Richard A. Cohen, MD

A professor of Medicine, Physiology, and Pharmacology and Experimental Thetherapeutics, Dr. Cohen is the Jay and Louise Coffman Professor of Medicine and Director of the Vascular Biology Unit. Dr. Cohen is also the Co-principal Investigator of the Boston University Cardiovascular Proteomics Center. Dr. Cohen is also a recipient of a MERIT award from the NHLBI. He is a former recipient of the American Heart Association Clinician Investigator and the Established Investigator awards. He is past president of the American Federation for Medical Research and founding president of the American Federation for Medical Research Foundation. Dr. Cohen is distinguished by his elections to the American Society for Clinical Investigation and the Association of American Physicians. He is an elected Fellow of the Cardiovascular Section of the American Physiological Society. He is a member of the editorial boards of the *American Journal of Physiology: Heart and Circulatory Physiology* and *Arteriosclerosis, Thrombosis, and Vascular Biology,* and *Free Radical Biology and Medicine*. Dr. Cohen is a past member of the NIH Experimental Cardiovascular Study Section and currently serves as an *ad hoc* reviewer for several NIH review groups. The research in the Vascular Biology Unit which Dr. Cohen directs focuses on the effect of vascular and metabolic disease on the function of blood vessels with special emphasis on the role of endothelium-derived nitric oxide and reactive oxygen species. The roles of redox mediated post-translational protein modifications of cardiovascular proteins including the sarcoplasmic reticulum calcium ATPase, sirtuin-1, and p21ras in cell signaling and disease are a current focus.

The Vascular Biology Unit of the Whittaker Cardiovascular Institute, led by Dr. Cohen, seeks to understand how nitric oxide and oxidants regulate the function of blood vessels. His group has found that in diabetes, hypertension, and atherosclerosis, the vasodilator, nitric oxide is inactivated by superoxide, an oxidant produced in diseased blood vessels. As a result of this reaction a potent oxidant, peroxynitrite, is formed. Sustained high levels of peroxynitrite cause irreversible chemical modifications, inducing nitrotyrosine or sulfonic acid cysteine modifications, and thus inactivate important proteins such as manganese superoxide dismutase or the sarcoplasmic reticulum calcium ATPase. At low levels, reactive oxygen and nitrogen species including peroxynitrite and hydrogen peroxide can induce reversible protein modifications such as *S*-glutathione cysteine adducts that redox regulate the function of many proteins including the sarcoplasmic reticulum calcium ATPase, p21ras, and sirtuin-1, thereby physiologically modulating cell signaling. As part of the BU Cardiovascular Proteomics Center, Dr. Cohen and his group are identifying chemical modifications of proteins formed by oxidants with mass spectrometric and protein tagging strategies. These modifications may serve as biomarkers for abnormal cell signaling and/or metabolic disease. Such markers have been found in diseased human arteries, platelets, and the blood.

OPTM (oxidative post-translational protein modifications) represent biomarkers for disease. Dr. Cohen has collaborated with the Center for Biological Mass Spectrometry to establish methods for proteomic detection of OPTM in cardiovascular tissues. He is Co-PI of the Cardiovascular Proteomics Center.

Perhaps just add a sentence at the end of my paragraph stating that “