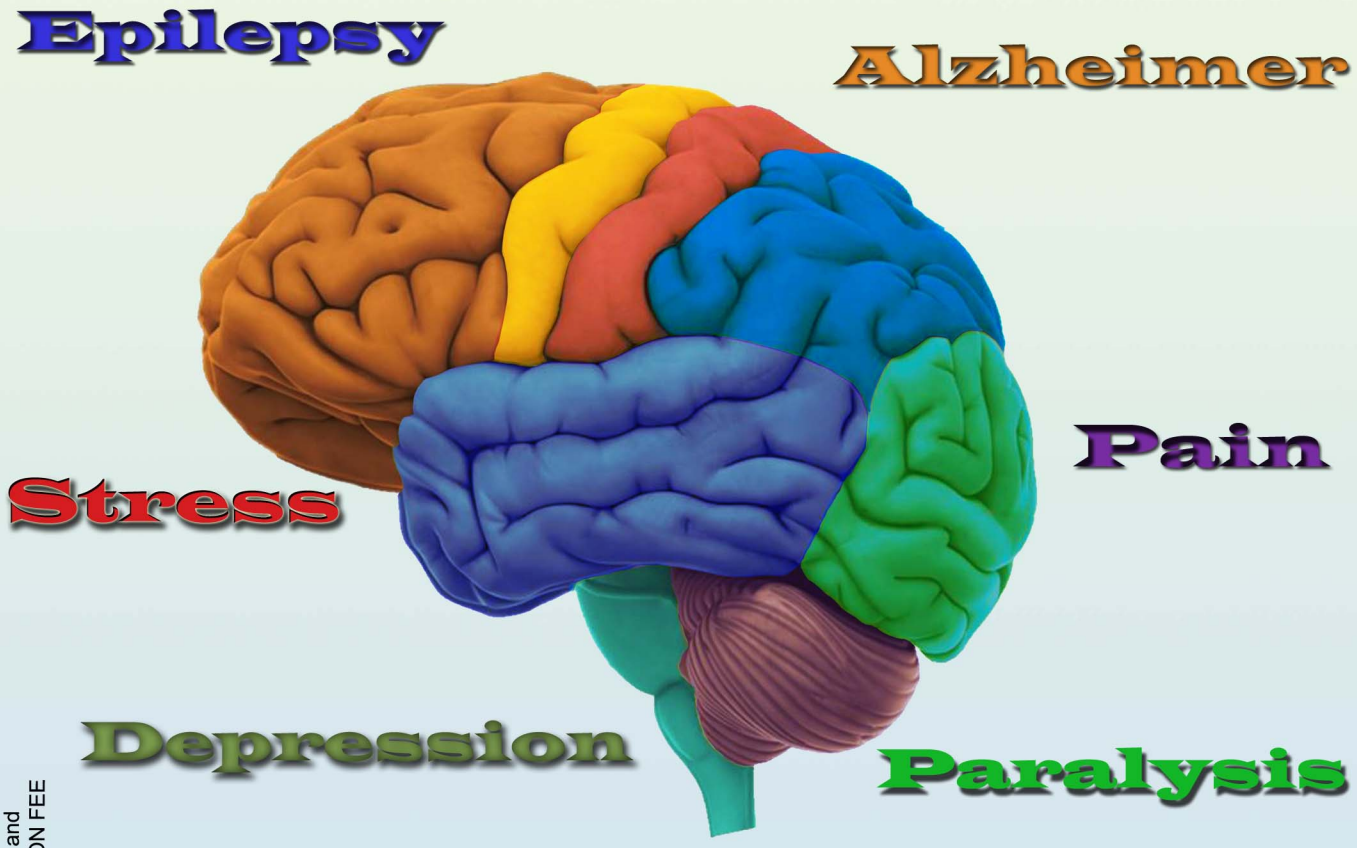
E-ISSN: 2149-7222 (Online)

Journal Cellular Neuroscience and Oxidative Stress

http://dergipark.gov.tr/jcnos

Former name; Cell Membranes and Free Radical Research

HSOD
Cellular Neuroscience and Oxidative Stress Society



OPEN ACCESS and NO PUBLICATION FEE

Brain Research School

Editor in Chief
Prof.Dr. Mustafa NAZIROĞLU

Volume 10, Number 3, 2018

Journal of Cellular Neuroscience and Oxidative Stress

http://dergipark.gov.tr/jcnos

An Official Journal of the Cellular Neuroscience and Oxidative Stress Society

http://hsord.org.tr/en/

Formerly known as:

Cell Membranes and Free Radical Research (2008 - 2014)

Volume 10, Number 3, 2018

3rd International Brain Research School

25 June – 1 July 2018 Isparta /TURKEY

2018.brs.org.tr

Volume 10, Number 3, 2018
E-ISSN Number: 2149-7222 (Online)
Indexing: Google Scholar, Index Copernicus, Chemical Abstracts, Scopus (Elsevier),
EBSCOhost Research Database, Citation Index Database,

AIM AND SCOPES
Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate cellular function, and the control or alteration of these processes by the action of receptors, neurotransmitters, second messengers, cation, anions, drugs or disease.

Areas of particular interest are four topics. They are;

A- Ion Channels (Na^+- K^+ Channels, Cl^- channels, Ca^{2+} channels, ADP-Ribose and metabolism of NAD^+, Patch-Clamp applications)

B- Oxidative Stress (Antioxidant vitamins, antioxidant enzymes, metabolism of nitric oxide, oxidative stress, biophysics, biochemistry and physiology of free oxygen radicals)

C- Interaction Between Oxidative Stress and Ion Channels in Neuroscience
(Effects of the oxidative stress on the activation of the voltage sensitive cation channels, effect of ADP-Ribose and NAD^+ on activation of the cation channels which are sensitive to voltage, effect of the oxidative stress on activation of the TRP channels in neurodegenerative diseases such Parkinson's and Alzheimer's diseases)

D- Gene and Oxidative Stress
(Gene abnormalities. Interaction between gene and free radicals. Gene anomalies and iron. Role of radiation and cancer on gene polymorphism)

READERSHIP

Biophysics	Biochemistry
Biology	Biomedical Engineering
Pharmacology	PhysiologyGenetics
Cardiology	Neurology
Oncology	Psychiatry
Neuroscience	Neuropharmacology

Keywords
Ion channels, cell biochemistry, biophysics, calcium signaling, cellular function, cellular physiology, metabolism, apoptosis, lipid peroxidation, nitric oxide, ageing, antioxidants, neuropathy, traumatic brain injury, pain, spinal cord injury, Alzheimer's Disease, Parkinson's Disease.

3rd International Brain Research School

The congress organization committee wishes thanks to the sponsors below

3rd International Brain Research School

Abstract Book

of

3rd International Brain Research School

25 June – 1 July 2018

Isparta, Turkey

with collaboration of

Cellular Neuroscience and Oxidative Stress Society & Neuroscience Research Center, Süleyman Demirel University

3rd International Brain Research School

_____ [Organization Committee] _____

Organization Chairman
Prof. Dr. Mustafa NAZIROĞLU
Department of Biophysics, School of Medicine
Suleyman Demirel University, Isparta, Turkey

Organization Vice Chairman
Assoc. Prof. Dr. Ömer ÇELİK
Department of Biophysics, School of Medicine
Suleyman Demirel University, Isparta, Turkey

Organization Secretariat
Ahmi ÖZ & Bilal ÇİĞ & Ramazan ÇINAR
Department of Biophysics, School of Medicine
Suleyman Demirel University, Isparta, Turkey

Accountant
Kenan YILDIZHAN &
Yener YAZĞAN *(Graphic Designer & Webmaster)*
Department of Biophysics, School of Medicine
Suleyman Demirel University, Isparta, Turkey

3rd International Brain Research School

[Scientific Committee]

3rd International Brain Research School

_____ [Scientific Committee] _____

3rd International Brain Research School

[CONTENTS]

Speakers

3rd International Brain Research School

Oral Presentations

3rd International Brain Research School

Poster Presentations

Speak No. 4

Intravenous NAD^+ effectively increased the NAD metabolome, reduced oxidative stress and inflammation, and increased expression of longevity genes safely in elderly humans

Nady BRAIDY[1], James CLEMENT[2], John STURGES[3], Yue LIU[1], Anne POLJAK[1,4,5], Perminder SACHDEV[1,6]

[1]Centre for Healthy Brain Ageing, School of Psychiatry, University of New South Wales, Sydney, Australia
[2]BetterHumans Inc, USA
[3]John A Sturges MD, 2170 W Ironwood Center Dr # A, Coeur d'Alene, ID 83814, USA
[4]Mark Wainwright Analytical Centre, University of New South Wales, Sydney, Australia
[5]School of Medical Sciences, University of New South Wales, Sydney, Australia
[6]Neuropsychiatric Institute, Euroa Centre, Prince of Wales Hospital, Sydney, Australia

Nicotinamide adenine dinucleotide (NAD^+) serves important roles in hydrogen transfer and as the cosubstrate for poly(ADP-ribose) polymerase (PARPs), the sirtuin (SIRT1-7) family of enzymes, and CD38 glycohydrolases. Recently, intravenous (IV) NAD^+ therapy has been used as a holistic approach to treat withdrawal from addiction, overcome anxiety and depression, and improve overall quality of life with minimal symptoms between 3-7 days of treatment.

We evaluated repeat dose IV NAD^+ (1000 mg) for 6 days in a population of 8 healthy adults between the ages of 70 and 80 years.

Our data is the first to show that IV NAD^+ increases the blood NAD^+ metabolome in elderly humans. We found increased concentrations of glutathione peroxidase -3 and paraoxonase-1, and decreased concentrations of 8-iso-prostaglandin F2α, advanced oxidative protein products, protein carbonyl, C-reactive protein and interleukin 6. We report significant increases in mRNA expression and activity of SIRT1, and Forkhead box O1, and reduced acetylated p53 in peripheral blood mononuclear cells isolated from these subjects. No major adverse effects were reported in this study.

The study shows that repeat IV dose of NAD^+ is a safe and efficient way to slow down age-related decline in NAD^+.

Keywords; Nicotinamide adenine dinucleotide; Oxidative stress: Inflammation: Longevity genes; Elderly humans.

References

Braidy N, Berg J, Clement J, Khorshidi F, Poljak A, Jayasena T, Grant R, Sachdev P. 2018. NAD^+ Precursors as Therapeutic Agents for Age-related Degenerative Diseases. Antioxidant and Redox Signaling. http://doi.org/10.1089/ars.2017.7269

Braidy N, Grant R, Sachdev P. 2017. Nicotinamide adenine dinucleotide and its related precursors for the treatment of Alzheimer's disease. Current Opinion in Psychiatry. http://doi.org/10.1097/YCO.0000000000000394.