NEUROPROTECTIVE STUDIES ON THE MPTP AND SOD1 MOUSE MODELS OF NEURODEGENERATIVE DISEASES

NEUROPROTECTIVE STUDIES ON THE MPTP AND SOD1 MOUSE MODELS OF NEURODEGENERATIVE DISEASES

Fontanilla, Christine V.

Permanent Link: http://hdl.handle.net/1805/2724
Date: 2012-02-29
Committee Chair: Farlow, Martin R.
Committee Members: Du, Yansheng; Jin, Xiaoming; Xu, Zao C.
Degree: Ph.D.
Degree Year: 2012
Department: Department of Medical Neuroscience
Grantor: Indiana University
Keywords: ALS ; MPTP ; SOD1 ; neurodegeneration
LC Subjects: Nervous system -- Degeneration -- Treatment -- Research ; Nervous system -- Diseases -- Treatment -- Research ; Amyotrophic lateral sclerosis ; Methylphenyltetrahydropyridine ; Superoxide dismutase

Abstract:

The main, underlying cause of neurodegenerative disease is the progressive loss of neuronal structure or function, whereby central and/or peripheral nervous system circuitry is severely and irreversibly damaged, resulting in the manifestation of clinical symptoms and signs. Neurodegenerative research has revealed many similarities among these diseases: although their clinical presentation and outcomes may differ, many parallels in their pathological mechanisms can be found. Unraveling these relationships and similarities could provide the potential for the discovery of therapeutic advances such that a treatment for one neurologic disease may also be effective for several other neurodegenerative disorders. There is growing awareness that due to the complexity of pathophysiological processes in human disease, specifically targeting or

inactivating a single degenerative process or a discrete cellular molecular pathway may be ineffective in the treatment of these multifaceted disorders. Rather, potential therapeutics with a multi-target approach may be required to successfully and effectively control disease progression. Recent advances in neurodegenerative research involve the creation of animal disease models that closely mimic their human counterparts. The use of both toxin- exposure and genetic animal models in combination may give insight into the underlying pathologic mechanisms of neurodegenerative disorders (target identification) leading to the development and screening of prospective treatments and determination of their neuroprotective mechanism (target validation). Taken together, ideal candidates for the treatment of neurodegenerative disease would need to exert their neuroprotective effect on multiple pathological pathways. Previous studies from this laboratory and collaborators have shown that the naturally-occurring compound, caffeic acid phenethyl ester (CAPE), is efficacious for the treatment against neurodegeneration. Because of its versatile abilities, CAPE was chosen for this study as this compound may be able to target the pathogenic pathways shared by two different animal models of neurodegeneration and may exhibit neuroprotection. In addition, adipose-derived stem cell conditioned media (ASC-CM), a biologically-derived reagent containing a multitude of neuroprotective and neurotrophic factors, was selected as ASC-CM has been previously shown to be neuroprotective by using both animal and cell culture models of neurodegeneration.

Description:

Indiana University-Purdue University Indianapolis (IUPUI)

This item appears in the following Collection(s)

- Medical Neuroscience Department Theses and Dissertations (/handle/1805/1721)

 Show Statistical Information (#)

My Account

- Login
- Register

Statistics

- Most Popular Items
- Statistics by Country
- Most Popular Authors