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编者按：2011 年，中国生理学会成立 85 周年之际，学会编辑出版了以王晓民理事长为主编的上下两本图书，上册为《根深叶茂 蔚然成荫——中国生理学人物记》，下册为《根深叶茂 蔚然成荫——中国生理学团队记》。从 2013 年第 3 期开始，《生理通讯》将陆续转载，以飨读者。

生理学家

张宗汉教授传略

翁恩琪　周绍慈

张宗汉
（1899年-1985年）

我国老一辈生理学家张宗汉教授，字真衡，1899年2月出生于浙江省嵊县一个贫寒的农民家庭。1918年考入之江大学，勤工俭学，时辍时续地学习。1926年毕业于南京东南大学，并应聘在南京生物研究所秉志先生领导下工作。1929年去美国芝加哥大学医学院留学，师从R. W. Gerard教授，攻读普通生理学。以优异的成绩获得哲学博士学位。1933年回国后，历任上海医学院教授、南京生物研究所研究员、江西中正医学院教授、江西中正大学教授兼系主任以及浙江大学人类学系、南通学院医科、同济大学理学院等院校教授。1951年华东师范大学建校后，任生物系教授并兼任主任。

张宗汉长期从事生理学教学和研究工作，兢兢业业为我国高等教育和生理科学的发展贡献出毕生的精力和热情。早年，他研究过动物学，曾描述过福州地区一种硬壳龟的新种，对南京地区的蛇类和海龟的种群分布进行过调查。其后，专门从事生理学的研究，尤其在神经代谢生理领域卓有成果，曾在《美国生理学杂志》、《比较神经学杂志》、《实验生物学和医学学会纪事》（纽约）和《中国科学社生物研究所学报》等刊物上发表过豚鼠大脑皮层运动区定位、龙虾肢体神经及神经节的代谢、离体神经的呼吸、乳酸对神经代谢的影响、电解质对神经呼吸的影响、血液组分对神经耗氧量的影响、部分窒息蛙的神经系统对水摄入及无尿机制的作用等论文。是我国最早从事神经代谢研究的工作者之一。张宗汉早年即加入中国生理学会，是终身会员，曾任理事。

张宗汉自20世纪30年代初任教以来，50多年如一日，始终把教育工作视为自己终身的崇高事业，在科学教育岗位上辛勤耕耘。抗日战争时期，他在极端困难的条件下，坚守岗位，在学校撤迁流亡途中，亲自带领学生下溪捕捞鱼蟹作为实验材料，讲解寄生虫和神经系统的结构功能，师生无不为之感动。新中国成立初期，他积极参与筹建华东师范大学的工作，是华东师范大学生物系的主要创建者之一，以后又出任系主任，并亲自兼任生理教研室主任，为生理学和生物学系诸多学科的建立和发展竭尽全力。张宗汉对待教学工作极端认真负责，授课思路清晰，论理透彻，更以严谨的治学态度和一丝不苟的工作精神博得师生们的尊敬和爱戴。他所培养的学生，已遍布祖国各地，其中有许多已成为高等院校生理学教学和科研的骨干。

张宗汉的一生，是追求真理的一生，他经历过清朝和民国，目睹旧社会的腐败。新中国成立前，他十分同情学生运动，帮助过地下党，并在陶行知先生主办的晓庄师范任教。新中国成立初期，为了支援抗美援朝战争，他废寝忘食地工作，同几位同事一起，成功地为人民志

愿军研制出新的干粮，受到人民政府的表彰。1959年，张宗汉实现了多年的夙愿，加入了中国共产党。他在工作中处处顾全大局，以身作则，严以律己，宽以待人，从不计较个人得失，因而享有很高的声誉。在“文化大革命” 期间，他刚直不阿，仗义执言，捍卫科学，维护老教师的声誉和尊严。

张宗汉于1972年因患脑血管疾患而瘫痪，但他身残志坚。这位白发苍苍的老人，以极大的毅力，克服半身不遂的不便和下肢浮肿病痛，每天俯身病榻旁的书桌，坚持阅读中外文献和各种报刊杂志。关心中青年教师的成长，经常找他们谈话，提出有关教学和科研的建议。直到生命的最后一息，他都关心着国家大事，惦记着教育和科学事业。由于病情恶化，医治无效，张宗汉于1985年12月23日与世长辞，终年87岁。噩耗传出，他的生前好友和学生，不胜悲痛。华东师大生物系全体师生撰写了一幅挽联：

滋桂培兰犹记先生勤灌溉

立言树德更教后辈仰声华

表达了对张宗汉一生业绩的崇敬之情。张宗汉为教育和科学事业奉献了他的一生，他永远活在后辈的心中。

生理学团队

中国科学院上海生命科学研究院 / 上海交通大学医学院 健康科学研究所分子心脏学实验室

健康科学研究所分子心脏学实验室组建于2000年，是中国科学院上海生命科学研究院与上海交通大学医学院共同组建的健康科学研究实体化运作成立的第一批实验室和中国科学研究院干细胞生物学重点实验室成员。团队带头人为杨黄恬研究员，为生物学一级学科生理学和细胞生物学二级学科硕士研究生和博士研究生培养点，分别依托中国科学院上海生命科学研究院和上海交通大学医学院招生研究生。团队的使命为以人口与健康研究为主导，瞄准生物医学领域的前沿课题和社会发展需求，围绕人类重大疾病，重点发展与临床结合的基础和应用性研究，加强整合生理学、细胞生理学等学科建设，培养一批出色的生物医学研究的复合型人才。

实验室由研究员2人、副研究员2人、助理研究员和技术员、博士后和硕博研究生组成，为分子生物学、细胞生物学、生理学、病理学、药理学等多学科交叉的团队。承担了研究生的教学任务，参与了“十一五”国家重点图书的编写，如《心血管生理学与临床》、《心血管肾脏生理学实验技术方法及其进展》及研究领域的有关书籍的编写，如Embryonic Stem Cells: Method and Protocols, Control of Celluar Physiology by E2F Transcription Factors,《心血管病前沿》等。

在科研方面，实验室在十多年的建设发展过程中，推崇崇德尚学、笃真求实、自强不息、探索进取的精神，坚持团队合作，围绕认识心脏生理功能调控和心脏疾病发生发展的分子机理、探寻防治心肌缺血、心衰的有效靶点和措施的研究目标，形成了如下主要研究方向：（1）多能干细胞向心肌细胞分化的调控和应用研究；（2）钙信号和G蛋白耦联受体介导的信号通路对心肌发育、收缩功能和心力衰竭的调控；（3）低氧适应心肌保护和内源性保护因子的作用和细胞分子机制和应用研究；（4）天然化合物抗心肌缺血损伤的作用和机制研究。在研究发展的过程中，实验室建立并发展了心肌细胞分离、成体心肌细胞培养和转基因、心肌细胞同步收缩和胞内钙瞬变检测平台；电生理记录与分析平台；离体心脏灌流与

缺血模型和动物心梗模型与分析体系；鼠和人胚胎干细胞与诱导性多能干细胞培养和分化诱导分析平台等。面向国家需求，积极申请各类竞争性项目，2005年来承担了包括科技部“973”和重大研究计划（课题组长和骨干）、国家自然科学基金重点、面上和青年项目、中国科学院创新工程方向性项目和干细胞先导项目、上海市科委重大项目（课题组长和骨干、国际交流科研项目等20余项；在本领域国际主流杂志发表研究论文20余篇，申请国家发明专利4项（已授权3项）；招收硕博研究生18人，毕业15人，其中2人获硕士学位，13人获博士学位。实验室1人获上海市科教系统“三八红旗手”，17名研究生分别获得了中科院院长优秀奖，中国科学院“地奥”二等奖，第六届、第七届海峡两岸心血管科学研讨会青年优秀论文奖，第九届谈家桢九源基金奖学金，中科院研究生院三好学生标兵，优秀学生干部和优秀毕业生，上海生命科学研究院院长奖等30项奖，为学科建设和发展输送了优秀人才。

附：学科带头人简介

杨黄恬，女，54岁，博士，研究员，博士生导师。

1982年毕业于南通医学院获医学学士学位；1988年获苏州医学院医学硕士学位；1994年获日本山形大学医学博士学位。先后在南通医学院任助教、讲师（1982—1992）、日本山形大学医学部助手和客座研究员（1993—1997）、美国NIH/NIA（1997—2001）从事心血管功能调控和药理研究。于2000年获中国科学院百人计划资助回国任现职，为国际心脏研究学会中国分会副主席、Biophysical Society和American Heart Association成员，中国病理生理学会心血管专业和受体专业委员会委员、中国细胞生物学学会理事、中华医学会高原医学学会理事、中国生理学会理事、中国生理学会心血管专业委员会委员和上海生理学会副理事长。为Frontier in Pharmacology, Cell Death & Differ, 中国药理学报、中国应用生理学杂志、中国细胞生物学等杂志编委、生理学报常务编委和Cardiovasc Res、Cell Death & Differ、JMCC、Stem Cell Dev等国际专业杂志的审稿者。获2001年NIH研究者优秀研究奖；2003—2004年度和2010—2011年度上海市科教系统“三八红旗手” 称号。长期从事心血管研究，尤其是心肌收缩功能调控、防治心肌缺血损伤和干细胞心肌细胞分化与应用研究，研究发现先后发表于PNAS，Circ Res，FASEB J，Cell Death Diff，Cardiovasc Res，Cell Calcium，Stem Cells Dev，Am J Physiol，Basic Res Cardiol，Cell Res等国际专业领先的杂志40余篇，近5年来在本领域国际主流杂志以通讯作者身份发表研究论文近20篇，带出了一支富有进取精神的以年轻人为主体的在分子生物学、细胞生物学、发育生物学、生理学、药理学、心脏科学研究方面多学科交叉的研究梯队。先后作为负责人和学术骨干主持／承当了国家重大科学研究计划和“973” 项目课题、国家自然科学基金委重大、重点和面上项目、中科院项目、上海市重大科技项目课题和重点项目。

公　告

王宪教授当选《生理科学进展》主编

《生理科学进展》本届编委会工作已满四年，应进行换届。经《生理科学进展》编委会主编推荐和中国生理学会常务理事会研究，现决定聘请北京大学医学部王宪教授担任《生理科学进展》新一届编委会主编，任期四年（从2015 年 4 月 16 日起），并由王宪教授组织新一届《生理科学进展》编委会。

中国生理学会

2015 年 4 月 16 日

张锡钧基金

编者按：2013 年 10 月 12-13 日中国生理学会张锡钧基金会第十二届全国青年优秀生理学学术论文交流会在湖南长沙顺利召开。由各省生理学会推荐的 37 名参赛选手的论文参加评选，会议展示了选手们近 3 年来在生理学研究方面所取得的最新研究成果。经过专家对参评者论文和现场报告的综合评判，评出一等奖、二等奖、三等奖、特别奖、最佳表达奖、最佳答辩奖和最佳图表奖共 11 名。从 2013 年第 6 期开始，《生理通讯》将陆续转载获奖者的参评论文各一篇，以飨读者。

Rg1 protects the MPP^+-treated MES23.5 cells via attenuating DMT1 up-regulation and cellular iron uptake

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Abstract

Ginsenoside-Rg1 is one of the pharmacologically active component isolated from ginseng. Our previous study observed the protective effect of Rg1 on iron accumulation in the substantia nigra (SN) in 1-methyl-4-phenyl-1,2,3,6- tetrahydropyridine (MPTP)-treated Parkinson's disease (PD) mice. However, the mechanisms of this neuroprotective effect of Rg1 are unknown. In this study, we elucidated possible mechanisms for this effect using 1-methyl-4-phenylpyridinium (MPP^+)-treated MES23.5 cells. Previous study showed MPP^+ treatment induced up-regulation of divalent metal transporter 1 without iron responsive element (DMT1-IRE) in MES23.5 cells. In the present study, we observed that pretreatment with Rg1 could inhibit MPP^+-induced up-regulation of DMT1-IRE in MES23.5 cells. Up-regulation of DMT1-IRE by MPP^+ treatment was associated with ROS production and translocation of nuclear factor-kappaB (NF-κB) to nuclei, both of which were significantly inhibited by Rg1 pretreament. The role of ROS and NF-κB in the up-regulation of DMT1-IRE was supported by application of an antioxidant NAC and BAY 11-7082, an inhibitor of IκBα phosphorylation. Furthermore, we also showed Rg1 could decrease DMT1-mediated ferrous iron uptake and iron-induced cell damage by inhibiting the up-regulation of DMT1-IRE. These results indicate that Rg1 protected the MPP^+-treated MES23.5 cells via attenuating DMT1-IRE up-regulation likely through inhibition of ROS-NF-κB pathway；Attenuation of DMT1-IRE expression decreased the iron influx and iron-induced oxidative stress.

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Keywords: Parkinson's disease; 1-methyl-4-phenylpyridinium (MPP^+); divalent metal transporter 1 (DMT1); NF-κB; Rg1.

1. Introduction

Parkinson's disease (PD) is a neurodegenerative disease, characterized by a selective degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNpc) and consequently a reduction in the striatal dopamine levels (Oertel and Ellgring, 1995). Recently, a growing body of evidence shows that selective high levels of iron and oxidative stress resulting from increased iron levels in the SNpc play a key role in PD development (Jiang et al., 2007; Youdim et al., 2004; Zecca et al., 2008). Misregulation of some iron transporters might be involved in this process (Burdo et al., 2001; Jiang et al., 2003; Salazar et al., 2008; Wang et al., 2007).

Iron could be transported into mammalian cells via the transferrin (Tf)-Tf receptor (TfR) and non-transferrin-bound iron (NTBI) pathways. The Tf-TfR was observed unchanged in PD (Faucheux et al., 1993; He et al., 1999), indicating the involvement of NTBI pathway in iron accumulation in PD. Divalent metal transporter 1 (DMT1) is one of the iron transporter involved in NTBI pathway (Gunshin et al., 1997)， and increased DMT1 expression was found in the SN of PD models (Burdo et al., 2001; Salazar et al., 2008). There are 4 isoforms of DMT1. At the 5′-end of the gene, alternative promoter usage leads to different transcription start sites at either exon 1A or exon 1B. In the 3′-untranslated region of the mRNA, one possesses iron responsive element (IRE) (termed +IRE), the other does not (termed −IRE) (Hubert and Hentze, 2002; Lee et al., 1998).

Due to the suggested involvement of DMT1 and iron accumulation in PD, some compounds that could regulate iron transporters and then alleviate iron accumulation might have pharmaceutical value in the treatment of PD. Ginsenoside-Rg1, the pharmacologically active component isolated from ginseng (Panax ginseng C.A. Meyer, Araliaceae), was shown to possess anti-inflammatory, antioxidant, anti-amnestic and anti-aging effects (Cheng et al., 2005; Leung et al., 2007; Wakabayashi et al., 1998). Our previous study showed Rg1 could substantially attenuate iron accumulation in the SN in 1-methyl-4-phenyl-1,2,3,6- tetrahydropyridine (MPTP)-treated PD mice (Wang et al., 2009). Since up-regulation of DMT1-IRE was shown to account for the iron accumulation in 1-methyl-4-phenylpyridinium (MPP^+)-treated MES23.5 dopaminergic cells (Zhang et al., 2009), we hypothesized that Rg1 might attenuate iron accumulation via regulating DMT1-IRE expression. By using MPP^+-treated MES23.5 cells as a model in the study, we elucidated the possible mechanisms how Rg1 affects iron levels. Results showed that Rg1 could attenuate MPP^+-induced up-regulation of DMT1-IRE probably through inhibiting ROS-nuclear factor-kappaB (NF-κB) pathway, which decreased the iron influx and iron-induced oxidative stress. This study has provided new evidence for Rg1 to be a potential therapeutic target for the treatment of PD.

2. Materials and methods

2.1 Materials

Unless otherwise stated, all chemicals were purchased from Sigma Chemical Co. (St. Louis. Mo, USA). The primary DMT1-IRE antibody was purchased from the ADI (ADI, San Antonio,

TX, USA). The primary NF-κB-p65, PCNA antibodies were from Santa Cruz (Santa Cruz Biotechnology Inc, CA, United States). Calcein-AM was from Molecular Probes (Molecular Probes Inc, Carlsbad, CA, USA). Dulbecco's modified Eagle's medium Nutrient Mixture-F12 (DMEM/F12) were from Gibco (Gibco, Grand Island, NY, USA). Rg1 was purchased from Baiqiuen Medical University. N-acetyl-L-cysteine (NAC) and BAY 11-7082 were from sigma. Other chemicals and regents available were from local commercial sources.

2.2 Cell culture

Dopaminergic cell line MES23.5 cells were offered by Dr. Wei-Dong Le (Baylor College of Medicine, TX, USA), which is a dopaminergic cell line hybridized from murine neuroblastoma-glioma N18TG2 cells with rat mesencephalic neurons exhibiting several properties similar to the primary neurons originated in the SN(Crawford et al., 1992). They were cultured in DMEM/F12 containing Sato's components with 5% FBS, 100 units/ml of penicillin and 100 mg/ml of streptomycin at 37°C, in a humid 5% CO_2, 95% air environment. Ginsenoside-Rg1 was dissolved in absolute alcohol as a concentrated stock and further diluted to their final concentration.

MTT assay showed that the viability of MES23.5 cells treated with MPP^+ (<5 μmol/L) for 24 hrs was unchanged compared to that of control. 10~160 μmol/L MPP^+ induced a significant reduction of cell viability (Zhang et al., 2009). In this study, we choose 10 μmol/L MPP^+, a relatively low dose, to keep most cells alive, which would help to detect subtle gene expression changes.

For experiments, cells were seeded at a density of $1\times10^5/cm^2$ in the plastic flasks or on glass cover slips and were allowed to attach for 24 hours before treatment. Then MES23.5 cells were pretreated with Rg1 for 24 h, and subsequently coincubated with MPP^+ (10 μmol/L) for 24 h. BAY 11-7082 is an irreversible inhibitor of IκBα phosphorylation, which results in the inhibition of the cytokine-induced NF-κB activation (Pierce et al., 1997; Woods et al., 2000). And NAC can act as a direct ROS scavenger of free radicals (Aruoma et al., 1989). In this study, BAY 11-7082 (2.5 μmol/L) or NAC (500 μmol/L) was added 30 min before insult with MPP^+ (10 μmol/L) for 24 h to detect the role of ROS and NF-κB in MPP^+ induced DMT1 up-regulation.

2.3 Total RNA extraction and semiquantitative PCR

Total RNA was isolated by using Trizol Reagent from MES23.5 cells according to the manufacturer's instructions. Then 2 μg of total RNA was reverse-transcribed in a 20 μl reaction using reverse-transcription system. Primers were designed using computer software (Primer Premier 5.0). The following primers were employed for DMT1-IRE: forward 5'-TGGCTGTCACGAGTGCTTACA-3', reverse 5'-CCATGGCCTTGGACAGCTATT-3'; Rat GAPDH gene was used as the reference: forward 5'-CCCCCAATGTATCCGTTGTG-3', reverse 5'-GTAGCCCAGGATGC CCTTTAGT-3', Amplification and detection were performed with the following conditions: an initial hold at 95°C for 10 s followed by 40 cycles at 95°C for 5 s and 60°C for 45 s.

2.4 Preparation of nuclear extract

For immunoblot analysis, the extraction and isolation of nuclear and cytoplasmic protein were performed according to the Nuclear and Cytoplasmic Protein Extraction Kit (Beyotime, Jiangsu, China). After washing with 1 mL ice-cold PBS, the cells were collected and then

centrifuged for 5 min at 1200 rpm at 4°C and the pellet was dissolved with cytoplasmic protein extraction agent A supplemented with PMSF. After 5 sec vortex, the tubes were incubated for 10-15 min on ice to promote lysis. Then the cytoplasmic protein extraction agent B was added, vortexed for 5 sec and incubated on ice for 5 sec. The samples were then centrifuged for 5 min at 14,000g at 4°C and the supernatant, containing the cytosolic fraction, was immediately frozen for further analysis. The pellet was resuspended in nuclear protein extraction agent supplemented with PMSF. After 15–20 times vortex for 30 min and 14,000 g centrifuging for 10 min, the supernatants containing the nuclear extracts were obtained.

2.5 Western blots

Cells were lysed directly on the culture dishes using lysis buffer (50 mmol/L Tris•HCl, 150 mmol/LNaCl, 1% Nonidet-40, 0.5% sodium deoxycholate, 1 mmol/L EDTA) plus 1 mmol/L PMSF and protease inhibitors (1 μg/ml pepstatin, 1 μg/ml aprotinin, 1 μg/ml leupeptin). The protein concentration was determined by the Bradford assay kit (Bio-Rad Laboratories, Hercules, CA). Sixty micrograms total proteins were separated using 10% SDS-polyacrylamide gels and then transferred to PVDF membranes. After overnight blocking with 5% non-fat milk at 4°C, the membranes were incubated with rabbit anti-rat DMT1-IRE antibody (1:2000, ADI, USA) or NF-κB polyclonal antibody (1:200, Santa Cruz) for 2 h at room temperature. β-actin was detected by mouse anti-β-actin monoclonal antibody (1:10000, Sigma, USA) according to similar procedures to ensure equal sample protein loading, and mouse anti-rat PCNA antibody (1:200, Santa Cruz) was used to ensure equal sample protein loading for nuclear extraction. Anti-rabbit and anti-mouse secondary antibodies conjugated to horseradish peroxidase were used at a dilution of 1:10000. Cross-reactivity was visualized using ECL western blotting detection reagents and then was analyzed through scanning densitometry by Tanon Image System.

2.6 Reactive oxygen species (ROS) assay

The production of ROS was assessed by the oxidation of 2′, 7′-dichlorodihydrofluorescein diacetate (H_2DCFDA; Sigma) as described before (Chwa et al., 2006; Yang et al., 2007; Yao et al., 2008; Zhang et al., 2009). After washing 3 times with PBS, cells were incubated in DMEM/F12 containing H_2DCFDA (10 μmol/L) for 30 min. The fluorescence signals were measured with excitation filter of 488 nm and 525 nm emission wavelengths.

2.7 Calcein loading of cells and ferrous iron influx assay

Ferrous iron influx into MES23.5 cells was determined by the quenching of calcein fluorescence as described before (Wetli et al., 2006; Zhang et al., 2009). Cells were incubated with calcein-AM (0.5 μmol/L final concentration) in HEPES-buffered saline (HBS, 10 mmol/L HEPES, 150 mmol/L NaCl, pH=7.4) for 30 min at 37°C. The excess calcein on cell surface was washed out 3 times with HBS. The coverslips were mounted in a perfused heated chamber. Calcein fluorescence was recorded at 488 nm excitation and 525 nm emission wavelengths and fluorescence intensity was measured every 3 min for 30 min while perfusing with 1 mM ferrous iron (ferrous sulfate in ascorbic acid solution, 1:44 molar ratio, pH=6.0), prepared immediately prior to the experiments (Picard et al., 2000). The mean fluorescence signal of 25-30 single cells in four separate fields was monitored at 200× magnification and processed with Fluoview 5.0 Software.

2.8 Detection of mitochondrial transmembrane potential (ΔΨm)

Changes in the mitochondrial membrane potential were measured by rhodamine123 using flow cytometry (Becton Dickinson, USA) in MES23.5 cells. Cells in different groups were incubated in 100 μmol/L ferrous iron (pH=6.0) for 3 hrs, and then incubated with rhodamine 123 in a final concentration of 5 μmol/L for 30 min at 37°C. After washing twice with HBS, fluorescence was recorded at 488nm excitation and 525nm emission wavelengths.

2.9 Statistical analysis

Each experiment was performed at least three times, and the results are presented as mean ± S.E.M. One-way analysis of variance (ANOVA) was used to compare the differences between means. Influx studies were carried out by the two ways ANOVA followed by Student-Newman-Keuls test and data were presented as mean ± S.D. A level of $P<0.05$ was considered to be significant.

3. Results

3.1 Rg1 decreased ROS production in the MPP^+-treated MES23.5 cells.

It was reported that the protective effect of Rg1 was mostly attributed to its antioxidant effect acting as a free-radicals scavenger (Chen et al., 2003; Lim et al., 1997; Lopez et al., 2007; Zhong and Jiang, 1997). Therefore, we observed the effect of different concentration of Rg1 on MPP^+-induced ROS production in MES23.5 cells to confirm whether Rg1 had protective effect on MPP^+ in MES23.5 cells and choose an optimal concentration of Rg1 to test its biological effects in MPP^+-treated cells.

As shown in Fig. 1, there was a significant 37.6% increase in the level of ROS in MPP^+-treated cells compared with that of control. However, cells pretreated with 10^{-5} mol/L, 10^{-6} mol/L, 10^{-7} mol/L and 10^{-8} mol/L Rg1 could cause a 31.8 %, 36.0%, 31.8%, 26.3% decrease in ROS production, respectively, compared to that of the MPP^+ treatment (Fig. 1). Therefore, 10^{-6} mol/L Rg1 which showed highest effect on reduction of ROS production was chosen for the following experiments.

No significant changes were observed in cell viability with individual 10-5～10-8 mol/L Rg1 for 48 hours (Data not shown), indicating that Rg1 had little cytotoxic effect on MES23.5 cells at the concentration range.

3.2 Rg1 attenuated up-regulation of DMT1-IRE in MPP^+-treated MES23.5 cells

Our previous study showed DMT1-IRE was up-regulated in MPP^+-treated MES23.5 dopaminergic cells and this up-regulation accounted for the iron accumulation in these cells (Zhang et al., 2009). Thus we hypothesized that Rg1 might attenuate iron accumulation in MPP^+-treated MES23.5 cells via regulating iron transporter DMT1-IRE gene expression.

To confirm this hypothesis, we detected the protein and mRNA levels of DMT1-IRE in MES23.5 cells with different treatments. As shown in Fig. 2 (2A and 2B), DMT1-IRE protein in MPP^+-treated MES23.5 cells was up-regulated to 54% compared to the control, while Rg1 could prevent this up-regulation of DMT1-IRE protein. To further investigate whether this reduced expression of DMT1-IRE protein was due to the decreased DMT1 mRNA transcription, semi-quantitative PCR was conducted to measure DMT1 mRNA levels. There was a 68% increase of DMT1-IRE mRNA in MPP^+-treated cells compared with that of the control. And Rg1 pretreatment reversed this MPP^+-induced increase of DMT1-IRE mRNA (Fig. 2C and 2D). This indicated Rg1 could attenuate the increase expression of DMT1-IRE at both mRNA and protein levels caused by MPP^+.

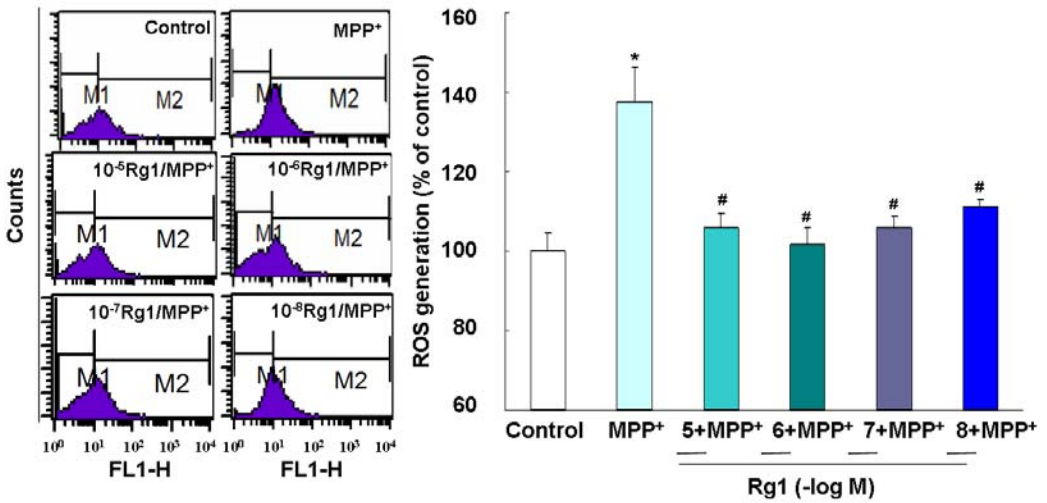

Fig. 1 Rg1 decreased the ROS production of MPP^+ treated MES23.5 cells.10 μmol/L MPP^+ increased the production of ROS in MES23.5 cells compared to the control, while cells pretreated with Rg1(10^{-5}～10^{-8} mol/L) showed a decreased levels of ROS compared to MPP^+. Fluorescence values of the control were set to 100%. Data were presented as mean±S.E.M. of 6 independent experiments. $^*P< 0.05$, compared to the control. $^\#P<0.05$, compared to MPP^+ treated cells.

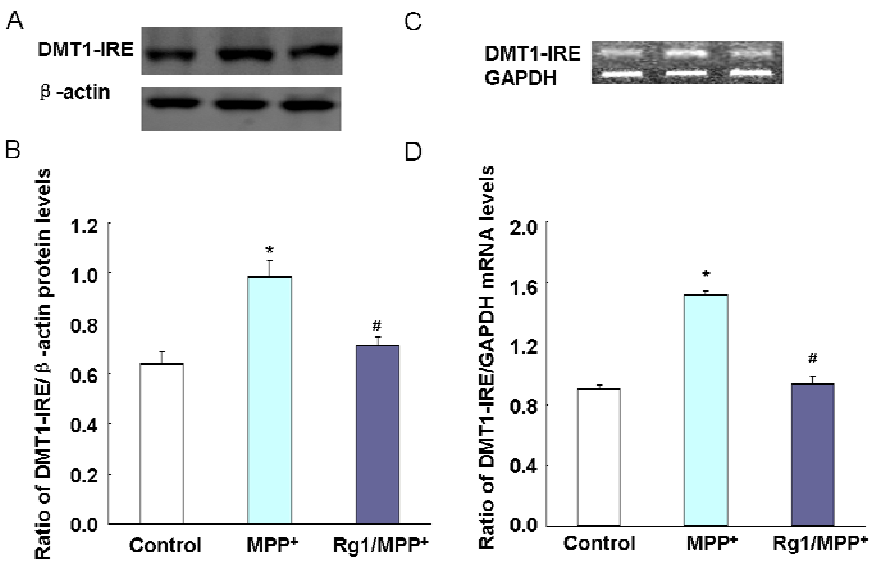

Fig. 2 Rg1 attenuated DMT1 up-regulation in the MPP^+-treated MES23.5 cells. A: Increased expression of DMT1-IRE protein was observed in MPP^+-treated cells. When pretreated with Rg1 (10^{-6} mol/L), the protein levels of DMT1-IRE decreased compared with MPP^+-treated cells. β-actin was used as a loading control. B: Bar graphs for protein levels of DMT1-IRE. C: mRNA levels of DMT1-IRE were increased in MPP^+-treated MES23.5 cells compared to the control. When pretreated with Rg1, the mRNA levels decreased significantly compared to MPP^+ group. D: Bar graphs for mRNA levels of DMT1-IRE. Data were presented as mean±S.E.M. of 6 independent experiments. $^*P<0.01$, compared to the control; $^\#P<0.01$, compared to MPP^+ group.

3.3 Rg1 prevented the MPP^+-induced translocation of NF-κB to nucleus.

NF-κB acting on NF-κB binding sites on the DMT1 promoter might play a role in the expression of this transporter (Huang et al., 2006). Then we hypothesized that Rg1 might regulation the expression of DMT1-IRE through inhibiting NF-κB activation in MPP^+-treated cells. To further confirm our hypothesis, we detected the expression of NF-κB-p65 in the cytoplasm and nucleus, respectively. As shown in Fig. 3, 10 μmol/L MPP^+ could reduce the protein levels of NF-κB-p65 in the cytoplama and increase its levels in nucleus, suggesting that MPP^+ could activate NF-κB translocation from cytoplasm to nucleus. However, when pretreated with Rg1, this MPP^+-induced decreases of NF-κB-p65 expression in cytoplasma and increases in nucleus were significantly inhibited, indicating the increased translocation of NF-κB was inhibited by Rg1 (Fig. 3).

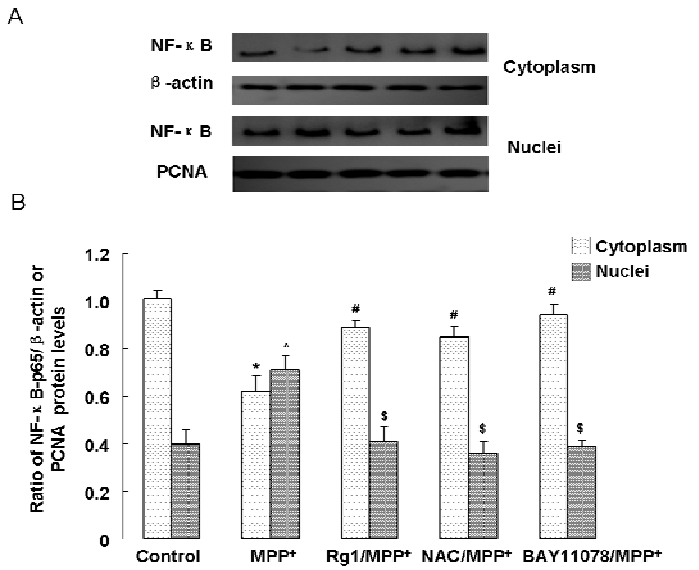

Fig. 3 Rg1 prevented the MPP^+-induced translocation of NF-κB to nucleus. A: The expression of NF-κB-p65 in cytoplasm and nucleus were both detected by western blots. Data showed 10 μmol/L MPP^+ could activate translocation of NF-κB from cytoplasm to nucleus, while when pretreated with Rg1, the translocation of NF-κB was inhibited. B: Bar graphs. Data were presented as mean±S.E.M. of 6 independent experiments. $^*P<0.01$, compared to the control; $^\#P<0.01$, compared to MPP^+ group.

3.4 NF-κB and ROS were involved in the MPP^+-induced up-regulation of DMT1-IRE.

To further confirm the role of NF-κB in the up-regulation of DMT1-IRE in MPP^+-treated cells, BAY 11-7082 (an irreversible inhibitor of IκBα phosphorylation which results in the inhibition of the cytokine-induced NF-κB activation) was chosen in this study. As shown in Fig. 4, DMT1-IRE protein levels in MPP^+-treated MES23.5 cells were significantly up-regulated compared to the control. Pretreated with 2.5 μM BAY 11-7082 could reverse the MPP^+-induced up-regulation of DMT1-IRE in MES23.5 cells, indicating the involvement of NF-κB activation in this process (Fig. 4).

Evidence proved that oxidative stress-induced ROS generation has been implicated in NF-κB activation (Pyo et al., 2008). Therefore, we hypothesized that Rg1 might have the regulatory effect on NF-κB and DMT1-IRE by its antioxidant effect. Thus a direct ROS scavenger of free radicals NAC was used in this study. Results showed 0.5 mmol/L NAC could also prevent the up-regulation of DMT1-IRE. This suggested ROS was also involved in this process.

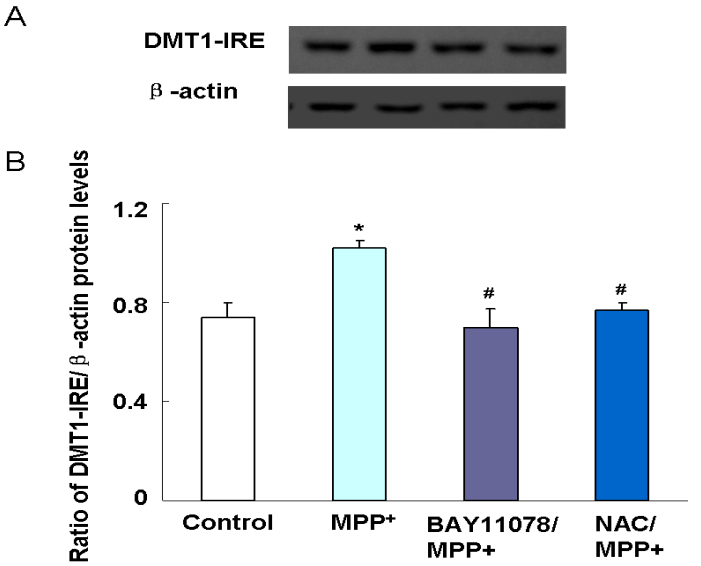

Fig. 4 BAY 11-7082 and NAC inhibited MPP^+-induced up-regulation of DMT1-IRE in MES23.5 cells. A. Western blots were applied to detect the protein levels of DMT1-IRE. Results showed BAY 11-7082 and NAC could abolish the MPP^+-induced up-regulation of DMT1-IRE in MES23.5 cells. B: Bar graphs. Data were presented as mean±S.E.M. of 6 independent experiments. $^*P<0.01$, compared to the control. $^\#P<0.05$, compared to MPP^+ group. $^\wedge P<0.01$, compared to the control; $^\$P<0.01$, compared to MPP^+ group.

3.5 Rg1 attenuated DMT1-mediated ferrous iron influx in MPP^+-treated MES23.5 cells

Up-regulation of DMT1-IRE could enhance iron uptake and increase the intracellular ferrous iron levels. To find out whether Rg1 has neuroprotective effect on DMT1-mediated iron uptake in MES23.5 cells, fluorescence dye calcein was used to measure the ferrous iron influx of MES23.5 cells after perfusing with 1 mmol/L ferrous iron. The fluorescence intensity decreased, indicating the extracellular ferrous iron was transported into cells. Results showed that there was more rapid fluorescence quenching and a significant decrease in the fluorescence intensity in MPP^+-treated cells compared with the control when perfusing with 1 mmol/L ferrous iron. The fluorescence intensity restored to the control level when pretreated with Rg1 (Fig. 5). This indicated that Rg1 could suppress the increased ferrous iron influx mediated by increased expression of DMT1.

3.6 Rg1 prevented increased iron-induced cell damage in MES23.5 cells.

MPP^+-induced up-regulation of DMT1-IRE could increase ferrous iron influx into cells. Excess ferrous iron could react with hydrogen peroxide and produce highly ROS; this aggravated the intracellular ROS levels and induced the dysfunction of mitochondrion. To further investigate if Rg1 could inhibit this damage, we measured ROS production and the mitochondrial membrane potential changes in MPP^+-treated cells followed by iron incubation. The intracellular ROS levels were detected using a fluorescence sensitive probe (H_2DCF-DA). As shown in Figure 6A and 6C, when MPP^+-treated cells incubated with 100 μmol/L ferrous iron, they showed a significant 58% increase in the level of ROS compared to the solely iron treatment. This could be suppressed by Rg1 pretreatment. Then changes in the mitochondrial membrane potential were measured by rhodamine123. As shown in Figure 6B and 6D, there was a significant 27% decrease of $\Delta\Psi m$ in MPP^+-treated cells when incubated with ferrous iron compared to the control. Cells solely

incubated in ferrous iron only showed 16% reduction. Pretreatment with Rg1 for 24 h could inhibit this reduction.

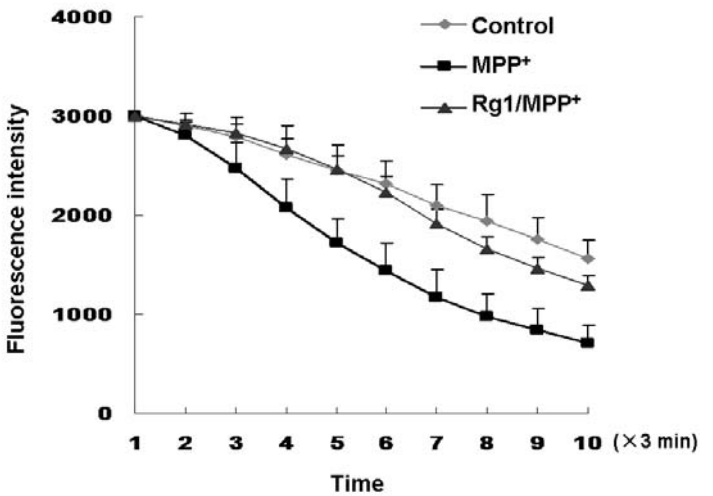

Fig. 5 Ferrous iron influx into MES23.5 cells in different groups. Ferrous iron influx function was detected by confocal using calcein fluorescence. The fluorescence intensity represented the mean value of 35 separate cells from four separate fields at each time point and was presented as mean±S.D. of 6 independent experiments. When perfusing different groups of MES23.5 cells with 1 mmol/L ferrous iron, fluorescence intensity in MPP^+ treated cells decreased more rapidly compared to the control. And there was a significant increase of fluorescence intensity in Rg1 pretreated cells compared with solely MPP^+ treated cells. (Two-way ANOVA, F=29.265, P<0.01, cells treated with MPP^+ compared to the control; P<0.01, cells pretreated with Rg1 compared to cells treated with MPP^+).

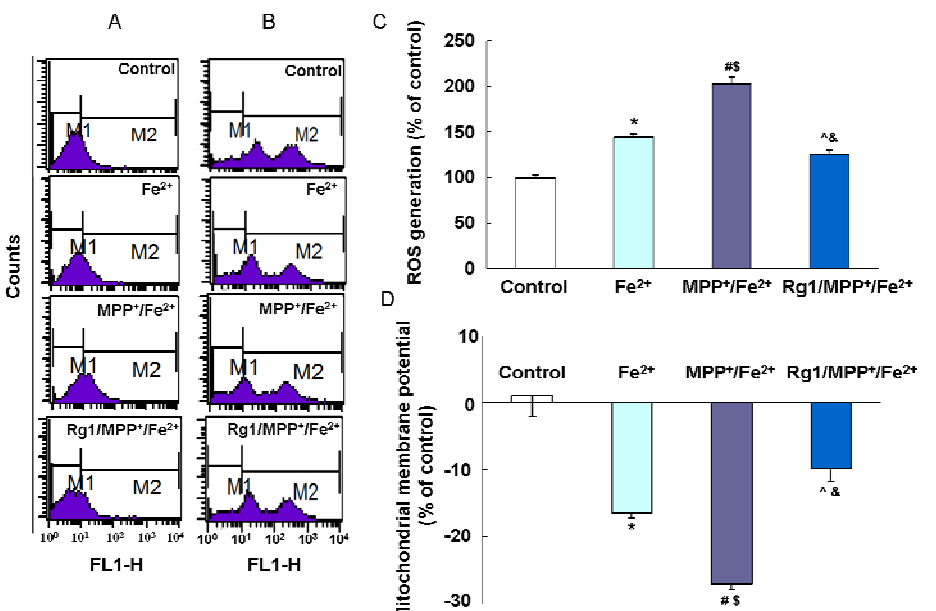

Fig. 6 Rg1 prevented increased iron-induced cell damage including ROS production (A, C) and ΔΨm (B, D) in MES23.5 cells followed by iron incubation. A: Representatives of the fluorometric assay on ROS. There was a significant increase in the levels of ROS when incubated with ferrous iron in MPP^+-treated cells. This increase of ROS could be suppressed

by Rg1 pretreatment. B: Representatives of the fluorometric assay on ΔΨm of different groups. When MPP^+-treated cells incubated in 100 μmol/L ferrous iron, they showed a significant decrease of ΔΨm. Pretreatment with Rg1 for 24 hrs could attenuate this reduction. Statistical analysis was presented as C and D. Data were presented as mean±S.E.M. of 6 independent experiments. Fluorescence values of the control were set to 100%. $^{*}P<0.01$, $^{\#}P<0.01$, compared to control; $^{\$}P<0.01$, $^{\&}P<0.05$, compared to Fe^{2+}; $^{\wedge}P<0.01$, compared to MPP^+/Fe^{2+}.

4. Discussion

This study showed the neuroprotective effects of Rg1 on MPP^+-treated dopaminergic MES23.5 cells and revealed the underlying mechanisms. Our results suggested that Rg1 could attenuate MPP^+-induced up-regulation of DMT1-IRE through inhibition of ROS-NF-κB pathway, resulting in the decreased ferrous iron influx and cell survival.

MPP^+ is a common neurotoxin used as an agent for inducing PD models. It could enter the cell through the dopamine reuptake system, and then inhibit complex I of the mitochondrial respiratory chain, and may induce oxidative stress (Cassarino et al., 1999; Desai et al., 1996). It was reported that the protective effect of Rg1 was mostly attributed to its antioxidant effect acting as a free-radicals scavenger (Chen et al., 2003; Lim et al., 1997; Lopez et al., 2007; Zhong and Jiang, 1997). Therefore, ROS production was first detected to confirm whether Rg1 had protective effect on MPP^+-treated MES23.5 cells. Results demonstrated that Rg1 significantly decreased the MPP^+-induced ROS production. This is also consistent with other previous work that Rg1 could suppress oxidative stress in MPTP-treated PD mice (Chen et al., 2005).

Iron is essential for vital cellular activities and also found throughout the brain for its important role including brain oxygen transport, electron transfer, neurotransmitter synthesis and myelin production. However, due to its highly reactive nature, excess iron could lead to the generation of oxidative stress, which is toxic to cells. Elevated iron levels in the SNpc have been found in PD. However, the underlying mechanisms of iron accumulation in this region are largely unknown. Previous studies showed that DMT1-dependent increase in iron levels played a key role in the death of dopaminergic neurons in PD. G185R mutation that impairs DMT1 iron transport decreases the susceptibility of microcytic mice (mk/mk) and Belgrade rats to MPTP-induced and 6-hydroxydopamine (6-OHDA)-induced neurotoxicity, respectively (Salazar et al., 2008). This convinced the important role of DMT1 and DMT1-dependent iron transport in MPTP or 6-OHDA-induced neurotoxicity. Therefore, DMT1 might be a new pharmacological target to treat PD. Some compounds that could attenuate the expression of DMT1 in the SNpc might have some pharmaceutical value in the treatment of PD.

Our previous study showed Rg1 could substantially attenuate iron accumulation in the SN in MPTP-treated PD mice (Wang et al., 2009). And up-regulation of DMT1-IRE (Divalent metal transporter 1, without iron responsive element) was shown to account for iron accumulation in 1-methyl-4-phenylpyridinium (MPP^+)-treated MES23.5 dopaminergic cells (Zhang et al., 2009). In this study, we investigated whether Rg1 had the protective effect on MPP^+-induced up-regulation of DMT1-IRE. Results showed Rg1 pretreatment could prevent the MPP^+-induced up-regulation of DMT1-IRE, suggesting Rg1 could affect the intracellular iron levels by regulating the expression DMT1-IRE.

However, the mechanisms underlying the

regulation of Rg1 on DMT1-IRE were not understood. The regulating of DMT1+IRE was proved to be dependent on IRE/IRP (iron regulatory protein) system (Lee et al., 1998). However, the regulation of gene expression of DMT1-IRE was not fully elucidated. It is postulated that transcription factors acting on nuclear factor κB (NF-κB) binding sites on the DMT1 promoter may play a role in the expression of the transporter (Huang et al., 2006). NF-κB is constitutively expressed as a cytoplasm p50/p65 heterodimer bound to an inhibitory subunit, IκB (Lenardo and Baltimore, 1989). Exposure of cells to various pathological stimuli activated NF-κB and promoted the translocation of NF-κB from cytoplasm to nucleus, and then regulated the target gene expression (Pahl and Baeuerle, 1997). Paradkar et al. found NF-κB nuclear translocation and the subsequent binding to the putative NF-κB response element (at −19 to −23) within the 1B promoter of DMT1 increased the expression of DMT1 in undifferentiated P19 embryonic carcinoma cells (Paradkar and Roth, 2006a, b). This indicated NF-κB might be involved in the regulation of DMT1 expression in MPP^+-treated cells, and Rg1 might regulate the expression of DMT1-IRE through inhibiting NF-κB activation. Therefore, we detected the protein levels of NF-κB in different group. As we expected, MPP^+ could activate NF-κB translocation from cytoplasm to nucleus. And this could be inhibited by Rg1, indicating Rg1 plays a role in NF-κB regulation and subsequent gene regulation, and this may be the mechanism of neuroprotective activity of Rg1 on DMT1 up-regulation. This is further supported by the application of BAY 11-7082, an inhibitor of IκBα phosphorylation and degradation, which can inhibit the activation of NF-κB. Pretreated with BAY 11-7082 could abolish the MPP^+-induced up-regulation of DMT1-IRE.

We have demonstrated that Rg1 could inhibit the ROS production in MPP^+-treated MES23.5 cells. Then whether the antioxidant effect of Rg1 contributes to its inhibition of NF-κB in MPP^+ treated MES23.5 cells? Evidence proved that oxidative stress-induced ROS generation has been implicated in NF-κB activation (Pyo et al., 2008). Therefore, MPP^+-induced oxidative stress may contribute to increased NF-κB nuclear translocation and binding activity response to MPP^+, and Rg1 might have the regulatory effect on NF-κB and DMT1-IRE by its antioxidant. To confirm this hypothesis, NAC, a free radical scavenger, was used in this study. As we expected, results showed NAC pretreatment led to an efficient inhibitory effect on MPP^+-induced translocation of NF-κB and the up-regulation of DMT1-IRE. This suggests that oxidative stress is involved in up-regulatioin of DMT1-IRE response to MPP^+ through activation of NF-κB, and Rg1 had the ability to regulate the expression of DMT1-IRE by inhibiting ROS-NF-κB pathway.

MPP^+ increased DMT1-IRE expression and enhanced ferrous iron influx in MES23.5 cells. The increased intracellular ferrous iron will interact with hydrogen peroxide to form hydroxyl radicals, which causes the mitochondria dysfunction. We observed increased ROS production and decreased mitochondria membrane potential in MPP^+ treated cells followed by iron incubation. Rg1 showed inhibitory effect on this increased iron influx and iron induced cell damage, indicating that Rg1 could exert its neuroprotective effect though regulating the cellular iron levels and inhibiting iron induced cell damage. This implicated the new pharmacological effect of

Rg1 on iron accumulation.

In conclusion, we reported that the neuroprotective effects of Rg1 against MPP^+ toxicity was through preventing the improper up-reulation of DMT1-IRE, decreasing the cellular iron influx and iron induced cell damage. And this effect may contribute to its inhibitory effect of Rg1 on the production of ROS and the activation of NF-κB. Further studies should be carried out to evaluate whether ginsenoside Rg1 could benefit as a future preventive and therapeutic drug of PD.

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List of Abbreviations

SN, substantia nigra; SNpc, substantia nigra pars compacta; PD, Parkinson's disease; MPP^+, 1-methyl-4-phenylpyridinium; DMT1, divalent metal transporter 1; Nramp2, natural resistance associated macrophage protein 2; IRE, iron responsive element; IRP, iron regulatory protein; NAC, N-acetyl-L-cysteine.

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中国生理学会张锡钧基金委员会关于第十三届全国青年优秀生理学学术论文评选及交流会议第一轮通知（再次刊登）

为鼓励我国青年学者创造性地开展高水平生理学研究，促进我国生理学的持续发展，中国生理学会接受张锡钧教授捐赠，并按照张锡钧教授的生前愿望设置张锡钧基金优秀生理学学术论文奖，奖励在生理学研究中做出突出成绩的、年龄在 40 周岁以下的中青年生理学工作者。张锡钧基金优秀生理学学术论文奖每两年评选一次，以下是张锡钧基金第十三届全国青年优秀生理学学术论文评选工作的通知，请各位中青年生理学工作者按照通知要求积极参与：

一、凡 1975 年 1 月 1 日以后出生的中国生理学会专业工作者或研究生，以其本人为主在国内完成的系列生理研究论文（最好三篇：其中至少两篇已正式发表，一篇已被接受刊登并有证明；或特别优秀的单篇论文）已在国内外公开发表的均可推荐，英文论文务必附详细的中文摘要。

二、候选论文应由两名副教授或副研究员以上本会会员推荐，除全部论文外，需附 1000 字左右的系列论文的综合摘要，上报至所在省市地区生理（科）学会或该会所属生理专业组集中，由省市学会依地区分配的名额（附后）选拔出最优秀的论文及其摘要，连同推荐书一式二份，于 2015 年 9 月 1 日前寄至学会张锡钧基金会。

三、所有被推荐论文的第一作者均将应邀出席第十三届全国青年优秀生理学学术论文交流会，并在会上作报告，由评委对报告的内容、图表制作、表达和答辩能力逐项评分，最后结合会前专家书面评审结果决出名次。

四、按名次评选出的等级包括一等奖 1 名；二等奖 2 名；三等奖 3 名及最佳图表；最佳表达；最佳答辩 3 个单项奖。获得名次的优秀论文获奖者，将颁发奖金、奖状和纪念品。凡未评上名次的青年代表，将颁发优秀论文证书及纪念品。

五、参加者往返旅费和食宿费由所在单位支付。

六、“推荐表”可直接在中国生理学会网站下载。

七、论文交流会将于 2015 年 10 月 24-25 日（23 日报到）在湖北武汉召开，会议报到事宜将另行通知。

联系人：肖 玲
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　　xiaoling3535@126.com
学会网址：http://www.caps-china.org/

中国生理学会

2015 年 4 月 20 日

各省、市、自治区参加第十三届张锡钧基金会全国青年优秀生理学学术论文交流会名额分配（共 51 名）

北京 5 人	天津 1 人	河北 1 人	内蒙古 1 人	山西 1 人
辽宁 2 人	吉林 1 人	黑龙江 2 人	江苏 3 人	浙江 1 人
上海 5 人	安徽 1 人	山东 2 人	江西 1 人	福建 1 人
河南 1 人	湖北 2 人	湖南 2 人	广东 3 人	广西 1 人
重庆 1 人	四川 2 人	云南 1 人	贵州 1 人	陕西 2 人
甘肃 1 人	宁夏 1 人	青海 1 人	新疆 1 人	西藏 1 人
海南 1 人	香港 1 人			

中国生理学会第十一届全国青年生理学工作者学术会议第一轮通知（再次刊登）

为推动我国从事生理学研究的青年学者和研究生的学术交流，促进我国生理学的持续发展和队伍建设，锻炼和培养未来生理学发展的学术带头人，中国生理学会于 1993 年决定举办首届全国青年生理学工作者学术会议，成为我国学术组织青年工作的创举。20 多年来，一代代青年学人从参加青年会议开始与中国生理学会结缘，展示自己的科研成果，逐步成为中国生理学发展和中国生理学会工作的中坚力量。

经中国生理学会常务理事会研究决定，第十一届全国青年生理学工作者学术会议将于 2015 年 10 月 24-25 日（23 日报到）在武汉召开。该会议将与张锡钧基金会第十三届全国青年优秀生理学学术论文交流会同期举行。欢迎从事生理科学及相关领域教学和研究的青年生理学工作者踊跃参会。

一、青年会议征文事项

1．青年会议应征论文的第一作者年龄应是 40 岁以下（1975 年 1 月 1 日以后出生），从事生理学或相近学科科研或教学工作的青年学者、博士后或研究生，也欢迎旅居和留学海外的青年学者。学会建议各单位特别支持在读的硕士生和博士生参加学术交流。

2．应征论文的内容包括生理科学及相关领域的基础研究、应用基础研究和实验技术等方面的学术论文。

3. 征文截稿日期为 2015 年 9 月 1 日。应征论文应在截稿日前未公开发表。

4. 应征稿件请发给：北京大学医学部生理与病理生理学系：马百荟 1219392522@qq.com

5. 会议将从评审通过的口头和墙报交流摘要中评出优秀论文奖各 6 名。

二、论文摘要的书写要求

1. 交流类别：请在左上角注明您希望交流的类别“口头”或“墙报”。

2．论文题目：应明晰地体现论文内容，不宜超过 20 字；用黑体 4 号居中。

3．作者姓名：与论文题目间空一行，用宋体 5 号字居中。

4．作者地址：格式为“（单位 城市 邮政编码）”，除括号外不要标点；不同作者的单位间可用分号“；”隔开，并用“* #”指明作者，用 5 号楷体居中。

5．摘要正文；限 600 字，言简意赅，述明研究的主要目的、方法、结果、结论，摘要中不要图表和文献；正文与作者地址之间空一行，用 5 号宋体。

6．为便于交流，在文摘后请附作者简介：包括姓名、出生年月、工作或学习单位、职务

职称或身份、专业领域、手机、电子邮箱等。

三、会议注册费（含会议期间统一用餐费）

1. 会议注册费在2015年9月1日前为600元，在2015年9月1日后为800元。

2. 注册费请汇至中国生理学会电汇账号：

开户单位：中国生理学会

开 户 行：工商行东四支行

银行帐号：0200004109014480653

3. 请在汇款时注明“姓名+青年会议注册费”，请不要经ATM机汇款，否则很难查收到银行单据。

4. 欢迎不投论文的青年朋友注册并参加会议。注册费与上述相同。汇款后请将姓名、出生年月、工作或学习单位、职务职称或身份、专业领域、手机、电子邮箱等信息发给北京大学医学部生理与病理生理学系：马百荟 1219392522@qq.com。

全国从事生理科学工作的青年朋友们，让我们携起手来，努力办好第十一届全国青年生理学工作者学术会议，共同托起祖国生理科学发展更加辉煌的明天！

中国生理学会青年工作委员会

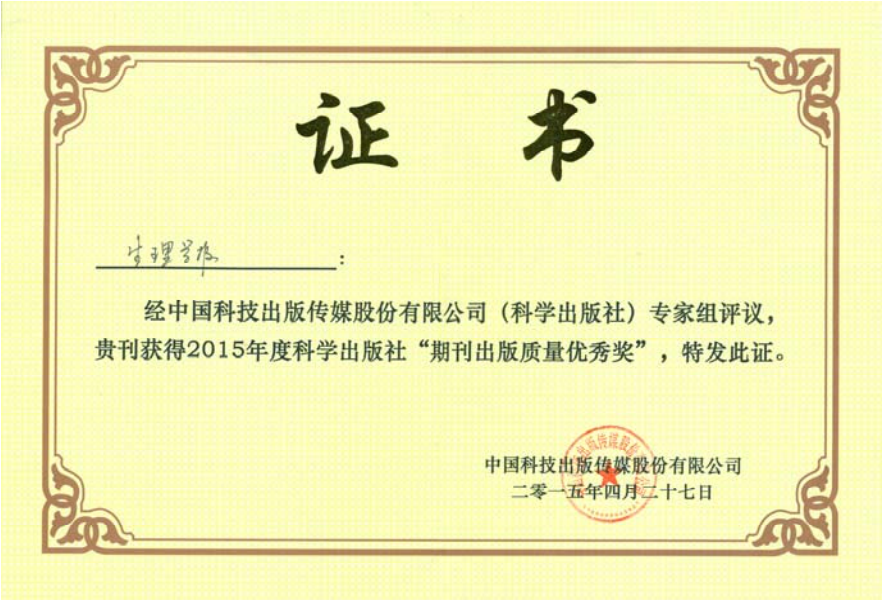
2015年4月21日

表彰奖励

《生理学报》荣获2015年度科学出版社“期刊出版质量优秀奖”

日前获悉，《生理学报》荣获2015年度科学出版社“期刊出版质量优秀奖”。

中国科技出版传媒股份有限公司（科学出版社）经对2013、2014年出版期刊（300余种，不含科学出版社自办期刊）进行严格评议，其中124种期刊荣获2015年度科学出版社“期刊出版质量优秀奖”。


证　书

生理学报：

经中国科技出版传媒股份有限公司（科学出版社）专家组评议，贵刊获得2015年度科学出版社“期刊出版质量优秀奖”，特发此证。

中国科技出版传媒股份有限公司

二零一五年四月二十七日

《生理学报》编辑部

2015年5月15日

讣　告

沉痛悼念印其章教授

中国共产党员，著名生理学专家、教授，原苏州医学院党委书记、院长，中国生理学会终身会员印其章教授，因病医治无效，不幸于 2015 年 6 月 4 日 20:00 分在苏州逝世，享年 89 岁。

谨此讣告

中国生理学会

2015 年 6 月 8 日

通　知

中国生理学会新型生理学实验技术平台培训班通知（再次刊登）

21 世纪，科技创新及科学技术迅猛发展，不断提高科技人员的职业技能，加速培养高层次、复合型高素质人才是时代的迫切需要。生理、药理和病生实验教学合并为机能实验教学在中国已经走过了十几年的道路，培养创新性的实验人才，需要有创新性的实验方法、技术和手段，更好地为实验课程开设提供科学合理的条件。提高实验教学师资队伍的业务素质，使他们在业务和专业技能方面有长足的发展，并在实验教学中发挥示范和带头作用，促进高新技术的应用，不断提高机能学实验这门课程的教学质量。

中国生理学会定于 **2015 年 7 月 27-8 月 2 日**在**安徽合肥**举办**“中国生理学会新型生理学实验技术平台培训班”**。届时将聘请国内具有丰富教学与实践经验的北京大学医学部王韵教授、中南大学湘雅医学院罗自强教授等授课。本次学习班还将展示一些比较先进的教学仪器，学员通过上机实践操作或动物实验可掌握较多的实验新理论和技术。并就如何构建基于 Internet 的网络教学平台和国家级医学虚拟仿真实验教学中心与专家进行讨论。

授课时间：2015 年 7 月 27-8 月 2 日（7 月 26 日报到）

地　　点：安徽医科大学基础医学院

收费标准：1680 元/人，包括教材，实验动物，上机操作。

提前交费和报到时交费均可。汇款请注明是交纳学习班费用。

学会电汇账号：

开户单位：中国生理学会

开 户 行：工商行东四支行

银行帐号：0200004109014480653

课程内容：

理论课程：1.生物医学中心的建立与发展；2.基础医学教学改革的机遇与思考，虚拟实验、人体实验是机能实验发展的方向；3.如何构建国家级医学虚拟仿真实验教学中心，下一代生物机能（集成化、网络化、信息化）实验系统的构想和实现；4.行为学研究设备在医学本科创新教育中的应用；5.Vascular oxidative stress in hypertension and diabetes；6.蛋白组学在心血管疾病研究中的应用；7.巨噬细胞活化在糖尿病肾病发病中的作用；8.遗传和环境因素对肿瘤发生发展的影响；9.神经心理学的机遇--从行为、影像到基因的研究；10.内质网应激在炎症性疾病中的作用；11.铅暴露对大鼠海马神经环路形成的影响和机理研究；12.电压门控钾离

子通道 KCNQ1 受辅助亚基及毒素多肽调控机制的电生理研究。

演示实验：1.膜片钳技术；2.钙成像技术；3.短路电流检测上皮细胞跨膜电位；4.微血管张力测定；5.大鼠离体海马脑片 CA1 区突触后动作电位的记录；6.大鼠在体海马 CA1 区突触后动作电位的记录；7.大鼠血管环张力实验；8.大鼠血流动力学记录；9.大鼠无创血压的测定；10.蛙类在体心肌细胞动作电位的观察；11.VMC-100 虚拟医学院展示（虚拟医学中心软件）；12.行为学实验设备的演示（学习记忆、抑郁类）。

教学实验：1.家兔血压的神经体液调节；2.Langendorff 心脏灌流；3.利多卡因抗心律失常作用；4.几种类型的缺氧；5.呼吸运动的调节；6.家兔失血性休克的复制及救治（微循环观测）。

参加学习班的学员在课程修满经考核合格后将颁发给Ⅰ类继续医学教育学分 7 分。欲参加学习班的老师请认真填写下列回执，并于 2015 年 7 月 20 日前发送电子版至学会电子邮箱（见下），学会将根据报名回执寄发报到通知。

欢迎从事机能实验教学与科研的老师踊跃报名。

联系人：肖 玲

电　话：010－65278802　010-85158602

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中国生理学会

2015 年 4 月 15 日

2015 年“中国生理学会新型生理学实验技术平台培训班”回执

姓 名		性别		年龄		职称或职务		
单 位								
联系地址							邮编	
办公电话						移动电话		
电子信箱								
备 注								

注：回执请于 2015 年 7 月 20 日前发送电子版至 lingxiao12341@sina.com

第四届全国呼吸系统重大疾病转化医学学术论坛会议通知

为进一步推动我国有关呼吸系统重大疾病基础研究和临床研究的多学科交叉融合与相互交流，中国生理学会呼吸生理专业委员会、中华医学会呼吸病学分会哮喘学组决定于 2015 年 9 月 18 日—9 月 20 日在江苏省常州市召开“第四届全国呼吸系统重大疾病转化医学学术论坛”，由中南大学基础医学院、湖南省重大呼吸疾病基础与临床研究实验室协办，常州大学生物医学工程与健康科学研究院承办此次论坛。本次学术论坛邀请从事生理学、病理生理学、药理学、内科学、儿科学、胸外科学以及呼吸力学、呼吸医学工程的同仁，围绕肺发育异常与疾病、呼吸调控与疾病、呼吸系统免疫与疾病、气道高反应与支气管哮喘和 COPD、急性肺损伤与肺保护、肺间质代谢与纤维化、肺循环及肺血管疾病、呼吸衰竭与呼吸支持等呼吸系统疾病的重大问题开展转化医学学术交流。**本次论坛还将特邀多名国内知名专家教授，就当前国内外呼吸疾病研究的重要进展和热点问题，结合自己的研究工作举行**

专题报告。这些专家均承担过多项国家自然科学基金等国家级课题，多数专家担任国家自然科学基金二审专家。这次论坛将是我国呼吸系统重大疾病基础与临床研究的一次学术盛会，其报告内容将反映目前我国呼吸领域的最新研究现状和未来发展趋势。本次会议也是国内呼吸研究领域同行相互了解、寻找合作伙伴的绝佳机会。热忱欢迎各位同仁及学生积极到会参加交流。非常感谢您的支持！

一、**会议主要内容与形式**

会议采用特邀专家专题报告、论坛学术报告、青年论文竞赛等。

二、**正文要求**

(1) 未在国内外公开发行刊物上发表的论（中文和英文两种语言均可，不接受其他语言）。

(2) 大会报告要求提交 1000 字左右摘要，会议论文摘要字数限 800 字以内。

(3) 论文格式为：论文题目、作者、单位(包括所在城市、邮政编码)。内容应包含目的(Objective)、资料与方法(Materials and Methods)、结果(Results)、结论(Conclusion)。以 Microsoft Word 编辑，A4 纸打印。字体格式：（英语）Times New Roman；(中文) 宋体。标题小四号字，正文五号字，行间距 1.5 倍。所有稿件以电子邮件方式投稿，稿件以附件(word 文档)发送至 hnsslkxh@163.com，并附单位名称和联系电话。

(4) 论文文责自负，不涉及保密内容。

(5) 会议期间将组织优秀论文、论文发言和优秀壁报评选，颁发证书。

会议地点：江苏省常州市

会议时间：2015 年 9 月 18 日-2015 年 9 月 20 日（9 月 18 日报到）

会议安排：2015 年 9 月 19 日专题报告及论文报告；2015 年 9 月 20 日上午青年论文竞赛。

会务费：800 元（学生 500 元）。报到时交纳，食宿统一安排，费用自理。

论文投送邮箱：hnsslkxh@163.com

论文摘要截稿日期：2015 年 8 月 10 日

联系人：学术组：冯丹丹（Tel：13975103143，邮箱：fengdandanph@163.com）；会务组（常州）：董进（Tel：0519-86330103、13813585120，邮箱：dongjin_2000@126.com）

主办：中国生理学会呼吸生理专业委员会，
中华医学会呼吸病学分会哮喘学组

承办：常州大学生物医学工程与健康科学研究院

协办：中南大学基础医学院，湖南省重大呼吸疾病基础与临床研究实验室

2015 年 5 月 25 日

全国呼吸系统重大疾病转化医学学术论坛 回执

姓 名		性 别		年 龄		职 称	
单 位						是否参加青年论文竞赛	
邮箱				手 机			

2015年中国生理学会运动生理学专业委员会会议暨“运动与心血管保护”学术研讨会通知

中国生理学会运动生理学专业委员会会议将于2015年9月在陕西师范大学召开，会议将研讨运动生理学在新形势下的发展与建设，同时将举行“运动与心血管保护”学术研讨会。在会上，将邀请国内知名心血管研究领域的专家和与会代表就“运动与心血管保护”等相关领域的研究和最新进展进行学术交流。会议欢迎从事相关领域研究的各位专家学者参加本

次研讨会。本次会议由中国生理学会运动生理学专业委员会（CSEP）主办，陕西师范大学体育学院承办。现将有关事宜通知如下：

一、会议宗旨

促进心血管研究在体育、医学、生物等学科发展，掌握国际及国内该领域研究的热点及方向。为广大相关领域工作者提供一个展示其最新研究成果和讨论当前研究热点的平台，分享先进经验和方法。促进CSEP与国内有关学术组织和机构的学术交流与合作。

二、会议时间

2015年9月18日—20日。 具体为：18日报道，19日全天、20日上午为研讨会时间，20日下午自由活动、晚上离会。

三、会议和报到住宿地点

会场设在陕西师范大学雁塔校区（老校区）重鎏楼（陕西省西安市长安南路199号），学校学术活动中心作为接待宾馆（报到地点：接待宾馆的启夏苑大厅）。

四、主要议题

本次研讨会的主题为“运动与心血管保护”，涉及以下主要议题：

1．运动性心脏结构与功能；
2．运动与心肌微损伤；
3．运动与心肌代谢及信号通路；
4．运动与心血管受体表征；
5．运动与心血管基因网络调节；
6．运动与心血管交感、副交感神经调控；
7．干细胞、细胞因子与心脏保护；
8．表观遗传学与心脏保护；
9．预适应与心脏保护；
10．运动与心血管氧化应激；
11．运动与心血管钙信号调控；
12．运动与心肌内分泌；
13．运动与心血管胰岛素抵抗；
14．运动与高血压；
15．运动与动脉粥样硬化；
16．运动与高血脂；
17．运动猝死防护；
18．运动性心率失常；
19．运动与心血管风险防控新方法、新手段；
20．运动与心脏损伤的流行病学；
21．缺血心脏的运动康复与应用；
22．心功能检测分析与发展；
23．航天医学中的心血管问题；
24．其它相关议题。

五、研讨形式

（一）特邀报告 ：特邀专家报告。

（二）主题报告与口头报告 ：按投稿评审结果安排

（三）书面交流 ：按投稿评审结果安排

六、参加人员

相关领域的科研院校或系教师、科研人员、博士生、硕士生等。

七、摘要要求

（一）出版论文集。选题范围参考研讨会“主要议题”部分，但又不局限于此。

（二）论文请勿涉及保密内容，请作者确保论文内容真实客观，文责自负。

（三）所有论文摘要将刊登在中国知网（CNKI）收藏和检索。凡作者未事先声明，视为已同意授权推荐。

（四）摘要格式要求

论文题目：三号黑体，居中排，文头顶空一行。

作者姓名与作者单位：小三号楷体，居中排，两字姓名中间空一全角格，作者之间用逗号区分。 作者单位换行，按省名、城市名、邮编顺序排列，五号宋体，居中排，全部内容置于括号之中。作者单位与省市名之间用逗号，城市名与邮编之间空一全角格。作者单位多于一个在作者姓名处用上角标注。

关键词：需列出3～5个。“关键词”三字小五号黑体，其他小五号宋体，中文关键词之间用分号。

摘要正文：中文摘要正文总字数严格控制在1500字以内。应征中文摘要必须具有科学

性、先进性、实用性，重点突出，文字力求准确、精练、通顺，分别按目的、方法、结果、结论四部分撰写。摘要正文不得有图表。摘要正文五号宋体通排；行间距为1.5倍。

（五）投稿截止时间：论文报送截止日期为2015年7月30日，大会只接受网上投稿，请将摘要文档发至大会邮箱：csep2002@126.com

八、会议费用

会务费每人为 700元，学生350元（凭有效学生证件）。会务费包括餐费、大会提供的文件袋、论文集等。差旅费和食宿费自理。陕西师范大学学术活动中心作为接待宾馆（报到地点：陕西师范大学 启夏苑大厅），标准间和单间的会议价格为 210-280元/间/天左右（含早餐）。

九、其它

其它未尽事宜，我们将在录取通知中详尽说明。

十、联系方式

联系人：周　越 13693593116
田振军 13572235280
宋　伟 15399425684
鲁文华 13087580629
电 话：010-62989582 ；029-85310156
电子邮件：csep2002@126.com

中国生理学会运动生理学专业委员会
2015 年 5 月 24 日

学会活动

第24届中国生理学会肾脏生理专业委员会2015年度委员会议暨湖北省肾脏科主任高峰论坛通讯

第 24 届中国生理学会肾脏生理专业委员会 2015 年度委员会议暨湖北省肾脏科主任高峰论坛于 5 月 8 日-10 日在湖北武汉召开，此次会议由中国生理学会肾脏专业委员会和华中科技大学协和医院肾内科联合主办，近 200 位来至肾脏领域的基础研究工作者和临床医生参加了会议。

在高峰论坛开幕式上，华中科技大学协和医院肾脏内科主任张春教授致开幕辞，并对与会同仁表示欢迎；湖北省医学会会长朱忠华教授、华中科技大学协和医院副院长黄恺教授分别代表湖北省医学会和华中科技大学协和医院到会祝贺并致辞；中国生理学会肾脏专业委员会主任委员管又飞教授代表肾脏专业委员会对会议召开表示祝贺和致谢。

此次高峰论坛把交流主题设定为基础与临床两大部分。基础部分的交流内容邀请了国内长期从事肾脏生理与疾病机制基础研究的专家到会作专题报告，他们把自己从事肾脏基础研究的成果、经验、进展，前沿和趋势同与会同仁进行了交流；临床部分的内容也邀请了国内长期从事肾脏临床工作的专家作专题报告，临床专家的交流内容涵盖诊断、机制、治疗、进展、存在的问题与困惑等。来自 New York Medical College 的王文辉教授、Emory University School of Medicine 的蔡辉教授，香港中文大学的蓝辉耀教授也参加了本次会议的交流。此次会议还特别设立“临床病理讨论”环节。

中国生理学会肾脏生理专业委员会 2015 年度委员会议是新一届中国生理学会肾脏专业委员会的首次委员会议。28 位现任委员（含 2 位海外委员）出席了此次会议，会议由专业委员会主任委员管又飞教授主持，与会委员就肾脏专业委员会的发展目标进行了讨论，对近期的主要工作进行了部署。会议就以下四方面

的内容进行重点讨论。

肾脏专业委员会组织机构调整工作。按照中国生理学会管理要求和章程的规定，中国生理学会的分支机构“中国生理学会肾脏生理专业委员会”进行了候任主任委员和副主任委员的推举工作，与会委员一致同意推荐复旦大学郝传明教授为候任主任委员，东南大学刘必成教授为候任副主任委员。为加强委员会的内部协调和组织管理，肾脏专业委员会决定设立学术、外事、组织和财务工作小组，并完成各工作组成员的推举工作。关注肾脏生理与疾病机制研究领域青年科研人才的培养和学术成长是肾脏专业委员会的工作宗旨之一，经委员会讨论决定，成立肾脏专业委员会青年工作委员会，旨在为青年工作者搭建平台，加强青年工作者的交流和相互学习，促进青年工作者的学术成长。

经为期两年的努力，中国生理学会肾脏生理专业委员会成功获得了国际肾脏病学会（International Society of Nephrology, ISN）2015 年前沿论坛的承办权，这是 ISN 前沿论坛首次在中国举办。会议将于 2015 年 10 月在深圳召开，这无疑是国内肾脏研究工作者与国际同仁沟通、学习和交流的机会，也是扩大中国肾脏研究国际影响力的机会。肾脏专业委员会专门成立 ISN 会议前沿论坛工作组，负责会议的筹备和组织相关事宜。

首届全球华人肾脏病大会将于 2015 年 12 月在香港举办，中国生理学会肾脏专业委员会是本次大会的协办单位之一，为积极筹划办好这次大会，肾脏专业委员会与会委员也就相关事宜进行了商讨。

经中国生理学会常务理事会讨论通过，肾脏专业委员会着手主办肾脏生理与疾病基础研究领域的专业学术期刊，本次委员会议专门成立了期刊工作小组，并就相关事宜进行了商讨及工作分工，拟本年度出版第一期。

本次会议开得圆满成功。高峰论坛报告精彩、内容丰富、会场讨论热烈。无论从事基础研究还是临床工作的与会人员都表示受益匪浅，是一次成功，高水平的基础与临床的深度交流。委员会议对中国生理学会肾脏专业委员会的努力方向在新一届委员中达成共识，对近期的工作进行了部署，会议实现预期目标。

这次会议得到华中科技大学协和医院肾脏内科的大力支持，全体与会同仁和到会委员会在此表示衷心感谢！

中国生理学会肾脏专业委员会

2015 年 5 月 11 日

中国生理学会消化与营养专业委员会筹建会议新闻

张弘弘　朱进霞　徐广银

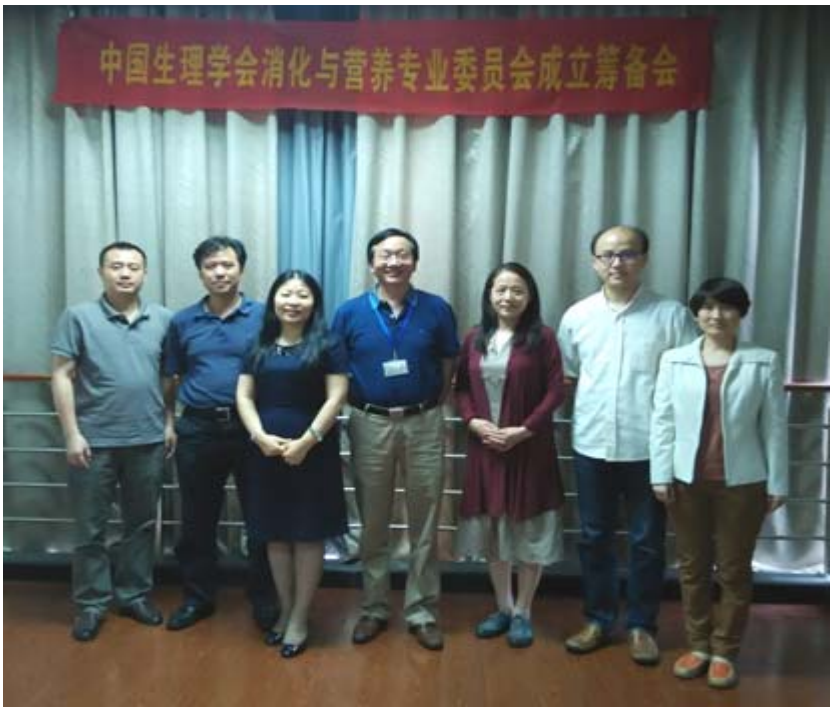
（苏州大学医学部神经科学研究所　苏州　215123）

（首都医科大学生理与病理生理学系　北京　100069）

（苏州大学医学部神经科学研究所　苏州　215123）

中国生理学会消化与营养专业委员会筹建会议于 2015 年 5 月 30 日在苏州大学神经科学研究所成功召开。会议由苏州大学特聘教授、神经科学研究所副所长、消化与营养专业委员会主任委员徐广银主持，参会人员有首都医科大学生理与病理生理学系朱进霞教授，上海交通大学生理学系戎伟芳教授，苏州大学生理与神经生物学系陶金教授，苏州大学附属第二医院内分泌科主任胡吉教授，以及中国生理学会消化与营养专业委员会秘书、苏州大学附属第二医院内分泌科副主任医师张弘弘博士。北京大学生理与病理生理学系张炜真教授因外出开会请假未能出席会议。中国生理学会秘书长王韵教授专程到会给予指导。

本次筹建会议的主要目的是启动中国生理学会消化与营养专业委员会组建工作，初步拟定本年度工作计划和要点。会议商讨了副主任委员拟定人选和委员分配方案，同时商议了有关召开“中国生理学会消化与营养专业委员会成立大会和第一届学术大会”等相关事宜。在经过两个多小时的讨论与部署后，本次筹建会议圆满结束。最后，全体与会人员合影留念。



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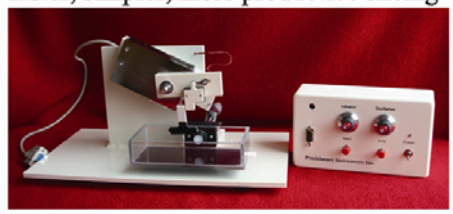

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在微处理器的精准控制下，阀门切换能够在0.01秒内完成。液流在长时间的实验中，能够保持持续的稳定供给。

ALPHA omega
Defining Neuroscience Technology

AlphaLab SnR™

在体多通道神经电生理实验系统

自由活动或受约束的动物皆适用

AlphaLab SnR™在体多通道神经电生理实验系统是目前市场上最先进的大规模神经元动作电采集系统，即将微电极植入大脑中采集神经元放电信号，用于自由活动和受约束动物，并带有同步刺激的数据采集综合工作站。

单个电极

EPS (Electrode Positioning System)
数字型在体电极定位系统

非常灵活的在体研究辅助设备，可用于所有在体研究

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北京新航兴业科贸有限公司

YP100 型压力换能器 测量范围：-50~300mmHg，最大耐压值：<400mmHg
YP100E 型压力换能器 测量范围：-50~300mmHg，最大耐压值：3800mmHg(免定标) 输出：12V/300mmHg/6V
YP200 型压力换能器 测量范围：-50~300mmHg，最大耐压值：<400mmHg(免定标)
YP300 型压力换能器 测量范围：-50~300mmHg，最大耐压值：2000mmHg
YP400 型压力换能器 测量范围：-50~70mmHg，最大耐压值：300mmHg
YP600 型压力换能器 测量范围：-50~300mmHg(无创型)
YP900 型压力换能器（免定标）注射器式 易排气泡 测量范围：-50~300mmHg
JZ100 型张力换能器 测量范围：0-10g 0-30g 0-50g 0-100g 0-300g 0-500g
XH1000 型等长张力换能器 测量范围：0-10g 0-30g 0-50g 0-100g 0-300g 0-500g
XH200 型等长收缩换能器 测量范围：0-3g 0-5g 0-10g 0-20g 0-30g 0-50g
DZ100 型等张力换能器（长度变化）测量范围：±20 mm
XH1000 型痛觉换能器（用于足底刺痛）测量范围：0-100g 0-200g 0-300g 0-500g 0-1000g
HX100 型呼吸换能器（人体胸带式）
HX101 型呼吸换能器（动物捆绑式）
HX200 型呼吸流量换能器（插管式）
HX300 型呼吸换能器（单咀式 连接丫字插管式或动物鼻孔）
HX400 型呼吸功能换能器（人体呼吸波、肺活量等测量用）
HX500 型插管式呼吸波换能器（用于兔子、大鼠、小鼠插气管或插鼻孔）
XH100 型小鼠呼吸实验盒（用于咳嗽药物实验）
WS100 型胃肠运动换能器（用于测量胃肠蠕动）
YL200 型力换能器（用于测量动物某个部位的折断力 最大拉力为 2000g）
CW100 型温度换能器（用于测量动物的肛温 探头为 Ø2×10mm）
CW200 型温度显示测量仪
CW300 型肛温换能器（用于测量动物的肛温，探头为 Ø3×50mm）
CW400 型片式体温换能器（用于测量动物表面体温）
XJ100 型心音换能器（用于人和动物的心音测量）
XJ200 型两用听诊器（用于教学实验 听声音与记录同步）
MP100 型脉搏换能器（用于测量人的指脉）
MP200 型鼠尾脉搏换能器（用于测量大鼠或小鼠的尾脉）
MP300 型腕部脉搏换能器（用于测量人的手腕部位的脉搏）
XH100 型脉诊换能器（用于测量人的手腕部位的脉搏 分析压力与脉搏的关系）
XH101 型恒温式大鼠无创血压测量装置（用于大鼠尾压无创血压测量）
XH200 型恒温式小鼠无创血压测量装置（用于小鼠尾压无创血压测量）
人体血压测量教学套件（用于无创血压测量 由血压表、压力换能器、电子听诊器组成）
其它附件：一维不锈钢微调器、二维微调器、神经屏蔽盒、进口三通、铂金电极、 记滴换能器、电极万向夹
以上产品都能与成都仪器厂、南京美易、成都泰盟、澳大利亚 BLOPAC 等国内外采集系统配套使用。

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