The lack of E2 conjugating enzyme (*Pfubc13*) effect on the growth and drug sensitivity in asexual blood-stage *Plasmodium falciparum*

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Abstract: The ubiquitination of protein is particularly essential in eukaryotic to control cellular integrity including protein degradation, stress response, cell signaling and DNA damage repair. This system comprises three enzymatic cascades, ubiquitin-activating enzyme (E1s), ubiquitin-conjugating enzyme (E2s) and ubiquitin ligase (E3s). In the human malaria parasite, *Plasmodium falciparum*, the protein quality control is very significant because of the high replication rate and the quick transformations during life cycle development. The Pfubc13 is a ubiquitin-conjugating (E2s) enzyme which is a homolog of human ubiquitin-conjugating enzyme 13 (HsUBC13). In this study, we identify the essentiality of the Pfubc13 in *P. falciparum* by using the inducible gene knockout, DiCre-recombinase system. The result shows that the *pfubc13*-knockout (*pfubc13*-KO) parasite has a deficiency in growth and delay phenotype compare to the control parasite. Moreover, the *pfubc13*-KO parasite shows more sensitivity to the DNA damaging agent, Methyl methanesulfonate (MMS), and Dihydroartemisinin (DHA). We are indicating that the Pfubc13 is essential for the growth and survival of asexual stage *P. falciparum*.