a process which itself is remarkably synchronous and tends to occur at a similar time each day (or second or third days) for any particular species. The synchronization of the asexual cycle was shown to be entrained by the host's circadian temperature cycle, itself governed by activity and hence (normally) by the diurnal rhythm of light and dark. All of this serves well, as the authors put it, «to make the gametocytes match the mosquitoes». This elegant phenomenon presumably does not apply to other genera of Haemosporina which lack erythrocytic schizogony.

Apart from this, nothing is known of pre-adaptations, if any, of haemosporine gametocytes for their subsequent development in the vector's midgut. They remain dormant in the vertebrate's blood and continue development only after withdrawal of the blood, either by the vector or in vitro; the factors controlling this are not well understood (Bishop and McConnachie, 1956; 1960; Sinden and Croll, 1975). Immature gametocytes have acristate mitochondria, but cristae develop in mature forms perhaps in preparation for life in the vector. Succinic dehydrogenase could not be detected, though it (and cytochrome oxidase) were found in the oocyst (Howells et al., 1972).

As stated in section 1b, little is known of the physiology of *Leishmania* spp. but the apparent cyclical changes in mitochondrial volume and complexity probably reflect a similar fluctuation in activity. Simpson (1968) has shown that the amastigote-promastigote transformation occurring when *L. donovani* is transferred from hamster spleen to culture in vitro involved a 5–7 fold increase in oxygen consumption (QO_2) but the respiration of both forms was sensitive to cyanide, sodium amytal and antimycin A. This implies a quantitative rather than qualitative change (increase) in mitochondrial activity, presumably associated with the increase in mitochondrial volume (section 1a). There is no direct evidence of similar changes in vivo, when amastigotes are ingested by a sandfly, but it seems likely that they do occur.

Much more is known of the physiological changes associated with transfer of *T. brucei* and other salivarian trypanosomes from vertebrate to invertebrate hosts, although most relevant studies have been made on the transfer of parasites from their vertebrate hosts to cultures in vitro, which may not fully mimic the natural transfer to the vector. Essentially, the changes are the reverse of those occurring during development of metacyclic trypomastigotes (section 1b). In *T. brucei* spp., the slender haematozoic forms, with an inactive and structurally reduced mitochondrion, transform in response to unknown exogenous and/or endogenous stimuli into shorter, stumpy forms, in which some mitochondrial development has occurred (Steiger, 1973) and some enzymic activity is demonstrable – e.g. oxidative decarboxylation, but not the cytochrome sys-

tem (Bowman et al., 1972). These forms are generally believed to be those which continue development in the vector (though this has been challenged by Ormerod et al., 1974), but develop no further in the vertebrate host. Under favourable conditions after ingestion by a tsetse fly, they undergo a two-stage physiological transformation; firstly to procyclic forms (Newton et al., 1973), probably begun in the crop (Harmsen, 1973) and completed in the endoperitrophic space of the midgut lumen (Evans and Brown, 1971; Vickerman, 1974c). The mitochondrion increases in volume and complexity (Steiger, 1973), succinoxidase activity appears and the parasites «switch» from glucose to proline as a substrate for oxidative metabolism (Brown et al., 1973). Secondly (after about 48 h), either in the endoperitrophic space or after reaching the ectoperitrophic space (perhaps by penetrating its soft anterior portion – Freeman, 1973; 1974), more mitochondrial proliferation occurs and a cyanide-sensitive cytochrome system appears; these «proventricular» forms revert to the use of glucose as their respiratory substrate (Brown et al., 1973; Baker et al., 1974; Vickerman, 1974c).

The factors producing the well-known insusceptibility of a majority of tsetse to the establishment of infections with *T. brucei* are little understood (Ward, 1968); genetic factors may be involved, as may the state of the peritrophic membrane. Geigy et al. (1971) have shown that the species, but not the individual, of the host providing the infective blood meal also plays a part in determining infectivity.

Although not strictly relevant to this review, the «classical» view that trypanosomes migrate from proventriculus to salivary glands of the tsetse via the food canal and hypopharynx of the proboscis has recently been challenged. Mshelbwala (1972) and Otieno (1973) recorded the presence of *T. brucei* in the haemocoel of infected *Glossina*, and Evans and Ellis (1975) provided electron microscopical evidence of the penetration of midgut cells by *T. b. rhodesiense*, perhaps en route for the haemocoel.

Little is known of the physiology of *T. congolense* and *T. vivax* within *Glossina*, but there is evidence (from NAD diaphorase reactivity and the oxidation of substrates such as glucose, glycerol, glycerophosphate and succinate) that the mitochondrion of the haematozoic trypomastigotes is partly functional, probably to a similar extent to that of stumpy haematozoic forms of *T. brucei* which they also apparently resemble in lacking a functional cytochrome chain – their respiration being insensitive to cyanide (Vickerman, 1969; Vickerman and Evans, 1974). There is no evidence of functional mitochondrial repression such as occurs in long slender haematozoic *T. brucei*, and therefore (as far as is known) no special haematozoic form pre-adapted to life in the vector. The morphological evidence (mitochondrial proliferation) suggests that a cytochrome system develops and respiration probably becomes cyanide sensitive in the forms of both species developing in *Glossina*, as they do in *T. brucei*. There is even less evidence for cyclical physiological changes in *T. cruzi* (or other sterco-

rarian species), though, as mentioned in section 1b, this may be due in part to lack of relevant studies. Recent evidence suggests that *T. congolense* and perhaps other species of *Trypanosoma* may show diurnal fluctuations in parasitaemia comparable to those of microfilariae, discussed in the next paragraph (Hawking, 1976).

I am not aware of any comparable studies on the respiratory physiology of microfilariae or subsequent larval stages of filarioid nematodes. Some of the former do, however, show par excellence the phenomenon of periodicity – not of maturity, as with *Plasmodium* spp., but of location in the vertebrate host's vascular system. This phenomenon has been known for many years – according to Hawking (1967) it was first recorded by Manson in 1879 – and it has long been related with the temporal feeding habits of the insect vectors. Amongst the Filarioidea parasitizing man, *Wuchereria bancrofti* and *Brugia malayi* exhibit pronounced nocturnal periodicity – i. e., their microfilariae are predominantly in the circulating peripheral blood at night and are restricted to the blood vessels of the lung during the day; *Loa loa* shows the reverse phenomenon, diurnal periodicity. This is correlated with the feeding habits of their vectors, the first two species being usually transmitted by night-biting mosquitoes (mainly *Culex pipiens fatigans* and *Mansonia* spp. respectively) while the last is transmitted by *Chrysops* spp. which feed only during daylight. A form of *W. bancrofti* on certain islands in the Pacific ocean (Polynesia, Fiji, Samoa and Tahiti) is transmitted by diurnally active mosquitoes (*Aedes* spp.) and shows a corresponding diurnal periodicity, though this is less clear-cut than the nocturnal periodicity of the commoner form (Muller, 1975). Duke (1972) has shown that a simian form of *L. loa* in West Africa has nocturnal periodicity, the largest number being present in the peripheral blood between 1900 and 2400 h and almost none between 0600 and 1700. This form is transmitted by species of *Chrysops* feeding almost entirely between 1700 and 2000 h. Human *L. loa* in the same region showed the typical diurnal periodicity (0700–1900), and was transmitted by different species of *Chrysops*, feeding mainly between 0900 and 1800. The differing periodicities of the two forms of *Loa* were maintained after their experimental transfer to drills (*Mandrillus leucophaeus*) and after passage through *Chrysops*. Microfilariae which were the progeny of hybridization between human and simian parasites showed «predominantly diurnal» periodicity, but with «a considerable extension... into the early hours of the night» (0800–2200); further studies involving back-crossing suggested that «the two strains were segregating on simple Mendelian lines with regard to periodicity...».

Thus it seems that there is a genetic mechanism controlling the periodicity of at least *L. loa*; a long series of papers by Hawking and his collaborators (reviewed by Hawking, 1967 and summarized by Worms, 1972) has culminated in the proposal of a hypothesis explaining the physiological mechanism regulating or «entraining» the periodicity of this and other species. Hawking observed, as has already been noted, that when absent from the peripheral circulation the

microfilariae were concentrated in the small blood vessels of the lungs. If the diurnal activity rhythm of the host was reversed the microfilarial periodicity was also reversed. Various experimental procedures (including induction of hypoxia and raising or lowering body temperature) indicated that the factor directly inducing aggregation in the lungs was the steep rise in oxygen tension encountered by the worms as they were carried by the circulating blood from the precapillary arterioles of the lung to the pulmonary capillaries; the difference in oxygen tension in the two classes of vessels in man may range from 55–71 mm Hg during day time (the higher values occurring during periods of activity), but falls to 42–47 mm Hg during sleep (Hawking, 1967). It was postulated that the higher oxygen tensions are in some way disadvantageous to the worms, and that their avoidance of them has survival value. However, the absolute necessity for the microfilariae to be present in circulating blood so that they may be ingested by their vectors has to be set against this, which accounts (teleologically) for their presence there during the hours of the vector's maximum activity only. The difference in oxygen tension was referred to by Hawking as the «oxygen barrier», and it is this barrier which must be overcome by the microfilariae to enable them to pass through the lungs and into the general circulation. With nocturnally periodic species (e.g. *W. bancrofti*) the natural lowering of this barrier as the host's activity is reduced during sleep at night is sufficient to allow the worms to cross it. With diurnally periodic species such as *L. loa*, it has been shown that periodicity is affected by changing the host's body temperature (normally lower at night). Hawking postulated that temperature change affects the sensitivity of the microfilariae to the oxygen barrier – the sensitivity increasing as the temperature falls, so that the worms can cross the barrier only during the daytime. It was suggested that this change in sensitivity may be mediated by «the accumulation (or disintegration) of some biochemical product» of the host's metabolism.

The physical means by which the worms are held up by the oxygen barrier may depend on their activity. Living microfilariae wriggle actively and continuously, and waves of contraction may pass along their bodies in either direction. In large blood vessels these contractions have no effect on the direction of (passive) movement of the worms, since there is no lateral resistance to provide purchase. In smaller vessels, however, contact with the walls would enable the direction of contraction to influence the direction of the worm's progression, posteriad contraction leading to anteriopad movement, and vice versa. It was proposed by Hawking (1967) that the normal posteriad contractions are initiated from an «excitatory centre» at the «head end» of the larva which «can be inhibited by a high oxygen tension», thus enabling or inducing reversed, anteriopad waves of contraction; thus the worms are retained in the narrow pulmonary pre-capillary arterioles as they approach the greatly increased oxygen tension of the pulmonary capillaries.

Conclusions

In preparing this brief review, I have been impressed by two aspects of the subject. Firstly, the beauty of some of the adaptations enabling parasites to complete their life cycles in very different environments in two different hosts; examples of these include the respiratory preadaptations of salivarian trypanosomes for their transfer from one host to another, the complex morphological specializations of the anterior end of haemosporine sporozoites, and the periodicity of malarial gametocytes and microfilariae. Secondly, the enormous gaps in our present knowledge have become apparent. A great deal has recently been revealed about, for example, the mitochondrial cycle and surface coat phenomenon of the salivarian trypanosomes, but how are these events controlled? What is the role of kinetoplast deoxyribonucleic acid in the former and of nuclear genes in the latter? Also in spite of 30 years' painstaking work by Hawking and his collaborators, the precise mechanism enabling microfilariae to «hide» within the lungs of their hosts for 12 h or more in every day is still unknown – Hawking's (1967) hypothesis of the effect of oxygen tension on an excitatory centre remaining elegant but unproven. In many groups of parasites we do not even know whether morphological and physiological adaptations related to transmission exist, let alone understand their significance and control. Clearly there remains a great deal of knowledge to be acquired about this aspect of parasite biology, knowledge which will be of great potential value in the control of the infections involved, since transfer from one host to another must always represent one of the more vulnerable stages of a parasite's life cycle.

- 1 Aikawa M.: *Plasmodium*: the fine structure of malarial parasites. *Exp. Parasit.* 30, 284–320 (1971).
- 2 Baker J. R., Cunningham I., Fromentin H., Vickerman K.: Development of salivarian trypanosomes *in vivo* and *in vitro*. In: *Les moyens de lutte contre les trypanosomes et leurs vecteurs. Actes du Colloque, Paris 12–15 mars 1974*, p. 368–369. Office International des Epizooties, Paris 1974.
- 3 Bishop A., McConnachie E.: A study of the factors affecting the emergence of the gametocytes of *Plasmodium gallinaceum* from the erythrocytes and the exflagellation of the male gametocytes. *Parasitology* 46, 192–215 (1956).
- 4 Bishop A., McConnachie E.: Further observations on the *in vitro* development of the gametocytes of *Plasmodium gallinaceum*. *Parasitology* 50, 431–448 (1960).
- 5 Böhringer S., Hecker H.: Quantitative ultrastructural investigations of the life cycle of *Trypanosoma brucei*: a morphometric analysis. *J. Protozool.* 22, 463–467 (1975).
- 6 Bowman I. B. R., Srivastava H. K., Flynn I. W.: Adaptations in oxidative metabolism during the transformation of *Trypanosoma rhodesiense* from bloodstream into culture form. In: *Comparative biochemistry of parasites* (H. Van der Bossche, ed.), p. 329–342. Academic Press, New York/London 1972.
- 7 Brack C.: Elektronenmikroskopische Untersuchungen zum Lebenszyklus von *Trypanosoma cruzi*. *Acta trop.* (Basel) 25, 289–356 (1968).
- 8 Brener Z.: Significance of morphologic variation of bloodstream forms. In: *New approaches in American trypanosomiasis research* (Pan American Health Organization Scientific Publication No. 318), p. 127–131. Pan American Health Organization, Washington 1976.

- 9 Brown R. C., Evans D. A., Vickerman K.: Changes in oxidative metabolism and ultrastructure accompanying differentiation of the mitochondrion in *Trypanosoma brucei*. *Int. J. Parasit.* 3, 691–704 (1973).
- 10 Burgdorfer W.: *Acta trop.* (Basel) 1977 (in press).
- 11 Creemers J., Jadin J. M.: Étude de l'ultrastructure et de la biologie de *Leishmania mexicana* Biagi, 1953. I. Les modifications qui surviennent lors de la transformation *leishmania – leptomonas*. *Bull. Soc. Path. exot.* 60, 53–58 (1967).
- 12 Cross G. A. M.: Identification, purification and properties of clone-specific glycoprotein antigens constituting the surface coat of *Trypanosoma brucei*. *Parasitology* 71, 393–417 (1975).
- 13 Desser S. S.: The fine structure of *Leucocytozoon simondi*. III. The ookinete and mature sporozoite. *Canad. J. Zool.* 48, 641–645 (1970).
- 14 Desser S. S., Wright K. A.: A preliminary study of the fine structure of the ookinete, oocyst and sporozoite formation of *Leucocytozoon simondi* Mathis & Leger. *Canad. J. Zool.* 46, 303–307 (1968).
- 15 Duke B. O. L.: Behavioural aspects of the life cycle of *Loa loa*. In: Behavioural aspects of parasite transmission (E. U. Canning and C. A. Wright, eds), p. 97–105. (*Zool. J. Linn. Soc.* 51, supplement.) Academic Press, New York/London 1972.
- 16 Duncan D., Eades J., Julian S. R., Micks D.: Electron microscopic observations on malarial oocysts (*Plasmodium cathemerium*). *J. Protozool.* 7, 18–26 (1960).
- 17 Evans D. A., Brown R. C.: Cyanide insensitive culture form of *Trypanosoma brucei*. *Nature* (Lond.) 230, 251–252 (1971).
- 18 Evans D. A., Ellis D. S.: Penetration of mid-gut cells of *Glossina morsitans morsitans* by *Trypanosoma brucei rhodesiense*. *Nature* (Lond.) 258, 231–233 (1975).
- 19 Faust E. C., Russell P. F., Jung R. C.: Craig and Faust's clinical parasitology, ed. 8. Lea & Febiger, Philadelphia 1970.
- 20 Freeman J. C.: The penetration of the peritrophic membrane of the tsetse fly by trypanosomes. *Acta trop.* (Basel) 30, 347–355 (1973).
- 21 Freeman J.: The penetration of the peritrophic membrane of the tsetse flies by trypanosomes. In: Les moyens de lutte contre les trypanosomes et leurs vecteurs: Actes du Colloque, Paris 12–15 mars 1974, p. 153. Office International des Epizooties, Paris 1974.
- 22 Garnham P. C. C.: The ultrastructure of Haemosporidia and allied protozoa with special reference to the motile stages. In: Progress in protozoology (J. Ludvík, J. Lom and J. Vávra, eds) (Proceedings of 1st International Conference on Protozoology, Prague, 1961), p. 427–433. Czechoslovak Academy of Sciences, Prague 1963.
- 23 Garnham P. C. C., Bird R. G., Baker J. R.: Electron microscopic studies of motile stages of malarial parasites. I. The fine structure of the sporozoites of *Haemamoeba* (= *Plasmodium*) *gallinacea*. *Trans. roy. Soc. trop. Med. Hyg.* 54, 274–278 (1960).
- 24 Garnham P. C. C., Bird R. G., Baker J. R., Bray R. S.: Electron microscopic studies of motile stages of malarial parasites. III. The fine structure of the sporozoites of *Laverania* (= *Plasmodium*) *falcipara*. *Trans. roy. Soc. trop. Med. Hyg.* 55, 98–102 (1961).
- 25 Geigy R., Kauffmann M., Steiger R., Brun R.: Influence of bloodmeals from different donors on the infection rates of *Trypanosoma brucei* in *Glossina*. *Acta trop.* (Basel) 28, 164–169 (1971).
- 26 Harmsen R.: The nature of the establishment barrier for *Trypanosoma brucei* in the gut of *Glossina pallidipes*. *Trans. roy. Soc. trop. Med. Hyg.* 67, 364–373 (1973).
- 27 Hawking F.: The 24-hour periodicity of microfilariae: biological mechanisms responsible for its production and control. *Proc. roy. Soc. Lond. B* 169, 59–76 (1967).
- 28 Hawking F.: Circadian rhythms in *Trypanosoma congolense*. *Trans. roy. Soc. trop. Med. Hyg.* 70, 170 (1976).
- 29 Hawking F., Worms M. J., Gammage K.: 24- and 48-hour cycles of malaria parasites in the blood; their purpose, production and control. *Trans. roy. Soc. trop. Med. Hyg.* 62, 731–760 (1968).
- 30 Hoare C. A.: The trypanosomes of mammals. Blackwell Scientific Publications, Oxford/Edinburgh 1972.

- 31 Hoare C. A., Wallace F. G.: Developmental stages of trypanosomatid flagellates: a new terminology. *Nature (Lond.)* 212, 1385–1386 (1966).
- 32 Howells R. E., Peters W., Homewood C. A.: Physiological adaptability of malaria parasites. In: *Comparative biochemistry of parasites* (H. Van der Bossche, ed.), p. 235–258. Academic Press, New York/London 1972.
- 33 Killick-Kendrick R., Molyneux D. H., Ashford R. W.: *Leishmania* in phlebotomid sandflies. I. Modifications of the flagellum associated with attachment to the mid-gut and oesophageal valve of the sandfly. *Proc. roy. Soc. Lond. B* 187, 409–419 (1974).
- 34 Klei T. R.: The fine structure of *Haemoproteus columbae* sporozoites. *J. Protozool.* 19, 281–286 (1972).
- 35 Lumsden W. H. R.: Infectivity of salivarian trypanosomes to the mammalian host. *Acta trop. (Basel)* 29, 300–320 (1972).
- 36 Molyneux D. H., Killick-Kendrick R., Ashford R. W.: *Leishmania* in phlebotomid sandflies. III. The ultrastructure of *Leishmania mexicana amazonensis* in the midgut and pharynx of *Lutzomyia longipalpis*. *Proc. roy. Soc. Lond. B* 190, 341–357 (1975).
- 37 Mshelbwala A. S.: *Trypanosoma brucei* infection in the haemocoel of tsetse flies. *Trans. roy. Soc. trop. Med. Hyg.* 66, 637–643 (1972).
- 38 Mühlpfordt H.: Elektronenmikroskopische Untersuchung eines im Rind gehaltenen *Trypanosoma vivax*-Stammes. *Tropenmed. Parasit.* 26, 1–8 (1975).
- 39 Muller R.: *Worms and disease*. Heinemann, London 1975.
- 40 Newton B. A., Cross G. A. M., Baker J. R.: Differentiation in Trypanosomatidae. In: *Microbial differentiation* (J. M. Ashworth and J. E. Smith, eds), p. 339–373. (Symposium 23 of the Society of General Microbiology.) University Press, Cambridge 1973.
- 41 Ormerod W. E., Venkatesan S., Carpenter R. G.: The effect of immune inhibition on pleomorphism in *Trypanosoma brucei rhodesiense*. *Parasitology* 68, 355–367 (1974).
- 42 Otieno L. H.: *Trypanosoma (Trypanozoon) brucei* in the haemolymph of experimentally infected young *Glossina morsitans*. *Trans. roy. Soc. trop. Med. Hyg.* 67, 886–887 (1973).
- 43 Rudzinska M. A., D'Alesandro P. A., Trager W.: The fine structure of *Leishmania donovani* and the role of the kinetoplast in the leishmania-leptomonad transformation. *J. Protozool.* 11, 166–191 (1964).
- 44 Rudzinska M. A., Vickerman K.: The fine structure. In: *Infectious blood diseases of man and animals* (D. Weinman and M. Ristic, eds), p. 217–306. Academic Press, New York 1968.
- 45 Simpson L.: The leishmania-leptomonad transformation of *Leishmania donovani*: nutritional requirements, respiration changes and antigenic changes. *J. Protozool.* 15, 201–207 (1968).
- 46 Sinden R. E., Canning E. U., Spain B.: Gametogenesis and fertilization in *Plasmodium yoelii nigeriensis*: a transmission electron microscope study. *Proc. roy. Soc. Lond. B* 193, 55–76 (1976).
- 47 Sinden R. E., Croll N. A.: Cytology and kinetics of microgametogenesis and fertilization in *Plasmodium yoelii nigeriensis*. *Parasitology* 70, 53–65 (1975).
- 48 Sinden R. E., Garnham P. C. C.: A comparative study of the ultrastructure of *Plasmodium* sporozoites within the oocysts and salivary glands, with particular reference to the incidence of the micropore. *Trans. roy. Soc. trop. Med. Hyg.* 67, 631–637 (1973).
- 49 Steiger R.: Some aspects of the surface coat formation in *Trypanosoma brucei*. *Acta trop. (Basel)* 28, 341–346 (1971).
- 50 Steiger R.: On the ultrastructure of *Trypanosoma (Trypanozoon) brucei* in the course of its life cycle and some related aspects. *Acta trop. (Basel)* 30, 64–168 (1973).
- 51 Steiger R.: Ultracytochemistry of the surface coat/pellicle complex in *Trypanosoma brucei*. *Acta trop. (Basel)* 32, 152–158 (1975).
- 52 Sterling C. R., Aikawa M., Vanderberg J. B.: The passage of *Plasmodium berghei* sporozoites through the salivary glands of *Anopheles stephensi*: an electron microscope study. *J. Parasit.* 59, 593–605 (1973).
- 53 Sterling C. R., De Guisti D. L.: Fine structure of differentiating oocysts and mature sporozoites of *Haemoproteus metchnikovi* in its intermediate host *Chrysops callidus*. *J. Protozool.* 21, 276–283 (1974).

- 54 Terzakis J. A.: Transformation of the *Plasmodium cynomolgi* oocyst. J. Protozool. 18, 62–73 (1971).
- 55 Terzakis J. A., Sprinz H., Ward R. A.: Sporoblast and sporozoite formation in *Plasmodium gallinaceum* infection of *Aedes aegypti*. Milit. Med. 131, 984–992 (1966).
- 56 Terzakis J. A., Sprinz H., Ward R. A.: The transformation of the *Plasmodium gallinaceum* oocyst in *Aedes aegypti* mosquitoes. J. Cell Biol. 34, 311–326 (1967).
- 57 Terzakis J. A., Vanderberg J. P., Weiss M. M. Viruslike particles in malaria parasites. J. Parasit. 62, 366–371 (1976).
- 58 Trefiak W. D., Desser S. S.: Crystalloid inclusions in species of *Leucocytozoon*, *Parahaemoproteus*, and *Plasmodium*. J. Protozool. 20, 73–80 (1973).
- 59 Vanderberg J. R., Rhodin J., Yoeli M.: Electron microscopic and histochemical studies of sporozoite formation in *Plasmodium berghei*. J. Protozool. 14, 82–103 (1961).
- 60 Vickerman K.: The mechanism of cyclical development in trypanosomes of the *Trypanosoma brucei* sub-group: an hypothesis based on ultrastructural observations. Trans. roy. Soc. trop. Med. Hyg. 56, 487–495 (1962).
- 61 Vickerman K.: Polymorphism and mitochondrial activity in sleeping sickness trypanosomes. Nature (Lond.) 208, 762–766 (1965).
- 62 Vickerman K.: The fine structure of *Trypanosoma congolense* in its bloodstream phase. J. Protozool. 16, 54–69 (1969 a).
- 63 Vickerman K.: On the surface coat and flagellar adhesion in trypanosomes. J. Cell Sci. 5, 163–194 (1969 b).
- 64 Vickerman K.: Morphological and physiological considerations of extracellular blood protozoa. In: Ecology and physiology of parasites (A. M. Fallis, ed.), p. 58–91. Toronto University Press, Toronto 1971.
- 65 Vickerman K.: The mode of attachment of *Trypanosoma vivax* in the proboscis of the tsetse fly *Glossina fuscipes*: an ultrastructural study of the epimastigote stage of the trypanosome. J. Protozool. 20, 394–404 (1973).
- 66 Vickerman K.: The ultrastructure of pathogenic flagellates. In: Trypanosomiasis and Leishmaniasis, with special reference to Chagas' disease (K. Elliott, M. O'Connor and G. E. W. Wolstenholme, eds). Ciba Found. Symp. 20, 171–190 (1974 a).
- 67 Vickerman K.: Antigenic variation in African trypanosomes. In: Parasites in the immunized host: mechanisms of survival (R. Porter and J. Knight, eds). Ciba Found. Symp. 25, 53–70 (1974 b).
- 68 Vickerman K.: Development of salivarian trypanosomes in the insect vector: changes in flagellate structure and physiology. In: Les moyens de lutte contre les trypanosomes et leurs vecteurs: Actes du Colloque, Paris 12–15 mars 1974, p. 157–158. Office International des Epizooties, Paris 1974 c.
- 69 Vickerman K., Evans D. A.: Studies on the ultrastructure and respiratory physiology of *Trypanosoma vivax* trypomastigote stages. Trans. roy. Soc. trop. Med. Hyg. 68, 145 (1974).
- 70 Ward R. A.: The susceptibility of *Glossina austeni* to infection with *Trypanosoma brucei*. Trans. roy. Soc. trop. Med. Hyg. 62, 672–678 (1968).
- 71 Wong T. C., Desser S. S.: Fine structure of oocyst transformation and the sporozoites of *Leucocytozoon dubreuilii*. J. Protozool. 23, 115–126 (1976).
- 72 Worms M. J.: Circadian and seasonal rhythms in blood parasites. In: Behavioural aspects of parasite transmission (E. U. Canning and C. A. Wright, eds), p. 53–66. (Zool. J. Linn. Soc. 51, supplement.) Academic Press, New York/London 1972.
- 73 Wright K. A., Hales H.: Cytochemistry of the pellicle of bloodstream forms of *Trypanosoma (Trypanozoon) brucei*. J. Parasit. 56, 671–683 (1970).

