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Injury-Induced Microvesicle Particle Release in Human Skin Explant Tissue

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Injury-Induced Microvesicle Particle Release in Human Skin Explant Tissue

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Microvesicle particles (MVP) are 100-1000 nm vesicles that bud from the cell membrane and transmit intercellular signals locally and systemically via their bioactive cargo (e.g. cytokines). We have previously shown that thermal burn injury or UVB radiation result in the formation Platelet Activating Factor (PAF), and UVB or a PAF agonist (CPAF) can promote MVP formation in keratinocytes. Thermal burn and UVB radiation can result in systemic effects, even though their injury is localized to the skin. We therefore propose that PAF-producing injuries, UVB irradiation or thermal burn injury, result in the formation of MVPs that can potentially transport cytokines through the blood stream and induce systemic effects. This study, in-part, addresses that proposed mechanism by demonstrating increased MVP release after thermal burn injury *in vivo* and *ex vivo*. *In vivo* there was a significant increase in MVP concentration in mouse skin that received thermal burn injury compared to untreated skin. This demonstrates that there is an increase in MVP release after injury in living models. Human skin explants that received suction blisters to separate the epidermis from the dermis exhibited increased MVP release into the blister fluid in response to CPAF, UVB or thermal burn injury as compared to controls, suggesting epidermal MVPs are mobile and can exit the epidermis. Acid Sphingomyelinase (aSMase), an enzyme that breaks sphingomyelin into ceramide and phosphorylcholine, has been found to be involved in MVP release in other cell types. Utilizing an FDA approved aSMase inhibitor, Imipramine, we found that UVB, CPAF and thermal burn-mediated MVP release is completely dependent on aSMase in keratinocyte cell lines and human explant skin. These results suggest that PAF-producing skin injuries (thermal burn and UVB) can induce MVP release, and the release can be therapeutically inhibited by imipramine.