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Activation of apoptotic pathways in the course of B cell infection with *Francisella tularensis*Z. Krocova¹, L. Zivna²¹FMHS UoD, Institute of Molecular Pathology, Hradec Kralove, Czech Republic, ²FMHS, UoD, Hradec Kralove, Czech Republic

Francisella tularensis is able to adhere and entry B lymphocytes and cause their apoptosis. We infected human B cell line Ramos with *F. tularensis* FSC200 *in vitro*. Killed bacteria *F. tularensis* FSC200, uninfected Ramos cells, cells influenced by 10 mM Staurosporine, and 200 ng/ml anti-CD95 antibodies were used as controls. Only live bacteria induced apoptosis measured by flow cytometry using AnnexinV in time and MOI manner. We measured activation of Caspase 8, Caspase 9 (western blot, colorimetric assay), caspase 3 (colorimetric assay), presence of Bcl-2 family proteins Bax and Bcl-xL in whole cell lysate, tBid, Cytochrome C and AIF (Western blot), in mitochondrial and cytosolic fraction and low-molecular form of protein PARP-1 that is cleaved by active caspase 3 in whole cell lysate by Western blot. To detect the changes on mitochondrial membrane potential we used flow cytometry and probe JC-1 that is specific for mitochondrial membrane depolarization. Live bacteria *F. tularensis* FSC200 activate both caspase pathways – receptor mediated and mitochondrial – as well as caspase independent pathway. Low level of pro-apoptotic protein Bax was expressed in the course of infection with or live either killed bacteria and vice versa huge amount of Bcl-xL was stimulated particularly after infection of B cells with killed bacteria. Killed bacteria also stimulated Caspase 3 and PARP but caspase 8, 9, tBid, AIF and cytochrome C were not activated and killed bacteria did not cause apoptosis measured by Annexin and JC-1. Results show that only live bacteria *F. tularensis* induce apoptotic process terminate by cell death.