

following the cadmium treatment. Both cadmium and ashwagandha-treated mice showed decrease in LPO in both the tissue homogenates when compared with that of mice treated with cadmium only. The results clearly indicate that ashwagandha is capable of reducing the toxicity caused by cadmium. Further, we found a significant decrease in SOD and CAT activities after cadmium administration and nearly normal values were obtained when ashwagandha was administered along with cadmium.

As in other studies, in the present investigation also cadmium increased tissue LPO and decreased the activities of antioxidant enzymes such as SOD and CAT which are known as endogenous antioxidative enzymes¹⁹. However, when ashwagandha extract was administered in the metal intoxicated mice, LPO was decreased and nearly normal values of antioxidative enzymes (SOD and CAT) were observed, indicating the ameliorating effect of this plant extract in metal toxicity.

Some antioxidants such as vitamin E, ascorbic acid and GSH decrease free radical generation and increase SOD and CAT activities. These have been found to protect the metal-induced oxidative damage²⁰. However, no plant product has been reported earlier to regulate the cadmium-induced toxic effects. The present findings clearly indicate the protective role of ashwa-

gandha extract on cadmium toxicity in mice.

It is thus suggested that ashwagandha extract may prove to be useful in the regulation of metal-induced clinical toxicity.

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Lambornella (a ciliate parasite) for biological control of *Anopheles stephensi*, the urban malaria vector

Lambornella is a hymenostome endoparasitic ciliate, two species of which namely, *L. clarki* and *L. stegomyiae*, are known to cause ciliotosis in mosquito larvae^{1,2}. We reported the natural infection of immatures of *Anopheles barbirostris*, *Anopheles hyrcanus* gp. and *Anopheles philippinensis* s.l. by ciliate of genus *Lambornella* for the first time in Assam from a forest fringe village of district Dibrugarh³. We isolated the endoparasitic *Lambornella* from the naturally occurring infected larvae and have been culturing it in our laboratory successfully on biphasic medium of 2.8% nutrient agar with hay infusion. In

our entomological laboratory the cyclical mosquito colony of the urban malaria vector, *Anopheles stephensi* (Delhi strain) is also being maintained and we report here the occurrence of epizootic of ciliate parasite belonging to *Lambornella* genus in the colony of *Anopheles stephensi* due to the accidental infection which wiped out the mosquito colony completely.

In the month of September 1996, we initially noticed the gradual mortality of IV instar larvae of *Anopheles stephensi* affecting the pupal output. The mosquito colony was being maintained in enamel pans at $27 \pm 2^\circ\text{C}$ temperature

and $70 \pm 10\%$ relative humidity and immatures were fed on a diet of dog biscuit + yeast (60:40). Change in larval food did not stop the mortality of immatures. Meanwhile the larval mortality was also noticed in II and III instar larvae. The growth of immatures was slowed down and the larval period between two instars was lengthened to 7-8 days as compared to the normal duration of 1-2 days. The larvae became sluggish by the time of attaining the III instar stage and would not come frequently to the water surface for respiration though on tapping the pan they wriggled at the bottom. It led to the suspicion of some

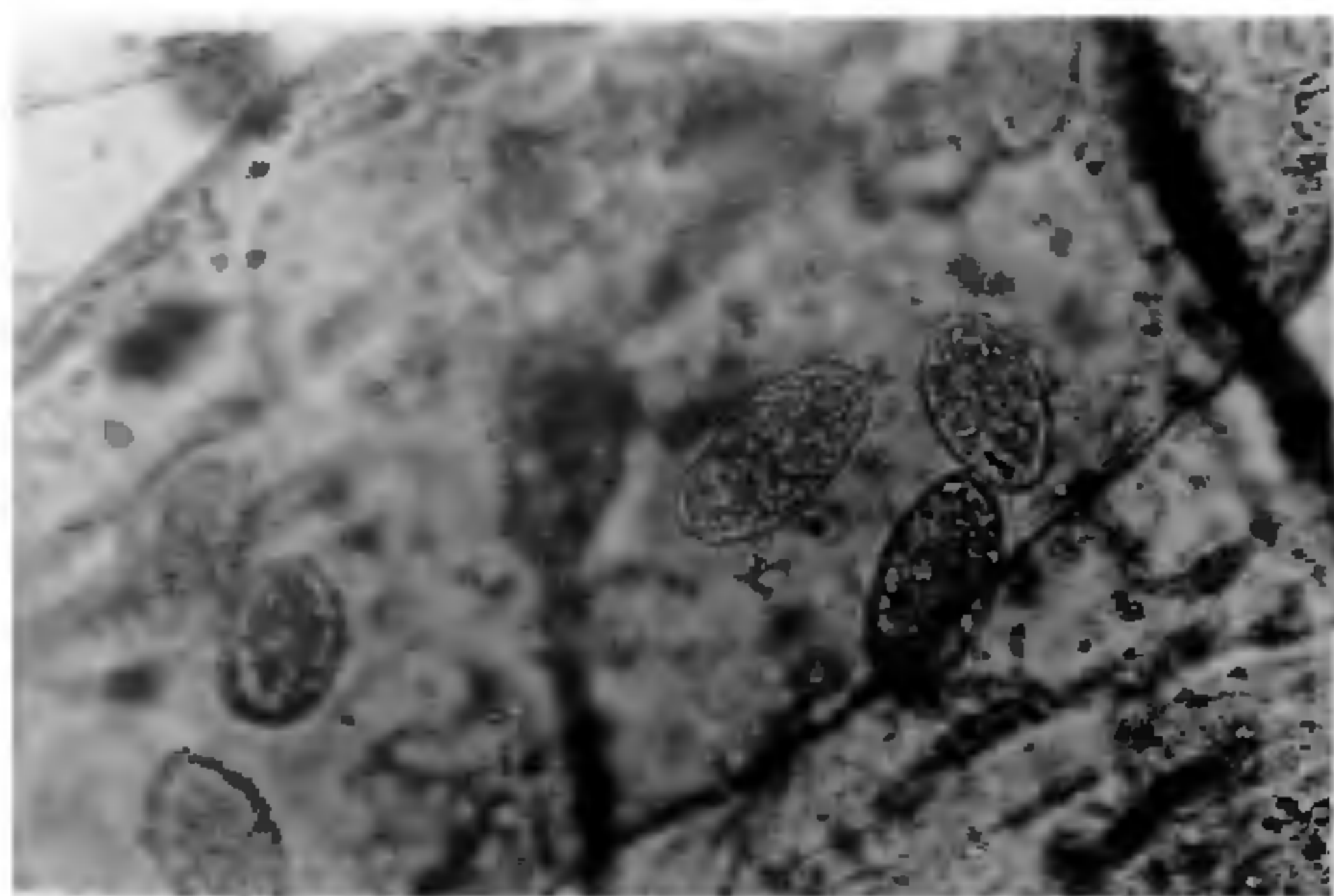


Figure 1. Infected mosquito larva with endoparasitic ciliate *in situ*. (Original magnification 1000 ×).

infection in the larvae. On examining under the microscope, mosquito immatures were found to be heavily infected with the ciliates belonging to genus *Lambornella* which were seen moving in the haemocoel (Figure 1). When examined thoroughly, the ciliate infection was noticed in all the larval instars and the extent of infection was more than 90%. Daily mortality of 10–15% larvae of all instars occurred and the total larval period from egg hatching to pupation was extended abnormally to about 25 days after which only 1–2% pupal yield could be obtained. Further, more than 95% pupae also died and finally only a few adults of *Anopheles stephensi* emerged in a nonsynchronized manner which failed to carry over the further progeny. This epizootic contin-

ued for 2–3 months and led to the collapse of the entire mosquito colony.

Our experience demonstrates that the present strain of *Lambornella* is highly pathogenic in laboratory conditions to the known urban malaria vector, i.e. *Anopheles stephensi* and that even a chance infection of it may adversely affect the colonization of *An. stephensi*. Hence it is necessary to maintain overall aseptic conditions in the insectory of this mosquito. *Lambornella* is known to form desiccation-resistant cysts to tide over the adverse conditions¹ and we suspect that the accidental infection of it in our mosquito colony passed through the cyst stages. Therefore, all laboratory ware in the mosquito insectory, particularly those used in handling of immatures, needs autoclaving to kill the cyst

stages of *Lambornella*, if present. As soon as the infection of this ciliate is noticed in the immatures of mosquito colony, it is advisable to destroy immediately the entire larval population, decontaminate all laboratory ware and start fresh generation from egg stage. However, while starting afresh, the seed eggs should be from original noninfected colony because we suspect even the few adults emerging out of infected immatures may pass on the infection to the colony during egg laying as also observed elsewhere⁴.

Though *Lambornella* appears to be a promising biological control agent, it will be interesting to see if it exhibits a similar degree of pathogenicity in the field conditions also. We are pursuing studies in order to work out further details to explore the true biocontrol potential of this ciliate in natural conditions and to see the various ecological limiting factors.

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