Title:	Role of single stranded DNA binding protein (Pf apSSB) plasmodium falciparum apicoplast replication
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Abstract:	An apicoplast is a non-photosynthetic plastid present in Plasmodium falciparum, which is a protozoan parasite known for causing malaria. Plasmodium belongs to the phylum Apicomplexa [1, 2, 3]. The apicoplast originated from an ancient bacteria through two endosymbiotic events which explains the presence of four membranes around the organelle [4]. Apicoplast plays an important role in the biosynthesis of isoprenoid precursors thus making it essential for the survival of P. falciparum [1]. It contains its own DNA of ~35kb (AT rich) but how its replisome works is not known very well [2, 3]. Its replication proteins are encoded by nuclear genome and gets imported to the organelle [3, 5, 6]. This project focuses on one of the key proteins of replisome, the P. falciparum apicoplast single stranded DNA binding protein (Pf apSSB). Pf apSSB (tetramer) is of bacterial origin and share sequence homology with Escherichia coli SSB. However a critical difference between bacterial and apicoplast SSB is the presence of a 28 amino acid C-terminal extension in Pf apSSB [3, 5, 6]. Due to this difference, interaction of Pf apSSB with other proteins might be different compared to those of E. coli SSB [3]. In E. coli replisome, χ subunit of DNA X interacts with E. coli SSB and this interaction is essential for replisome stability and delivers primers to lagging strand E. coli DNA polymerase [7]. If this complex is destabilized, it results in uncoupled leading and lagging strand synthesis and reduction in length of okazaki fragment [7]. But in apicoplast no such complex factor is known so far [3]. So how does apicoplast Polymerase (Pf apPol) replicates on lagging strand if Pf apSSB is protecting lagging strand ssDNA? So here we check for the possibility if Pf apSSB is having any interaction with Pf apPol that could be possibly influencing the activity of Pf apPol.
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