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3-Nitrotyrosine Used as Biomarker for The Vasculopathy.

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Introduction

The endothelial dysfunction of Fabry disease results from α-galactosidase A deficiency resulting in the buildup of globotriaosylceramide. Vasculopathy within the α-galactosidase A null mouse is manifested as oxidant-induced thrombosis, accelerated atherogenesis, and impaired arterial reactivity. to raised understand the pathogenesis of Fabry disease in humans, we generated a person's cell model by using RNA interference. Hybrid endothelial cells were transiently transfected with small interfering RNA (siRNA) specifically directed against α-galactosidase A. Endothelial gas synthase (eNOS) activity was correspondingly decreased by >60%. Levels of 3-nitrotyrosine (3NT), a selected marker for reactive nitrogen species and quantified using mass spectrometry, increased by 40- to 120-fold without corresponding changes in other oxidized amino acids, according to eNOS-derived reactive nitrogen species because the source of the reactive oxygen species. eNOS uncoupling was confirmed by the observed increase in free plasma and protein-bound aortic 3NT levels within the α-galactosidase A knockout mice. Thus, 3NT may function a biomarker for the vascular involvement in Fabry disease. Although this mouse doesn't exhibit a spontaneous vascular phenotype, several inducible models of vascular disease are reported.

These include oxidant-induced thrombosis,2 accelerated atherogenesis,3 and impaired vasorelaxation.4 a standard mechanism that would potentially link these experimentally observed abnormalities is endothelial gas synthase (eNOS) dysfunction.5 eNOS dysfunction may end in either decreased gas (NO) bioavailability or enzyme uncoupling, which generates a potent oxidant, peroxynitrite, a reactive nitrogen species.6,7 the connection between GLA and eNOS was explored by determining whether these changes might be recapitulated during a human endothelial cell line. We report that when the Gb3 content of EA.hy926 cells is increased with GLA knockdown, there's an associated uncoupling of eNOS with the formation of 3-nitrotyrosine (3NT), a selected marker for reactive nitrogen species. The eNOS dysfunction was specifically related to the loss of GLA activity therein comparable changes weren't observed with β-glucocerebrosidase (GBA) knockdown. High circulating levels of 3NT were measured within the plasma and aortic extracts of Gla knockout mice. Finally, the concentrations of protein-bound oxidized amino acids were measured in plasma samples from classic FD patients and compared with age- and gender-matched controls. A quite fivefold elevation in 3NT was observed within the FD samples compared with controls, raising the likelihood that 3NT represents an useful biomarker for vasculopathy in FD.

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