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Copper-Aβ induced erythrocyte oxidative stress and inhibition by polyphenolic antioxidants

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Oxidative stress is a primary trigger for neuronal dysfunction and is considered a causative factor contributing to Alzheimer's disease (AD) and the associated loss of cognitive function. Reactive oxygen species (ROS) generated by copper-bound amyloid-β peptide (CuAβ) are also thought to contribute to AD pathogenesis since insoluble proteinaceous deposits enriched with amyloid plaques and copper are found in AD brains. Previously, we reported that erythrocyte exposure to a partially aggregated form of CuAβ leads to a pronounced increase in red blood cell oxidative stress. These findings suggest that erythrocyte oxidative damage, which would result in improper tissue perfusion, may be involved in the etiology of AD. Antioxidants such as resveratrol and quercetin have been shown to incorporate into red cell membranes and increase the oxidant scavenging ability of the cell. Thus, the ability of these antioxidants to inhibit red cell oxidative stress promoted by CuAβ/ROS was investigated. The presented work will show that oxidative reactions promoted by CuAβ occur in close proximity to the red cell membrane and that membrane incorporated antioxidants can protect erythrocytes from the accompanying oxidative stress. Mitigation of ROS produced by CuAβ through natural antioxidants from our diets may subsequently reduce vascular inflammation, cognitive impairment, and/or neuro degeneration associated with AD.

Biography

Heather R. Lucas completed her Ph.D. at Johns Hopkins University in 2008 and conducted her postdoctoral studies at the National Institutes of Health. She will begin as an Assistant Professor at Virginia Commonwealth University during the fall of 2013.

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