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Persistence of memory B cells by inhibition of caspase-9-dependent and independent cell death

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Exposure to antigen stimulation leads to significant expansion of antigen-specific lymphocytes in adaptive immune responses. Following the clearance of antigens, most of these expanded lymphocytes are cleared by programmed cell death. However, some activated antigen-specific lymphocytes can survive and develop into memory cells. Memory B cells represent a heterogeneous population that can rapidly proliferate and differentiate into antibody secreting cells after re-encountering the antigens. It has been shown that the persistence of memory B cells is independent of the presence of antigens. To achieve long-term survival, memory B cells may suppress the intrinsic cell death pathways. The molecular mechanisms underlying the long-term survival of memory lymphocytes are investigated. Compared to germinal center B cells, memory B cells displayed inhibition in caspase signaling and were resistant to cell death during in vitro culture. Memory B cells produced less reactive oxygen species under stress and were resistant to oxidative stress-induced necrosis. Using memory B cells with conditional deletion of caspase-9, we demonstrated that the inhibition of both caspase-9-dependent apoptosis and oxidative stress-induced necrosis were important for the long-term survival of memory B cells. Simultaneously targeting distinct cell death pathways may promote the ling-term survival of memory B cells and improve the efficacy of vaccination.

Biography

Jin Wang received his Ph.D. from the University of Southern California and postdoctoral training at the NIH. He is an Associate professor at the Department of Pathology and Immunology of Baylor College of Medicine

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