P008 Bax localisation during neutrophils apoptosis triggered via the Fas pathway

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During inflammation neutrophils are rapidly recruited to the affected site, where they form a first line of defence against invading organisms. Resolution of inflammation requires that the environmental milieu returns to normal, a process which entails the safe removal of infiltrated neutrophils. Neutrophils undergo apoptosis which renders these cells unresponsive to exogenous secretagogues and marks them for recognition by phagocytes using an anti-inflammatory mechanism. Thus neutrophil apoptosis plays an important role in the resolution of inflammation. Many of the Bcl-2 family members, key regulators of apoptosis, are present in neutrophils. We have shown that the pro-apoptotic Bcl-2 family member Bax moves from the cytosol to the mitochondria during stress induced neutrophil apoptosis. However, it is unknown whether Bax is involved in death receptor-mediated and caspase-8 dependent neutrophil apoptosis. Thus using the anti-FasR mAb CH11 to induce apoptosis, we have investigated whether Bax translocates to the mitochondria during death receptor induced apoptosis in addition to showing caspase-8 recruitment to Fas as determined by confocal microscopy. We believe that Bax insertion into the mitochondrial membrane critically regulates neutrophil apoptosis.