

1: [EMBO J](#). 2006 Jul 12;25(13):3214-22. Epub 2006 Jun 15.



Toxoplasma gondii scavenges host-derived lipoic acid despite its de novo synthesis in the apicoplast.

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In contrast to other eukaryotes, which manufacture lipoic acid, an essential cofactor for several vital dehydrogenase complexes, within the mitochondrion, we show that the plastid (apicoplast) of the obligate intracellular protozoan parasite *Toxoplasma gondii* is the only site of de novo lipoate synthesis. However, antibodies specific for protein-attached lipoate reveal the presence of lipoylated proteins in both, the apicoplast and the mitochondrion of *T. gondii*. Cultivation of *T. gondii*-infected cells in lipoate-deficient medium results in substantially reduced lipoylation of mitochondrial (but not apicoplast) proteins. Addition of exogenous lipoate to the medium can rescue this effect, showing that the parasite scavenges this cofactor from the host. Exposure of *T. gondii* to lipoate analogues in lipoate-deficient medium leads to growth inhibition, suggesting that *T. gondii* might be auxotrophic for this cofactor. Phylogenetic analyses reveal the secondary loss of the mitochondrial lipoate synthase gene after the acquisition of the plastid. Our studies thus reveal an unexpected metabolic deficiency in *T. gondii* and raise the question whether the close interaction of host mitochondria with the parasitophorous vacuole is connected to lipoate supply by the host.

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