Macrophage-mediated tumor cell phagocytosis and subsequent neoantigen presentation are critical for generating anti-tumor immunity. A study aimed to uncover the potential clinical value and molecular mechanisms of MicroRNA-22 (miR-22) in tumor cell phagocytosis via macrophages and more efficient T cell priming. Tu et al. found that miR-22 expression was markedly downregulated in primary macrophages from glioma tissue samples compared to adjacent tissues. miR-22-overexpressing macrophages inhibited glioma cell proliferation and migration, respectively. miR-22 upregulation stimulated the phagocytic ability of macrophages, enhanced tumor cell phagocytosis, antigen presentation, and efficient T cell priming. Additionally, our data revealed that miR-22-overexpressing macrophages inhibited glioma formation in vivo, HDAC6 was a target, and NF-κB signaling was a pathway closely associated with miR-22 in tumor-associated macrophages (TAMs) of glioma. Our findings revealed the essential roles of miR-22 in tumor cell phagocytosis by macrophages and more efficient T cell priming, facilitating further research on phagocytic regulation to enhance the response to tumor immunotherapy [1)]

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Tu J, Fang Y, Han D, Tan X, Xu Z, Jiang H, Wang X, Hong W, Wei W. MicroRNA-22 represses glioma development via activation of macrophage-mediated innate and adaptive immune responses. Oncogene. 2022 Mar 12. doi: 10.1038/s41388-022-02236-7. Epub ahead of print. PMID: 35279703.