Questions concerning interactions of tumour with cells of the immune system are still current. One of the most intriguing participants playing an important role in these interactions are microvesicles. Our group focuses on the interactions of tumour-derived microvesicles (TMV) with human monocytes, which are precursors of tumour associated macrophages (TAM). Following their contact, TMV modulate phenotype of human monocytes, as shown by the transfer of tumour associated antigens. Moreover, TMV may modulate biological activity of monocytes by inducing production of ROI, cytokines and chemokines. Distinct subpopulations of monocytes (CD14++CD16-, CD14+CD16++) interact with TMV in a different manner, showing a pattern similar to the stimulation with tumour cells.

In the in vitro model of in situ TAM - tumour interactions, monocytes following short exposure to tumour cells respond to subsequent restimulation with tumour cells with decreased TNF and IL-12 but increased IL-10 secretion. The cytokine profile of these monocytes is equivalent to M2-like polarized macrophages. Our preliminary data indicate that also monocytes pre-exposed to TMV and restimulated with tumour cells show M2-like cytokine secretion. Taken together, TMV significantly modulate biological activity of monocytes and may affect their function during tumour progression, thus TMV mimicking the effect of tumour cells on monocytes. We postulate that TMV should be considered as a modulator of monocyte/macrophage functions in the tumour bed and in peripheral blood.