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Research Topics

- **Investigation of the *in vitro* and *in vivo* effects of extracellular vesicles (EVs) derived from viable, apoptotic, necroptotic, pyroptotic, and ferroptotic mesenchymal stem cells (MSC) in psoriasis models.**
  Psoriasis is a chronic, immune-mediated inflammatory disease characterized by pathological interactions between keratinocytes and immune cells and the sustained activation of the IL-23/IL-17 axis. Although current biological therapies effectively inhibit inflammatory cytokines, they do not target the early mechanisms that regulate the initiation and persistence of inflammation. Growing evidence suggests that damage-associated molecular patterns (DAMPs) released during regulated cell death of keratinocytes play a key role in sustaining immune activation by inducing persistent functional reprogramming of both innate and adaptive immune cells, thereby establishing a self-perpetuating inflammatory cycle. Apoptotic mesenchymal stromal cells (MSCs) are known to possess potent immunoregulatory activity. Our preliminary in vivo results obtained using an imiquimod-induced psoriasis model demonstrate that extracellular vesicles (EVs) derived from apoptotic and necroptotic MSCs reduce inflammation more effectively than EVs derived from viable MSCs, suggesting that the mode of regulated cell death determines the therapeutic function of MSC-derived EVs. The aim of this project is to elucidate how regulated cell death influences the composition and immunomodulatory properties of MSC-derived EVs. We will investigate the effects of EVs on dendritic cell and macrophage polarization, DAMP receptor expression, and T-cell responses, and validate their therapeutic efficacy in vivo. Our research introduces a novel mechanistic and therapeutic concept linking regulated cell death, EV-mediated immunoregulation, and chronic inflammatory diseases, and may provide the foundation for the development of new cell-free immunotherapeutic approaches.