Splenic CD11c (+) cells derived from semi-immune mice protect native mice against experimental cerebral malaria

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Background: Immunity to malaria requires innate, adaptive immune responses and Plasmodium-specific memory cells. Previously, mice semi-immune to malaria was developed. Three cycles of infection and cure (3-cure) were required to protect mice against Plasmodium berghei ANKA infection.

Methods: C57BL/6J mice underwent three cycles of P. berghei infection and drug-cure to become semi-immune. The spleens of infected semi-immune mice were collected for flow cytometry analysis. CD11c (+) cells of semi-immune mice were isolated and transferred into native mice which were subsequently challenged and followed up by survival and parasitaemia.

Results: The percentages of splenic CD4 (+) and CD11c (+) cells were increased in semi-immune mice on day 7 post-infection. The proportion and number of B220 (+) CD11c (+) cells (plasmacytoid dendritic cells, DCs) was higher in semi-immune, 3-cure mice than in their native littermates on day 7 post-infection (2.6 vs. 1.1% and 491,031 vs. 149,699 respectively). In adoptive transfer experiment, three months after the third cured P. berghei infection, splenic CD11c (+) DCs of non-infected, semi-immune, 3-cure mice slowed Plasmodium proliferation and decreased the death rate due to neurological pathology in recipient mice. In addition, anti P. berghei IgG1 level was higher in mice transferred with CD11c (+) cells of semi-immune, 3-cure mice than mice transferred with CD11c (+) cells of native counterparts.

Conclusion: CD11c (+) cells of semi-immune mice protect against experimental cerebral malaria three months after the third cured malaria, potentially through protective plasmacytoid DCs and enhanced production of malaria-specific antibody.

Biography
Bao Quoc Lam graduated PhD. in infection research in Nagasaki University, Japan. His study has been on cerebral malaria in semi-immune mice. In malaria transmission areas, the adults, as semi-immune individuals, were less vulnerable to cerebral malaria than children. Naturally acquired immunity to malaria minimizes malaria morbidity and mortality in older children and adults living in intensive Plasmodium sp. endemic regions. His mouse research has suggested that IL-10 producing B and plasmacytoid dendritic cells confer protection against cerebral malaria in semi-immune mice. In addition, he is currently a co-investigator in a cohort: Resistance to reinfection and pathogenesis of human schistosomiasis in Eastern Africa to identify the protective immune responses.

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