

The 146th RIKEN BRC SEMINAR



2017年 12月 1日 (金) 11:00~11:50

バイオリソースセンター1階 森脇和郎ホール

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Host response to malaria infection: From ENU mutagenesis to CRISPR/Cas9 and beyond

Malaria is a disease that kills more than 1 million children annually. In endemic areas, some people die from malaria while others survive the infection. To better understand this, our group has developed a strategy to study the interaction between the parasite and its host using ENU mutagenesis in mice or CRISPR/Cas9 technology in mice or human red blood cells. These technologies are used to introduce mutations into the germline of mice or human red blood cells progenitors. Mice or red cells carrying protective mutations will survive to a malarial infection whereas all other mice or normal red blood cells will succumb or will be cleared from the circulation. The genes harbouring the mutations are identified and assessed as potential antimalarial drug targets. The potential of CRISPR/Cas9 gene editing technology to combat infectious diseases such as malaria and the latest development of the technology will be discussed.

References

1. Hortle, E.J et al. (2016) Adenosine Monophosphate Deaminase 3 activation shortens erythrocyte half-life and provides malaria resistance in mice: *Blood* 128(9):1290-301.
2. Greth A et al. (2012) A Novel ENU-Mutation in Ankyrin-1 Disrupts Malaria Parasite maturation in Red Blood Cells of Mice. *PLoS ONE*. 7(6): e38999.
3. Smith CM et al. Red cells from ferrochelatase-deficient erythropoietic protoporphyria patients are resistant to growth of malarial parasites. *Blood* (2015) 125(3):534-41
4. McMorran BJ et al. (2012). Platelet factor 4 and Duffy antigen required for platelet killing of *Plasmodium falciparum*. *Science*, 338(6112), 1348-1351.
5. Dogovski C et al. (2015) Targeting the Cell Stress response of *Plasmodium falciparum* to overcome Artemisinin resistance. *Plos Biology* 13(4):e1002132.

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理化学研究所以外からの参加希望者は、所属する大学または研究
機関が発行する身分証をご持参し、守衛所にて掲示し、入講証を
お受け取りください。

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