Malaria caused by *Plasmodium* parasites remains a major infectious disease in tropical and subtropical parts of the world, and 300-500 million malaria cases and about 1 million deaths are recorded annually. *P. falciparum*, that causes most severe malaria, has a complex life cycle involving asexual multiplication in the red blood cells (RBCs) in human host and sexual reproduction in the mosquito host. In the blood stage, *P. falciparum* grows through ring, trophozoite, and shizont stages to produce daughter cells within 48 h, and remolds the host RBC. To transport the parasite proteins to the infected RBC (iRBC) membrane surface, membranous structures called Maurer’s clefts are constructed in the iRBC cytoplasm acting as a sorting compartment in trafficking. The cytoadherent ligand PfEMP1 is exposed at protrusions called knobs on the surface of the iRBC to adhere to several receptors on the vascular endothelium and to evade the host immune system and hence elimination from human body.

The molecular biology of *Plasmodium* infection is well studied, but the ultrastructural morphological characterization is still not sufficient. Although ultrastructural 3D reconstruction of the whole *Plasmodium*-iRBC by electron tomography and FIB-SEM has been reported recently [1-4], the quantitative 3D analysis of the whole structure has little been performed. In this study, we used SBF-SEM to image the 3D structure of multiple whole *Plasmodium*-iRBCs. The 3D organization showed that Maurer’s clefts are not sub-compartments or membrane extensions of the parasitophorous vacuole membrane but independent membrane structures as reported by using other microscopy techniques [1, 2, 5]. Moreover, we will discuss the quantitative analysis of the numbers and/or sizes of their cell components at the ultrastructural level.

References


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Fig. 1: SBF-SEM slice including *P. falciparum*-infected red blood cells.

Fig. 2: 3D reconstruction of the whole *P. falciparum*-infected red blood cell.