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Theoretical and in vitro studies of a C-terminal peptide from PGKC of Leishmania mexicana mexicana.

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Trypanosomatids cause deadly diseases in humans. Of the various biochemical pathways in trypanosomatids, glycolysis, has received special attention because of being sequestered in peroxisome like organelles critical for the survival of the parasite. This study focuses on phosphoglycerate kinase (PGK) from Leishmania spp which, exists in two isoforms, the cytoplasmic PGKB and glycosomal PGKC differing in their biochemical properties. Computational analysis predicted the likelihood of a transmembrane (TM) helix only in the glycosomal isoform PGKC-Lmex, of approximate length 20 residues in the 62-residue extension, ending at, arginine residues R471 and R472. From experimental studies using circular dichroism and NMR with deuterated sodium dodecyl sulphate, we find that the transmembrane helix spans residues 448 ±2 to 476. The significance of this observation is discussed in the context of glycosomal transport and susbtrate tunneling.

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