Mechanical stress facilitates wound healing in keratinocytes

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Epidermal keratinocytes are continuously exposed to a variety of external mechanical stimuli, which modulate fundamental cell events such as proliferation and differentiation. Actually mechanical stimuli facilitate proliferation of keratinocytes, which property has clinically been utilized to facilitate skin repair. However, detailed relationship and mechanisms linking between mechanical stress and ketinocyte proliferation remain unclear. We have explored the mechanosignaling pathways in cultured endothelial and mammary cells by measuring stretch-induced intracellular Ca^{2+} mobilization and ATP release. Furthermore, we found that wound healing in endothelial cells was significantly influenced by externally applied mechanical stress.

We used keratinocytes, HaCaT cell lines cultured on a flexible silicone chamber coated with collagen. Cells were subjected to a uni-axial stretch for a few seconds. We measured intracellular Ca^{2+} increase and ATP release using a newly developed real time imaging microscope and found that both of them were increased in a stretch-dependent manner. Wound healing assay under stretch was also carried out to explore therapeutic potential of mechanical stimuli for skin repair. Wound healing was significantly facilitated by stretch through the increases in the rate of cell migration and proliferation, which seemed to be mediated by the stretch-induced increases in the Ca^{2+} influx and ATP release.