



UNIVERSITY OF GOTHENBURG
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Abstract - Master Thesis Project, the Pharmacy Programme

Induction of neutrophil apoptosis by NK cells

Jenny Ousbäck, 2009

Neutrophils (PMN) are potent inflammatory cells essential for host defense, but may at the same time be responsible for tissue destruction during inflammatory disease. The removal of apoptotic PMN from inflammatory sites is a way to avoid cell leakage and unintended tissue damage. Thus, PMN apoptosis is crucial for resolution of inflammation and implicated in many disease states. Natural killer (NK) cells are mainly involved in the defence against viral infections and altered cell growth but also known to interact with other immune cells and modulate their functions; insufficient information exist on the ability of NK cells to alter neutrophil activation and survival. We found that NK cells significantly induced PMN apoptosis when the two leukocyte types were co-cultured. Co-culturing also induced degranulation of NK cells as well as production of reactive oxygen species from PMN, indicating physical interaction. Cell-cell interaction was crucial for mediating the PMN apoptosis that was caspase dependent and blocked by the general caspase inhibitor z-VAD-fmk. A mixed lymphocyte population had no effect on PMN activity or survival, indicating that PMN interaction was specific for NK cells. Our findings implicate that NK cells may accelerate PMN apoptosis through cell-cell interactions and such interactions could play an important part for the resolution of inflammation.