Alarming increase in the drug-resistant cases of malaria has emphasized the need for novel anti-malarials. In-depth understanding of molecular mechanisms involved in the development of human malaria parasite *Plasmodium falciparum*, may result in the identification of novel drug-targets. We have identified a novel signaling pathway in *Plasmodium falciparum*, which involves PfPKB, a protein kinase B (PKB) like enzyme. Calmodulin was identified as an activator of PfPKB; it activates PfPKB by interacting with its N-terminal region and promoting its autophosphorylation. Regulation of PfPKB by CaM and calcium could be demonstrated in the parasites. Additional studies indicated that Phospholipase C acts as an upstream regulator of this pathway as it controlled the calcium levels needed for this pathway to operate. To study the function of PfPKB, novel inhibitors were generated against it. Using inhibitors of PfPKB and other upstream components of this pathway, we could demonstrate that it may be involved in erythrocyte invasion, a key step in parasite life cycle.