Atmospheric pressure plasma induced changes in cellular phenotypes

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Atmospheric pressure plasma (APP) treatment has gained much attention in biomedical applications due to its selective activation of certain cell types. A number of attempts have been reported on skin therapy using APP to enhance wound healing or to treat cancers, most of which have been unfortunately limited to phenotypic observations. In this study, we compared metastatic cancer to normal cells from liver (SK-HEP-1 vs. THLE-2) and from mammary gland (MDA-MB-231 vs. MCF-10A) to investigate the plausible existence of signature characteristics in cellular responses to plasma in a cancer dependent manner [1].

When treated with APP, human liver cancer cells (SK-HEP-1) and normal cells (THLE-2) exhibited distinctive cellular responses, especially in relation to their adhesion behavior. We discovered the critical threshold voltage of 950 V, biased at the electrode of the micro-plasma jet source, above which SK-HEP-1 started to detach from the substrate while THLE-2 remained intact. Our mechanical and biochemical analyses confirmed the presence of intrinsic differences in the adhesion properties between the cancer and the normal liver cells, which provide a clue to the differential detachment characteristics of cancer and normal cells to the APP. Similar responses to APP were observed in mammary gland cells.

We also exposed the human dermal fibroblast (HDF) and human aortic endothelial cells (HAEC), to APP (970 V, 50 kHz) in vitro to observe dramatic phenotypic changes in relation to cellular transdifferentiation. Our results support the potential use of APP to control the cellular transformation to enhance wound healing or to suppress the growth of tumor mass by controlling the motility of these cell types.

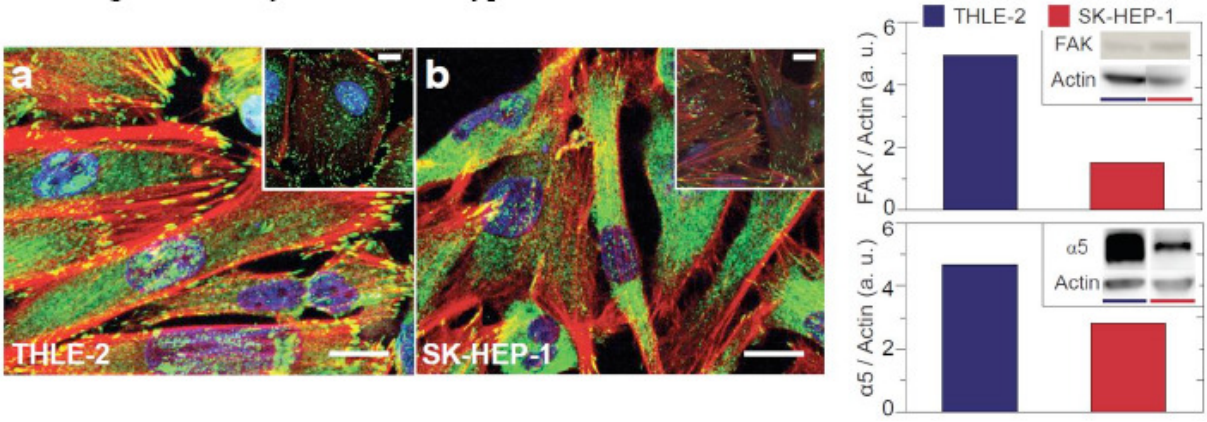

Figure 1: Immunofluorescence image of paxillin dots (green) and actin stress fibers (red) of (a) THLE-2 and (b) SK-HEP-1 (scale bar reads 20 μm). Right: Amount of FAK and α5 integrin proteins. The insets are the Western blot bands of FAK protein [1].

References

[1] Gweon B., Kim M., Kim D. B., Kim D., Kim H., Jung H., Shin J. H., and Choe W., Applied Physics Letters (2011), **99**, 063701-063704