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Circulation of cancer cells in blood ow is an important phase for distant cancer metastasis, during which cancer cells are exposed to hemodynamic shear stress. Recent studies identi ed shear stress as the primary factor that damages circulating tumor cells i blood ow. However, it remains unclear whether shear stress can modulate properties and functions of tumor cells in a manner that might help tumor cells to exit circulation. In our study, we demonstrate that uidic shear stress could positively regulate migration and extravasation of surviving tumor cells in circulation, and facilitate metastasis. We established a micro uidic circulatory system to apply physiological uidic shear stress on breast cancer cells and mimic the physical environment in blood ow. An arterial level of shear stress generated in the circulatory system signi cantly increased tumor cell migration in both transwell and wound healing assays. We also observed that shear stress enhanced extravasation of breast tumor cells in a transendothelial assay. mechanistic study identi ed the elevation of cellular ROS as an early molecular event induced by shear stress. e excessive cellula ROS subsequently activates ERK1/2 pathway, which leads to tumor cell migration and extravasation. Finally, by using a zebra sh model, we demonstrated that application of antioxidants could suppress shear stress-enhanced tumor cell extravasation in vivo. is new understanding of how uidic shear stress promotes metastatic potential of tumor cells has important implications in cancer treatment and can help us identify latent therapeutic targets for inhibiting tumor progression.

Biography

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