Hani Awad, Ph.D.

Assistant Professor, Research

Hani Awad, Ph.D.
Assistant Professor
Biomedical Engineering & Orthopaedics
Center for Musculoskeletal Research
URMC Box 639
University of Rochester
Rochester, NY 14642
Phone: 001 585 273 5268
Fax: 001 585 273 4746
Email: hani_awad@urmc.rochester.edu

My research program is focused on understanding the role of biophysical factors (such as mechanical forces and deformations, fluid flow, oxygen levels, and pH) and biochemical factors (such as growth factors, cytokines, and hormones) play in regulating the growth, differentiation, and metabolism of stem and primary (chondrocytes and osteoblasts) cells in tissue engineered cartilage and bone, respectively.

Specifically, there is growing evidence that the hydrodynamic environment provided by fluid flow is beneficial for growing engineered tissues. This has been attributed to a coupling of improved nutrient transport due to convection (which overcomes diffusion limitations *in vitro*) and metabolic stimulation of the cells due to shear (or stretch) activated signaling pathways. For example, there is evidence that flow-induced shear produces anabolic effects in chondrocytes mediated or amplified by growth factors such as TGF-beta1 and IGF-I. Further, the chondrocytes' response to shear stress appears to involve activation of MEK/ERK signaling pathways although the downstream transcriptional mechanisms involved remain to be uncovered. Using a novel bioreactor design, we study the effects of flow-induced shear stress and its interaction with growth factors and other vital proteins on the differentiation of adult stem cells in cartilage and bone tissue engineering applications. Our goal is to better understand the signaling pathways and the transcriptional mechanisms that regulate stem and primary cell responses (proliferation and differentiation) to fluid-induced shear, ultimately leading to the creation of functional tissue-engineered grafts.

In addition, I am interested in structurally and biomechanically evaluating models of human skeletal disease (*i.e.* OA and osteoporosis) and trauma (long bone fractures) using the various transgenic mice developed in our laboratories by colleagues. These skeletal phenotyping studies utilize non-invasive imaging using 3D quantitative micro-CT and biomechanical testing among other techniques. Transgenic mice also offer novel and exciting opportunities to study the roles that certain genes (deleted or overexpressed) play in specific differentiation pathways (*e.g.* Sox9 in chondrogenesis). Such studies could have a tremendous impact on the field of tissue engineering and on our ability to control the differentiated fate of stem cells and mesenchymal progenitor cells.