

## THE HAEMOGREGARINES OF MAMMALS AND REPTILES; A REJOINDER TO DR SAMBON.

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IN a recent paper Dr Sambon (1909) has made a violent attack on me for my gentle reminder to him that he has overstepped the boundary of established truths regarding the haemogregarines, and has let his imagination run wild. As he still persists in maintaining his position I feel I am bound to expose his methods, and in the present paper I propose examining in some detail his recent observations on the haemogregarines, and at the same time dealing with his reply to my paper.

I have read all of Dr Sambon's (1908—9) papers on the haemogregarines carefully, and as far as I can gather his knowledge of them is chiefly based on the work of others, much of which he has wrongly interpreted as I shall point out later. He has in addition studied some haemogregarines from the following snakes :

<i>Python molurus</i>	one specimen infected		
<i>P. spilotes</i>	"	"	"
<i>Boa constrictor</i>	"	"	"
<i>Corallus cookii</i>	"	"	"
<i>Eryx conicus</i>	"	"	"
<i>Tropidonotus fasciatus</i>	"	"	"
<i>Pseudaspis cana</i>	"	"	"
<i>Zamenis flagelliformis</i>	"	"	"
<i>Coluber corais</i>	"	"	"
<i>C. melanoleucus</i>	"	"	"
<i>Coronella getula</i>	"	"	"
<i>Psammophis sibilans</i>	"	"	"
<i>Naia tripudians</i>	"	"	"
<i>Lachesis lanceolatus</i>	"	"	"
<i>L. mutus</i>	"	"	"

Ten of the haemogregarines found in the above snakes have been made new species. Dr Sambon however tells us very little about the methods he employed when studying these haemogregarines; the snakes were examined at the Prosectorium of the Zoological Society's Gardens, and were dead, I suppose, at the time Dr Sambon made his discoveries. I presume therefore, that as Dr Sambon considers himself an authority on these parasites, he does not think it necessary to tell his readers about the methods which have led to his discovery of the sexual cycle of the haemogregarines.

In his classification of the Haemoprotozoa he tells us the haemogregarine oökinete encysts and produces sporozoites in secondary cysts or sporebags. I am quite at a loss to understand this very misleading statement, unless it is that Dr Sambon is anxious to be the first to predict the probable method of sexual reproduction of the haemogregarines. Judging from a recent leading article in the *Journal of Tropical Medicine and Hygiene*, Dr Sambon is said to have the gift of prophecy or the rare talent of drawing reasonable inferences from the analogy of established truths. Yet I hope to show, that his inferences regarding the life cycles of the haemogregarines are, at present at any rate, premature. Every Protozoologist now knows, that the great interest attached to these parasites lies in the discovery of their complete life cycles, yet there is not at present a single convincing description of such a cycle. The mere recording of new species is now of little interest, as these parasites are so numerous, that anyone who looks with some degree of care in the blood of mammals and reptiles can hardly fail to discover them.

In Dr. Sambon's opening paragraph on the haemogregarines of snakes he states that he proposes "to gather all the scattered information concerning haemogregarines in general." Had he only limited himself to this, and recorded the parasites he found in snakes, no one could have taken any exception to his statements, but when he proceeds to interpret the observations of others it is obvious he is not in a position to do so. Speaking of *Lankesterella minima* he says, "in 1871 Ray Lankester also noticed and figured the sporonts of *H. minima*." I would like to ask how Dr Sambon knows that the free vermicules of *L. minima* as figured by Lankester (1871) represent the sporonts of the parasite? By this term I understand that stage of a protozoon which is destined to undergo sporogony, wherever that may take place. Lankester himself says nothing to lead me to think he considered the free vermicules were sporonts. Surely Dr Sambon does not expect his readers to accept this

interpretation of Lankester's observations without convincing evidence to prove it; further I can find nothing in Dr Sambon's papers to show that he has even studied this parasite. These however are just his methods, and throughout his papers he interprets other workers' observations to suit his own ideas. The principle seems to be, Dr Sambon makes the free vermicules of haemogregarines sporonts, therefore they must be sporonts.

As I have studied *L. minima* I am in a position to examine any evidence in support of Dr Sambon's view. It is well known that on examining, in the fresh condition, the blood of a frog infected with this haemogregarine, the parasites soon leave the red blood corpuscles and may be seen as free vermicules moving about in the plasma. On feeding a species of *Glossiphonia* parasitic on *Rana tigrina*, and on examining its crop contents, it was found that the parasites had similarly liberated themselves, and were actively moving about in the fluid. Now if these parasites represent the sporonts it is only natural to expect that they would, after some unknown time, begin the process of sporogony somewhere in the leech's alimentary tract, and according to Dr Sambon we should expect to find them in couples lying side by side. Later they would have fused together and then produced an oökinete, which would eventually result in a cyst containing sporozoites. Now nothing of the sort takes place, the free vermicules (Dr Sambon's sporonts) do not undergo any such process, for they can be found in much the same condition in the crop of the leech for several days after it has sucked the blood of an infected frog. What then happens to them and how does the parasite complete its evolution? From some evidence I have been able to gather I believe the parasites make their way to the sheath of the proboscis, and are then inoculated into the next frog the leech bites. This is the only conclusion I can come to at present, and though it may seem strange to Dr Sambon that this parasite does not undergo sporogony in its invertebrate host, no amount of observations on the vermicules in the leech can demonstrate such a cycle. I suppose Dr Sambon would tell me, that had he examined the vermicules of *L. minima*, he would have been able to say whether in this case the sporonts exhibit any sexual differentiation, for he now tells me, that as he has not seen the leucocytic parasite of the hare, he is not in a position to say whether its sporonts do at any time exhibit sexual differentiation. I begin to wonder now whether there has been something wrong with the methods and technique I have employed in studying these parasites, because I am unable to find their sporonts. It is therefore to be regretted that Dr

Sambon does not give us at least some hints as to how he has been so fortunate in finding them, especially those of *L. minima*, even though he appears not to have studied this parasite.

When discussing the life history of the haemogregarines Dr Sambon begins by saying that, "like that of other haemoprotozoa, (it) is divided into two cycles; a schizogonic or 'vegetative' cycle spent in the blood of vertebrates and characterised by asexual multiplication, and a sporogonic or sexual cycle spent in the digestive organs of blood-sucking invertebrates and characterised by asexual reproduction." When I first read this statement I thought that Dr Sambon had been more fortunate than I have been, but as I read his papers further I discovered that he had not found anything whatever to show that the life history of a haemogregarine like that of other haemoprotozoa is divided into two cycles. As I have so far looked in vain for the sexual cycle of a haemogregarine thinking it would be something like that of the Coccidia, I was disappointed to find that after all Dr Sambon had discovered it was like that of other haemoprotozoa. Exactly what haemoprotozoa he refers to I am not sure of; as far as I am aware the malarial parasite is the only one whose life cycle we know with any certainty. Dr Sambon is evidently sure of the life cycles of some other haemoprotozoa, possibly he refers to the trypanosomes. I need hardly remind him that as far as we know at present these parasites do not pass through the two cycles referred to above.

Under the heading "schizogonic cycle," Dr Sambon says that "in examining *fresh blood* (the italics are mine) from vertebrates harbouring haemogregarines we find these parasites usually enclosed within the blood-cells they select for their development,..... For convenience of description we may distinguish three principal forms: *Young forms*, oval, fusiform, or club-shaped, with nucleus median, large, round or oval, homogeneous. At first they are free within the stroma of their host-cell; later encapsuled. *Adult sporonts*, club-shaped, more or less bulky, usually doubled up, always encapsuled, nucleus median, with chromatin filament forming a more or less open skein, or broken up into rods. Host's cells as a rule unaltered." Here we have a statement that certain of these parasites when seen in the fresh blood with appearances described above are young forms, but how does Dr Sambon know they are young forms? He gives no figures showing that the young merozoites just liberated, say, from a cyst in the lung of a snake, are exactly similar to the parasites he describes as young forms. Yet this description is apparently meant to hold good for all haemogre-

garines. As he has not studied the complete cycle of a haemogregarine in the blood and organs of his snakes, I fail to see how he is in a position to make the statements I have quoted above. In criticising my remarks on his schizonts and sporonts he says: "After declaring that he cannot recognise any specific difference between the haemogregarines from different genera and families of snakes, Captain Patton goes on to admit that he is unable to recognise any difference of stage in the development of any of the many examples examined." This is a misstatement and clearly exemplifies Dr Sambon's method of controversy. I have nowhere said I could not recognise any differences in the stages of development of snake or other haemogregarines, whether they be from the peripheral blood or from the organs of their hosts. If Dr Sambon will only read my paper carefully he will find it stated, that I have examined many examples of all these stages, but that I am not in a position at present to interpret them as young forms, adult sporonts, and adult schizonts; this is what I admitted, a very different thing to what Dr Sambon tries to infer. I also said that, "without infecting a snake through the agency of the right tick, and then studying the various forms of the parasites that appear in the blood and organs of the snake, I do not see how it is possible to speak of the parasites in the peripheral blood as schizonts, sporonts, etc." Dr Sambon has carefully avoided this sentence, and ridicules the previous one by saying that he fails to understand the significance of the presence or absence of ticks on snakes; he reminds me that we do not diagnose the species of malarial parasite by determining the kind of mosquito which ingests the parasite. This I should think is quite obvious to anyone; as Dr Sambon is not in a position to appreciate my remarks on ticks and snake haemogregarines I will explain it further.

The method of multiplication of the haemogregarines of snakes in their lungs and livers is a complicated process, two distinct cysts being formed which contain two forms of the parasites. On examining infected snakes, I have found that both these cysts are almost invariably present, and that it is quite impossible to say whether one or both represent schizogony, or a modified form of sporogony; at present I am inclined to the latter view. If it is impossible to express a definite opinion on the nature of these cysts and their contents, it is in my opinion equally impossible to say what stages the parasites in the peripheral blood represent. In order therefore to overcome this difficulty another method of studying these parasites must be adopted. A number of infected snakes harbouring ticks should first be collected;

in Madras I find the rat snake, *Zamenis mucosus*, is well adapted for this purpose. It is then necessary to obtain some uninfected snakes of the same species, and by careful feeding experiments with the ticks to transmit the parasites. Having ascertained the method of infection as well as the approximate time the parasites take to appear in the peripheral blood of the snakes, it is necessary to repeat the experiment, and after a short interval to examine the lungs of the snakes for the process of multiplication of the parasites. It will then be possible to see what type of cyst first appears in their lungs and livers, and what form of parasite is at the same time discharged into the general circulation. This is the plan I have adopted in studying these parasites of mammals and reptiles, and I believe it is the only way their complete life cycles can be worked out. It can be readily understood that it is very tedious work, requiring great patience as numerous initial difficulties have to be overcome.

Perhaps now Dr Sambon will understand my reason for referring to the presence or absence of ticks on snakes, and also why I consider it is impossible at present to say definitely, that certain parasites in the peripheral blood of snakes infected with haemogregarines represent schizonts, sporonts, and so on. Dr Sambon however believes that he is able to recognise these various stages, and mentions the well known forms of the malarial parasite in the peripheral blood of patients infected with this parasite, a truth I am well acquainted with; but have the haemogregarines analogous stages? Dr Sambon assumes they have, not a very remarkable discovery in itself, but an assumption at present quite unjustifiable to say the least of it. Dr Sambon, I note, also states, "that the multiplication forms in the lungs of snakes do belong to the schizogonic cycle there can be no doubt. The adult schizonts of snake haemogregarines as far as we know invariably and exclusively break up within the lungs of their hosts." The multiplication forms of snake haemogregarines do not exclusively develop in the lungs of snakes, but are just as common in their livers; Dr Sambon apparently is not aware of this fact. He need therefore hardly remind me, that these stages of snake haemogregarines in their vertebrate hosts are well known, and have been described by Lutz, Wenyon and himself<sup>1</sup>. Although Dr Sambon speaks of the schizogony of all his snake haemogregarines as occurring in the erythrocytes of the snakes, I can find no description of this cycle; he appears however to have seen the cysts of "*H. seligmanni*"

<sup>1</sup> To be accurate it is necessary to note, that Dr Sambon's description has yet to be published.

after he had written his papers. Dr Sambon then goes on to adopt his usual method of bringing in everything he knows that may have even the remotest bearing on the subject, regardless of the fact that most of what he says has nothing to do with the question I raised. What I have said is quite clear, and is in no way connected with the multiplication forms of other haemogregarines, yet Dr Sambon infers that I doubt whether other haemogregarines have such stages, and adds a list of names of observers, two of whom, as far as I know, have not described any of the multiplication forms of these parasites. I need hardly say that I have studied these stages of most of the haemogregarines.

Now with regard to the vexed question of the sexual cycle of haemogregarines, Dr Sambon says under the heading "conjugation," "I can fully confirm Labbé's observations, having witnessed it not only *in vitro* but also in blood taken from the gut of a tick fed on an haemogregarine-infected lizard"; nothing further is said about this parasite, which I presume is *H. ehrlichi*. Dr Sambon continues, "I have had the opportunity of examining the process of accouplement so frequently in *Haemogregarina seligmanni* that I have no doubt whatever about it." On the same page there are three figures which are not named, and which are not even referred to in the text, I can only presume therefore that they represent the sporonts of *H. seligmanni* in accouplement.

They appear to have proceeded only a little way towards conjugation, and Dr Sambon himself says, "I have not seen the nuclei of the conjugating haemogregarines unite." Yet he thinks this must take place in the gut of the invertebrate host, in this case I suppose in *Porocephalus crotali*. After having brought the reader breathlessly up to this point, eagerly expecting to have the mystery of the sexual cycle of these haemogregarines solved, Dr Sambon suddenly digresses to describe such uninteresting points as the structure and motility of the free sporonts. There is no proof that the parasites he figures are actually undergoing the process of conjugation, and I have certainly never seen any such process in snake haemogregarines, either *in vitro*, or in the alimentary tracts of ticks and linguatulids. Nothing more is said about the conjugation and further stages in the sporogony of "*H. seligmanni*"; Dr Sambon however says in a footnote that they will be described in a future paper. Under the heading "sporogony," Dr Sambon merely re-describes the observations of Simond (1904), Durham (1902), Billet (1904), Brumpt (1904), Christophers (1905), Laveran and Nègre (1905), and Prowazek (1908). With these conflicting statements and his limited observations on snake haemogregarines, Dr Sambon does

not hesitate to predict the method of sexual reproduction of all the haemogregarines.

When criticising my remarks on *L. leporis* in the tick *H. flava* Dr Sambon refers to Miller's (1909) recent paper on *Leucocytozoon* (*Hepatozoon*) *perniciosum*, and states that Miller has proved, "that the sporogony of this species occurs in a rat mite, *Lelaps echidninus*," and that Miller's investigations have "proved the correctness of the account I gave of the haemogregarines in my classification of the Haemoprotozoa which Sir Patrick Manson did me the honour of adopting and supporting in the fourth edition of his *Manual of Tropical Diseases*." Here we have it that in 1907 Dr Sambon prophetically predicted the probable method of sporogony of the haemogregarines, and that now Miller's work—though as yet unconfirmed—has proved the accuracy of Dr Sambon's prediction. This is however regardless of the fact that at the time Dr Sambon classified the haemoprotozoa, he had in my opinion no grounds whatever for saying that the haemogregarine oökinete encysts and produces sporozoites in secondary cysts or sporebags. This, I suppose, is what Dr Sambon calls "dabbling in zoological matters."

Miller summarises the sexual cycle of *L. perniciosum* as follows: "When the blood of an infected rat is swallowed by a mite the encysted trophozoites are set free in the stomach by solution of the cyst as free vermicules. Two similar vermicules become associated and conjugate. One, the macrogamete, grows larger and partly surrounds the other, the microgamete. The protoplasm becomes fused and later the nuclei conjugate and fuse to form a zygote. The zygote becomes a sluggishly motile oökinet, which penetrates the stomach wall of the mite and enters the body tissues and becomes encysted (oöcyst). Here a remarkable enlargement of the karyosome takes place. The parasite increases enormously in size. The nucleus of the spherical sporont thus formed undergoes division into many daughter nuclei, which migrate to the surface of the sporont. The surface of the latter becomes mammillated. The projections, each of which contains a nucleus, increase in size and length; later they are broken off and each becomes a sporoblast. The nucleus of the sporoblast undergoes division, the resulting nuclei being arranged at the poles. The sporoblast increases in size and a cyst wall develops. Around each nucleus a sporozoite is formed. In the ripe sporocyst, which measures 25 by 30 micra, the sporozoites, 16 in number (average) are arranged at the poles. The large cyst (oöcyst) contains from 50 to 100 of such sporocysts. When the mite is swallowed by a rat the cycle is repeated."



This account of the sexual cycle of the parasite at once recalls the appearances I have seen in *Porocephalus pattoni* from the lung of *Zamenis mucosus*, and are also very similar to those described by Christophers (1905) from the body cavity of *Haematopinus stephensi* from *Gerbillus indicus*. Not only are the ripe cysts in each case almost identical in that they contain small cysts full of sausage-shaped bodies, but the earlier stages I have seen in the linguatulid and those described by Christophers from the louse are very striking in their similarity. I have no doubt therefore that the parasites I have seen in *P. pattoni*, and those described by Christophers from the louse, and by Miller in the mite, represent stages in the development of different species of the same genus of a sporozoon. If we are to accept Miller's work as correct it follows that the parasites (cysts) seen by Prowazek, Sambon and myself in linguatulids represent the various stages in the sexual cycle of the haemogregarines of *Python reticulatus*, *Lachesis mutus* and *Zamenis mucosus*. The question then arises how are the sporozoites of these haemogregarines transmitted from an infected to an uninfected snake? According to Miller's conclusions it would be necessary for the uninfected snakes to swallow the infected linguatulids. It is not definitely known how snakes become infected with these arthropods, but it is believed they swallow their eggs or immature stages in their food; for instance the rat snake, *Zamenis mucosus*, probably swallows the eggs of *P. pattoni* in the frogs and toads, which are its principal food. At any rate it is well established that snakes harbour the adult stages of linguatulids, and are the definitive hosts of these arthropods. It is therefore most improbable that snakes swallow adult linguatulids, and if this is true, it is impossible at present to understand how snakes harbouring linguatulids can become infected with the sporozoites of haemogregarines in these arthropods. If it is impossible to understand how this method of infection can take place, it is equally as difficult to understand how an uninfected snake could become infected by the bite of an adult (haemogregarine-infected) linguatulid. Realising these difficulties in 1905, when I first found the cysts in *P. pattoni*, I came to the conclusion that these parasites did not represent stages in the sexual cycle of a haemogregarine. I have examined over 60 specimens of this linguatulid and could trace no connection between the haemogregarine of *Zamenis mucosus* and the parasites in *P. pattoni*. With regard to the cysts Prowazek (1908) has recently found in *P. moniliformis*, I pointed out that he suggests that they may represent a further development of *H. pythonis*; Dr Sambon however speaks of "Prowazek's discovery of

oökinetes and encapsuled oöcysts of *Haemogregarina pythonis*," as if it were absolutely proved by Prowazek that they represent the sexual stages of *H. pythonis*. Why Dr Sambon exaggerates Prowazek's statements I cannot understand, unless he thinks by doing so he will strengthen his own position. He now states that in consequence of Prowazek's announcement he has examined some of the specimens of *P. crocota* from *Lachesis mutus* infected with *H. seligmanni*, and has been able to confirm Prowazek's discovery, and thus complete the life history of this haemogregarine. He tells me that I am wrong in considering the haemogregarine oökinetes and oöcysts found in the stomach of *Porocephalus pattoni* as stages in the development of a parasite peculiar to the linguatulid. Dr Sambon surely does not think that I would have lost the opportunity of describing the sexual cycle of a snake haemogregarine which I found in 1905, and left it to Prowazek and himself to re-discover and describe. If Dr Sambon will refer to the Annual Report of the Bacteriological Section of the King Institute of Preventive Medicine for 1906 he will find the parasite of *P. pattoni* recorded there provisionally as a Gregarine; at present I see no reason to alter my opinion. I shall however look forward to reading Dr Sambon's description of the complete life cycle of *H. seligmanni*; I only hope that he will give us some definite proofs to support his statements.

Dr Sambon, in criticising my remarks on the developmental forms of haemogregarines in linguatulids, says, "Captain Patton is inclined to consider all the developmental forms of vertebrate haemoprotozoa found within the alimentary tubes of invertebrate hosts as totally independent parasites peculiar to these invertebrate hosts." It is now well known that blood-sucking invertebrates are infected with natural parasites whose life cycles are very imperfectly known, and as there is at present too great a haste to discover and describe the developmental forms (sexual cycles) of blood-inhabiting protozoa in the alimentary tracts of invertebrates, these natural parasites have been entirely overlooked. Recent protozoological literature is full of such mistakes. Knowing these facts it is necessary to observe great caution in interpreting the forms found in the digestive tubes of invertebrates fed on the blood of vertebrates infected with protozoa. This is what I wish to emphasise when I disagree with Dr Sambon in his interpretation of the parasites of linguatulids; I have however nowhere stated that *all* the developmental forms of vertebrate haemoprotozoa found in the alimentary tracts of blood-sucking invertebrates represent stages in the evolution of parasites peculiar to invertebrates. As for Dr Sambon's reference to

the herpetomonas stages of trypanosomes of vertebrates in the alimentary tracts of diptera, I hardly think he is in a position to criticise my work along these lines until he can prove that vertebrate trypanosomes have such stages; perhaps he may then be able to say I am wrong.

As I have so far been unable to find any developmental stages of haemogregarines in invertebrates I can only tentatively accept Miller's conclusions. It remains to be proved whether these blood parasites of mammals and reptiles will eventually be found to be transmitted in the extraordinary way suggested by Miller's recent work. I am certainly not so sanguine about it as Dr Sambon is. A great deal of work has yet to be done before it can be said for certain that the haemogregarine ookinete encysts and produces sporozoites in secondary cysts or sporebags.

I will now give the last example of how Dr Sambon interprets other workers' observations. In speaking of *Leucocytozoon funambuli*, he says, "In 1906, Captain Patton, in describing *Haemogregarina funambuli*, a parasite of the five-striped palm squirrel (*Funambulus pennantii*), stated that he had seen free sporonts in the stomach of the squirrel's louse, also a species of *haematopinus*." If Dr Sambon will refer to my (1906) memoir on this parasite he will find that I have nowhere stated that I had seen the free sporonts of the parasite; Dr Sambon apparently knows more about it than I do. In the last Report of the Bacteriological Section of the King Institute I stated, "I have again failed to find any extra-corporeal cycle in the lice found on *Funambulus pennantii*." Dr Sambon either does not know of this Government publication or else he chooses deliberately to ignore it. How can he then in face of these facts say the free vermicules of *L. funambuli* are the sporonts of the parasite?

In a footnote to his paper, Dr Sambon expresses great surprise at Professor Nuttall permitting the use of the name *Leucocytozoon* in his Journal, and says, "The name *Leucocytozoon* cannot be used for the haemogregarines of mammals whether they be parasites of leucocytes or not. It is the generic name of certain avian parasites discovered first by Danilewsky in 1884 in the blood of owls." Dr Sambon will find my reason for retaining these parasites in this genus in my paper on "Mammalian Leucocytozoa" in the Report of the King Institute for 1907. With Laveran I prefer to place the avian parasites in the genus *Haemamoeba*; the justification for this is fully supported by Mezincescu's (1909) recent work. In conclusion I may say I do not intend wasting my time in further discussing with Dr Sambon the points I have raised, even though he should choose to reply to this paper.

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