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Injury-Induced Microvesicle Particle Release in Keratinocyte Cell Line and Abdominoplasty Skin

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Ultraviolet B (UVB) radiation and burn injury are very common environmental stressors that can result in systemic effects. The mechanism by which these stressors which only impact the top layer of skin can induce systemic effects is an important unanswered question in photobiology. Microvesicle Particles (MVPs) are small fragments of the cell membrane, 200 nm to 1000 nm in diameter. It is currently believed that the shedding of MVP from various cells in response to stressors are involved in cellular communication. The current study was designed to see if epithelial cells can release MVP in response to UVB radiation and burn injury. The human keratinocyte-derived cell line HaCaT was used in our initial studies. HaCaT cells were burned in a 90° water bath, or treated with UVB. Given that both UVB and thermal burn injury also produce Platelet-activating Factor (PAF), we also treated HaCaT cells with the PAF agonist CPAF. After incubation, the MVPs were isolated analyzed. These studies demonstrated that thermal burn injury, UVB, and CPAF all increased MVP release from HaCaT cells. Of interest, only short (<45 second), but no longer exposures to heat resulted in increased MVP release. To define the effects of these agents on human skin, we instituted a novel methodology where suction-induced blisters were induced on human skin obtained from abdominoplasties. The blisters were treated with thermal burn, UVB, or topical CPAF and the fluid was obtained from the blisters for MVP analysis. Again, UVB and CPAF treatment induced MVP, yet thermal burn injury resulted in decreased levels of MVP. These studies demonstrate that various environmentally relevant stressors trigger MVP production in vitro as well as ex vivo. As MVP often contain signaling molecules, these findings suggest that MVP could be involved in the human response pathway to stressors like UVB radiation and burn.