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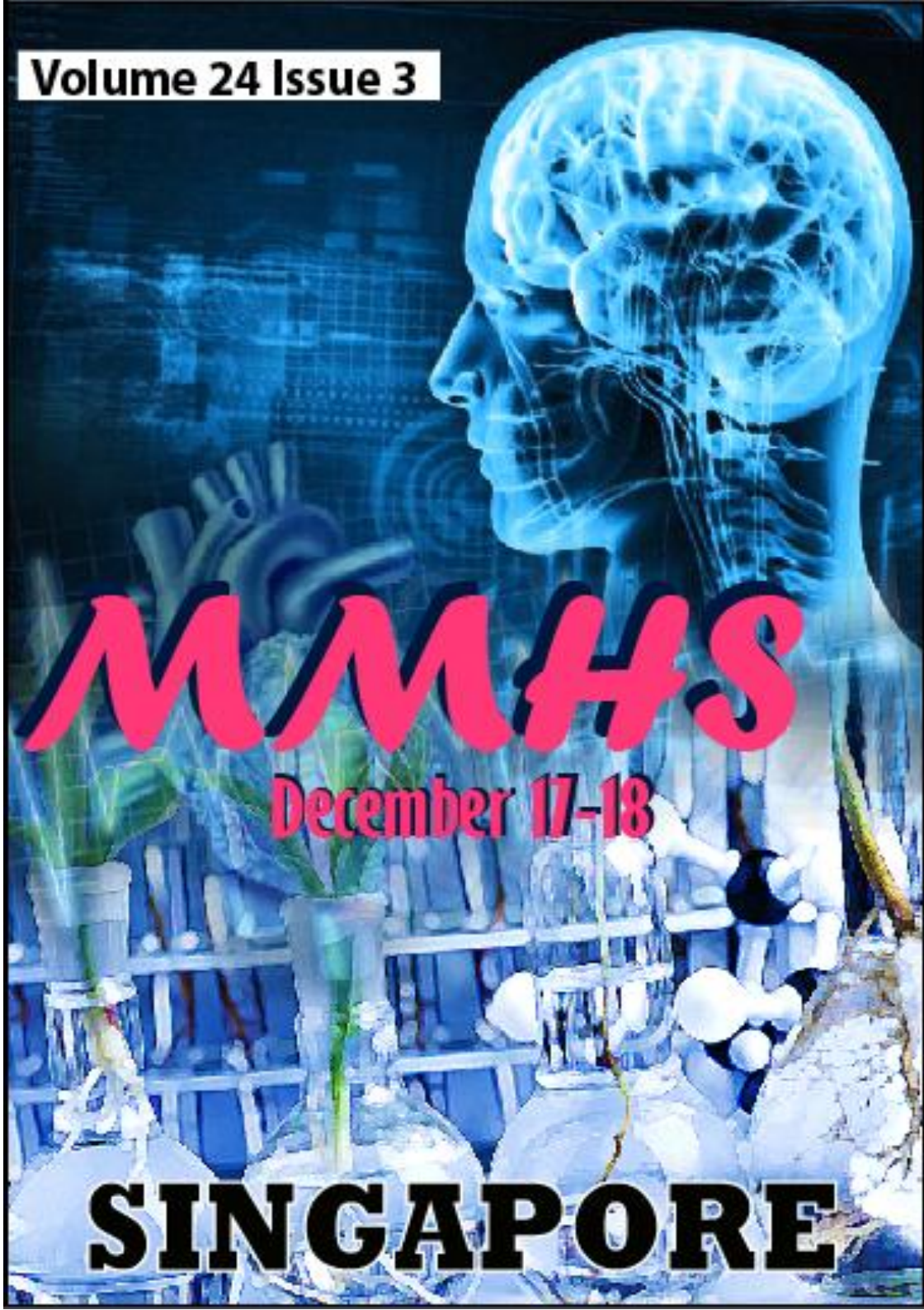

MMHS

December 17-18

SINGAPORE

Academic Fora

CONFERENCE PROCEEDINGS

BOOK OF ABSTRACT MMHS-2015

International Conference on
"Medical, Medicine and Health Sciences"
(MMHS-2015), Singapore

Book of Abstracts Proceedings

International Conference on
"Medical, Medicine and Health Sciences"
(MMHS-2015)
Singapore

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Proceedings of the International Conference on

"Medical, Medicine and Health Sciences (MMHS-2015)"

ISBN: ***978-969-670-070-8***

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International Conference on
"Medical, Medicine and Health Sciences
Singapore"

Venue: Hotel Grand Pacific Singapore

ORGANIZING COMMITTEE

1**. Ms. Petrel Qiu**

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PROGRAM COORDINATOR MESSAGE

Ms. Ani Wahyu

International Conference on Medical, Medicine and Health Sciences" serves as platform that aims to help the scholarly community across nations to explore the critical role of multidisciplinary innovations for sustainability and growth of human societies. This conference provides opportunity to the academicians, practitioners, scientists, and scholars from across various disciplines to discuss avenues for interdisciplinary innovations and identify effective ways to address the challenges faced by our societies globally. The research ideas and studies that we received for this conference are very promising, unique, and impactful. I believe these studies have the potential to address key challenges in various sub-domains of social sciences and applied sciences.

I am really thankful to our honourable scientific and review committee for spending much of their time in reviewing the papers for this event. I am also thankful to all the participants for being here with us to create an environment of knowledge sharing and learning. We the scholars of this world belong to the elite educated class of this society and we owe a lot to return back to this society. Let's break all the discriminating barriers and get free from all minor affiliations. Let's contribute even a little or single step for betterment of society and welfare of humanity to bring prosperity, peace and harmony in this world. Stay blessed.

Thank you.

Ms. Ani Wahyu

Program Coordinator
Email: aniwahyu@academicfora.com
MMHS-2015

CONFERENCE PROGRAM

DAY 01 Thursday (December 17, 2015)

Welcome Reception & Registration

08:00– 9:00 am

<u>Opening Ceremony (09:30 – 10:00 am)</u>
Venue: Room 1

09:00 – 9:20 am	Introduction of Participants
09:20 – 9:30 am	Welcome Remarks **– Felicia Chong –** Conference Chair Academic Fora
09:30 – 09.45 am	Group Photo Session

Grand Networking Session and Tea Break (09:45– 10:00 am)

DAY 01 Thursday (December 17, 2015)
Session 1 (10:00 am – 12:00 pm)
Venue: Room 1

Session Chair: Alkhaliel, Adeeb Abdullah

Track A: Business Management and Economics Studies

BCS-1215-114	Brand Extension: An Overview of the Concept and Research	Chin-Chiung Kuo
BCS-1215-123	Methodology To Study Sustainable Competitive Advantages For Ecotourism Development Of Phu Quoc Island	Pham Huy Hoang
BCS-1215-128	Comparative Analysis of Display Position in Smart Car among Countries	Hoonsik Yoo
BCS-1215-132	Web-based Analytic Hierarchy Process(AHP) Assessment Model for Information Security Policy of Commercial Banks	ShinaaMing Wu
BCS-1215-140	Low Cost Airlines Operating In The Ghanaian Airspace	Charles Andoh
BCS-1215-117	The Extent of the Internal Control Disclosure, the Executive Compensation, and the Timeliness Financial Reporting (A Case Study at Manufacturing Companies Listed at The BEI Jakarta 2013)	Julianti Sjarief
ECS-1215-126	A Case Study on Product Development Strategy in Consumption Value Structures	Soo Ah Cho

Lunch Break (12:00 - 1:00pm)

DAY 01 Thursday (December 17, 2015)
Session 1 (10:00am – 12:00 pm)
Venue: Room 2
Session Chairs: Dr Paramanantham & Deni Yasmara

Track D: Medical, Medicine & Health Sciences

MCS-1215-102	Design and Evaluation of AR-Based Serious Games with Clinical Knowledge for Promoting Health Fitness	Liao, Min-Wen
MCS-1215-103	Mitochondrial ROS manage the LPS-induced pro-inflammatory response in microglia cells by controling MAPK and NF-κB pathways	Un-bin chae
MCS-1215-104	Iron overload induces neuronal death via mitochondrial fission through Drp1-S637 dependent manner in HT-22 cells	Donggil Lee
MCS-1215-105	Peroxiredoxin/JNK axis regulate Stemness during neurogenesis from Embryonic stem cells	Jungbae Seong
MCS-1215-106	Depletion of mitofusin 2 is related to amyloid beta-mediated mitochondrial fragmentation and Cdk5-induced oxidative stress in neuron cells	Mi Hye Kim
MCS-1215-107	Peroxiredoxin 5 prevents amyloid-beta oligomer-induced neuronal cell death by inhibiting ERK-Drp1-mediated mitochondrial fragmentation	Bokyung Kim
MCS-1215-111	The influence of the knowledge about young women's cervical cancer screening who are doing physical activities on the screening attitude	Yun-Hwa Ko

Lunch Break (12:00 - 1:00pm

DAY 01 Thursday (December 17, 2015)
Session 2 (01:00 – 02:30 pm)
Venue: Room 1
Session Chair: Hoonsik Yoo

Track A: Business Management and Economics Studies

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BCS-1215-147A	Factors Affecting Consumers Decision to Purchase Vietgap Vegetable in Hanoi, Vietnam	Nhung Thi Thai
BCS-1215-103	Labour Legislation And Performance Of Small Enterprises In Gauteng Province of South Africa	Akhabue A Okharedia
BCS-1215-152	Education Inequality in Indonesia: Using Education Gini Index Measurement	Lilik Sugiharti
BCS-1215-153	Accelerating Economic Development Strategy Through Trade Sector Development In Surabaya City	Nurul Istifadah
BCS-1215-154	Technical efficiency and productivity growth: case of Indonesia's food and beverage manufacturing sector	Martha R Primanthi
ECS-1215-130	The Study on the Structural Changes of the Supplier Value Chain in the Convergence Industry	EunYoung Park

Tea Break (02:30 – 02:45pm

DAY 01 Thursday (December 17, 2015)
Session 2 (01:00 – 02:30 pm)
Venue: Room 2
Session Chair: Tzu-Wei Lin

Track B: Engineering & Technology, Computer, Basic & Applied Sciences

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ECS-1215-124	Design and Evaluation of Secure Digitally Signing Solutions for Hospital Consents	Wei-Cheng Wei
ECS-1215-134	Development of Miniature Planar Spring for Electrodynamics Vibration Energy Harvesting using Extra Thin Printed Circuit Board	Gandi Sugandi
ECS-1215-128	Application two-stage clustering method selected core functions of business and Recruitment and promotion of research	Jiun-Yi Li
ECS-1215-112	Design and Implementation of a Secure Cloud Platform for Protecting and Managing Healthcare Medical Information	Bo Yu Huang
ECS-1215-135	YII Framework Security Implementation On Application Study Of Energy Efficiency	Dewi Rosmala

Tea Break (02:30 – 02:45pm)

DAY 01 Thursday (December 17, 2015)

Session 3 (02:45 – 04:00 pm)
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BCS-1215-129	Comparative and Competitive Challenges of Active Learning in Indonesian University: What and How to Fix it	Pheni Chalid
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Closing Ceremony: 4:00 – 5:00 pm

DAY 01 Thursday (December 17, 2015)

Session 3 (02:45 – 04:00 pm)
Venue: Room 2
Session Chair: Sarala Joshi

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ECS-1215-104	Optimizing ZnS Buffer Layer of Cu(In,Ga)Se2 Thin Film Solar Cell with Tri-Sodium Citrate	Jun Chul Shin
ECS-1215-107	Effect of Ag Thickness and Heat Treatment on the structure, electrical and optical properties of GZO/Ag/GZO Multilayer Films.	SungHee Cho
ECS-1215-108	Effects of Surface Texturing Size on the Screen Printed Si Solar Cell	Dae Sung Kim
ECS-1215-109	Federated Anonymous Identity Management for Cloud Computing	Tzu-Wei Lin
ECS-1215-115	GUI PID Self-tuning System for Quadcopters	Chiao Tzu Huang

Closing Ceremony: 4:00 – 5:00 pm

LIST OF CONFERENCE ATTENDEES

The following Scholars/ practitioners/educationist who don't have any paper presentation, however they will attending the conference as delegates & observers.

Sr. No	Official ID	Name
1	BCS-1215-135A	Alkhaliel, Adeeb Abdullah
2	MCS-1215-109	Dr Paramanantham
3	BCS-1215-147	Dr. Kampanat Pensupar
4	MCS-1215-110A	Dong-Seok Lee
5	MCS-1215-102A	Chien-Lung, Hsu
6	MCS-1215-113A	Deni Yasmara
7	MCS-1215-114A	Sriyono

DAY 02 Friday (December 18, 2015)
City Tour and Shopping Day

All participants will be free to carry on their own tourism and shopping activities in Singapore. It's a free day for this purpose

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TRACK A: MEDICAL, MEDICINE& HEALTH SCIENCES

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Design and Evaluation of AR-Based Serious Games with Clinical Knowledge for Promoting Health Fitness

Liao, Min-Wen[1*], Hsu Chien Lung[2],
Chang Gung University, Taiwan

Abstract

Serious Games can be applied in various fields such as military, health care, aviation, and education. In evidence-base study, serious games based on clinical knowledge can be designed to enhance holistic care, elderly patient's safety, and health care delivery. This study uses augmented reality (AR) technology and considers clinical knowledge to design AR-based serious games with motion sensing controller for promoting health fitness. Users can not only paly the proposed serious grames enjoyly, but also imporve their health. Moreover, we consider "quality of game" and "perceived enjoymentand" constructs to design a new research model based on information system success model to evaluate user acceptance and statisfication of the proposed games. The results show that (i) "quality of game" construct could positively affect "perceived enjoymentand" one, (ii) "perceived enjoymentand" construct could positively affect "intention to use" and "Satisfaction" ones, and (iii) "Satisfaction" construct could positively affect " intention to use " one.

Keywords: Serious Game, Fitness, Information System Success Model, Clinical Knowledge

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Mitochondrial ROS manage the LPS-induced pro-inflammatory response in microglia cells by controling MAPK and NF-κB pathways

Un-Bin Chae[1*], Dong-Seok Lee[2]
[1, 2] kyungpook National University, Korea

Abstract

Activation of microglia cells in the brain contributes to neurodegenerative processes promoted by many neurotoxic factors such as pro-inflammatory cytokines and nitric oxide (NO). Reactive oxygen species (ROS) actively affect microglia-associated neurodegenerative diseases through their role as pro-inflammatory molecules and modulators of pro-inflammatory processes. Although the ROS which involved in microglia activation are thought to be generated primarily by NADPH oxidase (NOX) and involved in the immune response, mitochondrial ROS have also been proposed as important regulators of the inflammatory response in the innate immune system. However, the role of mitochondrial ROS in microglial activation has yet to be fully elucidated. In this study, we demonstrate that inhibition of mitochondrial ROS by treatment with Mito-TEMPO effectively suppressed the level of mitochondrial and intracellular ROS. Mito-TEMPO treatment also significantly prevented LPS-induced increase in the TNF-α, IL-1β, IL-6, iNOS and Cox-2 in BV-2 and primary microglia cells. Furthermore, LPS-induced suppression of mitochondrial ROS generation not only affected LPS-stimulated activation of MAPKs, including ERK, JNK, and p38, but also regulated IκB activation and NF-κB nuclear localization. These results indicate that mitochondria constitute a major source of ROS generation in LPS-mediated activated microglia cells. Additionally, suppression of LPS-induced mitochondrial ROS plays a role in modulating the production of pro-inflammatory mediators by preventing MAPK and NF-κB activation in microglia cells. Our findings suggest that a potential strategy in the development of therapy for inflammation-associated degenerative neurological diseases involves targeting the regulation of mitochondrial ROS in microglial cells.

Keywords: Lipopolysaccharide; MAPKs; Microglia; Mito-TEMPO; Mitochondrial ROS; NF-κB

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Iron Overload Induces Neuronal Death Via Mitochondrial Fission Through Drp1-S637 Dependent Manner in HT-22 Cells

Donggil Lee[1*], Dong-Seok Lee[2]

[1, 2] Kyungpook National University, Korea

Abstract

The accumulation of iron in neurons has been proposed to contribute to the pathology of numerous neurodegenerative diseases, such as Alzheimer's disease and Parkinson's disease. However, insufficient research has been conducted on the precise mechanism underlying iron toxicity in neurons. In this study, we investigated mitochondrial dynamics in hippocampal HT-22 neurons exposed to ferric ammonium citrate (FAC) as a model of iron overload and neurodegeneration. Incubation with 150 μM FAC for 48 h resulted in decreased cell viability and apoptotic death in HT-22 cells. The FAC-induced iron overload triggered mitochondrial fragmentation, which was accompanied by Drp1(Ser637) dephosphorylation. Iron chelation with deferoxamine prevented the FAC-induced mitochondrial fragmentation and apoptotic cell death by inhibiting Drp1(Ser637) dephosphorylation. In addition, a S637D mutation of Drp1, which resulted in a phosphorylation-mimetic form of Drp1 at Ser637, protected against the FAC-induced mitochondrial fragmentation and neuronal apoptosis. FK506 and cyclosporine A, inhibitors of calcineurin activation, determined that calcineurin was associated with the iron-induced changes in mitochondrial morphology and the phosphorylation levels of Drp1. These results indicate that the FAC-induced dephosphorylation of Drp1-dependent mitochondrial fragmentation was rescued by the inhibition of calcineurin activation. Therefore, these findings suggest that calcineurin-mediated phosphorylation of Drp1 (Ser637) acts as a key regulator of neuronal cell loss by modulating mitochondrial dynamics in iron-induced toxicity. These results may contribute to the development of novel therapies for treatment of neurodegenerative disorders related to iron toxicity.

Keywords: Calcineurin; Drp1; Iron overload; Mitochondrial Dynamics; Neurotoxicity

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Peroxiredoxin/JNK Axis Regulate Stemness During Neurogenesis from Embryonic Stem Cells

Jungbae Seong[1*], Dong-Seok Lee[2]
[1, 2] Kyungpook National University
Korea

Abstract

Redox balance has been suggested as an important determinant of \"stemness\" in embryonic stem cells (ESCs). In this study, we demonstrate that peroxiredoxin (Prx) plays a pivotal role in maintenance of ESC stemness during neurogenesis through suppression of reactive oxygen species (ROS)-sensitive signaling. During neurogenesis, Prx I and Oct4 are expressed in a mutually dependent manner and their expression is abruptly downregulated by an excess of ROS. Thus, in Prx I(-/-) or Prx II(-/-) ESCs, rapid loss of stemness can occur due to spontaneous ROS overload, leading to their active commitment into neurons; however, stemness is restored by the addition of an antioxidant or an inhibitor of c-Jun N-terminal kinase (JNK). In addition, Prx I and Prx II appear to have a tight association with the mechanism underlying the protection of ESC stemness in developing teratomas. These results suggest that Prx functions as a protector of ESC stemness by opposing ROS/JNK cascades during neurogenesis. Therefore, our findings have important implications for understanding of maintenance of ESC stemness through involvement of antioxidant enzymes and may lead to development of an alternative stem cell-based therapeutic strategy for production of high-quality neurons in large quantity.

Keywords: Antioxidants; Embryonic Stem Cells; Neurogenesis; Peroxiredoxin; Reactive Oxygen Species

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Depletion of Mitofusin 2 is Related to Amyloid Beta-Mediated Mitochondrial Fragmentation and Cdk5-Induced Oxidative Stress in Neuron Cells

Mi Hye Kim[1*], Dong-Seok Lee[2]
[1, 2] Kyungpook National University, Korea

Abstract

Mitochondrial dysfunction is implicated in age-related degenerative disorders such as Alzheimer's disease (AD). Maintenance of mitochondrial dynamics is essential for regulating mitochondrial function. Ab oligomers (AbOs), the typical cause of AD, lead to mitochondrial dysfunction and neuronal loss. AbOs have been shown to induce mitochondrial fragmentation, and their inhibition suppresses mitochondrial dysfunction and neuronal cell death. Oxidative stress is one of the earliest hallmarks of AD. Cyclin-dependent kinase 5 (Cdk5) may cause oxidative stress by disrupting the antioxidant system, including Prx2. Cdk5 is also regarded as a modulator of mitochondrial fission; however, a precise mechanistic link between Cdk5 and mitochondrial dynamics is lacking. We estimated mitochondrial morphology and alterations in mitochondrial morphologyrelated proteins in Neuro-2a (N2a) cells stably expressing the Swedish mutation of amyloid precursor protein (APP), which is known to increase AbO production. We demonstrated that mitochondrial fragmentation by AbOs accompanies reduced mitofusin 1 and 2 (Mfn1/2) levels. Interestingly, the Cdk5 pathway, including phosphorylation of the Prx2-related oxidative stress, has been shown to regulate Mfn1 and Mfn2 levels. Furthermore, Mfn2, but not Mfn1, over-expression significantly inhibits the AbO-mediated cell death pathway. Therefore, these results indicate that AbO-mediated oxidative stress triggers mitochondrial fragmentation via decreased Mfn2 expression by activating Cdk5-induced Prx2 phosphorylation.

Keywords: Alzheimer's Disease, Cdk5, Mitochondrial Fragmentation, Mitofusin 2, Oxidative Stress, Peroxiredoxin 2

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Peroxiredoxin 5 Prevents Amyloid-Beta Oligomer-Induced Neuronal Cell Death by Inhibiting ERK-Drp1-Mediated Mitochondrial Fragmentation

Bokyung Kim[1*], Dong-Seok Lee[2]
[1, 2] Kyungpook National University, Korea

Abstract

Alzheimer's disease (AD), a neurodegenerative disorder, is caused by amyloid-beta oligomers (AβOs). AβOs induce cell death by triggering oxidative stress and mitochondrial dysfunction. A recent study showed that AβO-induced oxidative stress is associated with extracellular signal-regulated kinase (ERK)–dynamin related protein 1 (Drp1)-mediated mitochondrial fission. Reactive oxygen species (ROS) are regulated by antioxidant enzymes, especially peroxiredoxins (Prxs) that scavenge H2O2. These enzymes inhibit neuronal cell death induced by various neurotoxic reagents. However, it is unclear whether Prx5, which is specifically expressed in neuronal cells, protects these cells from AβO-induced damage. In this study, we found that Prx5 expression was upregulated by AβO-induced oxidative stress and that Prx5 decreased ERK–Drp1-mediated mitochondrial fragmentation and apoptosis of HT-22 neuronal cells. Prx5 expression was affected by AβO, and amelioration of oxidative stress by N-acetyl-L-cysteine decreased AβO-induced Prx5 expression. Prx5 overexpression reduced ROS as well as RNS and apoptotic cell death but Prx5 knockdown did not. In addition, Prx5 overexpression ameliorated ERK–Drp1-mediated mitochondrial fragmentation but Prx5 knockdown did not. These results indicated that inducible Prx5 expression by AβO plays a key role in inhibiting both ERK–Drp1-induced mitochondrial fragmentation and neuronal cell death by regulating oxidative stress. Thus, Prx5 may be a new therapeutic agent for treating AD.

Keywords: Alzheimer's Disease, Amyloid Beta, Peroxiredoxin 5, Oxidative Stress, Mitochondrial Fragmentation, ERK, Drp

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The Influence of the Knowledge About Young Women's Cervical Cancer Screening who are Doing Physical Activities on the Screening Attitude

Yun-Hwa Ko*
Kongju National University, Korea

Abstract

This research observes the influence of the knowledge about cervical cancer screening on the screening attitude to provide necessary implications by drawing the method for preventing young women's cervical cancer. The research targets were extracted conveniently from Seoul and Gyeonggi-do area, it was total 281 people and the average age was 22.94 years old. The questions related to the cervical cancer knowledge are focused on investigating whether it is recognize the cause and the preventive potential through vaccination or not with the consideration that the cervical cancer has the possibility to prevent from happening through early detection. The 'Knowledge' related to cervical cancer is consists of 2 questions, and evaluated as 5-point Likert scale which means 1 point as 'Not at all' and 5 points as 'Absolutely'. In this research, as the total average scores higher, it evaluates the knowledge related to cervical cancer as higher. After examining how the knowledge related to cervical cancer effects on the cervical cancer screening attitude, the knowledge related to cervical cancer can't statistically and significantly affects on the cervical cancer screening attitude. To treat the cancer or to prevent, the early detection and preventive behavior has to be combined. It could be developed to the actual health behavior if it is with the related knowledge to happen these early detection and the preventive behavior. But in this research, the knowledge related to cervical cancer couldn't significantly effects on the cervical cancer screening attitude. However, with considering that young women has low seriousness about cervical cancer infection, the lack of the related knowledge or the low awareness of seriousness has steady effects on the result of this research, and it implies the necessity of the verification of continuous effects.

Keywords: Influence, Screening Attitude, Cancer

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FUTURE EVENTS

You can find the Details regarding our future events by following below:

Business, Economics, Social Science & Humanities (BESSH) Conferences:

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