Winter Symposium 2018
February 22, 2018
Eric P. Newman Educational Center
1:00-5:30pm

Featured Speaker (4:30pm):

Andre J. Van Wijnen, Ph.D.
Consultant, Department of Orthopedic Surgery
Joint Appointment
Consultant, Department of Biochemistry and Molecular Biology
Professor of Biochemistry and Molecular Biology
Professor of Orthopedics

Mayo Clinic

“Molecular Strategies for Musculoskeletal Regenerative Medicine”

Yousef Abu-Amer, Ph.D., Symposium Chair

Presentations by 2017 Pilot & Feasibility Study Awardees, Clarissa Craft and Tim Peterson
Podium presentations from top rated abstracts
Poster session

7 - $800 Travel Awards will be awarded.

Please check the website for continued updates and a sample agenda:

http://www.musculoskeletalcore.wustl.edu/content/Calendar/2998/Winter-Symposium.aspx

Please remember to include reference to support from the Musculoskeletal Research Center in your abstracts and publications.

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from the National Institute Of Arthritis And Musculoskeletal And Skin Diseases.
Research in the Meyer lab is focused on understanding physiological and pathological signaling between muscle and fat. Small stores of adipocytes (IMAT) are present between and within muscles in healthy individuals, but expand dramatically in the context of disease where they can occupy as much as 90% of the “muscle” volume. Across disease states, from aging to Diabetes to muscular dystrophies to orthopaedic injuries, the dogma is that the more fat present the worse muscle functions, but little else is known about it – especially at the cellular level. Our driving hypothesis is that, in the context of disease, signals from IMAT inhibit efficient muscle function and that understanding these signals will turn fat into a novel therapeutic target in the treatment of muscle pathology.

Currently, we are very interested in the role fat phenotype (i.e. white, brown or “beige”) plays in muscle-fat cross-talk. The discovery of beige fat, a type of fat that can be pharmacologically “browned”, has opened new doors for fat-targeted therapies and we think these have potential to be directed to muscle (Fig 1). IMAT around the muscles of the human rotator cuff is beige and we have new evidence from an ongoing study in the human lower extremity that IMAT within muscle is also beige. Our data also suggests that both of these fats shift toward a white phenotype with disease. To explore the significance of this shift for muscle and the potential for browning to promote muscle functional recovery, we have developed a mouse model (thanks to a pilot and feasibility award from the MRC!) where fats of different phenotypes are transplanted into the injured rotator cuff.

Data from this mouse show that brown fat has anabolic effects on muscle, mitigating atrophy during degeneration and increasing mass following regeneration. Ongoing work on this project is focused on employing proteomics to identify the signaling mediators that regulate this anabolic effect and developing an implantable biomaterial (in collaboration with the Zustiak lab at SLU) for sustained local delivery of a pharmacological browning agent.

Outside of the mouse, we are developing co-culture disease models of rotator cuff injury and diabetes using human muscle and fat progenitors to define how the cells that send and receive the signals change with disease. Finally, for anyone interested in quantifying IMAT in an animal model or human biopsy, we have developed methodology that is more comprehensive and precise than standard histology (Fig 2).

Come see us or e-mail to learn more or just to chat about muscle: 1748 West Building, meyerg@wustl.edu.