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IL-1 Generated Subsequent to Radiation-induced Tissue Injury Contributes to the Pathogenesis of Radiodermatitis

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Et al.

Title: IL-1 Generated Subsequent to Radiation-induced Tissue Injury Contributes to the Pathogenesis of Radiodermatitis.

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Radiation injury in the skin causes radiodermatitis, a condition in which the skin becomes inflamed and the epidermis can break down. This condition causes significant morbidity and if severe it can be an independent factor that contributes to radiation mortality. Radiodermatitis is seen in some settings of radiotherapy for cancer and is also of concern as a complication post-radiation exposure from accidents or weapons, such as a ‘‘dirty bomb’’. The pathogenesis of this condition is incompletely understood. Here we have developed a murine model of radiodermatitis wherein the skin is selectively injured by irradiation with high-energy electrons. Using this model we showed that the interleukin-1 (IL-1) pathway plays a significant role in the development of radiodermatitis. Mice that lack either IL-1 or the IL-1 receptor developed less inflammation and less severe pathological changes in their skin, especially at later time- points. These findings suggest that IL-1 pathway may be a potential therapeutic target for reducing the severity of radiodermatitis.