# Seattle Institute for Biomedical and Clinical Research

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## Feature

LeBris ("Lee") Quinn, PhD

LeBris ("Lee") Quinn, Ph.D. is a Research Biologist at the Geriatric Research, Education and Clinical Center (GRECC) at the VA Puget Sound, and a Research Associate Professor in the Division of Gerontology and Geriatric Medicine, University of Washington.

Research conducted in Dr. Quinn's laboratory has focused on the underlying cellular and biochemical mechanisms of the age-associated loss of skeletal muscle, termed "sarcopenia". Sarcopenia is a major cause of frailty, falls, and loss of independence in the elderly, affecting approximately one-third of the aged population. Much of Dr. Quinn's research to date has concerned the cellular and molecular mechanisms of action of the insulin-like growth factor (IGF) system on skeletal muscle. IGF is the effector of growth hormone action, a master hormone which declines with age. The ultimate goal of this research is to delineate molecular or biochemical strategies to mimic the trophic action of IGF on muscle without the negative effects of IGF and growth hormone on other functions. Funding for this work has come from federal agencies such as the Department of Veteran's Affairs (DVA), the US Department of Agriculture (USDA), and the Department of Defense (DOD).

In addition to the loss of skeletal muscle, another major change during normal aging is the increased deposition of adipose tissue. Together with sarcopenia, the increase in fat:lean body composition predisposes the elderly to development of insulin resistance metabolic syndrome, and type-2 diabetes, major factors in death and disability in the aged population. These, in turn, are risk factors for cardiovascular disease, cancer, and Alzheimer's disease. Recently, Dr. Quinn's lab has identified a novel factor, interleukin-15 (IL-15), which appears to regulate fat:lean body composition and insulin sensitivity. Dr. Quinn's laboratory has developed several strains of transgenic mice whose muscles produce increased amounts of IL-15, and are currently engaged in characterizing the body composition, aging patterns, and susceptibility to developing insulin resistance of these mice and their normal littermates. Modulation of this novel pathway may represent a new strategy to prevent or treat changes in body composition leading to insulin resistance. This work has been funded by the DVA, USDA, and Pfizer. Additionally, a proposal to the NIH to continue this work has received a very favorable scientific review score and a funding award is anticipated.

## What's New

### NIH TRANSITIONS TO ELECTRONIC SUBMISSIONS THROUGH GRANTS.GOV.

Initial plans for submission dates and mechanisms are as follows:

**December 1, 2005** – Small Business Innovative Research (SBIR) and Small Business Technology Transfer Programs (STTR) (R41, R42, R43, R44)

**December 15, 2005** - Support for Conferences & Scientific Meetings (R13 & U13)

**February 25, 2006** – Academic Research Enhancement Awards (AREA) (R15)

**June 1, 2006** – Small Grant Programs (R03) & Exploratory/Development Research Grant Awards (R21)

**October 1, 2006** – Research Project Grant Program (R01)

NIH will continue to communicate transition plans for other programs/mechanisms as they evolve and will provide the community with ample notice of impending events.

## IMPORTANT ANNOUNCEMENTS

- NIH will offer a videocast training on electronic submissions on Wednesday **January 11, 2006** from 9:30am - 1pm at the UW Hogness auditorium. No registration is required.

Please contact Danielle Belisle at 6-3971 or Marie Walters at 6-6487 for more information on electronic submissions.