

Preface

Mini-Forum “Mitochondria in Alzheimer Disease”

It is now well established that metabolic alterations and oxidative stress are key, early contributors in the pathogenesis of Alzheimer disease (AD). Since damaged mitochondria are less efficient producers of ATP and more efficient producers of reactive oxygen species, it is no wonder that substantial evidence supports a fundamental involvement of mitochondrial dysfunction in the disease process. In this issue of the *Journal of Alzheimer's Disease*, we are fortunate to have assembled key thought leaders to share their ideas and data for a Mini-Forum on “Mitochondria in Alzheimer disease”. First, in light of the recent demonstration that amyloid- β progressively accumulates within the mitochondrial matrix and provides a direct link to mitochondrial toxicity, Drs. Chen and Yan discuss how amyloid- β exerts its deleterious effect on mitochondrial function

and its potential therapeutic implications. Second, Drs. Picklo and Montine examine the effect of lipid peroxidation products on mitochondria as they relate to the development of AD. Third, Drs. Moreira, Perry, and colleagues present data suggesting the potential efficacy of antioxidant therapies based on lipoic acid and N-acetyl cysteine supplementation against mitochondrial oxidative stress and AD.

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