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The emerging role of ASC in dendritic cell metabolism and function during chlamydia infection

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Chlamydia trachomatis is a bacterial agent that causes sexually transmitted infections worldwide. The regulatory functions of dendritic cells (DCs) play a major role in protective immunity against chlamydia infections. The mechanisms underlying this immunomodulation are not fully understood. The inflammasome adaptor protein, apoptosis-associated speck-like protein containing a CARD (ASC) regulates the direction of immunity against such bacterial infections. Here, we investigate whether ASC, the critical components of inflammasome activation, participate in regulation of DC activation and function and the possible mechanisms during chlamydia infection. We observed that following chlamydia stimulation, the maturation and antigen presenting task of ASC-/- DCs were impaired compare to wild type DC (WT DC). Also, ASC deficiency induces a tolerogenic phenotype in chlamydia stimulated DCs, which may induce immune pathological response in infected host. We observed that following chlamydia stimulation of ASC-/- DCs prevented the chlamydia-induced increase in aerobic glycolysis, measured as extracellular acidification rates (ECARs) and had significantly reduced pyruvate production from the metabolism of glucose during glycolysis. To determine the effect of this reduction in pyruvate production in cellular respiration, we determined the morphology of the mitochondria. The results revealed that the mitochondria of infected ASC-/-DCs had their cristae disrupted compared to the normal narrow pleomorphic cristae found in un-stimulated WT, ASC-/- and stimulated WT DCs. In conclusion, the results suggest that ASC deficiency interrupts DC function through reprogramming of DC metabolism starting from glycolysis to the electron transport chain which occurs within the mitochondria, which controls the actions and functions of DCs during chlamydia infection. The interface between ACS and metabolism in innate immunity is of great interest. It may be possible for small molecules to reprogram the metabolism of immune cells to enhance vaccine efficacy against infectious diseases and tumors.

Biography

Qing He is an Associate Professor of Microbiology, Biochemistry & Immunology in the Morehouse School of Medicine, USA. She has a broad background in "Infectious disease and host innate and acquired immune responses" with specific training and expertise in key research areas. Her work focuses primarily on "Antigen processing, presentation and immunomodulation". She focuses on "The role of apoptosis-associated speck-like protein containing a CARD (ASC) in GT dendritic cell (DC), macrophage and T & B cell functions during genital chlamydia infection and to investigate the metabolic and immunologic plasticity of mucosal dendritic cells and T cells in the absence of the inflammasome. She has published several manuscripts that are related to this project.

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