

## Self-help -- tumors promote their own metastasis

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Current research suggests that tumor-secreted exosomes inhibit the immune response, enhancing tumor metastasis. The related report by Liu et al, "Contribution of MyD88 to the tumor exosome-mediated induction of myeloid derived suppressor cells," appears in the May 2010 issue of *The American Journal of Pathology*.

The immune system plays a critical role in identifying and destroying [tumor cells](#). Many tumors overcome this surveillance by inhibiting local immune responses, often leading to metastasis.

One potential method of tumor-mediated [immune suppression](#) is secretion of exosomes - small, membrane-enclosed sacs that can be used for storage or transport. Researchers led by Dr. Huang-Ge Zhang and colleagues at the University of Alabama at Birmingham and the University of Louisville, KY therefore examined the effect of tumor-secreted exosomes in lung metastasis. They found that treatment with tumor exosomes increased the number of myeloid-derived suppressor cells, which inhibited immune activation and accelerated tumor metastasis in the lung. This effect was mediated by the molecule MyD88, which plays a key role in the innate immune response.

In this study, Liu et al "identified the role of tumor exosomes in the enhancement of [tumor metastasis](#) through the expansion of myeloid-derived suppressor cells. A tremendous amount of information remains to be discovered about the mechanisms of cellular machinery that regulates the sorting of immune suppressor molecules into tumor

exosomes. [Their] goal will now be to develop not only strategies to interfere with these pathways, but to transform tumor exosomes from immune suppressors to immune stimuli with the objective to ultimately use these modified exosomes as a tumor vaccine."

**More information:** Liu Y, Xiang X, Zhuang X, Zhang X, Liu C, Cheng Z, Michalek S, Grizzle W, Zhang H-G: Contribution of MyD88 to the tumor exosome-mediated induction of myeloid derived suppressor cells. *Am J Pathol* 2010, 176: 2490-2499

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