

Malaria, toxoplasmosis: Toward new lines of research?

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A study realized by teams from the Institut Pasteur, the Institut Cochin and the Wellcome Trust Centre for Molecular Parasitology of the University of Glasgow, could redefine part of the present lines of research toward a treatment against the parasites responsible for malaria and toxoplasmosis.

This work, published on 10th October on the website of *Nature Communications*, concerns the role of one [protein](#) which is common to these parasites. Called AMA1, it has been at the heart of many years' research on upgrading treatments, such as trying out vaccination against [malaria](#). However, the present authors have reservations about the success of therapeutic strategies which rely solely on the blockage of AMA1, by demonstrating that the malaria and [toxoplasmosis](#) parasites, without the protein, can develop normally.

With 1 million victims every year, malaria is the most dangerous parasitic disease in the world, while Toxoplasmosis, often asymptomatic, represents a danger to pregnant women and those with weak immune systems.

Plasmodium falciparum and *Toxoplasma gondii*, the parasites responsible respectively for malaria and toxoplasmosis, belong to the group of Apicomplexa. This group, wholly composed of parasite organisms, share a common protein called AMA1. This protein is described in many studies as being indispensable for entry into the [cells](#) they infect. As a result, since its discovery, many research teams have made AMA1 a

major therapeutic target in the improvement of treatments.

However, a collaboration between the teams of Robert Ménard at the Institut Pasteur in Paris, Isabelle Tardieux at the Institut Cochin, and Markus Meissner at the University of Glasgow, has just shown that *Plasmodium falciparum* and *Toxoplasma gondii* can survive and multiply in the infected cells totally without the action of AMA1. This discovery will have an important impact on the search for a treatment for malaria and toxoplasmosis.

The team generated parasites totally lacking AMA1 thanks to a technique of "reverse genetics", never before used in the field. The scientists thus made the following observations: in the absence of AMA1, *Plasmodium falciparum*, at all human stages (blood and liver), is capable of invading the host cells. The same is true of one of the two human stages of *Toxoplasma gondii*. On the other hand, for both [parasites](#), the attachment to the host cells, which precedes cellular invasion, is affected.

As a result, the scientists have deduced that AMA1, short of being indispensable to the process of cellular invasion, is in fact a protein implicated in the adhesion to host cells.

As a result of their observations, the researchers are recommending ways of optimising research which targets AMA1 with a view to improving treatments. In particular, they suggest that [therapeutic strategies](#) should be based on the blockage of other proteins complementary to AMA1.

Provided by University of Glasgow

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