Kinases are central to the control of cell cycle in several biological models, including Plasmodium falciparum, the ethiological agent of malaria infection. Unraveling the mechanisms of signal transduction pathways engendered by the malaria parasite is a fundamental question, considering the resistance to artemisin and the urgence for the development of new strategies to combat the disease. It is now accepted that, Plasmodium senses the environment and exploits calcium and cAMP signaling pathways to modulate cellular functions. The question then arises, to the identification of the downstream molecular targets followed by second messenger generation. Our study is focused on the Plasmodium falciparum kinase: PfeIK. By using western blotting, confocal microscopy assays and cell sorter we have identified that phosphorylation of the Plasmodium falciparum kinase (PfeIK) increases with melatonin in a time-dependent manner. Moreover, a PfeIk knock-out cell line lacks the ability to respond to the hormone treatment. Our results indicates that phosphorylation of PfeIK is a downstream molecular effector of melatonin-inducing changes in Plasmodium falciparum cell cycle. The present study could help us to dissect the Plasmodium falciparum signaling at a kinase level.

Key words: Plasmodium PfeIK and signal transduction
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