1. Record Nr. UNISA996393439303316

Autore	Mather W (William), <fl. 1695.>
Titolo	Of the Quakers despising the Holy Scriptures [[electronic resource]] : as appears in their preachers printed books as follows
Pubbl/distr/stampa	London, : Printed for B. Aylmer ... and C. Brome ..., 1700
Descrizione fisica	1 sheet ([1] p.)
Soggetti	Society of Friends Broadsides17th centuryEnglandLondon
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia
Note generali	Signed at end: W. Mather. Reproduction of original in Huntington Library.
Sommario/riassunto	eebo-0113

2. Record Nr. UNINA9910220056703321

Autore	Manoj Kumar Jaiswal
Titolo	The Role of Mitochondria, Oxidative Stress and Altered Calcium Homeostasis in Amyotrophic Lateral Sclerosis: From Current Developments in the Laboratory to Clinical Treatments
Pubbl/distr/stampa	Frontiers Media SA, 2017
Descrizione fisica	1 online resource (336 p.)
Collana	Frontiers Research Topics
Soggetti	Neurosciences
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia

Sommario/riassunto

Amyotrophic lateral sclerosis (ALS) is a rapidly progressive, devastating and fatal disease characterized by selective loss of upper and lower motor neurons of the cerebral cortex, brainstem, spinal cord and muscle atrophy. In spite of many years of research, the pathogenesis of ALS is still not well understood. ALS is a multifaceted genetic disease, in which genetic susceptibility to motor neuron death interacts with environmental factors and there is still no cure for this deleterious disease. At present, there is only one FDA approved drug, Riluzole which according to past studies only modestly slows the progression of the disease, and improves survival by up to three months. The suffering of the ALS patients, and their families is enormous and the economic burden is colossal. There is therefore a pressing need for new therapies. Different molecular pathways and pathological mechanisms have been implicated in ALS. According to past studies, altered calcium homeostasis, abnormal mitochondrial function, protein misfolding, axonal transport defects, excessive production of extracellular superoxide radicals, glutamate-mediated excitotoxicity, inflammatory events, and activation of oxidative stress pathways within the mitochondria and endoplasmic reticulum can act as major contributor that eventually leads to loss of connection between muscle and nerve ultimately resulting to ALS. However, the detailed molecular and cellular pathophysiological mechanisms and origin and temporal

progression of the disease still remained elusive. Ongoing research and future advances will likely advance our improve understanding about various involved pathological mechanism ultimately leading to discoveries of new therapeutic cures. Importantly, clinical biomarkers of disease onset and progression are thus also urgently needed to support the development of the new therapeutic agents and novel preventive and curative strategies. Effective translation from pre-clinical to clinical studies will further require extensive knowledge regarding drug activity, bioavailability and efficacy in both the pre-clinical and clinical setting, and proof of biological activity in the target tissue. During the last decades, the development of new therapeutic molecules, advance neuroimaging tools, patient derived induced stem cells and new precision medicine approaches to study ALS has significantly improved our understanding of disease. In particular, new genetic tools, neuroimaging methods, cellular probes, biomarker study and molecular techniques that achieve high spatiotemporal resolution have revealed new details about the disease onset and its progression. In our effort to provide the interested reader, clinician and researchers a comprehensive summaries and new findings in this field of ALS research, hereby we have created this electronic book which comprises of twenty seven chapters having various reviews, perspective and original research articles. All these chapters and articles in this book not only summarize the cutting-edge techniques, approaches, cell and animal models to study ALS but also provide unprecedented coverage of the current developments and new hypothesis emerging in ALS research. Some examples are novel genetic and cell culture based models, mitochondria-mediated therapy, oxidative stress and ROS mechanism, development of stem cells and mechanism-based therapies as well as novel biomarkers for designing and testing effective therapeutic strategies that can benefit ALS patients who are at the earlier stages in the disease. I am extremely grateful to all the contributors to this book and want to thank them for their phenomenal efforts. Amyotrophic lateral sclerosis (ALS) is a rapidly progressive, devastating and fatal disease characterized by selective loss of upper and lower motor neurons of the cerebral cortex, brainstem, spinal cord and muscle atrophy. In spite of many years of research, the pathogenesis of ALS is still not well understood. ALS is a multifaceted genetic disease, in which genetic susceptibility to motor neuron death interacts with environmental factors and there is still no cure for this deleterious disease. At present, there is only one FDA approved drug, Riluzole which according to past studies only modestly slows the progression of the disease, and improves survival by up to three months. The suffering of the ALS patients, and their families is enormous and the economic burden is colossal. There is therefore a pressing need for new therapies. Different molecular pathways and pathological mechanisms have been implicated in ALS. According to past studies, altered calcium homeostasis, abnormal mitochondrial function, protein misfolding, axonal transport defects, excessive production of extracellular superoxide radicals, glutamate-mediated excitotoxicity, inflammatory events, and activation of oxidative stress pathways within the mitochondria and endoplasmic reticulum can act as major contributor that eventually leads to loss of connection between muscle and nerve ultimately resulting to ALS. However, the detailed molecular and cellular pathophysiological mechanisms and origin and temporal progression of the disease still remained elusive. Ongoing research and future advances will likely advance our improve understanding about various involved pathological mechanism ultimately leading to discoveries of new therapeutic cures. Importantly,

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