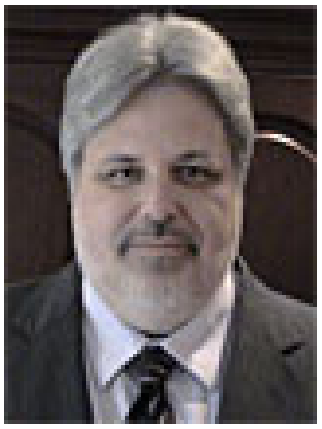
MORE Science at UTSA
Environmental Science and Engineering
Spring 2007 Seminar Series

Where: **Loeffler room (3.03.02) in the BioScience Building**
When: **4:00 PM – 5:00 PM on April 6, 2007**

Refreshments will be provided

Speaker: Dr. Victor Sylvia



Dr. Victor L. Sylvia received his B.S., M.S., and Ph.D. degrees from Texas A&M University in College Station, He is currently an Associate Professor in the Department of Orthopaedics at the University of Texas Health Science Center in San Antonio. His laboratory examines angiogenic factor expression by giant cell tumor stromal cell cultures and matrix metalloproteinase expression by soft tissue sarcomas pre- and post-irradiation. He is also investigating mechanisms to improve chronic wound healing using a diabetic mouse model and skin fibroblast cultures, specifically focusing on regulation of matrix metalloproteinases by nitric oxide. He collaborates with Dr. Sarkar and Dr. Datta at UTSA examining how bioremediation methods affect bioavailability of arsenic. Dr. Sylvia is also on the faculty of the Biomedical Engineering program at UTSA. His laboratory is part of the Center for Clinical Bioengineering investigating the growth and development of osteoblasts on various biomaterials by measuring changes in cell viability, cytokine production, proliferation, differentiation and mineralization.

Topic: Matrix Metalloproteinases and their Role in the Wound

Wound repair is a physiological event, in which tissue injury results in a repair process, which finally leads to restoration of structure and function of the tissue. Cutaneous wound repair can be divided in to three overlapping phases: (i) formation of fibrin clot followed by inflammation (early or late); (ii) re-epithelialization and granulation of tissue formation; and (iii) matrix formation and remodeling. In the first phase of wound repair, a fibrin clot is formed as a result of platelet aggregation and blood coagulation. Various growth factors and chemotactic factors released from activated coagulation pathways, injured cells, and platelets attract inflammatory cells to the wound area. Proteolytic degradation of extracellular matric (ECM) is required in many stages of wound repair, such as degradation of the provisional matrix, angiogenesis and keratinocyte migration. Following injury, MMP-1 is expressed by basal keratinocytes at the migration front of epidermis in several types of cutaneous wounds including incision wounds and blistering skin diseases. The expression of MMP-1 in basal keratinocytes is rapidly induced after dermal injury, persists during healing, and subsides at re-epithelialization. MMP-1 expression is most abundant at the very edge of the wound, and it diminishes progressively away from the wound edge. In chronic wounds, such as those common in diabetic complications, MMP-8 and MMP-9 are over-expressed, contributing to premature breakdown of the ECM, and interfering with normal healing. Certain nitric oxide-generating compounds can reduce MMP expression and promote healing.

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