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Tentative topic of the invited talk

Microfluidics Based Study to Understand EMT and MET Transitions

Abstract of the invited talk

The authors take recourse to interdisciplinary research in the area of bio-microfluidics applicable for diagnostics of cancer metastasis. While the epithelial-to- mesenchymal transition (EMT) is known to be an important factor in cancer spread, how the converted cells travel through the blood vessels and undergo reverse transition (mesenchymal-to-epithelial, MET) at the secondary sites has hitherto not been completely understood.

EGF exists in the mammary glands. EGF is a pivotal secretory growth factor produced by various epithelial tissues, including mammary epithelium, and primarily promotes cellular growth and proliferation. In this study, MDA-MB-468 breast cancer cells induced to undergo EMT by treatment with specified amount of epidermal growth factor (EGF) were initially passed through several blockages and then through a constricted microchannel, mimicking the flow of invasive metastatic cells through constricted blood microcapillaries. EMT cells acquired enhanced migratory properties and retained 50% viability, even after migration through wells and a constricted passage of 7 μm and 150 microns in length at a constant flow rate of 50 $\mu\text{L/h}$. The hydrodynamic properties revealed cellular deformation with a deformation index, average transit velocity, and entry time of 2.45, 12.3 mm/s, and 31,000 μs , respectively for a cell of average diameter 19 μm passing through one of the 7 μm constricted sections.

The cells collected at the channel outlet regained epithelial character, undergoing reverse transition (mesenchymal to epithelial transition, MET) in the absence of EGF. The number of colonies formed from EMT cells and paclitaxel-treated EMT cells after passing through a constriction were found to be 95 ± 10 and 79 ± 4 , respectively, confirming that the EMT cells were more drug resistant with a concomitant two-fold higher expression of the multidrug resistance (MDR1) gene.

The bio-microfluidics-based interdisciplinary research initiatives were successful to understand how the cells lose their EMT phenotype and revert back to epithelial format through a process termed MET.