In the November issue of our newsletter, we would like to highlight one of our Cores, and one of our Core users. We have also included the November/December schedule for the Musculoskeletal Seminar Series. As always, we welcome your comments, please feel free to call or send an email.

Core Highlight

Core B - Musculoskeletal Structure and Strength

The goal of Core B is to facilitate assessment of musculoskeletal structure and/or strength in animal models. Our main focus is bone imaging and mechanical testing, although we have worked with tendon, ligament and cartilage. To assess structure, we have several x-ray imaging options ideally suited for bone imaging and density measurement. Most of these can be used for either post-mortem specimens or live animal imaging. Investigators can use these systems themselves or you can hire us to do the work. Instruments we have include: Faxitron plane radiography (MX20); GE/Norland Piximus Dual-Energy X-Ray Absorptiometry (DXA, for bone density scanning); Norland/Stratec pQCT scanner; Scanco uCT40 microCT scanner specimen; Scanco ViaCT40 microCT scanner. To assess strength, we can test your bone or other tissue samples using one of three Instron mechanical testing systems. These are used to provide quantitative measures of stiffness, strength and other mechanical properties.

Examples of work done in Core B recently include: in vivo microCT scanning of mouse bones to measure changes in bone volume in response to temporary paralysis, in vivo DXA and microCT scanning of mutant mice to assess skeletal phenotype (increased or decreased bone mass), in vivo microCT scanning to assess club foot deformity in mice, post mortem microCT to assess bone changes after knee injury in mice, bending tests to assess mechanical properties of femurs from ~1000 mice to identify genes related to bone strength.

For more information on the Cores, please click on the links below:
Core A—Administrative Core
Core B—Structure and Strength Core
Core C—In Situ Molecular Analysis Core
Core D—Mouse Genetics Models Core

Save the Date
1st Annual
Winter Symposium
January 27, 2011
(abstracts due 12/29/10)
Mice created by our laboratory types. have fundamentally opposite pheno-
posing signals and would therefore standing hypothesis that growth directly supports our long-
posing effects on human skeletal control of skeletal growth, these op-
these two genes were critical to the our more general hypothesis that
Gpc3 overgrowth of bone and tall stature. Mice created by our laboratory bearing mutations in
Gpc6 of
causes an X-linked disorder in hu-
mans known as Simpson Golabi Behmel syndrome (SGBS). This human disor-
der is associated with several abnormalities including defects in skeletal pat-
tering, overgrowth of bone and tall stature. Mice created by our laboratory bearing mutations in
Gpc3 model this disease. Recently, loss-of-
mutations in Gpc6 in humans have been shown to cause autosomal recessive Omodysplasia, a disease associated with restricted growth of bone and short stature. In addition to supporting our more general hypothesis that these two genes were critical to the control of skeletal growth, these oppo-
posing effects on human skeletal growth directly supports our long-
standing hypothesis that Gpc3 and Gpc6 have evolved to balance oppo-
sing signals and would therefore have fundamentally opposite pheno-
types.

Loss-of-function mutation in GPC3 causes an X-linked disorder in hu-
mans known as Simpson Golabi Behmel syndrome (SGBS). This human disorder is associated with several abnormalities including defects in skeletal patterning, overgrowth of bone and tall stature. Mice created by our laboratory bearing mutations in Gpc3 model this disease. Recently, loss-of-function mutations in GPC6 in humans have been shown to cause autosomal recessive Omodysplasia, a disease associated with restricted growth of bone and short stature. In addition to supporting our more general hypothesis that these two genes were critical to the control of skeletal growth, these opposing effects on human skeletal growth directly supports our long-standing hypothesis that Gpc3 and Gpc6 have evolved to balance opposing signals and would therefore have fundamentally opposite phenotypes.

Remember to include reference to support from the Center in your abstracts and publications.

Each publication, press release or other document that cites results from NIH grant-supported research must include an acknowledgment of NIH grant support and disclaimer such as:

“The project described was supported by Award Number P30AR057235 from the National Institute Of Arthritis And Musculoskeletal And Skin Diseases. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute Of Arthritis And Musculoskeletal And Skin Diseases or the National Institutes of Health.”