Plasmodium falciparum, an apicomplexan parasite and the causative agent of human malaria, has a complex life cycle within multiple hosts. More than a decade ago the P. falciparum genome was sequenced and gave us numbers of surprising features. The genome stands out in being extremely AT-rich (80%) and 60% of transcripts carry polyA stretches within genes distinguishing them from other model organisms. Protein translation is an essential field of Plasmodium research because most of the phylogenetic neighbors that have characterized translational machinery appear to differ in important aspects from what is known for Plasmodium.

Our recent work showed that genes with polyA tracks usually coding for Lysine are attenuated in the majority of prokaryotic and eukaryotic organisms. In most eukaryotes, polyA sequences are hot spots for ribosome stalling and frameshifting which elicits no-go (NGD) or non-sense mediated (NMD) mRNA surveillance system. Here we show that P. falciparum, in contrary to other tested organisms can efficiently translate mRNAs with polyA tracks without notable effects on mRNA and protein stability. Biochemical analysis of endogenous genes containing polyA tracks as well as introduced reporter sequences points out clear differences in translational abilities of Plasmodium compared to other eukaryotes. We show here that even eukaryotes with high AT-rich content, specifically, Tetrahymena, cannot efficiently translate polyA stretches, giving us a unique opportunity to compare human and malaria protein translation machinery and look for promising targets for new drug devilment against malaria parasites.