Project description

Project Title: Mechanisms of innate immune cell imprinting during protozoan-nematode co-infection

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Project Description:

Co-infections reflect the natural situation in wildlife and domestic animals as well as human populations in endemic areas. But co-infections impose a hurdle for the immune system to efficiently mount protective immune responses against both infectious agents in particular when the pathogens induce contradictory immune responses.
Based on our previous work showing a defect in helminth-specific Th2 differentiation in protozoan-nematode co-infected mice (Ahmed et al. Frontiers in Cellular and Infection Microbiology, 2017*)*, the project aims to decipher the imprinting of innate immune cells early during co-infection and its impact on helminth-specific T cell responses in primary and secondary infection.
The following questions will be addressed: i) What is the phenotypical difference of innate cells during single versus co-infection? ii) By which mechanisms do innate cells affect Th2 differentiation in protozoan-nematode co-infected mice and how long does the imprinting of innate cells last? This is of particular interest, as Th2-driven responses are central for protection against secondary nematode infections. In conclusion, this project will detect how innate immune cells initiate the inadequate priming of T helper cell responses during co-infection and if the defect in helminth-specific Th2 differentiation in co-infected hosts is maintained upon challenge translating into inefficient nematode control.

Reference

Ahmed, N., T. French, S. Rausch, A. A. Kühl, K. Hemminger, I. R. Dunay, S. Steinfelder, S. Hartmann. 2017. Toxoplasma co-infection prevents Th2 differentiation and leads to a helminth-specific Th1 response. *Frontiers in Cellular and Infection Microbiology*, doi:10.3389/fcimb.2017.00341.