



July - September 2018



Seattle Institute for Biomedical and Clinical Research Spotlight Feature

JAMES (ERNIE) BLEVINS, PHD



James (Ernie) Blevins, Ph.D., is a Research Biologist at the VA Puget Sound Health Care System (VA Puget Sound) and a Research Associate Professor at the University of Washington. He received his Ph.D. from Creighton University in 1997 and completed a 3-year postdoctoral fellowship under the mentorship of Dorothy Gietzen, Ph.D., in the Department of Nutrition at University of California (UC) Davis before undertaking a subsequent postdoctoral fellowship under the mentorship of Denis Baskin, Ph.D., at VA Puget Sound.

He initially received pilot and feasibility funding through the Clinical Nutrition Research Unit at the University of Washington (2002-2004) prior to receiving his first VA Merit Award in 2004. He has found that the neurohypophyseal hormone, oxytocin, which is a downstream target of the adiposity signal, leptin, can be used as a therapeutic strategy to treat obesity in leptin resistant diet-induced obese (DIO) rats. He recently translated these findings to a nonhuman primate (rhesus monkey) model through a UC Davis California National Primate Research Center Pilot Project (2013-2014) and found that oxytocin elicits weight loss in a DIO rhesus monkeys, in part, by reducing food intake and increasing energy expenditure.

His recent findings suggest that oxytocin may elicit weight loss by activating brown adipose tissue (BAT) to increase energy expenditure. These findings helped provide preliminary data to obtain a 4-year NIH R01 award titled “Role of Brown Adipose Tissue Thermogenesis in Oxytocin-Elicited Weight Loss” as well as a 4-year VA Merit Award titled “Role of Brown Adipose Tissue Thermogenesis in Oxytocin-Elicited Weight Loss in Rodents and Nonhuman Primates.”

His current work focuses on whether oxytocin-elicited changes in sympathetic outflow to interscapular BAT is required for oxytocin-elicited changes in energy expenditure and weight loss and if reducing oxytocin signaling in a CNS site that controls BAT contributes to leptin resistance, obesity and the anti-obesity effects of chronic systemic oxytocin. He will further investigate the translational question of whether chronic intranasal oxytocin elicits weight loss, increases sympathetic outflow to BAT and improves glucose tolerance in DIO nonhuman primates. These findings will direct future studies to address the extent to which intranasal oxytocin may reverse obesity in humans by stimulating BAT thermogenesis.

IMPORTANT GRANT APPLICATION INFORMATION

MAJOR NIH APPLICATION CHANGES FOR HUMAN SUBJECTS RESEARCH

These new requirements continue to catch applicants by surprise. Contact your SIBCR Grant Manager to discuss new requirements for 2018 submissions. Read more here: <https://grants.nih.gov/grants/guide/notice-files/NOT-OD-17-119.html>

Be aware for October 2018, NIH Biosketch approved forms are changing again (10/31/18 versions can be used until current expiration date). <https://grants.nih.gov/grants/forms/biosketch.htm>