Chlamydia trachomatis can protect host cells against apoptosis in the absence of cellular Inhibitor of Apoptosis Proteins and Mcl-1.

Abstract:
Infection with Chlamydia protects mammalian host cells against apoptosis. Hypotheses have been proposed to explain this molecularly, including the up-regulation of host anti-apoptotic proteins such as cellular Inhibitor of Apoptosis Protein (IAP) 2 and the Bcl-2 protein Mcl-1. To test for the importance of these proteins, we used mouse embryonic fibroblasts from gene-targeted mice that were deficient in cIAP1, cIAP2, cIAP1/cIAP2, XIAP, or Mcl-1. Infection with Chlamydia trachomatis protected all cells equally well against apoptosis, which was induced either with tumour necrosis factor/cycloheximide (IAP-knock-out cells) or staurosporine (Mcl-1-knock-out). Therefore, these cellular anti-apoptotic proteins are not essential for apoptosis-protection by C. trachomatis.