The protozoan parasite *Toxoplasma gondii* heads out of its host cell when Ca\(^{2+}\) levels peak in its cytoplasm. Now, Arrizabalaga et al. (page 653) find that proper regulation of these Ca\(^{2+}\) levels, and thus of *Toxoplasma* exit, requires a Na\(^+/H^+\) exchanger (NHE), suggesting a link between proton and Ca\(^{2+}\) regulation.

The increase in Ca\(^{2+}\) levels, which can be induced artificially with an ionophore, triggers a series of morphological changes that primes *Toxoplasma* for both exit and a speedy entrance into a neighboring cell. Death can ensue, however, if the ionophore sticks around while there is no target cell for the parasite to enter; presumably the over-stimulated *Toxoplasma* cannot sustain itself in an activated state.

The authors used this death assay to isolate a mutant defective in activation. One mutant with a disruption in a sodium/hydrogen exchanger (NHE) showed both an activated state and Ca\(^{2+}\) export, which is required for a speedy exit. Toxoplasma lacking this plasma membrane pump also had normal internal pH, but their proton efflux was no longer prevented with an NHE inhibitor.

Resting Ca\(^{2+}\) levels were higher in mutant parasites. This would make it harder to detect any exit signal that increased these Ca\(^{2+}\) levels even further, perhaps explaining why these mutants are slow to exit. The increase in resting Ca\(^{2+}\) levels may come about if a Ca\(^{2+}\) exporter (Ca\(^{2+}\) out/H\(^+\) in) relies on prior action of the NHE (H\(^+\) out/Na\(^+\) in). Such a hypothetical linkage may be part of regulating *Toxoplasma* mobility or simply an intersection between the exit system and *Toxoplasma*’s normal ion homeostasis.

**An exit exchanger**

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Ever since the introduction of the fluid mosaic model, proteins have been viewed as rather hyperactive ships in a sea of lipids. Cholesterol-rich rafts were thought to be at least a partial exception to this trend. Although raft proteins move, their movement was predicted to be dampened by their inclusion in rafts, which act as stages for signaling.

Now, Kenworthy et al. have tested this idea directly (page 735). They used fluorescence recovery after photobleaching (FRAP) to follow green fluorescent protein (GFP)-tagged raft and nonraft proteins. The team then calculated the diffusion coefficients of the tested molecules. They found that raft-associated proteins and those not bound to rafts move similarly—neither faster nor slower.

This conflicts with some studies using single-particle tracking. There are several possible explanations for the new data. Meaningful rafts may not exist at all, at least in resting cells that are not actively signaling. The number of proteins in rafts and their mobility variation may be so small that the differences escape detection by the averaging methods used during FRAP. Or, say the authors, the proteins may not sit statically in rafts but rather move rapidly in and out.

**Fast rafting**

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Centrioles and surrounding centrosomal material nucleate both mitotic spindles and interphase microtubule asters, but centrioles are not an easy structure to dissect with either biochemistry or genetics. There are budding yeast mutants that arrest at various stages of maturation of the spindle pole body (SPB), but the SPB and mammalian centriole bear no physical resemblance to each other despite sharing some proteins and fulfilling the same function.

Matsuura et al. (page 663) therefore resort to a trick of *Chlamydomonas* biology. In this unicellular green algae, the basal body has its normal function of nucleating flagellar microtubules during interphase, but it also switches to act as a centriole during cell division. This dual role gives the authors an easily identifiable phenotype for the isolation of mutants defective in centriole function.

Using insertional mutagenesis, the team isolated 74 *Chlamydomonas* mutants lacking flagella. Most of the mutants lack flagellum-specific proteins, but one mutant, *bld10*, which grew very slowly and exhibited abnormal cell division, piqued their interest. Upon closer examination, they found that *bld10* mutants had no basal bodies. Before this discovery, it was not known if the complete elimination of basal bodies would result in death. These findings suggest that insertional mutagenesis could be used to find and characterize the more than 200 other potential players involved in basal body assembly.

Immunogold labeling showed that Bld10p localizes to the cartwheel, a complex circular structure where microtubules originate. The authors speculate that Bld10p’s localization means that the cartwheel plays a critical role in putting together basal bodies.