Peripheral blood monocytes phagocytose and kill pathogens; they are recruited into inflammatory sites and are responsible for triggering the symptoms of sepsis. We focused on the consequences of apoptosis of monocytes, spontaneous or induced, on their function.

Monocytes acting as antigen-presenting cells (APC) are eliminated by activated T-cells via the mechanism that triggers their apoptosis. This represents an important immunoregulatory mechanism, since subsets of monocytes less sensitive to T-cell mediated killing are more efficient as antigen presenting cells. Furthermore, blocking CD95 or interfering with the intracellular pathways of apoptosis signaling increase monocyte antigen-presenting capacity, indicating that measures which interfere with monocyte apoptosis can be used to increase T-cell responses.

Recently, we also found that monocytes recognize apoptotic cells, and that this has an impact on the monocytes’ ability to produce cytokines and to present antigens. In particular, monocytes in contact with apoptotic cells (monocytes or PMNs) produce more IL-10, while the production of proinflammatory cytokines is rather inhibited. A small monocyte subpopulation CD16$^{\text{high}}$ CD14$^{\text{low}}$ produces very little if any IL-10. By contrast, the CD16$^{\text{high}}$ CD14$^{\text{high}}$ subpopulation is a predominant producer of IL-10. Both CD16-positive monocytes populations are increased in proportion and number during sepsis. Interestingly, the CD16$^{\text{high}}$ CD14$^{\text{high}}$ population easily associates with apoptotic cells that are known to increase in number during sepsis. Monocyte contact with apoptotic cells also compromises the formers’ ability to present the antigen (PPD) to CD4+ Th1 memory cells. By contrast, the antigen present in apoptotic monocytes may be re-used by bystander non-apoptotic APC.

We conclude that monocyte survival and functional activity at the site of local inflammation or during sepsis have an important role in the regulation of the inflammatory response and of the specific immune responses.