Chiravat Memorial Lecture

ENHANCING THE RESOLUTION OF INFLAMMATION: A THERAPEUTIC TARGET?

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ABSTRACT

Over-recruitment, uncontrolled activation and defective removal of inflammatory cells (especially neutrophil and eosinophil granulocytes) from inflammatory loci likely play a prominent role in the development of chronic inflammatory conditions such as rheumatoid arthritis and asthma. During resolution of inflammation granulocytes undergo apoptosis thereby allowing their recognition and clearance by phagocytes using mechanisms that down-modulate the inflammatory response. Although there is little doubt that apoptosis plays a critical role in embryological morphogenesis and tissue remodelling, it's precise role in inflammatory diseases is still unclear. Evidence will be presented showing that inflammatory mediators and pharmacological agents can differentially modulate neutrophil and eosinophil apoptosis and alter macrophage phagocytosis of apoptotic cells. For example, we believe that the powerful anti-inflammatory glucocorticoids may be using the above processes to selectively induce eosinophil (as well as lymphocyte) apoptosis and importantly enhance the clearance of the apoptotic cells. Thus the hypothesis that selective induction of neutrophil or eosinophil apoptosis and augmented non-phlogistic removal of apoptotic granulocytes by phagocytes as a potential therapeutic target will be discussed.