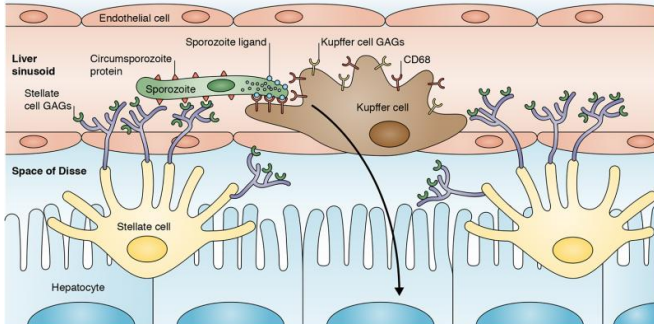


Malaria's key to the liver uncovered

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protein called CD68 is needed for parasite passage and efficient liver infection, as mice lacking CD68 had 70% fewer parasites in their livers compared to intact animals. If this reduction is sufficient to substantially limit blood infections (and thus disease), CD68 may represent a potential new drug target in the fight against malaria.

More information: Cha, S.-J., et al. 2015. *J. Exp. Med.* DOI: [10.1084/jem.20110575](https://doi.org/10.1084/jem.20110575)

This illustration shows how the protein receptor CD68 acts as a gateway for a malaria parasite (sporozoite) to enter the liver through a Kupffer cell, as revealed by Cha et al. Credit: Cha et al.

Provided by Rockefeller University Press

Scientists uncover a port of liver entry for malaria parasites in a report published in *The Journal of Experimental Medicine*. If these results hold up in humans, drugs that target this entry protein might help prevent the spread of disease.

Malaria is caused by a parasite called *Plasmodium falciparum*, which is transmitted to humans via mosquito bite. Recent efforts to limit parasite transmission and increase treatment coverage has reduced the number of malaria-related deaths, but the parasite still causes roughly 200 million new infections and half a million deaths worldwide each year.

The first stop for malaria parasites in humans is the liver, where a few organisms multiply into tens of thousands, which are then released into the bloodstream. Passage through the liver is essential for the parasite to establish a productive infection and cause disease, and the bug's entry route has been traced to specialized [liver cells](#) called Kupffer cells. But exactly how the parasite traverses these cells is not clear.

A team of scientists at Johns Hopkins Bloomberg School of Public Health now find that a Kupffer cell

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