# Vitamin E but not 17?-estradiol protects against vascular toxicity induced by ?-amyloid wild type and the dutch amyloid variant

Muñoz, Francisco J.

Opazo, Carlos

Gil-Gómez, Gabriel

Tapia, Gladys

Fernández, Virginia

Valverde, Miguel A.

Inestrosa, Nibaldo C.

Amyloid ?-peptide (A?) fibril deposition on cerebral vessels produces cerebral amyloid angiopathy that appears in the majority of Alzheimer's disease patients. An early onset of a cerebral amyloid angiopathy variant called hereditary cerebral hemorrhage with amyloidosis of the Dutch type is caused by a point mutation in A? yielding A?Glu22?Gln. The present study addresses the effect of amyloid fibrils from both wild-type and mutated A? on vascular cells, as well as the putative protective role of antioxidants on amyloid angiopathy. For this purpose, we studied the cytotoxicity induced by A?1-40 Glu22?Gln and A?1-40 wild-type fibrils on human venule endothelial cells and rat aorta smooth muscle cells. We observed that A?Glu22?Gln fibrils are more toxic for vascular cells than the wild-type fibrils. We also evaluated the cytotoxicity of A? fibrils bound with acetyl-cholinesterase (AChE), a common component of amyloid deposits. A?1-40 wild-type-AChE fibrillar complexes, similar to neurona