ABSTRACT:

Vesicants such as sulfur mustard (SM) and nitrogen mustard (NM) cause blistering of skin, and microblistering of the cornea. These blisters are highly similar to those caused by ultraviolet (UVB) exposure, showing epithelial separation at the same blister plane as that from UVB, i.e., at the basal surface of the basal epithelial cells, where they sit on their basement membrane. Physicians have noted for almost 100 years that mustard injuries take longer to heal than blisters incurred by other injuries. To examine this delay in healing after mustard injury, we have followed wound closure over the course of 7 days in corneal organ cultures exposed to doses of NM and UVB that induce equivalent epithelial-stromal separation. Our data show that matrix deposited in the wound bed (the provisional matrix) is an important player in the delay. The anti-adhesive provisional matrix components deposited in the wound bed are removed at least a day later in the healing mustard wound than in the UVB-induced wound. In addition, fibronectin, a pro-migratory provisional matrix component, was found to be delayed in its deposition into the wound bed of a mustard injury as compared to an equivalent UVB injury. The demonstration of delayed healing after mustard injury highlights the importance of considering the provisional matrix when developing potential therapies for mustard injury.

LOCATION: Life Sciences Building Rutgers - The State University of New Jersey, 145 Bevier Road, Piscataway, New Jersey 08854, New Jersey Center for Biomaterials Suite - Conference Room 102

TIME: 5:30 PM

HOST: Bozena B. Michniak-Kohn, Ph.D., M.R.Pharm.S. Director, Center for Dermal Research, Professor of Pharmaceutics, Ernest Mario School of Pharmacy