Symposium

Introduction: The co-morbidity of stress and disease: effects of chronic stress on metabolism, cardiovascular disease and behavior

Susan E Mulroney and Yvette Taché

Physiologic responses to acute stress contribute to maintenance of overall homeostasis, and are necessary for survival. The appropriate interplay between the sympathetic nervous system (and associated neuropeptides) and the hypothalamic-pituitary-adrenal axis keeps the system primed to keep responding to isolated stressors. In contrast, chronic stress has been shown to dysregulate the systems responding to stress, and is rapidly becoming thought of as a key contributor to the development and progression of metabolic, cardiovascular, gastrointestinal and psychological diseases. The objective of this symposium was to bring together work from both classic and more recent stress mechanisms, and highlight the ways chronic stress can exacerbate development of diseases.

The reviews herein expand on symposium talks given at the annual FASEB Experimental Biology meeting, held Sunday, April 21, 2009, in San Diego, California. The speakers were Ann Rasmusson, MD, VA Boston Healthcare System, National Center for PTSD, Women's Health Science Division, and Boston University School of Medicine, Boston, MA; Yvette Taché, PhD, CURE: Digestive Diseases Research Center and Center for Neurobiology of Stress, Digestive Diseases Division, David Geffen School of Medicine at University of California Los Angeles and Veterans Affairs Greater Los Angeles Healthcare System, Los Angeles, CA; Annika Thorsell, PhD, Laboratory of Clinical and Translational Studies, National Institute on Alcohol Abuse and Alcoholism, National Institutes of Health, Bethesda, MD; and Zofia Zukowska, MD PhD, Department of Physiology & Biophysics, Georgetown University Medical Center, Washington, DC.

These articles provide excellent insight on the basis of stress physiology, and focus primarily on the contributions of corticotrophin-releasing hormone and neuropeptide Y during chronic stress. They develop the concept that inappropriate stimulation of these factors impacts end-organ function, and give compelling evidence that stress-related changes in brain hormones and peptides contribute to physiological and psychological dysfunction, including post-traumatic stress disorder. In light of the recent focus on the increased exposure to chronic stress in daily life, the evidence that chronic stress is a co-morbid factor in diseases is all the more relevant and timely, especially when considering potential therapeutic interventions based on recent knowledge of underlying mechanisms. The Society of Experimental Biology and Medicine extends their appreciation to the speakers for these insightful articles.

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