Miscellanea : The peritrophic membrane of blood sucking diptera in relation to their role as vectors of blood parasites

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Objekttyp: **Article**

Zeitschrift: **Acta Tropica**

Band (Jahr): **18 (1961)**

Heft 3

PDF erstellt am: **12.07.2024**

Persistenter Link: https://doi.org/10.5169/seals-310951

The Peritrophic Membrane of Blood Sucking Diptera in Relation to their Role as Vectors of Blood Parasites.*

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Usually the peritrophic membrane (PM) of insects is only known and described morphologically as a part of the midgut and its presence and function is not clearly understood (DAY & WATERHOUSE 1953, WIGGLESWORTH 1953). It is generally agreed that the PM protects the cells of the midgut from hard or sharp particles of food, though in many insects which suck blood or fluids of plant origin, a PM can be found. The PM consisting mainly of chitin is readily permeable to digestive enzymes and products of digestion, and a number of dye-feeding experiments suggest that the membrane acts as an ultrafilter. Thus the PM forms a barrier permeable only to extremely small particles. Three different types of PM are found in insects and can be characterized as follows:

Type I of the membrane consists of a series of thin concentric lamellae, independent or loosely attached to one another. The formation of these lamellae is not quite clear but it is thought that they are produced periodically by the separation of thin sheets from the surface of the cells throughout the length of the midgut.

Type II of the membrane consists of a single uniform layer. It is secreted continuously in viscous form by a group of cells at the anterior end of the midgut. It soon becomes solid and is of uniform circumference throughout its length. Both these types are produced irrespective of the intake of food.

Type III of the membrane is formed only after a meal (STOHLER 1957). The material is secreted in viscous form by a part of or the whole midgut epithelium. It stiffens, completely enclosing the gut content, and is excreted at the end of digestion.

After this introduction we come to the point of this survey. Under discussion is the way parasites, transmitted by blood sucking dipteras, deal with the PM, which evidently forms a barrier in their way from the lumen to the gutwall and other parts of the insect's body. An answer to the following questions will be attempted:

1. Is there a PM in important vectors of insectborne diseases?
2. Does the PM form any barrier or obstacle for parasites? and if so:
3. Can parasites readily penetrate the PM or are there ways to by-pass the membrane?

To begin with we take the example of the different trypanosome cycles in the tsetse fly (GEIGY & HERBIG 1955). These cycles show an increasing adaptation to the morphologic conditions in the insect gut, where a PM of type II is found. The cycle of *Trypanosoma vivax* is the simplest as only trypanosomes which can fix themselves in the hypopharynx are transmitted, while those taken into the gut are digested. But already with *T. grayi* the PM of the Glossina has a certain effect. While the blood is digested in the endoperitrophic space, the trypanosomes pass to the end of the PM, penetrate the ectoperitrophic space where they finish their evolution and finally come to rest in the

* Read before the XIth International Congress of Entomology, Vienna, Austria, 17th—25th August, 1960, during the Symposion on "Arthropods in relation to blood parasites, especially those of wild animals". It is foreseen to be also published in the Congress Report.

hindgut still outside the PM. From there the metacyclic forms are excreted little by little with the rest of the digested blood. A further complication is found in the case of *T. congolese.* As before the end of the membrane is by-passed and the trypanosomes travel in the ectoperitrophic space to reach the region where the PM is formed near the proventriculus. Here the membrane is still soft and can be penetrated actively by the trypanosomes. Finally they reach the hypopharynx from where they are transmitted to another host on the occasion of a blood meal. In the case of the trypanosomes of the brucei-group, i.e. *T. rhodesiense, gambiense,* and *brucei,* the route is the same as before but prior to reaching the infective stage, they must pass to the salivary gland from where they are injected into a new host. Summarizing the facts we can say that the PM of tsetse flies cannot be penetrated with the exception of a short region at the place of formation, however trypanosomes find opportunities enough to by-pass the membrane at its open end.

Another example of interactions between membrane and parasites is found in the case of phlebotomes which act as vectors for Leishmaniasis. Here we find a PM of type III, i.e. after a blood meal a membrane forms around the whole content of the midgut. In this way the parasites are enclosed in the peritrophic sac. In three different species of phlebotomes what happens next has been studied (FENG 1951). In *Phlebotomus mongolensis* the PM forms a complete sac throughout its existence, the flagellates envelopped within. As the digestion of the blood goes on, the flagellates decrease in number. At the end the peritrophic sac is discharged together with the remains of the blood and the flies become free of infection. In contrast to this the PM in *P. chinensis* also forms a sac, but later it breaks especially at its posterior end and the flagellates are set free in the midgut, they invade the proventriculus in which they become established. Finally in *P. squamirostris,* vector of the toad trypanosome *T. bocagei,* the PM also forms a sac initially. But in this case it is not tightly closed, so that after some time portions of the digested blood are discharged and flagellates begin to pass down the end gut, they attach themselves to the gutwall, multiply and develop into the infective stage.

No corresponding studies have been made with other species of sandflies. Anyway, from these results we can conclude that the PM of sandflies cannot be penetrated by parasites, at least when the membrane has become solid. Later the condition of the membrane decides further progress of an infection.

Also simuliids possess a PM of type III. Its role in parasitic infections was studied in *Simulium damnosum* infected with *Onchocerca volvulus* (LEWIS 1950 and 1953). The PM formed at the time of the blood meal envelops the content of the midgut and remains unbroken for at least 24 hours. The average number of microfilariae ingested was usually found to be considerably greater than that of larvae completing the development. Subsequently most of the ingested microfilariae were seen imprisoned in the peritrophic sac. Only few remain in the tubular part of the midgut where no membrane is formed and it is thought that only these few can make their way to the thoracic muscles. Further it is possible that some of the microfilariae can escape from the PM during its viscous phase soon after the formation. But still the majority must be too late to penetrate. Only exceptionally in very heavily infected flies does it appear that the numerous microfilariae can prevent the formation of a proper membrane. In this case the flies become overinfected and die.

From these facts it is possible to conclude that, once formed, the PM of *Simulium damnosum* is normally unpenetrable for enclosed microfilariae and thus protects the fly from heavy infections without preventing transmission of the disease.

The last example of relations between parasites and PM in dipteras is found

in mosquitoes. Experimental studies were made with *Aedes aegypti* and the parasite of chicken malaria *Plasmodium gallinaceum* (STOHLER 1957). The PM of Aedes is also secreted in fluid form following each blood meal. In the course of 20-30 hours it grows more and more solid. Up to the 30th hour after the infective meal ookinetes readily penetrate the membrane and reach the cells of the midgut. The rest finds themselves captured in the peritrophic sac and die, usually lining the now solid membrane. Therefore it seems that as in the case of Simulium the PM of Aedes has a regulative function on the infection with the malaria parasite.

At the end a rather speculative idea about the influence of the PM on infections shall be introduced. It was found that it was impossible to infect certain species of mosquitoes with viruses of Murray Valley encephalitis and western and eastern equine encephalitis when the mosquitoes were fed on infected animals. But when suspensions of viruses were injected into the body cavity or when the gut filled with blood was perforated with a needle, infections took place and the mosquitoes were able to transmit the disease normally by bite (HURLBUT 1951, MCLEAN 1953, 1955, MERRILL & TENBROEK 1935). These results could be easily explained supposing that in certain cases an intact PM would intercept a natural infection.

Bearing in mind all cases where connections between PM and parasites have been found it seems permissible to summarize the results and to answer the questions put earlier as follows:

In most vectors of parasites which must fulfil part of a cycle in an insect a PM is found. It forms a real barrier and cannot be penetrated. But as was to have been expected from acknowledged vectors of parasites, membranes can be either by-passed at the open end (type II) or parasites force their way through the membrane just after its secretion in viscous form (type II and III). Eventually a solid membrane breaks and sets the enclosed parasites free. Therefore the PM does not prevent the infection of an insect, at best the degree of an infection is influenced.

On the other hand the possibility that the PM prevents a transmission can not be excluded, though it has not yet been found. In this respect one must think of numerous cases where related species of insects are either good, bad or no vectors, without any obvious reason for such behaviour being found. Possibly a study of the respective circumstances of the PM could bring some light to these cases.

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