SUPPLEMENT INFORMATION

Fig S1: *Pathways for synthesis of three major phospholipids in human, Plasmodium and Toxoplasma.* The *H. sapiens* and *P. falciparum* pathways are adapted from literature, and of *T. gondii* are constructed based on the reported enzyme activities and annotations in the parasite database (www.ToxoDB.org). The pathways, common to all organisms, are shown in black; and those specific to human, are depicted in green. The SDPM-pathway, shown in purple, denotes a plant-type route for PtdEtn and PtdCho synthesis, and is exclusive to *P. falciparum*. Initial precursors are shown in blue; the intermediates of lipid synthesis are in black; phospholipids are in red, and the enzymes are depicted in brown color. DME is metabolized *via* the CDP-choline route and produces PtdDME, which is not methylated to PtdCho in *T. gondii* causing disruption of membrane biogenesis. The treatment of parasite cultures with DME leads to a reduction in PtdCho and an accumulation of PtdDME. CK, choline kinase; PCT, phosphocholine cytidylyltransferase; CPT, CDP-choline phosphotransferase; EK, ethanolamine kinase; PET, phosphoethanolamine cytidylyltransferase; EPT, CDP-ethanolamine phosphotransferase; PEMT, phosphatidylethanolamine methyltransferase; PMT, phosphoethanolamine methyltransferase; SD, serine decarboxylase; PSS, phosphatidylserine synthase; PSD, phosphatidylserine decarboxylase; DME, dimethylethanolamine.

Fig S2: *The TgCK cDNA encodes a choline kinase with 630 residues, which shows 19%, 16% and 10% identity with HsCKα, PfCK and ScCK1, respectively.* Sequence alignment **(A)** and percentage identity **(B)** of *Tg*CK cDNA. The best homologies are found in the Brenner's (red box) and choline kinase (blue box) motives. *Tg*CK also harbors an N-terminal hydrophobic peptide (first 20 amino acids; magenta box) with no homology to a known protein in the NCBI database. The NCBI accession: *Hs*CKα, NP_001268.2; *Pf*CK, PF14_0020; *Sc*CK1, YLR133W.

Fig S3: *The TgEK cDNA encodes for an ethanolamine kinase with 547 residues, which shows 21%, 20% and 14% identity with HsEK1α, PfEK and ScEK1.* . Sequence alignment **(A)** and percentage identity **(B)** of *Tg*EK cDNA. The NCBI accession: *Hs*EK1α, NP_061108.2; *Pf*EK, PF11_0257; *Sc*EK1, YDR147W.

Fig S4: *TgCK shows a punctate intracellular distribution in T. gondii.* **(A)** Indirect Immuno-fluorescence assay performed with tachyzoite-infected human foreskin fibroblasts or axenic parasites using the mouse anti-*Tg*CK sera and the Alexa488 antibodies. **(B)** *Tg*CK expression in COS-7 cells. The *Tg*CK with a C-terminal V5-tag (*Tg*CK-V5) was over-expressed under the control of the *pCMV* promoter and localized using anti-V5 antibody (green). Cells were co-stained with phalloidin to visualize the cytosolic actin (red).

Fig S5: *The Δtgck$_i$ mutant is not impaired in its in vitro growth, which can, however, be inhibited by a choline analog, dimethylethanolamine (DME).* **(A)** Quantification of plaques formed by the *Δtgck*$_i$ mutant and the parental strain. In total, 100 plaques from three independent assays were scored for their sizes. **(B)** Parasites in their vacuoles were counted in the parasitized HFF (29 hrs post-infection) following anti-*Tg*Gap45 staining. Values are means ±S.E. for 3 independent experiments, each with 50 parasitophorous vacuoles. **(C)** *Identification of 53-kDa and 44-kDa protein isoforms using anti-TgCK sera*. Immuno-blot of cell-free extract (CFE) prepared from *T. gondii* tachyzoites and uninfected HFF (negative control) using anti-*Tg*CK (1:200) and anti-*Tg*Actin (control for parasite proteins; 1:1000) antibodies.