Research Article Open Access

Keywords: Alzheimer's Diseases; APP/PS1; mnSOD; HIF; P-53; Cu-ZnSOD

Abbreviations: AD: Alzheimer's disease; Mn-SOD: Manganeso-Super-Oxido-Dismutase; Cu/Zn-SOD: Cu/Zn-Super-Oxido-Dismutase; HIF: Hypoxia Inducible Factor; P-53: P-53 protein; PPAR-γ: Peroxisoma Proliferator-Activated Receptor

Introduction

Alzheimer's disease is a common multifactorial neurodegenerative disorder that occurs with aging. e neuropathological hallmarks of Alzheimer's disease (AD) are amyloid plaques, intra-neuronal tangles, and activation of glial cells [1-3]. Glial swelling and astrogliosis are a characteristic response of astrocytes to in ammation, oxidative stress and trauma, leading to secretion of several potentially toxic products, including in ammatory [4] and oxidative stress mediators [5].

Beta amyloid (Aβ) deposition can result in brain damage and neurodegeneration, but whether astrocytes activation participates in the Aβ-induced brain damage, and furthermore, its intervention in in ammation and in oxidative stress in AD is poorly known. Also in vivo studies have been demonstrated the involvement of oxidative stress and in ammation in the induction of Alzheimer phenotypes. We demonstrate here oxidative stress and in ammation changes in APP/PS1 transgenic mice [transgenic mice, APP (amyloid precursor protein) and PS1 (Preseniline 1) protein] compared with wild type mice. We determine by western-blot MnSOD and Cu/ZnSOD, such as pro-oxidant proteins, PPAR-γ such as anti-in ammatory protein, p-53 and Sir-2 proteins, in transgenic compared with wild type mice. We previously found that oestradiol or genistein attenuate in ammation and oxidative processes, preventing expression of in ammatory mediators and production of peroxide levels, demonstrating antioxidant and anti-in ammatory e ects of oestrogens in neurons and astrocytes in primary culture [6]. Furthermore, these compounds promote PPAR-γ activation as a regulator of in ammatory responses and consequently protecting cells from Aβ deleterious e ects [7]. Our results here, demonstrate a decreased expression of Mn-SOD, HIF and p-53 with increase in Sir-2 and without changes in Cu/Zn-SOD and PPAR-γ expression in transgenic compared with wild type mice.

Material and Methods

Materials

Dulbecco's modi ed Eagle's medium (DMEM) and fetal bovine serum (FBS) were obtained from Gibco (Gibco Invitrogen Corparation, Barcelona, Spain). Western Blot Chemiluminescent Detection System (ECL) was from Amersham (Amersham Biosciences, Barcelona, Spain). Antibodies: monoclonal anti-peroxisome proliferator-activated receptor antibody (anti-PPARγ) (1:250) and monoclonal anti-α-tubulin antibody (1:1000) (Santa Cruz Biotechnology, Madrid, Spain), polyclonal anti-Mn-SOD protein antibody (anti-Mn-SOD) (1:500), polyclonal anti-Cu/Zn-SOD antibody (anti-Cu/Zn-SOD) (1:500), polyclonal anti-HIF protein antibody (anti-HIF) (1:250), monoclonal anti-p-53 antibody (anti-p-53) (Sigma Aldrich, Madrid, Spain). All other reagents are analytical or culture grade purity.

Western blot analysis

Protein extracts from brain cells were mixed with equal volumes of SDS bu er (0.125 M Tris-HCl, pH 6.8, 2% SDS, 0.5% (v/v)

***Corresponding author:** Soraya L Valles, Ph.D., Department of Physiology,

Citation: Aguirre-Rueda D, Aldasoro M, Santonja S, Guerra S, Iradi A, et al. (2013) Pro-Oxidant and Inþammatory Mediators Produced In Transgenic Mice (App/Ps1) . Biochem Physiol 2: 121. doi:10.4172/2168-9652.1000121

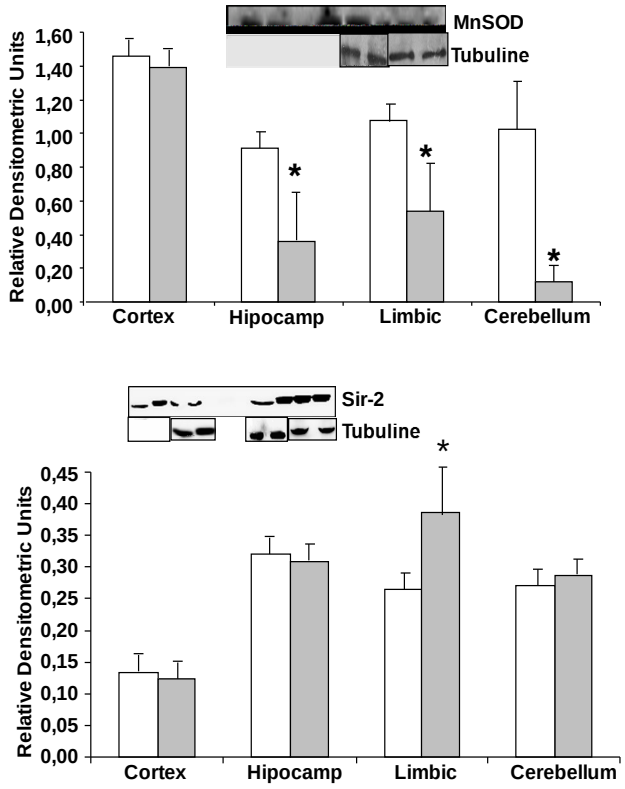
MnSOD
Tubuline
Relative Densitometric Units
1,60
1,40
1,20
1,00
0,80
0,60
0,40
0,20
0,00
Cortex
Hipocamp
Limbic
Cerebellum
Sir-2
Tubuline
Relative Densitometric Units
0,45
0,40
0,35
0,30
0,25
0,20
0,15
0,10
0,05
0,00
Cortex
Hipocamp
Limbic
Cerebellum

Figure 2: