The 6th FAONS Congress &
The 11th Biennial Conference of CNS

Plenary Lecture

- PL-1 The Molecular Logic of Neural Circuits: Implications for Autism and Schizophrenia (Thomas Südhof)
- PL-2 Neural Basis of Emotional and Social behavior (Hailan Hu)
- PL-3 Control of Social Aggression by the Habenula (Hitoshi Okamoto)
- PL-4 Regulation and Function of Adult Neurogenesis in the Mammalian Hippocampus (Fred H. Gage)
- PL-5 Genetic variation in S100B modulates neural basis of human spatial navigation (Jia Liu)
- PL-6 Molecular and Synaptic Mechanism of Paroxysmal Kinesigenic Dystonia (Zhiqi Xiong)
- PL-7 Dopamine neurons, synapses and susceptibility in Parkinson’s disease (Paul Bolam)
- PL-8 Mapping global patterns of connectivity in the mammalian brain (Liqun Luo)

Satellite Meeting

- SM1 7th MCCS-Asia Meeting (Organizer: Weidong Li)
- SM2 Cross-Strait Neuroscience Symposium (Organizer: Y. Henry Sun, Shumin Duan)
- SM3 Mental Disorders and Their Biological Contributions Symposium (Organizer: Lingjiang Li)
- SM4 Disorders of Consciousness Symposium (Organizer: Ruxiang Xu)

Symposium

- S1-FAONS Symposium-From Synaptic Transmission to Circuit Function (Sun Jianyuan)
- S2-Recent progress in Pain and Itch Circuit and Signaling (Ji Rurong)
- S3-Neural Regeneration and Functional Reconstruction (Gu Xiaosong)
- S4-Understanding the Motor System-Synapses, Circuits and Disorders (Wang Jianjun)
- S5-Neuroimmunology and CNS Diseases (Peng Yuping)
- S6-Autophagy in Neuronal Functions and Neurodegenerative Diseases (Le Weidong)
- S7-Metal Metabolism and Neurodegenerative Disorders (Xie Junxia)
- S8-Sensory Systems Neuroscience (Anna Wang)
- S9-FAONS Symposium-Cognitive Functions in Health and Diseases(Saeed Semnanian)
- S10-ISN Symposium-Recent Advance in Basic Neurochemistry in Brain Diseases (Zhu Xiongwei)
- S11-Emotional and Cognitive Modulation of Pain (Zhang Yuqiu)
- S12-Glial Biology in Health and Diseases (He Cheng)
- S13-Dopaminergic Neurotransmission in Health and Disease (Zhou Jiawei)
- S14-Brain Banking in China-Progress and Research Updates (Yan Xiaoxin)
- S15-Neural Plasticity and Learning and Memory (Luo Jianhong)
P-184 Ginsenoside Rg1 attenuates lipopolysaccharide-induced inflammatory responses via G protein-coupled estrogen receptor in murine BV2 microglial cells

Wen-Fang Chen*, Xian-Qi Gao
Department of Physiology, Medical College of Qingdao University, Qingdao 266071, PR China. Shandong Provincial Collaborative Innovation Center for Neurodegenerative Disorders, China.
*Corresponding author. E-mail: chenwenfangqd@163.com

Abstract: Objective Accumulating clinical and experimental evidence suggests that chronic neuroinflammation plays an important role in neurodegenerative diseases. The G protein-coupled estrogen receptor (GPER) was reported to be a novel membrane estrogen receptor, which responds to estrogen and mediates estrogen’s rapid cellular effects. Studies have shown that activation of GPER could account for some of the protective effects of estrogen against inflammatory responses in animal model of multiple sclerosis. Our previous studies demonstrated that ginsenoside Rg1, a steroidal saponin of high abundance in ginseng, could mediate its action via ER in human breast cancer. The present study aimed to evaluate the involvement of GPER in the inhibitory effects of Rg1 on lipopolysaccharide (LPS)-induced microglia activation. Methods Murine BV2 cells were treated with LPS and/or Rg1 in the absence or presence of GPER antagonist G15. Real time RT-PCR and western blot were used to determine the mRNA and protein expressions of proinflammatory cytokines. Lentivirus-mediated siRNA interference was used to knockdown the expression of GPER. Results (1) Rg1 treatment significantly inhibited the LPS-induced production of proinflammatory cytokines such as iNOS, COX2, IL-1β and TNFα. These effects could be abolished by GPER antagonist G15.(2) Lentivirus mediated GPER siRNA knocked down the GPER expression in BV2 microglial cells, which led to
cytokines caused by HSP60 decreased significantly. These results suggest that HSP60 may mediate the OPC apoptosis through binding to TLR4 on the surface of OPC so to activate TLR4-NFκB signaling pathway. HSP60 may be a potential target for treatment of myelin-related neurodegenerative diseases that are accompanied by microglia activation. **Keywords:** heat shock protein 60; OPC; microglia; TLR4

---

**P-188 Inhibition of neuroinflammation in LPS-activated microglia by genistein**

Zhong-Rei Du, Xiao-Fan Ren, Wen-Fang Chen*

*Department of Physiology, Medical College of Qingdao University, Qingdao 266071, PR China. Shandong Provincial Collaborative Innovation Center for Neurodegenerative Disorders, China*

*Corresponding author. E-mail: chenwenfangqd@163.com*

**Abstract:** **Objective** Genistein, a natural isoflavone phytoestrogen present in soybeans, have significant estrogenic properties in various tissues. Several lines of evidence suggest that genistein can bind to G protein coupled estrogen receptor (GPER) and activate the non-genomic estrogen signaling pathway. This study aimed to investigate the anti-inflammatory effects of genistein and the potential mechanism in BV2 microglial cells. **Methods** LPS was used to induce inflammatory response in BV2 microglial cells. BV2 cells were treated with genistein in the absence or presence of GPER antagonist G15. Real time PCR was used to determine the mRNA expression of proinflammatory cytokines. The protein expressions of proinflammatory cytokines and the activation of mitogen-activated protein kinases (MAPKs) cascades were measured by western blot. **Results** (1) Pretreatment of genistein could significantly inhibit the IL-1β gene expression in LPS-stimulated microglia. (2) LPS-induced protein expressions of inducible nitric oxide synthase (iNOS),
LPS-stimulated BV2 and primary microglia cells. The promoting effects of telmisartan on M2 polarization were attenuated by an AMP-activated protein kinase (AMPK) inhibitor or AMPK knockdown, indicating that AMPK activation participates on telmisartan effects. Furthermore, telmisartan enhanced brain AMPK activation and M2 gene expression in a mouse model of LPS-induced neuroinflammation. Conclusion Our results indicate that telmisartan can be considered as a novel AMPK activator, suppressing neuroinflammation by promoting microglia M2 polarization. Telmisartan may provide a novel and safe therapeutic approach for the treatment of brain disorders associated with neuroinflammation.

**Keywords:** Telmisartan; Microglia polarization; AMP-activated protein kinase; Neuroinflammation

**P-212 Involvement of glucocorticoid receptor in the anti-inflammatory effects of ginsenoside Rg1 in BV2 microglial cells**

Xiao-Fan Ren, Zhong-Rei Du, Wen-Fang Chen*

*Department of Physiology, Medical College of Qingdao University, Qingdao 266071, PR China. Shandong Provincial Collaborative Innovation Center for Neurodegenerative Disorders, China.*

*Corresponding author. E-mail: chenwenfangqd@163.com*

**Abstract:** Objective Ginsenoside Rg1 is the major pharmacological ingredient of ginseng. Studies have shown that Rg1 possess a variety of beneficial effects on human health, including anti-inflammation, antioxidant, anti-neurotoxin. The present study aimed to evaluate the protective effects of Rg1 on lipopolysaccharide (LPS)-induced microglia