CAAE·青年思行

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中国抗癫痫协会青年委员会

CHINA ASSOCIATION AGAINST EPILEPSY YOUTH COMMITTEE

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在李世绰创会会长追思会上的发言

梁树立

中国抗癫痫协会青年委员会 首都医科大学附属北京儿童医院

尊敬的张阿姨、李妍姐及各位家属，尊敬的各位领导、各位同仁：

今天，我们怀着无比沉痛与不舍的心情，齐聚于此，深切缅怀协会的创始人、我们敬爱的李世绰会长。

3 月 7 日，那个我们始终不愿接受的日子，李会长永远离开了我们。他离开了毕生挚爱、倾尽心血为之奋斗的抗癫痫事业，离开了他始终牵挂、悉心栽培的后辈学子与同仁。他的离去，是国际抗癫痫领域的巨大损失，更是我们心中无法弥补的伤痛与空缺。

作为一名青年医生，我有幸与李会长有过无数次温暖而珍贵的交流。那些点滴过往，如今想来，字字句句都萦绕耳畔、刻进心底。

2008 年，协会送我出国学习，李会长叮嘱我：之所以选派外科医生去学习，是因为外科更需要规范。这句话，我始终铭记在心，时刻不管懈怠。

2024 年，一次晚餐会，他特意让我坐在身旁，勉励我们青年人要有长远眼光、要做正确的事。当我为他夹菜时，他轻声告诉我，身体其实非常难受，几乎吃不下东西，但为了癫痫事业发展、为了支持协会工作，他再难受也要到场。这份担当与情怀，让我至今动容。

2025 年 12 月起，李会长在天坛医院、宣武医院住院治疗，我多次前去探望。看着他日渐虚弱，我满心心疼。常务理事会后我去看他，他说还有好多未竟的事业，特别是癫痫患者的公平就业。最后一次陪他做 CT 时，他已虚弱到需要持续吸氧，说话都很费力，却依然紧紧拉着我的手，眼神里满是执着与期盼。叮嘱我：一定要把青年委员会带好，打造一支国际化的优秀青年抗癫痫团队，既要脚踏实地做好临床，守护好每一位患者，又要仰望星空、敢于创新，为行业发展注入新的力量。

这些嘱托，是前辈对后生的厚望，是长者对事业的坚守，我一刻也不敢忘记。如今，李会长走了，但他的精神永存，他的嘱托我们永远牢记。在此，我代表青年委员会全体成员、代表全体青年癫痫专家，向李会长郑重承诺：我们一定**不忘**

初心、不负嘱托、砥砺前行、勇担使命，拼尽全力完成他未竟的夙愿。2026 年，我们将把国际化建设作为青委会核心工作，努力打造一支让李会长放心、让行业认可的优秀青年队伍。

我们坚信，在周会长和协会领导的带领下，在段老师及秘书处的鼎力支持下，我们必将传承李会长的高尚风范与医者初心，深耕抗癫痫事业、关爱广大患者、全力培育青年人才，圆满完成他交付的使命，不辜负他一生的栽培与期望。

敬爱的李世绰会长，您一生医者仁心、大爱无疆，为抗癫痫事业鞠躬尽瘁。您虽远行，音容宛在，精神永存！愿您在天堂没有病痛，一路走好，安息千古！

在青委会筹备会议合影及首次西部行遵义站合影

长风送行 心灯长明

——纪念中国抗癫痫协会创会会长李世绰教授

林一聪

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可敬又可爱的阳光老会长，永远离开了我们。

天堂再无病痛，愿您安息。

记得第一次见到阳光般温暖的老会长，是在筹备癫痫中心规范化建设工作委员会的会议上。整场交流开放、自由，没有半分居高临下的距离感，却自然流露出长者的笃定与风度。那一刻便知，老会长不仅是学术路上的前辈，更是一位可亲可信的良师益友。

近日我翻遍了与老会长的全部微信，一页页翻看，一幕幕重现眼前：从共同设计癫痫中心调研问卷，到逐条斟酌癫痫中心分级标准的修订；从对我辩论赛发言的由衷赞许，到鼓励我勇敢站上国际会议的讲台；从审慎打磨“北斗计划”中国际视野课程的课件，到满怀兴致地分享初试 ChatGPT 的惊喜与思考……字句之间，仿佛老会长仍在身侧。那种对中国抗癫痫事业的赤诚热爱、对晚辈的倾心栽培，历经岁月，依旧温润有力。

老会长常跟我们年轻人讲他独特而丰饶的人生旅程。从西北黄土高原的厚土，到南太平洋岛国的海风；从旧时农村的土炕，到法国顶级酒店的灯火。岁月如梭，沧海桑田，他的初心从未更改，那份情怀始终扎根于中国抗癫痫事业的沃土之上。走过世界，看过万象，他仍回到这里，把一生的心力与智慧，默默浇灌在这份救疾济困的事业上。视野宏阔、精神丰沛、脚步遍及四方，却又始终扎根一处，这是老会长独有的气象与格局。

即便在生命的最后时光，老会长的心仍牵挂在倾注了他无尽热情的工作上。他挂念着国际癫痫中心评价体系的推进，挂念着中国癫痫中心分级体系的完善；兴致盎然地设想“痊愈之后”要做的事，第一件、第二件、第三件……细数未尽的心愿。面容日渐消瘦，昔日洪亮的声音渐渐远去，可思维依旧敏锐，目光依旧澄澈，话语依旧一语中的。我深信，倘若光阴再慷慨十年，这位智者必能把未来

的画卷铺展得更辽阔、更精彩。

斯人已逝，风范长存。老会长的笑颜与教诲，会如一盏温厚的灯，照亮我们前行的路。作为晚辈，唯有铭记这份深情与嘱托，接过前辈手中的薪火，承续未竟之志，守正拓新，笃行致远，为中国抗癫痫事业竭尽所能，方不负您一生所托。

愿您安息，我们会在路上，替您看见那更美好的春天。

灯塔不灭，薪火相传

——敬悼李世绰会长

谭泊静

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惊闻李世绰会长仙逝的消息，作为中国抗癫痫协会青委委员，北京抗癫痫协会理事，心中满是难以言喻的悲痛与不舍。在八宝山告别，也许是绵绵细雨，湿了眼眶；在北展追思，总感觉眼角微痒，扭头瞥见擦拭泪水，再也无法控制泪水流淌，那是无法抑制的思念。这位我国抗癫痫事业的奠基者、中国抗癫痫协会创会会长、北京抗癫痫协会发起人首任会长，用毕生心血为千万癫痫患者点亮生命之光，更以长者的仁爱与远见，为我们后辈筑牢成长的根基。他的离去，是行业的巨大损失，但那座温暖而坚定的“灯塔”，永远照亮着我们前行的航程。

初识李会长，是在第三届 CAAE 国际癫痫论坛上。彼时初入行业，对临床工作、学科发展与社会服务的平衡满是困惑。是他循循善诱的开导：“青年是发展的未来，要做好眼前事，把每一个病人诊好，把每一项基础工作做实，多抬

头看看远方，路自然就越走越宽。”这句朴实的叮嘱，如春风化雨，驱散了我心头的迷雾，也成为我此后职业生涯中始终坚守的信条。

李会长一生心系青年，提携后学不遗余力。即便耄耋之年，他依然活跃在行业一线，亲临青年学术会议分享从医历程，为基层癫痫中心建设悉心指导，用国际视野为青年医生搭建成长平台 。在他的推动下，中国抗癫痫事业实现跨越式发展，学科建设日臻完善，人才培养体系不断健全，而他始终以长者之爱，默默守护着年轻一代的成长。他常说，医学不仅是科学，更是人文关怀，这份初心与担当，深深烙印在每一位受他教诲的青年心中。

斯人已逝，精神永存。李会长用一生践行了医者的使命与担当：牵头完成全国大规模神经系统疾病流行病学调查，摸清癫痫防治需求；推动成立中国抗癫痫协会，搭建全国防治网络；斩获国际抗癫痫联盟终生成就奖，让中国经验走向世界。他开创的事业，我们必将薪火相传。此刻化悲痛为力量，我将铭记李会长“做好眼前事，深耕抗癫痫路”的嘱托，以他为榜样，秉持仁心、精进医术，在抗癫痫事业的航程中坚定向前。愿李会长一路走好，灯塔不灭，薪火永续！

悬壶济世

“悬壶济世”的典故出自《后汉书·方术列传·费长房》。费长房，“汝南人，曾为市掾。”此人见一老翁，老翁素日里售药于人，将一壶挂在门前。这位老人被后世称为“壶翁”，据说他的壶里有药丸，用以普济病患。费长房曾见过老翁在人群散尽后跳进壶中，便备以酒食、多次拜访老翁，询问奥妙。老翁邀其共入于壶，见壶中“玉堂华丽”，别有洞天。后费长房随老翁入深山修行学习，出山后，长房已成为可治疗诸多疾患的“方士”。

此一传说具有神秘色彩，然而从古籍的文本中，我们已能读到“药”与“医疗众病”等字眼。及今世，我们常以“悬壶济世”指代医者疗疾行医的行为。

---摘自国家中医药博物馆

山河同念

——追忆李世绰会长

丁瑶 王爽

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三月的风，仍带着初春的凉意。

当李世绰会长离世的消息传来，一时让人难以相信。那位总是精神矍铄、言语从容、眼中闪着光芒的老人，仿佛永远站在讲台上、会场中、学术讨论的中心。可如今，他却悄然远行。中国癫痫防治事业失去了一位奠基者，也失去了一位始终为患者点灯的人。

曾无数次聆听李会长的讲课。从全球癫痫的疾病与经济负担，到癫痫相关猝死等重大问题，他总能以开阔的视野和清晰的逻辑，把复杂的医学与公共卫生议题娓娓道来，语气总是平和，但却蕴含着高屋建瓴的洞见，常常令人深受启发。令我印象最为深刻的，是最后一次听他回顾自己的人生与事业。那场讲述平静而真诚，也让我不由得翻阅了李会长的自传《悬壶济》。读罢不禁感慨：李会长的一生，不只是学者的一生，更是公共卫生事业推动者的一生：他早年从基层卫生工作起步，后来进入国家卫生管理体系，曾任卫生部国际合作司司长，并在世界卫生组织担任助理总干事，他很好的发挥了他的人格魅力，大力推进了卫生领域的中外交流。这样的经历，使他拥有宽广的国际视野和深厚的公共卫生情怀。也正因为如此，他始终将中国癫痫防治事业置于全球视野之中思考与推进，在连接中国与世界的过程中，为我国癫痫防控事业的发展开辟了更为广阔的道路。

在他的推动与全球同行的共同努力下，癫痫问题逐渐被提升到全球公共卫生的重要议题。2015 年，第 68 届世界卫生大会通过决议，首次系统关注全球癫痫负担；2020 年，世界卫生大会再次通过《2021-2031 年癫痫和其他神经系统疾病跨部门全球行动计划》。这些重要进展的背后，都体现了李会长始终坚持的理念——癫痫不仅是医学问题，更是公共卫生与社会问题。

对许多地方学会和基层医生来说，李会长不仅是学界的领路人，更像一位温和而坚定的长者。

在浙江，许多人都清楚地记得他的身影：

2013 年，他赴温州参加温州医学会神经病学年会，发表题为"中国癫痫防控事业的格局与未来"的学术讲座；2015 年，又专程赴浙江台州讲学，作"全球癫痫负担及国家层面协调应对其卫生、社会与公众知识影响的必要性"专题报告。没有华丽的辞藻，却视野宏阔。他从全球公共卫生角度讲述癫痫的疾病负担、社会歧视以及健康体系的责任，让许多医生第一次意识到，癫痫防治不仅仅是临床治疗，更是一项需要政府、社会与公众共同参与的系统工程。

2016 年，浙江省抗癫痫协会成立时，李会长亲临杭州。那一天的会场并不宏大，却格外庄重。对浙江的癫痫学界而言，这不仅是一场成立大会，更是一种象征——中国抗癫痫事业的火种，正在各个省份生根发芽。李会长在会上说，建立学会的意义，不只是学术交流，更重要的是把医生、患者与社会连接起来，让更多患者得到规范治疗。这句话，至今仍被许多人记在心里。

2018 年，他又来到浙江大学医学院附属第二医院，参加脑电图培训基地的评审工作。那时他年事已高，但依然认真听取汇报、细致询问教学与临床细节。

有人记得，他在讨论时反复强调一句话：“人才培养，是事业最重要的基础。”正是这样的坚持，使得许多年轻医生在他的鼓励与指导下逐渐成长起来。

这些往事历历在目，李会长从不只是站在宏大的历史舞台上。他更愿意走进具体的地方、具体的医院、具体的人群之中。无论是学术会议的讲台，还是基层

医院的走廊，他都乐于与医生、患者和学生交谈。那是一种真诚而朴素的情怀——把一件事情做好，并且让更多人一起把它做好。

许多与他接触过的人，都记得他的风度。他说话时总是娓娓道来，很少看稿，却条理清晰；他既有外交家的沉稳，也有学者的谦逊。更重要的是，他总能把不同背景、不同专业的人凝聚在一起。有人说，学术界像一个小江湖，而李会长却能让各路专家彼此信任、共同合作。这种能力，既来自他的格局，也来自他的胸怀。

而在工作之外，他也是一个真实而温暖的人：他爱旅游、爱摄影，珍视家人，也格外关心晚辈。每当刷到李会长的朋友圈，总能感受到一种积极而从容的力量——既有对事业的热情，也有对生活的热爱。那一张张风景照片、几句简短的话语，常常让人感到满满的正能量。正因为如此，他在人们心中不仅是一位德高望重的学者，更是一位亲切可敬、令人倍感温暖的长辈。

如今，李世绰会长虽已远去，但他开创的事业仍在延续。中国抗癫痫协会从初创时的筚路蓝缕，成长为深具影响力的学术组织；各地癫痫中心相继建立，基层防治网络日趋完善；越来越多患者得以接受规范治疗，走出疾病投下的阴影。这些变化，正是他当年播下的种子。

斯人已逝，精神长存。

在中国癫痫防治事业的历史长河中，李世绰教授不仅是一位开拓者，更是一盏照耀大家前行的长明灯，它的光芒依然会在照亮后来者的道路，指引大家穿越许多困难。我想，对李会长最好的纪念，就是秉持他对事业的执着，学习为人宽和包容的精神，把我们的抗癫痫事业做好！

灯火不灭，薪火相传

——追忆李世绰会长与基层抗癫痫事业

郭崇伦

CAAE 青年委员会浙皖赣大区第一、二届青年委员

南昌大学第三附属医院（南昌市第一医院）

三月十三日，北京，八宝山东礼堂。

春寒料峭，细雨如丝。我伫立在送别队伍里，看着眼前缓缓移动的人流，喉头哽咽。数天前，那个消息如晴天霹雳——我们抗癫痫事业的掌灯人、中国抗癫痫协会创会会长李世绰教授，于三月七日永远离开了我们。来自全国的神经内外科及癫痫学界的同仁齐聚于此，送李会长最后一程。

三月十四日上午，在协会组织的追思会上，听着周东会长、创会理事们及李会长的同学、好友和家人们回忆他的往事，讲述创建协会初期的筚路蓝缕，我的思绪一次次被拉回到那些与李会长为数不多的珍贵接触中。通过大家点点滴滴的讲述，我不仅感受到一个从基层奋斗出来、最后站上世界卫生讲坛的高大形象，更感受到他作为行业领袖却又平易近人的超凡人格魅力，以及他对下属、对同事、特别是对青年人的关爱和扶持。作为一名从基层走出来的医生，我虽然与李会长的接触不算太多，但有幸在职业生涯的关键节点，得到了他的亲自指点与勉励。

初识印象：情系基层的学术大家

2005 年 6 月，受导师张明教授指派，我到北京协和医院参加"脑电图与临床癫痫诊治新进展"学习班。此后，我经常参加中国抗癫痫协会的学习班及学术，从而认识了李会长，当然，当时他还不认识我。李会长给我的第一印象是能力全面——不仅学术造诣深厚，英语也非常出色。我印象最深刻的是他对基层的深厚感情，比如他在学术演讲中展示的一张照片：在云南，他与省市县乡村各级癫痫医生在癫痫患者家门口合影，照片中还有患者养的一条狗。我记得他不止一次说过，这是他"最值得显摆"的一张照片。

近距离接触：慈祥长者的关怀

自从 2013 年 9 月入选首届中国抗癫痫协会青委会后，我与李会长接触的机会多了起来。在我印象中，只要李会长在国内，他都会亲自参加我们的青委会年

会。

2014 年 5 月，在北京举行的 CAAE 青年委员会"西部行"活动启动会上，我第一次有机会近距离站在李会长身旁。那时，作为一个基层青年医生要承办中国抗癫痫协会西部行活动，我心里还是忐忑不安的。李会长在启动仪式后注意到了这一点，特地走到我身旁，仔细询问遂川县人民医院的具体情况——有多少床位、脑电图设备是什么型号、有多少青年医生愿意投身癫痫专业。

当他听说我们是县级医院，正在积极开展癫痫诊疗工作时，老人家眼睛发亮，拍着我的肩膀说："小同志，从基层做起好啊！癫痫防治的难点在基层，希望也在基层。你们年轻人敢想敢干，一定要做好癫痫诊治工作，造福一方百姓！"也许是为了鼓励我，李会长特意与我单独合影。那一刻，我感受到了长辈对青年医生的真切关爱。那不是高高在上的指示，而是发自内心的期许与鼓励。

云端对话：难忘的勉励

2021 年 12 月 14 日，丁玎教授主持了一场特别的线上讲座，邀请远在澳大利亚的李会长为青年医生们讲述个人成长经历和中国抗癫痫事业的发展历程。那天，年届八旬的李会长端坐镜头前，整整两个小时，从红寺卫生院到世界卫生组织，从协会 2005 年创建的艰辛讲到目前中国抗癫痫水平的现状，他丰富的人生阅历让我们大开眼界。

在讲座的提问环节，李会长谈到，详细介绍自己的经历，主要是想告诉青年医生如何给病人解除病痛，希望大家看清自己努力的方向，不管遇到什么困难，不管环境如何变化，不管工作职务有什么调动，一定要坚定不移地走下去。当我提问"基层医生如何成长"时，李会长语重心长地说："基层工作的医生确实有自己的困难，因为基层的医疗设备、检查设施不可能那么健全，不可能有大医院或者大癫痫中心那样的条件。但只要全心全意地投入到这项工作，加强自身学习，关注国内国际学术动态，与上级癫痫中心和专家建立联系，加强沟通，向他们学习，就能使自己的工作不断提高。关键还是自己的志向和努力。"

透过镜头，他深情地说："我一辈子从基层来，知道县医院、乡镇卫生院的苦。你们青年医生在一线，要耐得住寂寞，守得住初心。"屏幕前的我热泪盈眶。那种被理解、被看见的温暖，让我深知自己选择的道路没有错。

在这次网络会议中，我向李会长发出邀请，希望他到我负责创建的遂川县人

民医院癫痫中心来指导。李会长答应，回国后只要走得动，一定来看看。那时，遂川县人民医院的癫痫中心已成功获批中国抗癫痫协会首批一级癫痫中心（全国共 64 家）。此后，在医院的支持和同事的不断努力下，2023 年 12 月，中心又获批组建吉安市癫痫临床医学研究中心。2024 年 12 月，中心通过了市相关部门的中期检查，已获得市县相关部门 40 万元的资金支持。目前，癫痫中心已添置了一台能同时完成多导睡眠监测的进口视频脑电图仪和脑电图质控软件，诊疗人次已超过 5000 人次，已开展癫痫的基因诊断、MRI-Harness 序列等影像诊断及新型抗癫痫发作药物治疗等新技术。

多少次，我想邀请李会长到井冈山来看看——这里是革命老区，也是我院开展癫痫事业的精神高地。我想带他看看我们县医院的癫痫中心，看看那些从山区赶来、经过规范治疗后重获新生的病友，看看我们为基层癫痫防治搭建的三级诊疗网络。我想当面告诉他：您当年拍我肩膀的嘱托，我们都做到了。

但因为疫情，因为李会长日益繁忙的学术事务，更因为老人家年事渐高、行动不便，这个愿望终究未能实现。如今，这成了永远的遗憾。

灯火不灭，薪火相传

三月十四日，返赣的高铁上。

怀中紧抱着协会赠送的纪念品——李会长的个人传记《悬壶记》。翻开扉页，那些沉甸甸的文字如重锤击心。从河北沙河的一个普通农家子弟，到乡镇卫生院的院长，再到县医院、市卫生局、北京市神经外科研究所、北京市卫生局，直至卫生部国际合作司司长、世界卫生组织助理总干事……

读到他在乡镇卫生院时期，白天看病、晚上挑灯夜读医学外文；读到他在进修时，为了弄懂一个神经解剖问题，泡在图书馆直到闭馆；读到他在 WHO 任职期间，依然心系国内癫痫防治，利用假期回国调研农村癫痫患者的生活状况……高铁穿过一个个隧道，光影明灭间，我仿佛看见那个从基层一路走来的身影，始终未曾忘记为何出发。

李会长在《悬壶记》中写道："出身平凡，基层起步，环境常改，专业屡变，竭尽全力，不断攀登。"这二十字，正是他一生的写照，也是激励我从县医院走到今天的精神灯塔。

如今，我已离开遂川，在南昌大学第三附属医院（南昌市第一医院）老年医

学科任职，但仍兼任遂川县人民医院癫痫中心主任、吉安市癫痫临床医学研究中心主任。虽然今后工作的重点将转向老年病友，但那些年在遂川县人民医院创建癫痫中心的日子，那些承办"西部行"活动的经历，始终是我职业生涯中最珍贵的底色。解除更多基层患者的病痛，仍是我的初心。每当看到那些曾经每月发作十几次、如今已能正常上学务工的病友，我就想起李会长当年拍我肩膀时的温度。

车过长江，暮色四合。我望向窗外渐亮的灯火，想起追思会上协会宣布即将成立"李世绰基金"、召开"李世绰教授癫痫学术思想研讨会"的消息。老人家走了，但那盏灯不能灭，他的精神永远照耀我们前行。

作为一名从基层走出的癫痫医生，我或许永远无法达到李会长那样的学术高度和国际视野，但我可以守住他最深情的牵挂——把基层抗癫痫事业做下去。正如数年前他在网络会议中所表达的愿望：无论是在南昌，还是在吉安，还是在祖国大地的千山万壑间，让规范化的癫痫诊疗触手可及，让每一位病友都有尊严地生活，让每一位青年医生都能看到成长的希望。

灯火不灭，薪火相传。李会长，您放心吧。从乡镇卫生院到世界卫生组织，您用一生诠释了什么是"悬壶济世"；而今，我们接过这盏灯，一定让它在基层的土壤中继续燃烧，照亮万千癫痫病友的前行道路。

待到明年杜鹃花开满井冈山时，我们还会再来看您，用癫痫中心的诊疗数据告诉您：基层的抗癫痫事业，后继有人。

谨以此文，送别敬爱的李世绰会长。

一盏灯 一行路 一辈情

---深切缅怀中国抗癫痫协会阳光老会长李世绰先生

张春青

CAAE 青年委员会川渝滇黔藏大区 陆军军医大学新桥医院

惊闻李世绰先生与世长辞，噩耗传来，整个癫痫领域沉浸在无尽的悲痛之中。作为在先生悉心关怀、谆谆教诲、全力扶持下成长起来的晚辈医者，久久无法平静。过往与先生相处的点点滴滴，如电影般在脑海中回放：他温和的笑容、恳切的话语、严谨的态度、温暖的关怀，清晰如昨，历历在目。先生一生深耕医学、心系患者、栽培后学、治家严谨，用毕生心血奠基中国抗癫痫事业，用仁心大爱温暖无数医者与患者。如今先生虽已远行，但他的精神风范、教诲嘱托、人格魅力，永远镌刻在我们心中，成为照亮医学道路、指引人生方向的不灭明灯。

李世绰先生是中国抗癫痫事业当之无愧的开拓者、奠基人与领航者。他毕生投身神经疾病防治、卫生事业管理与国际医学交流合作，从临床一线到管理岗位，从国内深耕到国际发声，每一步都走得坚定而扎实，每一份付出都饱含对医学事业的赤诚与担当。2005 年，先生牵头创建中国抗癫痫协会，结束了我国癫痫领域缺乏全国性专业组织的历史，为全国癫痫防治工作搭建起权威、专业、温暖的平台，让万千癫痫患者有了依靠，让无数癫痫领域医者有了“家”。

回顾我的学习与工作经历，李世绰先生的身影始终相伴。印象深刻的是，研究生时期撰写论文，发现我国癫痫发病率的数据报道不一，从千分之七到百分之一均见于学术刊物。数值虽然看似差别不大，但是考虑到我国巨大的人口基数，便是近 400 万的偏差，因此一直揣着这个问题，始终想得到一个准确的数字。直到 2009 年李世绰先生主编出版了《中国癫痫预防与控制绿皮书》，指出我国癫痫发病率为千分之七，使得我们在撰写论文、项目书时有了依据与底气。后来才知道，李世绰先生虽然是我国神经外科的前辈，但是先生的研究生论文是神经系统疾病的流行病学调查工作，曾牵头在全国 7 市 22 省开展 30 万人群神经系统疾病流行病学调查，获取了中国癫痫流调的第一手珍贵资料。此后，先生陆续主编了《临床诊疗指南·癫痫病分册》、《癫痫患者经济负担蓝皮书》等一系列权威

著作，系统规范癫痫诊疗标准，普及癫痫防治知识，为我们一众后学筑牢了学术根基。

先生对青年医生的关爱，不是流于形式的鼓励，而是实实在在的指引、真真切切的扶持、无微不至的关怀。他常说，青年医生是医学事业的未来，是抗癫痫事业的希望，一定要用心培养、全力支持，让他们站稳脚跟、快速成长、勇担重任。已经不能清晰回忆是哪年初见李世绰先生，可能是国际癫痫论坛，也可能是中抗年会，那个时候，先生是学界泰斗，我们则是青年学生，只能在会场仰望。印象中与先生第一次长时间“线下”接触是我参与承办 2016 年 ASEPA-ANZAN 脑电图培训班。作为亚太地区癫痫领域学术盛会 ASEPA-ANZAN 会议汇聚了国内外众多顶尖专家学者，学术氛围浓厚而热烈。已过古稀之年的李世绰先生，不顾舟车劳顿专程赴会指导。重庆的夏天热情似火，先生身着正装，精神矍铄，面容慈祥，眼神温和而坚定。反复叮嘱我们注重会议细节，安排专家小班课辅导的流程，同时强调了与外籍专家交流的要点，满是慈爱、期许与鼓励，没有一丝疏离与威严。更让我们感动的是，会议间隙，先生特意抽出宝贵时间，召集我们几位青年医生与国际抗癫痫联盟（ILAE）亚澳区事务委员会 Andrew Bleasel 教授、John W.Dunne 教授以及来自马来西亚、印度的专家一同座谈，询问我们的临床工作、科研进展、学习困惑和国际交流情况，认真倾听我们在癫痫诊疗、学术研究、职业发展中遇到的难题与迷茫。

没有居高临下的说教，没有空洞乏味的讲话，先生是一位慈祥的长辈，用深厚的学术素养、开阔的国际视野，为我们注入思想力量。可能是有感于我们磕磕巴巴的英文专业词汇和战战兢兢的外宾座谈，李世绰先生格外叮嘱我们：“中国抗癫痫事业要发展，必须紧跟国际前沿，你们一定要沉下心来学好英文，主动参与国际学术交流，学习先进理念、技术与经验，再结合中国患者的实际情况，走出属于我们自己的抗癫痫之路”。此外，我还清晰的记得，先生鼓励我们癫痫领域大有可为，要敢于申报各类学术奖项，用成绩证明自己的努力与实力。

先生的话语朴实无华，却字字千钧、直击人心，如春雨般滋润我们的心田，如明灯般照亮我们前行的道路。正是在先生的鼓励、支持与悉心指导下，我备受鼓舞、倍感振奋，更加坚定了在癫痫领域深耕不辍、精益求精的决心。我始终牢记先生的教诲，把对患者的责任、对事业的热爱，融入每一次临床诊疗、每一台

手术操作、每一项学术研究中。在临床工作中，我坚守医者仁心，用心对待每一位癫痫患者，耐心倾听他们的病痛，精心制定诊疗方案，尽全力为他们解除痛苦、重拾希望；在学术道路上，我勤奋钻研、勇于探索，积极参与国内外学术交流，努力提升专业素养与科研能力；在协会工作中，我担当作为、履职尽责，用心投身 CAAE 青年委员会与外科专委会的各项工作，传承先生的育人精神，助力更多青年医生成长。

在工作中，李世绰先生严谨务实、精益求精，是令人敬仰的行业领航者；在生活中，他治家严谨、家风淳厚，是重情重义、热爱生活的温情长者。我时常能在先生的朋友圈，看到他分享的家人幸福时光：或是与老伴相伴漫步的温馨瞬间，或是与儿孙团聚的欢乐画面，或是节日里阖家团圆的幸福剪影，没有华丽的辞藻，没有刻意的修饰，只有最朴实、最真挚的情感流露。字里行间，满是对家人的疼爱、对家庭的珍视、对生活的热爱。

那些温暖的文字、温馨的照片，让我们看到了先生褪去学界泰斗光环后，最真实、最柔软、最温情的一面。他治家严谨，注重家风传承，用言传身教教会家人真诚待人、踏实做事、坚守本心、懂得感恩；他珍惜家庭温暖，把工作与生活平衡得恰到好处，用爱守护着家人的幸福，用责任诠释着为人夫、为人父、为人长辈的担当。先生常说，一个人只有经营好家庭、心怀温情，才能更好地肩负起社会责任，才能以更饱满的热情、更仁爱的心怀对待患者、对待事业。他的言传身教，如春风化雨、润物无声，让我们深刻懂得，行医先做人，做人先修身，修身先齐家，唯有坚守本心、心怀大爱、珍视亲情，方能行稳致远、无愧于心。

与先生相处的岁月里，他的一言一行、一举一动，都彰显着高尚的人格魅力与医者仁心。他一生淡泊名利、甘于奉献，始终把患者放在首位，把中国抗癫痫事业的发展放在心上，从不计较个人得失，默默耕耘、无私奉献；他待人真诚、谦和有礼，对待前辈敬重有加，对待同辈真诚相待，对待晚辈关怀备至，用博大的胸怀温暖着身边每一个人；他治学严谨、精益求精，对待学术一丝不苟，对待工作认真负责，用专业与坚守诠释着医者的使命与担当；他心怀大爱、心系患者，始终关注癫痫患者的生存现状，积极推动社会关爱，为患者争取权益、消除偏见，用仁心守护着万千家庭的幸福。

先生的一生，是为医学事业奋斗的一生，是为癫痫防治奉献的一生，是为青

年后辈引路的一生，是为家庭尽责、为世人表率的一生。他用毕生热忱与坚守，点亮了中国抗癫痫事业的火炬；用渊博学识与高尚品格，铸就了行业丰碑；用温厚善良与无私大爱，温暖了无数心灵。他不仅是中国抗癫痫事业的领航者，更是我们青年医生的精神榜样，他的风范、他的教诲、他的情怀，将永远激励着我们砥砺前行。

如今，先生悄然离去，中国抗癫痫事业失去了一位奠基人、领航者，我们失去了一位良师、一位益友、一位长辈，医学界失去了一位德高望重、仁心仁术的前辈。回想与先生相处的点点滴滴，他温和的笑容、恳切的嘱托、严谨的态度、温暖的关怀，依旧萦绕在心头，从未远去。我们悲痛于先生的离去，更感恩于先生的付出与栽培，铭记着先生的期望与嘱托。

斯人已逝，风范长存；薪火相传，初心不改。作为受先生恩泽、被先生栽培的青年一辈医者，我们唯有化悲痛为力量，继承先生的遗志，传承先生的精神，沿着他开辟的道路坚定前行。我们将坚守医者仁心，精进医术、厚德笃行，用心守护每一位癫痫患者的健康与尊严，不辜负先生对我们的殷切期望；我们将秉持先生的学术精神，勇于创新、不断探索，深耕癫痫诊疗与科研事业，推动中国抗癫痫事业不断迈向新高度。

一盏灯，照亮前行之路；一行路，坚守医者初心；一辈情，铭记先生风范。李世绰先生，您从未远去，您的精神永远与我们同在，您的风范永远激励我们前行。愿您在天之灵安息，我们定不负您的期望，将您毕生致力的中国抗癫痫事业发扬光大，以告慰先生的在天之灵！

李世绰先生，我们永远怀念您！

追忆老会长：温厚如灯，步履如炬

郭强

CAAE 青年委员会粤桂琼港大区　广东三九脑科医院癫痫中心

与李会长相识的十余年间，诸多片段常萦绕心头，他是治学严谨的前辈，是提携后辈的长者，更是心怀大爱、步履不停的医者、斗士，其温厚之姿、睿达之思，如明灯照路，让我辈青年感念至深，久久难忘。

“青年是八九点钟的太阳，是行业的未来，更是抗癫痫事业的主力军。”

2012 年韩国仁川举办癫痫年会（KEC），我有幸与姜玉武老师、王艺老师等一同，随李会长带领的中国专家团队远赴海外交流。会后晚餐席间，李会长召集我们畅谈，首次向我们袒露了成立中国抗癫痫协会青年委员会的构想。言语之间，满是对青年医师成长的殷切期许，更有对中国抗癫痫事业长远发展的深谋远虑。尤其是对姜玉武老师委以重托，反复叮嘱这份事业的初心与方向。为了这份期许，他为青委会定下了创新与研究、横向合作、交流沟通、走向国际的发展方向，高屋建瓴的指导，为彼时的我们指明了前行的道路。这份从构想到落地的行动力，这份心系青年、擘画行业的格局，让我深深动容。2013 年，青委会应势成立，姜玉武老师任首任主任委员。十余年间，一代又一代青年医师循着指引阔步前行。

“咱国内不少领域不做则已，一做就过热，大家一哄而上，一定要谨防过度医疗，让技术健康发展。”

2015 年，我院在广州承办立体定向脑电图与脑定位国际论坛，海内外顶级专家齐聚一堂，李会长的一番致辞，至今仍让我记忆犹新。他先是肯定了国内学者在 SEEG 领域不懈探索的努力。话锋一转，李会长说了上述那番话，他以行业长者的睿智，温和却恳切地提醒着在场众人，言语间满是对行业发展的清醒与思虑。彼时国内 SEEG 技术正处于发展上升期，这份清醒的提醒，如醍醐灌顶，让在场的我们深知，技术发展的初心是为患者谋福祉，唯有理性克制、稳步前行，方能让技术真正服务于临床，这份对专业的敬畏、对行业的坚守，成为我此后行医治学的重要标尺。

“我问个比较外行的问题呀。”

2019 年，我院承办第三届国际癫痫病理学习班，虽因疫情防控临时转为线上，却在李会长的大力支持下，收获了超出预期的规模与效果。开幕式致辞后，李会长笑着向我抛出一个问题：“我问个比较外行的问题呀，现在癫痫病理有没有到电子显微镜水平？如果没有，是为什么？”彼时的我，一时语塞。这个问题看似浅显，细想之下却一点也不“外行”—— 现代神经病理诊断早已从常规病理，直接迈入分子病理与组学病理（基因组学、蛋白组学、代谢组学）的新阶段，恰恰绕过了电子显微镜水平下的超微结构病理研究。彼时的李会长，已身居行业高位，却依旧在具体亚专业领域保持着孜孜不倦的学习与思考，这份对知识的渴求，对专业的执着，让我们晚辈深感汗颜。我随即向病理科专家请教答案，再向他反馈时，他认真聆听，与我细细探讨的模样，至今历历在目。那一刻，我真切懂得，真正的学者，永远心怀谦卑，永远在探索的道路上步履不停。

“你要坚持做下去，让更多人重视癫痫猝死的风险。”

近些年，我尝试做新媒体癫痫科普，希望能用通俗的语言，让更多人了解癫痫、规范化诊疗，本是一份朴素的初心，却没想到常能得到李会长的关注与鼓励。他会饶有兴致地翻看每一条科普内容，偶尔还会与我交流看法，这份对

新生事物的包容，对科普事业的支持，让我备受鼓舞。记得有一次，我分享了一位女大学生患者，本可通过积极治疗控制病情，却因一次突发的癫痫发作猝死（SUDEP）离世的消息，李会长看到后，满是痛心，特意私信鼓励我，要坚持做下去，让更多人重视癫痫猝死的风险，更呼吁国内尽快组织发起多中心关于 SUDEP 的研究，为更多患者筑起生命的防线。寥寥数语，藏着他对患者的悲悯，对医学事业的责任，也让我更加坚定了做科普的初心。

“昨儿的男排，可惜赢不了。”

2025 年 6 月 28 日国际癫痫关爱日，活动结束后，我护送李会长返回酒店，彼时他已行动不便，大多时候坐着轮椅。路上他忽然问及，昨天是否看了凌晨五点中国男排对阵巴西的比赛。我诧异于他年事已高，仍有这般兴致与精力，坦言未看后，他虽稍显失望，却依旧饶有兴致地与我谈起比赛细节。那一刻，他褪去了行业领路人的光环，如一位亲切的长辈，有着自己的热爱与欢喜，那份鲜活与温暖，让我至今想起，仍觉心头温热。

青委十余载岁月，从仁川的一席畅谈，到论坛上的谆谆提醒，从病理学习班的谦卑探问，到科普路上的暖心鼓励，李会长的身影，早已深深印在我辈青年医师的心中。他以高瞻远瞩的格局，为中国抗癫痫事业擘画未来，搭建青委会平台，托举青年一代成长；以睿智清醒的专业素养，洞悉行业发展规律，警醒后辈谨防技术浮躁，坚守医疗初心；以孜孜不倦的探索精神，始终保持对知识的渴求，躬身治学，步履不停；以悲天悯人的医者仁心，心系患者安危，关注科普事业，为癫痫患者的健康奔走呼吁；更以温厚谦和的长者风范，平等对待后辈，真诚分享热爱，如春风一般，温暖着身边的每一个人。

如今，李会长虽已远行，但他为我辈留下了巨大的精神财富，指引青年医师们继续前行。我们必将以李会长为榜样，带着他的期许与嘱托，坚守行医治学的初心，心怀大局，心怀大爱，心怀大为，在抗癫痫的道路上稳步前行，以点滴努力，让他为之倾注一生的事业生生不息，薪火相传，不负他的厚爱，不负医者的使命。

会长，请一路走好！

读《悬壶记》有感

——以初心赴使命，以坚守护安康

孙林琳　　姚丽芬

CAAE 青年委员会黑吉辽大区　哈尔滨医科大学附属第一医院

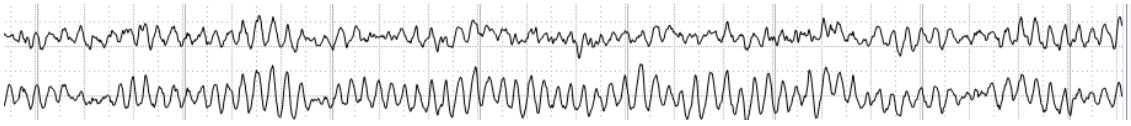
细读李世绰会长的《悬壶记》，字里行间满是一位医学家的济世情怀与责任担当。作为哈尔滨医科大学附属第一医院神经内科癫痫亚专业的医生，我自 2006 年起便深度参与中国农村地区癫痫防治管理项目，同时也多次参加中国抗癫痫协会的各项学术活动，如今身为中抗理事，更是亲身见证了李会长为中国癫痫防治事业付出的心血与坚守。书中关于中国抗癫痫协会创立与中国农村地区癫痫防治管理项目的记述，令我倍感亲切与振奋，也让我更加明晰了自身所肩负的行业使命，读懂了“悬壶济世”四个字的千钧重量。更难忘的是，李会长多次莅临黑龙江，作为陪同亲身感受了老会长平易近人、谦逊温和，毫无大家架子，其人格魅力与专业担当，深深影响着我从医路上的每一步。

中国农村地区癫痫防治管理项目，是李会长用初心浇灌的民生之花，更是中国癫痫防治史上的里程碑式实践，其意义远超医疗本身。作为深度参与该项目十余年的从业者，我深知在项目起步阶段，农村医疗资源匮乏、癫痫认知极度落后的困境——相当一部分癫痫患者被困在乡村，因贫困和缺医少药，承受着病痛与歧视的双重折磨。李会长带领老一辈癫痫领域专家目睹这一切后，摒弃了高端学术的“象牙塔”，扎根基层推出了“低成本、可及、可推广”的防治模式，将诊疗服务下沉到县、乡、村三级医疗网，用廉价有效的药物、常态化的随访，为农村患者点亮了希望之光，而我也有幸成为这份希望的传递者，亲身参与到基层癫痫患者的诊疗与随访工作中。这个项目的意义，不仅在于让数十万农村癫痫患者得到规范治疗、控制发作，更在于打破了“癫痫是不治之症”“抽风是鬼神附体”的愚昧偏见，让科学的诊疗理念走进田间地头。它用最接地气的方式，破解了农村癫痫防治的痛点难点，被世界卫生组织誉为典范，更让“医者仁心”不再是一句口号，而是化作实实在在的守护，惠及最需要帮助的底层群体，为后辈们树立了“面向基层、服务患者”的榜样。

中国抗癫痫协会的创立，则为中国癫痫防治事业搭建了坚实的发展平台，奠定了行业规范化发展的根基。作为中抗理事，我多次在协会学术会议上感受到行业发展的蓬勃活力，也深知上世纪八九十年代，国内癫痫诊疗乱象丛生，缺乏统一的诊疗标准、专业的交流平台，患者误诊误治现象普遍，行业发展举步维艰。正是在这样的困境中，李会长联合多学科专家挺身而出，历经重重困难发起成立协会，扛起了推动行业发展的大旗，而协会也成为了我们癫痫领域从业者交流学习、共同进步的核心平台。

协会的创立，结束了中国癫痫领域“各自为战”的局面，通过制定诊疗指南共识、开展科普宣传、搭建学术交流桥梁、推动国际合作，让癫痫诊疗走向规范化、专业化。更重要的是，协会推动设立“6·28 国际癫痫关爱日”，全力消除社会对癫痫患者的歧视，为患者营造了更包容的环境，也为我们后辈提供了学习、成长、交流的平台，推动中国癫痫防治事业逐步与国际接轨。

李会长用一生践行了“悬壶济世”的誓言，他的坚守与付出，不仅改变了无数癫痫患者的命运，更照亮了中国癫痫防治的前行之路。作为后辈，我有幸追随李会长的脚步，参与农村癫痫防治项目、投身协会各项工作，也亲身感受过他的平易近人与专业坚守。未来，我将继续以李会长为榜样，铭记初心，传承担当，深耕癫痫亚专业、扎根基层，用专业能力守护患者安康，用责任与坚守，续写中国癫痫防治事业的新篇章。



薪火映冰城 仁心照龙江

——追忆李会长与黑龙江癫痫事业的深情羁绊

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春寒未褪，冰城仍留余霜，我们却永远失去了那位为中国抗癫痫事业点亮明灯的领路人——李世绰会长。2026 年 3 月 7 日，这位深耕神经疾病防治数十年的奠基者溘然长逝，享年 84 岁，留给世间无尽的思念与未竟的牵挂。在黑龙江这片广袤的黑土地上，他的足迹遍布哈尔滨的医院诊室、佳木斯的基层医疗机构，他的话语温暖了无数癫痫患者的心灵，他的坚守为龙江癫痫事业的崛起铺就了坚实道路。作为中国抗癫痫协会（CAAE）新的青年一代，我们循着他的足迹，追忆他与龙江癫痫事业的点点滴滴，读懂一位医者的赤诚与担当，传承那份跨越山海的仁心与坚守。

黑龙江，作为我国癫痫患病率较高的省份之一，曾面临着诊疗资源匮乏、规范水平不足、患者认知滞后的困境，约 30 万癫痫患者在病痛与偏见中艰难前行，其中农村地区患者因经济负担重、就医不便，更是陷入“看病难、治不起 ”的绝境。上世纪 80 年代，李世绰教授在全国开展 30 万人群神经系统疾病流行病学调查时，便将目光投向了这片黑土地，龙江患者的疾苦，从此成为他心中难以割舍的牵挂。那时的他，已是神经流行病学领域的先驱，却始终保持着“ 出身平凡，基层起步 ”的自勉，一次次踏上北上的列车，穿越冰雪，奔赴龙江大地，用脚步丈量这片土地上的癫痫防治之路。

冰城哈尔滨，是李会长与龙江癫痫事业结缘的重要起点，也是他推动区域诊疗规范化的前沿阵地。他深知，癫痫防治的关键在规范，核心在人才，根基在基层。2011 年在李会长的大力支持下，哈尔滨医科大学附属第二医院成功承办 CAAE 癫痫分类与诊治国际研讨会；2018 年 7 月，第三届 CAAE 全国癫痫外科学术盛会在哈尔滨隆重举行，作为中国抗癫痫协会创会会长，李世绰先生亲临现场致辞，为龙江癫痫外科事业的发展指明方向。彼时，他已年过七旬，却不顾旅途劳顿，全程参与学术研讨，与来自国内外的专家学者、龙江本地医护人员深入交流，探讨癫痫外科术前评估、手术技术的最新进展，为哈尔滨乃至黑龙江的癫痫诊疗搭建起高水平的学术交流平台。他反复强调：“癫痫不是‘不治之症 ’，更不是‘ 中邪 ’，规范诊疗能让七成患者症状得到控制，我们要让每一位龙江患者都能享受到科学的治疗，让他们重获尊严与希望。” 2024 年李会长更是心系龙江癫痫事业发展，为黑龙江省抗癫痫协会副理事长单位哈尔滨医科大学附属第二医院朱延梅教授团队颁发万众 e 心奖。

李会长的身影被许多医护人员深深铭记。他多次前往院内指导癫痫中心建设，从学科布局、人才培养到诊疗规范制定，每一个细节都亲力亲为。2021 年 11 月，黑龙江省医学会脑电图专科联盟暨哈尔滨医科大学神经病学博士论坛举行，李会长再次亲临致辞，鼓励龙江青年医护人员深耕癫痫领域，脚踏实地钻研技术，用专业力量守护患者健康。他常常与青年医生促膝长谈，分享自己在美国国立卫生研究院研修的经历，传授流行病学调查的经验，叮嘱大家“医者仁心，既要懂医术，更要懂患者”。在他的感召下，一批批龙江青年医护人员投身癫痫防治事业，龙江综合癫痫中心不断发展壮大，现已成立两家集临床、教学、科研为一体的国家级综合癫痫诊治中心，为龙江患者提供了优质的诊疗服务。

黑龙江的广袤大地，不仅有冰城的学术盛会，更有基层的迫切需求。李会长深知，龙江地域辽阔，基层医疗机构的癫痫诊疗水平直接关系到千万患者的福祉。他始终关注基层防控，积极推动“癫痫的社会控制“项目在龙江落地，助力搭建六级防治网络，让优质医疗资源下沉到县乡。佳木斯作为黑龙江东部的医疗重阵，李会长曾多次前往佳木斯指导工作，为中心的建设出谋划策，鼓励团队坚守初心，克服困难，让基层患者在家门口就能享受到规范诊疗。他牵挂着农村癫痫患者的经济负担，呼吁推广廉价有效的治疗方案，推动农村癫痫防治项目落地，让更多贫困患者能免费获得药物治疗，摆脱病痛的折磨。

在龙江的岁月里，李会长用行动诠释着“为国为民，永不停息“的誓言。他

走遍了黑龙江的主要城市与重点乡镇，每到一处，都要深入诊室查看患者、指导诊疗，与医护人员交流经验，向群众普及癫痫防治知识，破除“癫痫传染”“治不好”的偏见。有医护人员回忆，李会长待人谦和，没有丝毫架子，面对患者的咨询，总是耐心细致地解答，用温和的话语缓解他们的焦虑；面对基层医护人员的困惑，他倾囊相授，毫无保留地分享自己的经验与智慧。他曾说：“癫痫防治事业不是一个人的战斗，需要一代又一代人的坚守与传承，龙江的癫痫事业有很好的基础，有一群有情怀、有担当的医护人员，我相信未来一定会越来越好。”

作为国际癫痫领域的领军人物，李会长用国际视野为龙江癫痫事业赋能。他推动龙江癫痫诊疗与国际接轨，鼓励本地医护人员参与国际学术交流，支持我们考取国际脑电图资质证书，提升专业水平。在他的推动下，龙江的癫痫诊疗逐渐走向规范化、国际化，越来越多的患者走出病痛的阴霾，重新拥抱生活。他荣获国际抗癫痫联盟（ILAE）和国际癫痫病友会（IBE）“终生成就奖”，成为中国首位获此殊荣的学者，这份荣誉的背后，也凝聚着他对龙江癫痫事业的深情付出。

如今，李会长已离我们而去，但他播下的希望种子，早已在龙江大地上生根发芽。学术平台的日益完善，基层诊疗网络的不断健全，青年医护人员接续奋斗，龙江癫痫患者就医环境不断改善，这一切，都离不开李会长的远见卓识与无私奉献。他用一生践行医者使命，用仁心温暖万千患者，用坚守传承医学薪火，他的精神，如冰城的星光，如龙江的沃土，滋养着一代又一代 CAAE 青年前行。

作为新时代的 CAAE 青年，我们追忆李会长，不仅是缅怀他的功绩，更是要传承他的初心与担当。我们要铭记他“竭尽全力，不断攀登 ”的追求，深耕癫痫防治领域，脚踏实地钻研技术；我们要传承他“心系基层，关爱患者 ”的仁心，扎根岗位，用专业力量守护每一位患者的健康；我们要延续他“薪火相传，勇担使命 ”的信念，接过他手中的接力棒，为龙江乃至全国癫痫事业的发展贡献青年力量。

冰城依旧，薪火相传；仁心永驻，精神长存。李会长与黑龙江癫痫事业的深情羁绊，将永远镌刻在我们心中。他的音容宛在，他的精神永存，指引着我们在癫痫防治的道路上，不忘初心，勇毅前行，用青春与奋斗，续写中国抗癫痫事业的新篇章，不负他的嘱托，不负时代的使命。

薪火永继，风范长存

——深切缅怀李世绰会长

王剑虹

CAAE 青年委员会黑吉辽大区 复旦大学附属华山医院神经内科

惊闻中国抗癫痫协会创会会长李世绰先生与世长辞，我悲痛难抑。作为一名深耕癫痫领域的青年医者，先生于我，是引路明灯，是宽厚长者，更是毕生追随的精神标杆。

与先生相识相知的点滴，此刻清晰浮现。2020 年全国抗癫痫年会上，我怀着赤诚之心登台朗诵，诉说青年一代传承抗癫痫事业的决心。话音刚落，先生便投来赞许目光，会后特意走到我身边，温声鼓励：“你们年轻人有情怀、有担当，中国抗癫痫事业的未来，就靠你们了。”这句肯定，是我前行路上最珍贵的力量，时刻提醒我不忘初心、坚守使命。

曾有幸随先生一同出国参会，亲眼见证他的双重风采。学术场上，他与国际抗癫痫事业的先驱前辈畅谈行业发展，目光坚定、言辞恳切，以开阔视野为中国癫痫学科走向世界铺路搭桥，尽显学科领路人的格局与担当；闲暇间隙，他褪去学者光环，化作慈祥长辈，细心为孙辈挑选礼物，眉眼间满是温柔与疼爱。这份严谨治学与温情待人的完美融合，让我读懂了何为“大医精诚”。

先生毕生以仁心护佑万千癫痫患者，以赤手搭建中国抗癫痫事业的根基，用一生践行“为患者谋福祉”的誓言。他的离去，是中国抗癫痫领域不可估量的损失，但他留下的精神火种，必将生生不息。

作为后辈，我定将铭记先生教诲，传承他的医者初心与家国情怀，在癫痫诊疗与患者关爱之路上勇毅前行，以实干告慰先生在天之灵，让中国抗癫痫事业薪火相传、蒸蒸日上。

李世绰先生，一路走好！

编号：EYX-2026-1-1

引用格式：Xiao L, Qin L, Jiang T, Qu M, Hou M, Tang Y, Hu S, Feng L. Neuroimmune activation in temporal lobe epilepsy patients with worsening seizure following the COVID-19 pandemic: A [18F]DPA-714 PET/MR study. Sci Adv. 2026;12(5):eadu5874. doi:10.1126/sciadv.adu5874

共同通讯：冯莉

Abstract

Patients with temporal lobe epilepsy (TLE) frequently experience worsening epilepsy following COVID-19, referred to as post-COVID-19 active TLE. While neuroinflammatory changes are suspected in these patients, measurements of both central and systemic inflammation in the brain remain unexplored. We investigate whether the translocator protein standardized uptake value ratio (TSPO SUVr), a quantifiable marker of neuroinflammation using positron emission tomography (PET), is elevated in the brains of patients with post-COVID-19 active TLE. In addition, we examine correlations between TSPO SUVr and inflammatory factors to identify potential peripheral blood inflammatory predictors of post-COVID-19 active epilepsy. Our study highlights the presence of widespread neuroinflammation in the brain and increased levels of inflammatory cytokines in the plasma of individuals with post-COVID-19 active TLE. Furthermore, strong correlations between plasma levels of interleukin-1β (IL-1β), IL-10, and interferon-γ (IFN-γ) and neuroimmune activation suggest the potential for integrating plasma inflammatory factors with TSPO PET as a dependable approach for clinical diagnosis, dynamic monitoring, and assessment of immune-based therapeutic efficacy in TLE-associated neuroinflammation.

编号：EYX-2026-1-2

引用格式：Wang J, Du Y, Su G, Tang W, Long X, He Y, Feng L. Dual-functional pH-sensitive nanoliposomes modified with CD47 mimicry peptide enhance icariin delivery to attenuate neuroinflammation and oxidative stress in epilepsy. Mater Today Bio. 2025;35:102528. doi: 10.1016/j.mtbio.2025.102528

共同通讯：冯莉

Abstract

Around 30 % of patients fail to achieve seizure-free with existing anti-seizure medications (ASMs), and developing drugs that target neuroinflammatory and oxidative stress may benefit patients with refractory epilepsy. The bioactive compound icariin (ICA) isolated from the traditional Chinese medicine Epimedium has strong anti-inflammatory and antioxidant effects. Nonetheless, the clinical application of ICA is restricted by its low bioavailability and limited ability to penetrate the blood-brain barrier (BBB). This research developed a pH-sensitive nanoliposome delivery system modified with CD47 mimicry peptide(ICA@LipD-CD47) to enhance the therapeutic efficacy of ICA for epilepsy. Through the immune escape mechanism of CD47 mimicry peptide, the phagocytosis of the drug-loaded nanoparticles by macrophages could be reduced, thereby facilitating the prolonged circulation of ICA. Additionally, considering the acidic microenvironment present in the epileptogenic foci, pH-sensitive liposomes could enhance the efficiency of ICA's targeted entry into the epileptogenic foci. In this study, network pharmacology, animal experiments, and transcriptomics analysis confirmed the effect of ICA in alleviating epilepsy-induced inflammation and oxidative stress. Moreover, ICA@LipD-CD47 demonstrated prolonged circulation and effective targeting of epileptogenic foci, significantly alleviating neuronal damage and cognitive dysfunction in epileptic mice. As a novel nano-delivery system, ICA@LipD-CD47 presents a promising approach to improve the therapeutic effectiveness of ICA for treating epilepsy.

编号：EYX-2026-1-3

引用格式：Xiong J, Duan H, Chen J, You X, He F, Zhang C, Yang L, Chen C, Deng X, Yang L, Mao L, Wang G, Chen S, Zhang W, Yin F, Xiao Z, Peng J. Integrated genotype-phenotype function analysis reveals distinct pathogenic mechanisms for cognitive impairment in KCNQ2-related disorders. Epilepsia. 2026. doi: 10.1002/epi.70075

共同通讯：彭镜

Abstract

Objective: Pathogenic variants of KCNQ2 lead to a spectrum of disorders including self-limited familial neonatal-infantile epilepsy (SeL(F)NIE), developmental and epileptic encephalopathies (DEEs), and neurodevelopmental disorders (NDDs) with intellectual disability (ID). This study aimed to delineate the clinical progression and underlying pathogenesis of these disorders. Particularly, we unraveled the role of gain-of-function (GoF) variants in neurodevelopmental impairment. **Methods:** We conducted a longitudinal study of a 90-patient Chinese cohort with KCNQ2-related disorders, classified into SeL(F)NIE, DEEs, and NDDs subgroups. Clinical phenotyping was integrated with functional analyses (electrophysiology, biochemistry) of five missense variants in homomeric and heteromeric (with Kv7.3/Kv7.5) channel assemblies. **Results:** Despite comparable seizure control to SeL(F)NIE (96% vs 100%), the NDDs group exhibited significant cognitive impairment. All deceased patients (8/90) were in the DEEs group. Functional profiling revealed a spectrum from loss-of-function (LoF) to GoF. LoF variants (e.g., S247L in DEEs) were linked to severe epilepsy. Crucially, we identified strong GoF variants (P335A/L in NDDs) in the S6-HelixA domain that confer insensitivity to phosphatidylinositol 4,5-bisphosphate (PIP2) regulation and are associated with a primary neurodevelopmental phenotype, distinct from the severe epileptic phenotype of established voltage-sensing domain (VSD) GoF variants. **Significance:** Our integrated clinical and functional analysis showed that the clinical outcome relies on the functional consequence of a KCNQ2 variant (LoF vs GoF) and its behavior in heteromeric complexes, rather than its mere location. We defined a novel class of strong GoF KCNQ2 variants that are mechanistically and phenotypically distinct, highlighting aberrantly enhanced channel function as a key driver of cognitive dysfunction and presenting new targets for precision medicine.

编号：EYX-2026-1-4

引用格式：Chen S, Quan Y, Yin F, Peng J. Clinical and genetics spectrum of 392 Chinese patients with genetic epilepsy with febrile seizures plus. J Neurol. 2025;273(1):18. doi: 10.1007/s00415-025-13544-9.

通讯作者：彭镜

Abstract

Background: Genetic epilepsy with febrile seizures plus (GEFS+), an inherited epilepsy syndrome, is characterized by broad genotypic and phenotypic heterogeneity, with causative genes remaining unidentified in approximately 70% of cases. The aim of this study was to explore the clinical and genetic profile of GEFS+. **Method:** We retrospectively analyzed the genotypic spectrum and clinical characteristics of 133 GEFS+ families. Probands were divided into two groups based on their degree of fever sensitivity to compare the phenotypic and genetic differences. A PPI analysis was conducted to declare the interaction of involved genes. **Results:** 392 affected patients were identified from 133 GEFS+ families. FS (288/392, 58.2%) and FS+ (70/392, 17.9%) consisted of the majority of phenotypes. Other phenotypes included FS/FS+ with generalized seizures (21/392, 5.4%), FS/FS+ with focal seizures (8/392, 2.0%), Dravet syndrome (8/392, 2.0%), afebrile GTCS (17/392, 4.3%), complex phenotypes (5/392, 1.3%) and unclassified seizures (35/392, 8.9%). Patients who were mildly sensitive to fever were more likely to have focal, myoclonic, and tonic seizures, and have two or more seizure types, whereas patients being highly sensitive to fever were more inclined to have only one type of seizures ($P < 0.05$). Meanwhile, patients being mildly sensitive to fever have more probability to require antiseizure medications ($P < 0.05$). WES or WGS was undertaken in 127 families. 83 variants of 43 different genes (31 P/LP variants and 52 VUS variants) were identified in 78 GEFS+ families, with a diagnostic yield of 23.6%. The most commonly implicated genes were predominantly voltage-gated channels genes (SCN1A, SCN1B, KCNT1, CACNA1A, CACNA1H), and GABA receptor-related genes (GABRA1, GABRB2, GABRB3, GABRG2). Venn diagram analysis showed that voltage-gated channel genes distributed across different fever-sensitivity groups, while GABA receptor-related genes were more frequently in high- and moderate-sensitivity groups. **Conclusion:** The phenotypic spectrum of GEFS+ was broad and FS and FS+ constituted the main phenotypes. Multiple types of seizures and intellectual disability/developmental delay (ID/DD) were more likely to occur in children with low fever sensitivity. Sodium voltage-gated channel genes, especially SCN1A, and GABA receptor-related genes were frequent in the genotypic

spectrum of GEFS+. GABA receptor-related genes, but not voltage-gated channel genes, were closely related to fever-sensitivity.

编号：EYX-2026-1-5

引用格式：Chen B, Chen S, Xiong J, Kessi M, Peng J, Yin F, He F. The integration of plasma non-target metabolomics and lipidomics analysis for the discovery of global developmental delay/intellectual disability biomarkers. Front Cell Neurosci. 2026;20:1688339. doi: 10.3389/fncel.2026.1688339

通讯作者：何芳

Abstract

Background: This study aimed to identify metabolic signatures and potential biomarkers for global developmental delay (GDD) and intellectual disability (ID) using plasma metabolomics and lipidomics. The research sought to evaluate the feasibility of these methods for early identification and to explore the underlying metabolic pathways associated with GDD/ID. **Methods:** A liquid chromatography coupled with tandem mass spectrometry (LC–MS/MS)-based method integrated with multivariate data analysis was employed to comprehensively characterize plasma metabolomics and lipidomics profiles in children diagnosed with GDD/ID compared to typically developing (TD) children. The study focused on identifying distinct metabolites and lipids that could differentiate GDD/ID from TD children. **Results:** The analysis revealed that a combination of 11 metabolites and lipids could effectively discriminate between GDD/ID and TD children. Receiver operating characteristic (ROC) analysis identified several potential biomarkers for GDD/ID. In positive ion mode, glycerophosphocholine (AUC = 0.899) and sphinganine (AUC = 0.859) showed diagnostic potential. Negative ion mode analysis revealed five biomarkers, notably 2-ketohexanoic acid (AUC = 0.912) and N-acetyl-L-aspartic acid (AUC = 0.870). Lipidomics analysis highlighted two high-performance biomarkers: diacylglycerol (DAG) (16:0/16:0) (AUC = 0.956) and DAG (16:0/18:0) (AUC = 0.949). Key metabolic pathways associated with GDD/ID included D-glutamine, D-glutamate,

alanine, aspartate, glutamate, sphingolipid, histidine, arginine, and proline metabolisms. Furthermore, lysine metabolic pathways, including degradation and biosynthesis, as well as aminoacyl-tRNA biosynthesis, were implicated in GDD/ID pathogenesis. **Conclusion:** This study identified putative biomarkers and metabolic pathways associated with GDD/ID, highlighting the potential of combined plasma metabolomics and lipidomics for early screening of GDD/ID and providing tentative insights into its pathophysiology. The biomarkers show strong diagnostic performance as screening tools, but future studies are needed to validate their prognostic value and clinical utility in multi-center cohorts.

编号：EYX-2026-1-6

引用格式：Kessi M, Pan L, Chen B, Yang L, Yang L, Bamgbade OA, Wang G, Peng J, Yin F, He F. Mitochondrial and lysosomal dysfunctions might be involved in the pathogenesis of the CACNA1A-related neurodevelopmental disorders according to in vitro studies. Biol Res. 2025;58(1):76. doi: 10.1186/s40659-025-00655-w

通讯作者：何芳

Abstract

Background: CACNA1A variants are associated with severe neurodevelopmental disorders (NDDs), but the underlying mechanisms remain unclear. Our goal was to investigate the molecular mechanisms through which these variants lead to intellectual disability (ID), autism spectrum disorder (ASD), epilepsy, and ataxia. **Methods:** Clinical information was collected from six pediatric patients. Molecular experiments were performed on transfected human embryonic kidney and Chinese hamster ovary cells to study the effect of these variants on mitochondrial and lysosomal function. RT-qPCR, Western blot, apoptosis assay, mitochondrial and lysosomal tracker fluorescence intensity, and mitochondrial calcium concentration tests were performed. Additionally, we examined the levels of reactive oxygen species (ROS), adenosine triphosphate (ATP), and mitochondrial enzymes and copy numbers.

Results: We identified six variants that downregulated CACNA1A mRNA: p.D1644N, p.Y62C, p.G701R, p.R279C, p.R1664Q, and p.L1422Sfs*8. Five variants down-regulated Cav2.1 protein expression, whereas, the p.R279C variant up-regulated it. All variants led to dysfunctions in the autophagy-lysosomal system: p.D1644N, p.R279C, and p.G701R variants blocked the fusion of autophagosomes and lysosomes while p.Y62C, p.R1664Q, and p.L1422Sfs*8 variants displayed increased lysosomal expression. The p.Y62C, p.G701R, p.R279C, p.R1664Q, and p.L1422Sfs*8 variants exhibited defective autophagy. The p.Y62C and p.D1644N variants disrupted mitochondrial function by downregulating mitochondrial enzyme activities and ATP levels, as well as by upregulating mitochondrial copy numbers, calcium levels, and ROS levels. Furthermore, the p.Y62C variant increased mitochondrial expression, fusion, and fission. In contrast, the p.D1644N variant decreased mitochondrial expression, fusion, fission, and mitophagy. The p.G701R, p.R279C, and p.R1664Q variants also interrupted mitochondrial function. These variants down-regulated mitochondrial enzyme activities, fusion and fission, the mitophagy process, and ATP levels while up-regulating mitochondrial copy numbers and ROS levels. The p.L1422Sfs*8 variant increased the expression, fusion and fission of mitochondrial proteins, while decreasing mitochondrial calcium levels and the mitophagy process. The p.R279C variant increased mitochondrial expression and calcium levels while enhancing apoptosis. The p.G701R variant decreased mitochondrial expression and calcium levels while enhancing apoptosis. The p.R1664Q variant increased mitochondrial calcium levels and enhanced apoptosis without changing mitochondrial expression. **Conclusions:** CACNA1A variants may alter mitochondrial and lysosomal function, resulting in the development of NDDs.

编号：EYX-2026-1-7

引用格式：Wu H, Dai W, Cheng J, Li A, Peng Y, Guo P, Kong Z. T7 peptide-engineered liposomal Irisin mitigates PND progression through AMPK/PGC-1α

signaling: multi-omic evidence of metabolic and epigenetic modulation. J Nanobiotechnology. 2026;24(1):243. doi: 10.1186/s12951-026-04109-7

通讯作者：孔朝红

Abstract

This study explored the molecular mechanisms by which T7 peptide-modified liposomal irisin (T7@Lipo@Irisin) alleviates perioperative neurocognitive disorders (PND) via regulation of the AMPK/PGC-1α metabolic pathway. T7@Lipo@Irisin nanoparticles were prepared by thin-film hydration and ultrasonic dispersion and showed favorable physicochemical performance, with an encapsulation efficiency of approximately 85%. Serum analysis of healthy donors (n = 10) and PND patients (n = 6) showed higher IL-6 and TNF-α and lower brain-derived neurotrophic factor (BDNF) in PND. In vitro, T7@Lipo@Irisin restored mitochondrial membrane potential, reduced reactive oxygen species (ROS) accumulation, enhanced Neuro-2a hippocampal neuron viability, and activated the AMPK/PGC-1α axis under oxidative stress. In a PND mouse model, it improved Garcia neurological scores, preserved neuronal morphology, and decreased apoptosis. Multi-omic integration of scATAC-seq/scRNA-seq and TMT-based proteomics demonstrated enhanced neuro-glial crosstalk, epigenetic activation of metabolic/antioxidant genes (e.g., Sirt1, Nfe2l2), and upregulated pathways (mitochondrial function, NAD-dependent metabolism, synaptic homeostasis). Proteomics confirmed upregulation of SIRT1, NDUFS2, and BDNF, forming a network linked to energy metabolism and neural repair. Collectively, T7@Lipo@Irisin mitigates PND by activating AMPK/PGC-1α to enhance mitochondrial function and stabilize the neuro-microenvironment.

编号：EYX-2026-1-8

引用格式：Kong Z, Jiang J, Deng M, Deng M, Wu H. Astrocyte-targeted nanovesicle delivery of resveratrol activates SIRT1 to suppress neuroinflammation and restore neural homeostasis in epilepsy. Nano Res. 2026;19(1): 94908190. doi: 10.26599/NR.2025.94908190

第一作者：孔朝红

Abstract

Epilepsy is a complex neurological disorder aggravated by chronic euroinflammation largely driven by reactive astrocytes. These cells promote epileptogenesis through persistent cytokine secretion and glial scar formation. Current antiepileptic drugs remain ineffective in targeting these mechanisms due to limited blood–brain barrier (BBB) permeability and poor astrocytic specificity. A transferrin-functionalized biomimetic nanotherapeutic loaded with resveratrol (RN@RTA) was developed to regulate astrocyte-mediated inflammation by activating SIRT1 and suppressing the MAPK/NF-κB axis. Using *in vitro* BBB models, primary astrocytes, and a ilocarpine-induced chronic epilepsy mouse model, we evaluated the capacity of RN@RTA to cross the BBB, inhibit inflammatory signaling, and reduce seizure activity. Mechanistic assays included immunoprecipitation of NF-κB complexes, cytokine quantification, RNA sequencing, and histopathological assessments of glial and synaptic markers. RN@RTA achieved 82% uptake by hippocampal astrocytes and significantly reduced *Il6*, *Tnf-α*, and *Nlrp3* expression. SIRT1 activation disrupted the NF-κB p65/p300 complex, leading to transcriptional repression of inflammatory genes and enhancement of autophagy. *In vivo*, seizure frequency decreased by 67%, synaptic structure was preserved, and astrogliosis was markedly alleviated. The findings demonstrate a dual regulatory mechanism in which RN@RTA suppresses neuroinflammatory signaling and restores neural homeostasis, offering a promising molecularly targeted approach for refractory epilepsy.

编号：EYX-2026-1-9

引用格式：Xia Z, Liu H, Guo P, Chen C, Ge L, Tang L, Zhang Y, Ma Y. EEFSEC deficiency underlies a human selenopathy with primary neurodevelopmental origins via midbrain-hindbrain hypoplasia. HGG Adv. 2026;7(2):100563. doi: 10.1016/j.xhgg.2026.100563.

通讯作者：马燕丽

Abstract

Bi-allelic mutations in EEFSEC, a key factor in selenoprotein synthesis, cause a severe human selenopathy characterized by developmental delay, spasticity, and profound cerebellar atrophy. While previous studies in invertebrate models framed this condition as an early-onset neurodegenerative disorder, the contribution of primary developmental defects to the severe brain malformations in patients has remained a critical unanswered question. Here, we address this gap using a zebrafish model of EEFSEC deficiency. We discovered that loss of eefsec function does not impair global somatic growth but instead causes specific and significant hypoplasia of the midbrain and hindbrain-the embryonic precursors to the human cerebellum and brain stem. These structural defects directly correlate with robust behavioral impairments, including diminished locomotion and blunted escape responses, mirroring the severe motor dysfunction in patients. Critically, our findings provide the in vivo evidence from a vertebrate model that this disorder involves a primary neurodevelopmental defect, which underlies the severe brain malformations and creates a structurally vulnerable nervous system. This establishes a developmental basis for understanding this condition. We propose that this initial failure in brain construction, which we term a developmental selenopathy, creates a structurally vulnerable nervous system, providing a plausible mechanistic explanation for the human phenotype and proposing a framework for understanding this devastating condition.

欢迎大家引用上述论文

编号：HJL-2026-1-1

引用格式：Fu J, Zhang H, Yu X, Liu P, Pan J, Duan Q, Liu W, Wang Y, Li X. Multi-omics reveals that genes linked to succinylation regulate the onset of epilepsy through metabolic reprogramming. Mamm Genome. 2025;37(1):8. doi: 10.1007/s00335-025-10180-6

通讯作者：王莹

Abstract

The relationship between succinylation modification and epilepsy is not yet well defined, and the potential mediation of metabolic imbalance in its regulatory pathways requires deeper investigation. This study combines Mendelian randomization (MR) and single-cell transcriptomic techniques to investigate the causal interplay between succinylation-related genes, plasma metabolites, and epilepsy. Specifically, the eQTLGen and plasma metabolite databases are utilized for two-sample MR analysis, which evaluates genetic instrumental variables and quantifies causal effects. The two-step MR approach is applied to identify potential metabolic pathways mediating these genetic effects. This study integrates single-cell data from the temporal lobe of epilepsy patients to delineate cell-type-specific gene expression and regulatory networks. MR analysis identified that elevated expression of the CTBP1 gene significantly increases the risk of epilepsy (OR=1.052, p=0.0026). This pathogenic effect is mediated through the dysregulation of eight metabolites: a reduction in six neuroprotective sphingolipids and ceramide (β<0), coupled with an accumulation of the pro-epileptic metabolite Methylsuccinate (β>0). Among these, Sphingomyelin (d18:1/21:0, d17:1/22:0, d16:1/23:0) exhibited the highest mediation ratio (25.71%). Single-cell transcriptomics further revealed that CTBP1 is specifically highly expressed in excitatory neurons. In the epileptic temporal lobe, these neurons displayed rewired intercellular communication, primarily characterized by enhanced signaling via the NRG3-ERBB4 axis, alongside alterations in neuroimmune and metabolic pathways. This study provides the first integrated multi-omics evidence that CTBP1 may promote epileptogenesis through metabolic reprogramming and neuronal heterogeneity

regulation, suggesting a potential role for CTBP1-mediated metabolic reprogramming in temporal lobe excitatory neurons in the disorder's pathology.

编号：HJL-2026-1-2

引用格式：Cui J, Zhai Q, Tan Z, Zou Z, Zhang M, Gao N, Sun J. Resveratrol–loaded self–assembled tetrahedral framework nucleic acids reshape the epileptic microenvironment by regulating oxidative stress and neuroinflammation via the SIRT3/SOD2 pathway. J Nanobiotechnol. 2026. doi: 10.1186/s12951-026-04145-3

通讯作者：孙家行

Abstract

Background: Epilepsy management remains a significant clinical challenge, as conventional antiseizure medications primarily mitigate symptoms without addressing the core pathological drivers, specifically the vicious cycle formed by neuroinflammation and oxidative stress. Furthermore, the therapeutic efficacy of potential neuroprotective agents is severely compromised by the blood–brain barrier (BBB), poor stability, and insufficient accumulation at epileptic lesions. Therefore, engineering a BBB–penetrating delivery strategy that simultaneously disrupts the vicious cycle of neuroinflammation and oxidative stress is critical for achieving disease–modifying effects in epilepsy treatment. **Results:** Here, we developed a biomimetic, brain–targeting nanosystem (R–tFNAs@PB) by anchoring resveratrol–loaded tetrahedral framework nucleic acids (tFNAs) onto a Prussian blue (PB) core. This nanosystem effectively traversed the BBB and exhibited precise accumulation within hippocampal epileptic foci. Mechanistically, R–tFNAs@PB acted as a dual–function modulator. The PB core and resveratrol synergistically scavenged reactive oxygen species (ROS) and activated the SIRT3/SOD2 signaling pathway, thereby increasing the mitochondrial antioxidant capacity. This cascade effectively inhibited NLRP3 inflammasome activation and promoted the polarization of microglia from the proinflammatory M1 phenotype to the anti–inflammatory M2 phenotype. In a mouse

model of kainic acid–induced epilepsy, the nanosystem significantly reduced neuronal damage, reduced the seizure frequency and severity, and ameliorated cognitive deficits. **Conclusions:** This study presents a novel nanotherapeutic strategy that integrates microenvironment remodeling with neural repair. By leveraging the specific brain–targeting capability of tFNAs and the synergistic antioxidant properties of the core–shell structure, R–tFNAs@PB represent a promising approach for treating refractory epilepsy through the precise regulation of the oxidative stress–neuroinflammation axis via the SIRT3/SOD2 pathway.

编号：HJL-2026-1-3

引用格式：You Z, Huang C, Gao X, Fan Z, Wei F, He Y, Zhao S, Sun J. Dihydroartemisinin Alleviates Neuronal Damage and Seizures in Epileptic Mice by Inhibiting Ferroptosis via the SIRT1/FOXO1/SLC7A11/GPX4 Pathway. CNS Neurosci Ther. 2026;32:e70798. doi: 10.1002/cns.70798

通讯作者：孙家行

Abstract

Background: Epilepsy represents a prevalent neurological disorder. Currently, ferroptosis has been reported to be intricately linked to epilepsy onset and progression. Dihydroartemisinin (DHA) can inhibit the level of ferroptosis in various diseases. Therefore, the present study investigated whether DHA could inhibit seizures and display neuroprotective impacts by impeding ferroptosis. **Methods:** In the KA-induced epileptic mouse model, the effects of DHA on epileptic behavior, cognitive function, and hippocampal neuronal damage were observed. Using both in vivo and in vitro models, the impact of DHA on neuronal injury and ferroptosis-related markers was investigated. Techniques including molecular docking, Western Blot, immunofluorescence, and CHIP-qPCR were utilized to analyze the regulatory mechanism of DHA on ferroptosis in epilepsy. Finally, brain tissue samples from patients with temporal lobe epilepsy (TLE) were collected to validate the expression of ferroptosis-related markers. **Results:** Our experimental results showed that DHA

attenuated seizures, hippocampal neuronal damage, and memory and learning deficits in epileptic mice. Moreover, DHA inhibited ferroptosis by activating solute carrier family 7 member 11 (SLC7A11) and glutathione peroxidase 4 (GPX4) expression in vivo and in vitro. Subsequently, we found that DHA activated SIRT1 expression in the mouse hippocampus, leading to a decrease in the acetylation level of forkhead box O1 (FOXO1), thereby increasing the transcriptional activity of SLC7A11. Finally, our findings provide preliminary clinical support for the association between ferroptosis and TLE. **Conclusion:** In summary, our findings indicate that DHA may have antiepileptic and neuroprotective benefits by suppressing ferroptosis through the SIRT1/FOXO1/SLC7A11/GPX4 signaling pathway.

编号：HJL-2026-1-4

引用格式：Zhai Q, Cui J, Tan Z, Wu H, Yu Y, Sun J. Chimeric antigen receptor macrophages therapy for glioblastoma: challenges and opportunities from preclinical evidence to clinical translation. Front Immunol. 2026;17:1726329. doi: 10.3389/fimmu.2026.1726329

通讯作者：孙家行

Abstract

Treatment failure in glioblastoma (GBM) is primarily attributed to the convergence of multiple barriers, including an immunosuppressive tumor microenvironment (TME), intratumoral heterogeneity, and the blood-brain barrier. Chimeric antigen receptor macrophages (CAR-M) therapy presents a promising new avenue for GBM treatment, leveraging its inherent tumor-homing capacity, TME reprogramming function, and potential to bridge innate and adaptive immunity. However, despite promising preclinical data, clinical efficacy in GBM remains unproven. This review critically analyzes the translational gap. We first outline the theoretical rationale and inherent advantages of CAR-M therapy in overcoming the core barriers of GBM. We then critically assess the limitations of current preclinical evidence and the uncertainties associated with its extrapolation to the clinical setting. We then focus on bottlenecks

such as target selection strategies, engineering design, and TME-driven issues like phenotypic inactivation and antigen escape, discussing corresponding optimization approaches like armoring modifications, logic-gated designs, and convection-enhanced delivery. Finally, we propose a pragmatic clinical translation pathway prioritizing mechanistic validation. This pathway emphasizes integrating CAR-M therapy with combinatorial approaches and smart technologies in early-phase clinical trials, supported by biomarker analyzes, to address fundamental biological questions regarding the homing, survival, and function of these cells in patients. This review aims to provide a systematic and critical reference to guide the translation of CAR-M therapy from concept to clinical application, a path characterized by both opportunities and challenges.

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编号：JJJ-2026-1-1

引用格式：Liao J, Zuo J, Dai Y, Zhang J, Wang Q, Cui T, Liu M, Lv R. Efficacy and safety of transcutaneous auricular vagus nerve stimulation in drug-resistant epilepsy: a single-center prospective real-world study. Ther Adv Neurol Disord. 2025;18:17562864251396022. doi: 10.1177/17562864251396022

通讯作者：吕瑞娟

Abstract

Background: Drug-resistant epilepsy (DRE) imposes a heavy disease burden and urgently requires new, effective, and safe treatment options. **Objective:** To evaluate the efficacy and safety of transcutaneous auricular vagus nerve stimulation (ta-VNS) in patients with DRE. **Design:** Ongoing, single-center, prospective real-world study. **Data sources and methods:** Patients diagnosed with DRE and undergoing ta-VNS treatment at Beijing Tiantan Hospital, affiliated with Capital Medical University, were prospectively enrolled in this study between January 2023 and December 2024. The follow-up period lasted 1-2 years. The frequency of seizure reduction was assessed using seizure diaries, with a 50% decrease in seizure frequency deemed indicative of efficacy. Adverse reactions and comorbidities were recorded concurrently. All patients were maintained on stable anti-seizure medications during this study. **Results:** Ninety-nine patients were enrolled, among whom 16 were lost to follow-up and 18 refused follow-up. Ultimately, 65 patients were successfully followed up for analysis. The overall efficacy rate was 61.54%. Specifically, 15 patients experienced seizure reductions >90%, 8 achieved a reduction between 75% and 90%, 17 demonstrated a reduction ranging from 50% to 75%, and 1 exhibited a reduction of <50%. There were no severe adverse events, although 10 patients reported mild side effects (e.g., ear tingling and tinnitus). The efficacy rate was found to be independent of variables such as age, sex, treatment frequency, and type of epilepsy. However, it demonstrated an association with baseline seizure frequency prior to treatment and the etiology of epilepsy. Patients exhibiting a higher baseline frequency of seizures (>30 episodes per month) demonstrated significantly improved response rates, with an efficacy rate of

90.9%. Etiologies, including posttumor surgery, congenital brain dysplasia, and genetic mutations, demonstrated efficacy rates >90%. **Conclusion:** The efficacy of ta-VNS was 61.54% in patients with DRE. Patients with high baseline seizure frequency or identifiable etiologies tended to experience greater therapeutic benefits. Owing to its noninvasive nature and favorable safety profile, it presents a promising alternative for the management of DRE.

编号：JJJ-2026-1-2

引用格式：Liu Y, Lv K, Guo H, Wu H, Gong P, Chen Z, Deng J, Wang X, Guan Y, Zhou J, Wang R, Liu Q, Cai L, Ji T, Tang C, Luan G. Clinical characteristics and surgical outcomes of Rasmussen encephalitis: A retrospective dual-center cohort study. Epilepsia. 2026. doi: 10.1002/epi.70175

通讯作者：唐重阳

Abstract

Objective: Rasmussen encephalitis (RE) is a rare progressive disorder causing drug-resistant epilepsy. Hemispheric surgery is an established treatment, but comprehensive data on postoperative seizure, motor, and cognitive outcomes are limited. We aimed to evaluate these outcomes and identify associated prognostic factors. **Methods:** This dual-center retrospective study included RE patients who underwent hemispheric surgery at two tertiary epilepsy centers in China. Seizure outcomes were classified by Engel class, with Engel class I regarded as seizure-free. Motor outcomes were evaluated using the Motricity Index (MI), Gross Motor Function Classification System (GMFCS), and Manual Ability Classification System (MACS), with stable outcomes defined as improvement or no change between baseline and follow-up. Cognitive function was evaluated using standardized scales. Multivariable Cox regression identified factors associated with seizure and motor outcomes. **Results:** Eighty-five patients (52 female, 63%) with median follow-up of 2.75 years were included. At last follow-up, 79% were seizure-free after initial hemispheric surgery. Hemispheric disconnection showed better seizure control (92%) than anatomic (67%) or functional hemispherectomy (57%), with

shorter surgery and less blood loss. Stable gross motor function was achieved in 94%, whereas 64% experienced worsening fine motor skills. Generalized seizures (hazard ratio [HR] 5.48, 95% confidence interval [CI] 1.63-18.42), contralateral magnetic resonance imaging (MRI) abnormalities (HR 6.43, 95% CI 1.14-36.28), contralateral interictal electroencephalography (EEG) discharges (HR 4.61, 95% CI 1.57-13.51), and type of hemispheric surgery (HR 5.25, 95% CI 1.57-13.51) were associated with seizure persistence. Postoperative seizure-free (HR 4.43, 95% CI 1.61-12.17) and baseline MI (HR .94, 95% CI .91-.96) predicted overall motor stability, whereas fine motor stability was related to epilepsia partialis continua (EPC) duration (HR .50, 95% CI .25-.98), preoperative immunotherapy (HR 3.30, 95% CI 1.13-9.59), and baseline MACS (HR 2.45, 95% CI 1.68-3.57). **Significance:** This study suggests that hemispheric surgery is effective in achieving seizure-free and favorable gross motor recovery in RE. Early surgery, attention to contralateral abnormalities, and preoperative immunotherapy may further improve outcomes.

编号：JJJ-2026-1-3

引用格式：Ren G, Hannan S, Schiller K, Thomas J, Moye M, Avigdor T, Jaber K, Wei X, Ye H, Ho A, Ghosn NJ, Conrad EC, Southwell D, Hall J, Shao X, Wang Q, Radtke R, Gotman J, Frauscher B. Impact of antiseizure medication taper on electroencephalographic dynamics in focal epilepsy: A stereoelectroencephalographic study. Epilepsia. 2026. doi: 10.1002/epi.70149

第一作者：任国平

Abstract

Objective: Tapering of the antiseizure medication dosage in the epilepsy monitoring unit can provoke seizures, but its effects on seizure dynamics remain poorly characterized. This study addresses three questions: (1) Does antiseizure medication tapering influence spatiotemporal dynamics of seizures? (2) Does the tapering rate affect these dynamics? (3) Does tapering have a similar effect on interictal epileptic discharges as it does on seizures? **Methods:** Patients with drug-resistant epilepsy

undergoing stereoelectroencephalographic (stereo-EEG) presurgical evaluations at Duke University Medical Center (n = 104) and the Montreal Neurological Institute and Hospital (n = 80) were screened. We included patients in whom the antiseizure medication dosage was tapered from the highest daily dosage (high dosage) to ≤ 50% (low dosage) during stereo-EEG monitoring, and at least one seizure from the same focus was recorded in both conditions. Using an intrapatient design, we compared seizure onset-zone, onset pattern, and propagation dynamics between the two conditions. Given the intrinsic seizure variability, comparisons were made between same-dosage and cross-dosage seizure pairs. We further assessed effects of tapering rates and examined the characteristics of interictal epileptiform discharges. **Results:** Among 30 patients, the proportion of channels in the seizure onset zone did not differ between high-dosage and low-dosage conditions (7.25% vs. 8.95%, p = .50, d = -.04). Similarly, no differences were observed in the overlap ratio of seizure-onset regions (62% vs. 64%, p = .72, d = -.01), or the cross-correlation of seizure-onset patterns (.36 vs. .35, p = .54, d = .04) when comparing same-dosage versus cross-dosage seizure pairs. Conversely, seizures at low dosage involved more channels (40.71% vs. 81.49%, p = .001, d = -.39) and lasted longer (33.36 s vs. 74.30 s, p < .01, d = -.47). Tapering rate did not affect seizure dynamics. The mean interictal epileptiform discharge rate and number of propagation channels also remained unchanged. **Significance:** Despite seizure exacerbation during antiseizure medication tapering, seizure-onset location remained stable. This supports the robustness of seizure-based localization even under reduced medication levels and rapid tapering regimens.

编号：JJJ-2026-1-4

引用格式：Leng X, Yang X, Xiang J, Wang R, Dong H. Resting-state MEG of whole-brain functional network in cingulate gyrus epilepsy. Front Neurol. 2026;17:1646021. doi:10.3389/fneur.2026.1646021

第一作者：冷雪荣

Abstract

Objective: To investigate the connectivity and formation mechanism of the whole brain resting-state network in cingulate gyrus epilepsy and to identify biological markers and potential neuromodulation targets for this condition. **Methods:** Fifteen patients with cingulate gyrus epilepsy and 15 healthy controls underwent resting-state magnetoencephalography (MEG). To compute functional network connectivity at the source level, we used MEG Processor software. Twenty regions of interest (ROI) were selected from both cerebral hemispheres, and connectivity was assessed across four frequency bands: theta (4-7.5 Hz), alpha (8-13 Hz), beta (14-30 Hz), and gamma (31-80 Hz). **Results:** The number of neocortical-related functional connectivity differences increased with the frequency band, being smallest in the theta (θ) band and largest in the gamma (γ) band. The connections between the angular gyrus (AG) and the occipital gyrus (OG) and between the OG and the superior temporal gyrus (STG) were the most influential in terms of functional connectivity within the neocortex. The connectivity between the anterior cingulate cortex (ACC) and the inferior frontal gyrus (IFG) showed the most pronounced differences in the α, β, and γ bands. Among the functional connectivities to the posterior cingulate gyrus (PCC), those involving the AG-PCC and STG-PCC were the most significant. The hippocampal-related functional connectivity differed from neocortex-related functional connectivity, and the number of differential functional connections was greater in the θ-band than in the α-band. **Conclusion:** Enhanced functional connectivity (AG-OG and OG-STG) of the neocortical surface may be characteristic features of the resting-state network in cingulate gyrus epilepsy and could serve as potential biological markers for this condition. The IFG's close relationship with the ACC suggests it may be a candidate target for neuromodulation therapy in anterior cingulate gyrus epilepsy. Similarly, the AG and STG's connections with the PCC make them potential candidates for neuromodulation therapy in posterior cingulate gyrus epilepsy for future investigation.

编号：JJJ-2026-1-5

引用格式：Fan X, Dai J, Liu J, You G, Li K, Fang S, Dong J, Shi J, Wang J.

Glioma-related epilepsy in patients with newly diagnosed and recurrent glioblastoma, IDH-wildtype: a study under the 2021 WHO CNS tumor classification. BMC Cancer. 2025;26(1):53. doi: 10.1186/s12885-025-15351-x

第一作者：樊星

Abstract

Background: The 2021 classification of central nervous system tumors significantly alters the defined category of glioblastoma (GBM). This study aims to evaluate the clinical relevance of glioma-related epilepsy (GRE) in patients with the newly classified GBM, IDH-wildtype. **Methods:** A single-center retrospective cohort study was performed. The correlation of GRE with clinicopathological features was explored via appropriate inter-group statistical methods. Kaplan-Meier analysis was employed to assess the prognostic value of preoperative GRE with respect to overall survival (OS) and progression-free survival (PFS). Multivariate binary logistic regression analysis was carried out to identify potential risk factors associated with inadequate seizure control. **Results:** The final cohort included 294 patients. Preoperative GRE was observed in 22.4% of newly diagnosed and 19.7% of recurrent GBM cases. In patients with newly diagnosed GBM, GRE was significantly associated with younger age ($p < 0.001$). Moreover, survival analysis confirmed the prognostic significance of preoperative GRE for improved OS and PFS ($p = 0.012$ and 0.004, respectively) in the same population. Moreover, in newly diagnosed GBM cases, preoperative GRE was identified as the independent risk factor for inadequate postoperative seizure control (OR 4.009, 95% CI 1.447–11.106, $p = 0.008$). **Conclusion:** The current study comprehensively describes the clinical correlations of GRE in GBM, IDH-wildtype. The incidence of preoperative GRE in GBM patients is approximately 20%. Among patients with newly diagnosed GBM, IDH-wildtype, preoperative GRE is more likely to occur in younger individuals and is associated with prolonged survival outcomes. Furthermore, a history of preoperative GRE may increase the risk of inadequate postoperative seizure control in this population.

编号：JJJ-2026-1-6

引用格式：Dong J, Liu L, Zhao Z, Huang G, Chen Q, Liu X, Wang J, Fan X, Ge S, Jiang T. China National Glioma Registry (CNGR): protocol for a prospective observational registry study. J Neurooncol. 2025;176(1):83. doi: 10.1007/s11060-025-05333-1

通讯作者：樊星

Abstract

Purpose: Gliomas present a significant public health challenge owing to their complex management and poor prognosis. Robust real-world datasets are essential for advancing glioma research and informing clinical practice. To address limitations of existing resources, we established the China National Glioma Registry (CNGR), a comprehensive, glioma-specific registry designed to systematically capture multidimensional data. **Methods:** The CNGR is a prospective, longitudinal, observational registry initiated at Beijing Tiantan Hospital. Adult-type diffuse glioma patients are consecutively enrolled following informed consent. Clinical, pathological, imaging, functional, and long-term follow-up data are collected at predefined time points by trained specialists using standardized procedures and recorded in a secure electronic data capture system. All aspects of data acquisition and quality control are overseen by a dedicated multidisciplinary team to ensure accuracy, consistency, and integrity. The study protocol has been approved by the Ethics Committee of Beijing Tiantan Hospital and registered with the Chinese Clinical Trial Registry. **Conclusion:** The CNGR offers a structured framework for high-quality real-world data acquisition across the glioma disease trajectory. Although currently single-center, its nationwide recruitment ensures cohort diversity and broad applicability. The CNGR is intended to serve as a scalable model for future multi-center studies in neuro-oncology and as a robust research infrastructure to enhance diagnostic precision, inform therapeutic decision-making, and support prognostic evaluation. It is poised to become a valuable resource for translational research, evidence-based practice, and the development of personalized treatment strategies in glioma care.

编号：JJJ-2026-1-7

引用格式：Yang J, Yang X, Liu H, Liu J, Li K, Shi J, Liang Y, Song Y, Fan X, Qiao H. The application value of intraoperative lateral spreading response monitoring during microvascular decompression in patients with primary hemifacial spasm. Eur J Med Res. 2025;30(1):1095. doi: 10.1186/s40001-025-03293-w

通讯作者：樊星

Abstract

Background: The application value of intraoperative lateral spreading response (LSR) during microvascular decompression (MVD) is always disputed. The current study aimed to explore the predictive value of intraoperative LSR monitoring for the long-term outcome in patients with primary hemifacial spasm (pHFS). **Methods:** The data from 312 pHFS patients were retrospectively reviewed. The zygomatic LSR (ZYG-LSR) and mandibular LSR (MAN-LSR) monitoring were performed during surgery. The correlations of ZYG-LSR and MAN-LSR disappearances with patients' long-term outcomes (one year after surgery) were retrospectively investigated. Consequently, binary logistic regression analysis was applied to explore their predictive value. Finally, the implications of their combined utilization for predicting the long-term outcome were explored. **Results:** Patients with either persistent ZYG-LSR or MAN-LSR exhibited a higher incidence of spasms one year after surgery ($p < 0.001$). Persistent ZYG-LSR (odds ratio 7.721, $p < 0.001$) and MAN-LSR (odds ratio 10.729, $p < 0.001$) were both identified as independent predictive factors for an unfavorable long-term outcome. Taking both ZYG-LSR and MAN-LSR into consideration, patients with simultaneously disappeared two waves had the highest long-term recovery rate (97.2%), followed by patients with either persisted wave (76.5%) and patients with simultaneously persisted two waves (27.3%). The differences among all pairwise comparisons were statistically significant ($p < 0.001$ for all). **Conclusion:** The current study confirmed the application value of intraoperative LSR monitoring during MVD

in patients with pHFS. In addition, the clinical significance of two-branch LSR monitoring was also described. The findings can provide important insights for optimizing the application of intraoperative LSR monitoring in clinical practice.

编号：JJJ-2026-1-8

引用格式：Ma S, Liang Y, Shi J, Yang L, Liu J, Li K, Yang J, Qiao H, Jia W, Fan X. Can intraoperative facial nerve motor evoked potential monitoring predict postoperative facial synkinesis following vestibular schwannoma surgery? A retrospective study. J Neurooncol. 2025;176(2):141. doi: 10.1007/s11060-025-05369-3

通讯作者：樊星

Abstract

Background: Facial synkinesis is a distressing late-onset complication of vestibular schwannoma (VS) surgery. The current study aimed to explore the predictive value of intraoperative facial nerve motor evoked potential (FNMEP) monitoring for postoperative facial synkinesis in VS patients. **Methods:** The data of 170 VS patients were retrospectively analyzed. The final-to-baseline amplitude ratio (FBR) of mentalis FNMEP was selected as a representative metric to assess FNMEP alterations before and after tumor resection. The presence of facial synkinesis was evaluated one year after surgery. Group comparison and logistic regression analysis were performed to assess the predictive value of mentalis FBR for synkinesis. A predictive nomogram was subsequently established. The optimal FBR threshold for predicting synkinesis was obtained by the receiver operating characteristic curve analysis. **Results:** Among the cohort, 44.12% (75/170) of patients developed postoperative facial synkinesis. Patients with synkinesis showed higher FBR ($p < 0.001$) and in-hospital House-Brackmann (HB) grades ($p < 0.001$). Moreover, both FBR (Odds Ratio 1.107, $p < 0.001$) and in-hospital HB grade (Odds Ratio 1.731, $p < 0.001$) were identified as independent predictors for synkinesis. The constructed nomogram showed good calibration and net benefit. The optimal FBR threshold for predicting postoperative facial synkinesis was 11.42% ($p <$

0.001). **Conclusions:** The FBR of mentalis FNMEP was identified as an independent indicator for the risk of postoperative facial synkinesis. A corresponding predictive model with good predictive performance and significant clinical applicability was developed. Furthermore, a two-step warning strategy for FNMEP monitoring was proposed.

编号：JJJ-2026-1-9

引用格式：Liu T#, Wang Q#, Kuang S, Cao D, Ding P, Zhang S, Wei H, Wei Z, Xu J, Huang X, Liu B*, Liang S*. Predicting Epileptogenic Tubers in Patients With Tuberous Sclerosis Complex Using a Fusion Model Integrating Lesion Network Mapping and Machine Learning. Ann Clin Transl Neurol. 2025 Dec 13. doi: 10.1002/acn3.70277.

第一作者：刘婷红　通讯作者：梁树立

Abstract

Objective: Accurate localization of epileptogenic tubers (ETs) in patients with tuberous sclerosis complex (TSC) is essential but challenging, as these tubers lack distinct pathological or genetic markers to differentiate them from other cortical tubers. Approximately 60% of patients fail to have their ETs identified through noninvasive preoperative evaluations, creating an urgent clinical need for effective, noninvasive localization strategies. **Methods:** A novel fusion model was developed, integrating lesion network mapping-based risk assessment with a machine learning prediction model that utilizes brain functional connectivity and random forest algorithms. The model was built based on magnetic resonance imaging data. Retrospective analysis was conducted on patients with TSC-related epilepsy who had undergone resective surgery and achieved seizure freedom at the 1-year follow-up; tubers were classified as true epileptogenic tubers (true ETs) or true non-epileptogenic tubers (true non-ETs) according to the resected regions. The model calculated and ranked the probability of each tuber being an ET for every patient. **Results:** A total of 47 patients were enrolled in the study. The fusion model successfully ranked the true ETs within the top three in

91% of the cases. Significant differences in the probability rankings of ETs were observed among true ETs, true non-ETs, and random tubers ($p < 0.01$). Receiver operating characteristic curves were plotted to evaluate the accuracy of true ET localization across different methods, and the fusion model exhibited an area under the curve of 0.86. This performance significantly outperformed that of scalp electroencephalography, semiology, and positron emission tomography based on structural magnetic resonance imaging in the same cohort. Cross-validation in three independent epilepsy centers confirmed the model's high generalizability. **Interpretation:** Overall, this fusion model demonstrates high accuracy and robust clinical utility as a noninvasive tool for the localization of ETs. It effectively addresses the current challenges in identifying ETs, providing valuable support for surgical planning in patients with TSC-related epilepsy.

编号：JJJ-2026-1-10

引用格式：Liu C[#], Ding P[#], Zhao T[#], Liu T, Wu S, Yan Y, Yuan L, Zhang L, Liang S. Impact of Anesthesia With Propofol on Epileptic Discharges Recorded by Stereo-Electroencephalography in Pediatric Epilepsy Surgery. CNS Neurosci Ther. 2026;32(1):e70767. doi: 10.1002/cns.70767. PMID: 41589347.

第一作者：刘畅　通讯作者：梁树立

Abstract

Objective: This study aimed to evaluate the influence of anesthesia with propofol on epileptic discharges recorded by stereo-electroencephalography (SEEG) during pediatric epilepsy surgery. **Methods:** We enrolled 30 pediatric patients (aged 2-14 years) undergoing epilepsy surgery between June 2022 and November 2023. SEEG recordings were obtained under different states of consciousness: awake, sleep, and anesthesia. We analyzed the power spectral density (PSD) across frequency bands and quantified the number of interictal spikes and high-frequency oscillations (HFO) including ripples and fast ripples (FRs). **Results:** There was a significant reduction in the number of spikes,

particularly within the seizure onset zone (SOZ), while ripples and FRs increased under general anesthesia, but the extent of the increase did not differ between the SOZ and other detected brain regions (NSOZ). Analysis of PSD showed increased energy during anesthesia, particularly in high-frequency bands. Meanwhile, no statistical differences were observed in energy changes between the SOZ and NSOZ when compared to wakefulness or sleep. HFOs emerged as more robust and consistent interictal biomarkers for localizing the SOZ under propofol anesthesia, particularly in the frontal cortex and cingulate cortex, and were not influenced by age. **Conclusions:** General anesthesia with propofol significantly affects the number and frequency of epileptic discharges, particularly by reducing spikes while increasing HFOs. Notably, HFOs-particularly in the frontal and cingulate cortices-emerged as reliable biomarkers for delineating the SOZ and guiding surgical resections. Furthermore, no significant age-related variability was identified, supporting the applicability of intra-operative electroencephalography in pediatric epilepsy surgery.

编号：JJJ-2026-1-11

引用格式：Liang S[#,*], Fan X[#], Kuang S[#], You G[#], Liu T[#], Zhang J[#], Zhang W, You Y, Deng Y, Ma W, Lin Y, Qian R, Lin W, Wang L, Zhang C, Guan Y, Yan Z, Yang X, Li W, Yue W, Zhang H, Sun D, Liu Y, Wang Z, Li H, Han X, Wang Y, Wang Y, Liao W, Cai L, Huang G, Chen Y, Chen J, Liu X, Peng J, Chen Z, Chen L, Zhang G, Zhang J, Qiu X, Zhao G, Jiang C, Chu L, Mou Y, Chen L, Zhu S, Wu A, Zhang N, Li S, Wu J, Wang S, Wang Y, Li S, Zhou D[*], Jiang T[*]. Clinical practice guidelines for the diagnosis and treatment of diffuse glioma-related epilepsy: 2025 update. Cancer Lett. 2026;645:218360. doi: 10.1016/j.canlet.2026.218360.

第一作者：梁树立 樊星 刘婷红 张静文； 通讯作者：江涛 周东 梁树立

Abstract

Diffuse glioma-related epilepsy (dGRE) frequently presents with epilepsy as the initial symptom and is closely associated with tumor progression or recurrence, imposing

significant social and psychological burdens on patients. The pathogenesis of dGRE is highly complex, involving both peritumoral microenvironmental mechanisms and tumor-intrinsic factors. Diagnosis requires a comprehensive approach integrating neuroimaging, EEG, molecular biomarkers, and spatial correlation between the tumor and the epileptogenic zone. Management aims to control seizures and improve prognosis. Non-enzyme-inducing anti-seizure medications (ASMs), such as levetiracetam and lacosamide, are recommended as first-line therapy, while valproic acid serves mainly as a second-line agent. Surgical resection, particularly maximal safe and supratotal removal guided by electrophysiological monitoring, significantly improves seizure outcomes. Radiotherapy, chemotherapy, and targeted agents further contribute to seizure control. The updated 2025 Chinese clinical practice guidelines incorporate recent advances in ASM use, postoperative withdrawal strategies, and multidisciplinary treatment algorithms. These updates provide an evidence-based reference for standardized diagnosis and management of dGRE.

编号：JJJ-2026-1-12

引用格式：Li J[#], Liu T[#], Liu C[#], Deng J, Wu S, Kuang S, Li X, Wei Z, Liang S. Treatment of pediatric epilepsy. Pediatr Investig. 2026 Jan 29;10(1):86-100. doi: 10.1002/ped4.70043.

第一作者：刘婷红 刘畅； 通讯作者：梁树立

Abstract

Pediatric epilepsy is a neurological disorder arising from various etiologies, including structural, genetic, immune, infectious, metabolic, and unknown causes. Anti-seizure medications remain the primary treatment; however, in cases of drug-resistant epilepsy, surgical interventions, ketogenic diet, and emerging therapies have become increasingly effective options. Disease-modifying treatments, such as antisense oligonucleotides and adeno-associated virus-mediated gene replacement, have shown promise in some epilepsy treatments, with early trials reporting moderate seizure reduction. Minimally invasive surgical approaches, including magnetic resonance-

guided laser interstitial thermal therapy, have also demonstrated favorable outcomes, showing a 68% seizure-free rate at 2 years in the largest pediatric series. Although the ketogenic diet is effective in some patients, demonstrating superiority over conventional management for >50% seizure reduction, long-term use may be associated with metabolic risks; careful monitoring is warranted. Future treatment strategies are expected to emphasize personalized medicine through the integration of genetic, electrophysiological, and neuroimaging data to optimize therapeutic decision-making and enable targeted interventions based on the underlying etiology.

坚定不移走中国特色卫生与健康发展道路推动“十五五”时期健康中国建设取得决定性进展（摘要）

建设健康中国是一项系统工程。面对人民群众日益增长的多元化卫生健康需求，必须突出重点，紧紧抓住那些惠及面广、牵一发而动全身的工作，在健全公共卫生体系、建设优质高效医疗服务体系、倡导健康文明生活方式等方面集中力量和资源、采取有效措施加以推动，不断取得新的成效。要健全党委统一领导、党政齐抓共管的工作格局，完善健康促进政策制度体系，为健康中国建设提供有力保障。进一步深化改革，完善医疗、医保、医药协同发展和治理机制。推动科技创新成果转化运用，推进全民健康数智化建设。加强卫生健康行业党建工作，调动广大医务人员的积极性主动性创造性，加大人才培养力度，弘扬优良医德医风，着力营造风清气正的行业环境。

---2026 年 3 月 6 日，习近平主席在看望参加全国政协十四届四次会议的的农工党九三学社医药卫生界社会福利和社会保障界委员时讲话

编号：JJM-2026-1-1

引用格式：Ju Y, Ji TY. Electro-clinical features of Mowat-Wilson syndrome: A retrospective study of 31 children in mainland China. Epileptic Disord. 2025. doi:10.1002/epd2.70149

通讯作者：季涛云

Abstract

Objective: To summarize the electro-clinical and genetic characteristics of children with Mowat-Wilson syndrome (MWS). **Methods:** This study is a hospital-based case series analyzing clinical data from 31 pediatric patients with MWS and epilepsy treated at Peking University First Hospital between June 2020 and December 2024. Information on seizures, electroencephalographic features, genetic characteristics, treatment, and prognosis was summarized and analyzed using descriptive statistics. **Results:** Among the 31 children (16 males and 15 females), seizure onset occurred at a median age of 25.5 months (range: 1-113 months). Eighteen cases (58.1%, 18/31) began with fever-induced seizures; all 31 children experienced focal seizures, and 16 (51.6%, 16/31) exhibited atypical seizure presentations. Twelve (38.7%, 12/31) experienced seizures accompanied by gastrointestinal (GI) symptoms. Two children had myoclonic seizures, one had epileptic spasms, and another had atypical absence seizures. Ten (32.3%, 10/31) experienced convulsive status epilepticus. Electroencephalographic findings evolved from posterior head-dominant discharges to multifocal or anterior head-dominant discharges, with a significant increase in discharges during sleep. All 31 children had de novo ZEB2 variants, including 27 with single-nucleotide variants (SNVs) or insertions/deletions (indels) and four with copy number variants. Among the SNVs/indels, nonsense (13) and frameshift (12) variants predominated. One patient with rare seizures did not receive anti-seizure medication (ASM). Thirty received ASMs; both levetiracetam and valproic acid, used as monotherapy or in combination, proved effective. Sixteen children achieved seizure control for more than 6 months, and seven maintained seizure control for over 1 year. **Significance:** Our findings reveal the

electro-clinical characteristics, genetic variants, and effective treatments associated with MWS, providing an important basis for clinical diagnosis and management.

编号：JJM-2026-1-2

引用格式：Li G, Ren J, Zheng M, Wu X, Li W, Wang Y, Wu N. Polymorphic low-grade neuroepithelial tumor of the young and treatment of epilepsy: a case report. Front Oncol. 2026;16:1797276. doi: 10.3389/fonc.2026.1797276

通讯作者：吴楠

Abstract

Background: Polymorphic low-grade neuroepithelial tumor of the young (PLNTY) is a rare central nervous system tumor. Clinical manifestations often start with seizures, and the lesions are often located in the superficial parts of the cerebral hemisphere, especially in the temporal lobe. Patients with PLNTY can be cured via surgical treatment, but whether the seizure can be controlled by simply removing the tumor through surgery still needs to be determined. **Case description:** The patient was a 6-year-old boy with clinical manifestations of recurrent epileptic seizures. Preoperative standardized antiepileptic drug treatment failed to control seizures. The patient's electroencephalogram showed was right parietal and central regions is the main slow wave and spike slow wave emitting area, and Magnetic Resonance Imaging s(MRI) showed was structural abnormalities in the right parietal lobe cortex. After multidisciplinary preoperative evaluation at the epilepsy center of Tianjin Children's Hospital, lesion enlargement resection was performed with the assistance of multimodal imaging and electrocorticography (ECoG) monitoring. There were no epileptic seizures during the 6-month follow-up after surgery. **Conclusions:** For this patient with PLNTY accompanied by epilepsy, surgical resection can be the first line of treatment. Meanwhile, a comprehensive multidisciplinary preoperative evaluation should be conducted. Additionally, appropriate enlargement and resection can effectively eliminate epileptic seizures.

编号：SGQNX-2026-1-1

引用格式：Li Z, Zhu N, Chen Y, Chen B, Dong Q, Gan L, Zhao S, Yan Z, Zhang T. EpilepsyFM: A domain-specific foundation model for epileptic representation learning using EEG signals. Neural Netw. 2026;193:108060.

通讯作者：闫志强

Abstract

Epilepsy with its complex seizure mechanisms and diverse clinical manifestations, presents numerous challenges for clinical diagnosis and treatment, while electroencephalography (EEG) plays a crucial and irreplaceable role in its diagnosis. Although general-purpose foundation models have demonstrated some capability in knowledge processing, they still face challenges in capturing specific disease features and dealing with data scarcity in highly specialized domains such as epilepsy. To address these issues, we propose a domain-specific foundation model for epilepsy-EpilepsyFM, designed to learn generalized representations of epilepsy to support various downstream tasks. EpilepsyFM utilizes self-supervised pre-training, integrating clinical EEG data from top-tier hospital neurosurgery departments with large-scale public datasets such as TUH EEG Corpus, covering a variety of patient conditions to enhance the model's representation capacity. The model employs a discrete neural tokenizer to construct a domain-specific neural codebook for epilepsy and proposes a brain region masking strategy based on the mechanisms of clustered neuronal discharges during seizures, allowing for more effective capture of the spatiotemporal features of seizures. Furthermore, EpilepsyFM integrates temporal, spectral, and spatial encoding modules to fully exploit the multidimensional propagation patterns of epilepsy. Experimental results show that EpilepsyFM achieves state-of-the-art performance in six downstream tasks, including seizure detection, seizure type detection, short- and long-term signal forecasting, frequency-phase forecasting, anti-seizure medication efficacy analysis, and radiofrequency thermocoagulation surgery analysis, demonstrating outstanding generalization ability and broad clinical application potential.

编号：SGQNX-2026-1-2

引用格式：Zhang W, Shi Z, Kong W, Gong Y, Lin W, Wu Z, Han Y, Liu Y, Wang T. Locating the epileptogenic zone for drug-resistant epilepsy through neuroelectrophysiological brain network topology. Front Neurosci. 2026;20:1781032. doi: 10.3389/fnins.2026.1781032

通讯作者：刘亚青

Abstract

Introduction: Drug-resistant epilepsy (DRE) constitutes approximately one-third of the epilepsy population, posing a significant challenge due to low seizure freedom rates. Accurate localization of the epileptogenic zone (EZ) is the prerequisite for successful surgery. However, the limitations of conventional visual inspection underscore an urgent need for novel localization strategies based on quantitative brain network topology. **Methods:** This study established a hierarchical analytical framework to independently analyze neuroelectrophysiological signals from scalp EEG (used for macroscopic hypothesis formulation) and stereoelectroencephalography (SEEG, used for mesoscopic confirmation). We included 25 patients with favorable surgical outcomes and constructed brain networks from ictal and interictal recordings. Subsequently, we evaluated the diagnostic value of these network features using machine learning classifiers [including Support Vector Machine (SVM), Random Forest, etc.]. **Results:** In SEEG, the EZ exhibited significantly reduced topological metrics (specifically node degree, clustering coefficient, and local efficiency) compared to non-EZ regions ($P < 0.001$), indicating that the epileptogenic focus is a functionally isolated node. The SVM model based on interictal scalp EEG features achieved superior diagnostic performance (AUC = 0.927, Accuracy = 85.7%, Sensitivity = 85.7%, Specificity = 85.7%). In the SEEG modality, we applied Log-transformation and Z-score normalization to overcome individual variations in implantation schemes. This processing significantly boosted the performance of the interictal SEEG model (SVM)

(AUC = 0.872, Accuracy = 81.9%, Sensitivity = 83.1%, Specificity = 80.7%). **Discussion:** These findings confirm the stability of the EZ's topological signature in the resting state and demonstrate a stepwise workflow: scalp EEG provides coarse localization of the potential EZ to guide SEEG implantation, while SEEG offers more precise surgical recommendations for EZ localization.

编号：SGQNX-2026-1-3

引用格式：Maimaiti A, Feng Y, Abulizi A, Han D, Maimaitituerxun Y, Kasimu M, Pei Y, Feng Z, Abudukeyimu F, Wang Y, Jiang L, Wang X. Unraveling the shared genetic foundations of neurodevelopmental and psychiatric disorders: Insights from comprehensive genome-wide analyses. J Affect Disord. 2025;390:119826. doi: 10.1016/j.jad.2025.119826

通讯作者：王西宪

Abstract

This research explores the shared genetic landscape between neurodevelopmental and psychiatric disorders, uncovering significant genetic correlations and overlaps that hint at a common genetic foundation and potential pleiotropy. Utilizing advanced analytical tools and integrating extensive GWAS data and QTL projects, we identified pleiotropic gene loci bridging the two disorder types. Our findings revealed substantial genome-wide genetic correlations in most trait pairs analyzed, with 6203 SNPs, 94 pleiotropic loci, and 19 colocalized loci pinpointed. Through further analyses, 226 pleiotropic genes were recognized, implicated in critical pathways related to neuronal functions and development. Notably, seven of these genes were validated for their associations across both disorder classes, illustrating their complex roles in gene expression and methylation patterns. This study not only highlights the genetic interconnections between neurodevelopmental and psychiatric disorders but also emphasizes the significance of pleiotropic genes in their pathogenesis, offering new insights into their shared biological basis and paving the way for innovative therapeutic strategies.

编号：YCQDZ-2026-1-1

引用格式：Mao J, Zhang LM, Zhu YL, Gao SJ, Liu MW. The role and mechanism of IL-35 in myasthenia gravis (Review). Int J Mol Med. 2026;57(4):98. doi: 10.3892/ijmm.2026.5769

通讯作者：张林明

Abstract

Myasthenia gravis (MG) is a chronic autoimmune disorder characterized by impaired neuromuscular junction transmission, leading to fluctuating muscle weakness and fatigue. This condition is driven primarily by autoantibodies targeting the acetylcholine receptor at the neuromuscular junction. These antibodies are predominantly generated through a T-cell-dependent pathway, initiating immunomodulatory responses via complement activation. Cytokines and inflammatory mediators also play pivotal roles in the pathogenesis of MG. Recently, increasing attention has been given to the involvement of cytokines in autoimmune diseases. Interleukin-35 (IL-35), an immunoregulatory cytokine, is critical in various inflammatory and autoimmune conditions. It modulates immune responses by promoting Treg proliferation, enhancing their immunosuppressive functions, inhibiting Th17 cell differentiation, and reducing proinflammatory cytokine levels. IL-35 is thus pivotal in the onset and progression of MG. The present review outlines the key functions of IL-35 in MG pathogenesis and the impact of IL-35 on the treatment and prognosis of myasthenia gravis, explores its therapeutic potential, and assesses its prognostic value, offering insights into its mechanisms and implications for treatment.

编号：YCQDZ-2026-1-2

引用格式：Wu Y, Wang J, Wen J, Zhang L. Proteome-Wide Mendelian Randomization Implicates Shared Necroptosis-Ferroptosis Effectors in Causal Pathways of Multiple Sclerosis Susceptibility. Ann N Y Acad Sci.

2026;1556(1):e70162. doi: 10.1111/nyas.70162

通讯作者：张林明

Abstract

Multiple sclerosis (MS) is a chronic neurodegenerative disorder for which dysregulated ferroptosis and necroptosis have demonstrated pathological associations but these lack causal validation in disease susceptibility. This study employed proteome-wide Mendelian randomization (MR) to investigate causal links between ferroptosis/necroptosis pathways, their upstream regulators, immune interactions, and MS risk. Transcriptomic validation utilized bulk RNA-seq and single-cell RNA-seq data. MR identified IFNA4 (OR = 0.24) and TNFAIP3 (OR = 2.0) as key causal ferroptosis/necroptosis-related proteins for MS risk. Analysis revealed 15 upstream regulators significantly associated with MS (FDR < 0.05; e.g., GZMA, CXCL3, APOE, CFB, CA6, KIR2DL2/3). Transcriptomic validation consistently identified ceruloplasmin (CP) as upregulated in MS microglia and lesions. Mediation analyses established two complete causal pathways: an IFNA4-mediated pathway wherein five upstream immune regulators (KIR2DL2, KIR2DL3, CFB, GZMA, and CA6) influence MS susceptibility through IFNA4 regulation, with all component effects statistically significant; and an APOE-driven pathway operating via TNFAIP3, demonstrating significant total effects and near-significant mediator-outcome effects on MS risk. While 59 immune traits were MS-associated, only TNFAIP3 showed a suggestive association with $CD27^{+}$ memory B cells. This study establishes ferroptosis/necroptosis pathways as causal drivers of MS susceptibility, highlighting TNFAIP3, IFNA4, CP, and APOE as therapeutically actionable targets.

编号：YCQDZ-2026-1-3

引用格式：Yan X, He J, Cheng M, Hong S, Jiang L, Han W. Efficacy differences between tocilizumab and ketogenic diet during acute phase of febrile Infection-Related epilepsy syndrome in children: A retrospective cohort study. Epilepsy Behav. 2026;177:110898. doi: 10.1016/j.yebeh.2026.110898

通讯作者：韩慰

Abstract

Febrile infection-related epilepsy syndrome (FIRES) is a rare and devastating subtype of new-onset refractory status epilepticus (NORSE), characterized by rapid progression, high mortality, and poor response to conventional antiseizure therapies. The underlying pathophysiology remains elusive, and no standardized treatment has been established. This retrospective study evaluated 36 pediatric patients diagnosed with FIRES at the Children's Hospital of Chongqing Medical University from January 2019 to December 2024. Patients received first-line immunotherapy alone (n = 17), combined with tocilizumab (n = 8), or with ketogenic diet (KD) (n = 11). Compared to other groups, the tocilizumab cohort showed more rapid seizure control within the first week, improved EEG background activity, and earlier normalization of the neutrophil-to-lymphocyte ratio (NLR), an inflammatory biomarker. At three months, KD demonstrated superior EEG improvement, suggesting a delayed but sustained therapeutic effect. These findings suggest that early initiation of tocilizumab as a second-line therapy following first-line immunotherapy may effectively reduce seizure burden and improve clinical outcomes in children with FIRES.

编号：YCQDZ-2026-1-4

引用格式：Yang M, Hu Y, Wu J, Zhang H, Li J, Wang X, Xu X. Stathmin 1 regulates epileptic seizures by influencing GABAA R-mediated inhibitory synaptic activity. Neurobiol Dis. 2026;220:107310. doi: 10.1016/j.nbd.2026.107310

通讯作者：徐馨

Abstract

Epilepsy is a common neurological disease, and approximately 30% of patients with epilepsy do not respond adequately to antiseizure medications. STMN1 (Stathmin 1) is a potent regulator of microtubule stability, and previous research has shown that STMN1 expression is increased in epileptic brain. However, the effect of STMN1 on

epileptic seizures remains unclear. Our study found that STMN1 expression was associated with GABAA receptor (GABAAR), inhibitory GABAergic neurons and the inhibitory postsynaptic scaffold protein gephyrin. STMN1 downregulation decreased the susceptibility to epileptic seizures in a kainic acid-induced model and pentylenetetrazole-induced kindling model of epilepsy, whereas the overexpression of STMN1 showed opposite effects. More specifically, whole-cell patch-clamp recordings indicate that STMN1 regulated inhibitory synaptic transmission, potentially due to STMN1-mediated postsynaptic GABAAR transport. These results provides the demonstration that STMN1 plays a critical role in the regulation of epileptic seizures, suggesting a molecular mechanism for new therapeutic strategies.

编号：YCQDZ-2026-1-5

引用格式：Li J, Mao Y, Zhang H, Xu X. The associations between epilepsy, metabolism, and their clinical implications. Front Endocrinol (Lausanne). 2026;17:1694550. doi: 10.3389/fendo.2026.1694550

通讯作者：徐馨

Abstract

Epilepsy can cause metabolic disorders, and metabolic abnormalities can also trigger epilepsy, forming a bidirectional pathological cycle. Over the past century, from the earliest use of ketogenic diets to treat epilepsy, it has been confirmed that metabolic intervention can control seizures. Subsequent studies have gradually revealed that metabolic disorders such as glucose abnormality and vitamin B6 deficiency can directly induce epilepsy, while epileptic seizures themselves can cause lactic acidosis, electrolyte imbalance and other internal environment disorders. With the breakthroughs in metabolomics technology, the research on epilepsy and metabolism has entered a systematic stage, and their relationship has attracted increasing attention. However, current reviews mostly focus on the isolated analysis of a single metabolic element (such as iron, vitamin D), lacking a systematic integration of multiple metabolic elements. This review for the first time integrates the changes of seven major metabolic

elements (glucose, lipids, vitamins, minerals, water, adenosine triphosphate, uric acid) in the onset, progression and treatment of epilepsy; summarizes the clinical associations between metabolic diseases (diabetes mellitus, alcoholism, uremia) and epilepsy; reveals the specific metabolic changes in childhood epilepsy; and emphasizes the importance of epilepsy metabolomics data. It provides a reference for basic research and a metabolic monitoring framework for clinicians.

编号：YCQDZ-2026-1-6

引用格式：Liang S, Wang L, Shen K, Wang Z, Zheng X, Duan Q, Shi X, Zhang L, Dai Y, Zou Y, Deng J, Zhang X, Jia H, Liu S, Yang H, Mao Y, Liao X, Zhang C, Chen X. Targeting brain hubs of ictal fast ripple activity to reduce seizures in patients with drug-resistant epilepsy. Sci Transl Med. 2025;17(830):eadq4423. doi: 10.1126/scitranslmed.adq4423

通讯作者：张春青

Abstract

Brain stimulation therapies have been increasingly applied to treat patients with drug-resistant epilepsy or other neuropsychiatric disorders, but identifying effective stimulation targets appropriate for individual patients remains challenging. Using intracranial electrophysiological recordings, we found that fast ripple (FR) activity was tightly correlated with the severity of consciousness impairment during seizures in patients with drug-resistant, consciousness-staged epilepsy. Epileptic network analysis based on FR coincidence across brain regions revealed hubs of ictal FR activity, defined as highly connected nodes, specific to individual patients. A small, exploratory study on eight patients with drug-resistant epilepsy showed that stimulating these hubs reduced FRs, ameliorated consciousness impairments, and reduced seizures during a poststimulation time window of up to 11 days. Moreover, FR hub stimulation showed a lower risk of evoking seizures than stimulating seizure onset zones, a now approved treatment option. These results suggest a potentially safe and effective strategy to alleviate epileptic seizures by stimulating patient-specific FR network hubs.

编号：YCQDZ-2026-1-7

引用格式：Guo Y, Wu J, Luo TG, Song JP, Shi XJ, Zhu M, Shen KF, Yang H, Wang ZJ, Zhang CQ. Brain network characteristics of favorable outcomes following radiofrequency thermocoagulation for drug-resistant epilepsy in periventricular nodular heterotopia patients. BMC Neurol. 2026;26(1):89. doi: 10.1186/s12883-025-04611-9

通讯作者：张春青

Abstract

Objective: This study investigated the brain network characteristics that are associated with favorable outcomes following radiofrequency thermocoagulation (RF-TC) treatment in patients with drug-resistant epilepsy related to periventricular nodular heterotopia (PNH). The goal of the study was to elucidate the potential alterations exhibited by the epileptic networks of PNH patients and provide insights for optimizing surgical strategies. **Methods:** Seven PNH patients were enrolled. Stereoelectroencephalography data collected from four patients were analyzed. The channels were categorized as thermocoagulated (TC; n = 107) or non-thermocoagulated (non-TC; n = 368). Two 200-second epochs were selected for analysis purposes: one preceding RF-TC and one commencing 15 min after RF-TC. The power spectral density (PSD) and functional connectivity (FC) changes were compared between these epochs. To mitigate the potential volume conduction effects amplified by post-RF-TC lesions, the phase lag index (PLI) was employed alongside mutual information (MI) for the FC analysis, providing a more robust assessment of network changes. **Results:** At the 12-month follow-up point, all seven patients achieved Engel class I outcomes. After RF-TC, the PSD decreased significantly across all frequency bands in both TC channels ($p < 0.01$ for all bands) and only θ band ($p < 0.001$) in non-TC channels with a more pronounced reduction observed in the TC channels. An MI-based FC analysis revealed increased FC across almost all bands in the TC channels after RF-TC (δ band: $p = 0.542$; other bands: $p < 0.01$). Conversely,

the non-TC channels exhibited decreased FC in all bands except δ and ripple bands. A PLI-based analysis revealed a significant differences between TC and non-TC channels in the β (p = 0.001) and low-ripple band (p = 0.011) for the post-TC period. **Conclusion:** Nodules and/or the overlying cortex exhibiting epileptic network connectivity may function as a seizure generation unit, potentially facilitated and augmented by other networks. PLI-detected β and ripple band changes may constitute the key phase-synchronous pathway through which RF-TC modulates brain networks.

编号：YCQDZ-2026-1-8

引用格式：Zhu M, Song J, Shi X, Shen K, Wang L, Wang Z, Liu L, Wu Y, Guo Y, Yang X, Liu S, Yang H, He X, Zhang C. Connectivity-based localization and mapping of central epilepsy semiology. Epilepsy Behav. 2025;172:110662. doi: 10.1016/j.yebeh.2025.110662

通讯作者：张春青

Abstract

Objective: Semiology-based preoperative anatomical hypotheses are necessary, yet comprehensive reports on the semiology and its correlation with central subregions in central epilepsy has still lacked. We wished to identify semiologic subgroups and their correlations with central subregions. **Methods:** We retrospectively included 21 patients with central epilepsy identified by stereoelectroencephalography (sEEG). The central region was segmented into 12 subregions using the Human Brainnetome Atlas, and both sEEG data and semiology underwent quantitative analysis. **Results:** We defined three patient groups based on semiologic pattern similarities. Several intriguing anatomical-electroclinical correlations were initially observed, including the involvement of paracentral lobule (PCL) subregion 2 in upper-limb sensations, PCL1 in autonomic signs, and notably, postcentral gyrus (PoG) 2 in orofacial motor signs and precentral gyrus (PrG) 1 in hand sensations, which may be explained by the overlap among motor and sensory cortices, suggesting a reexamination of traditional localizations of somatosensory or motor signs to the PoG or PrG. Furthermore, anatomic structures

initiating ictal signs constructed specific early spread networks. While all patient groups exhibited propagation to the parietal (P) and cingulate cortices (CG), ictal discharges originating from the superior, the posterior-inferior (near the lateral sulcus), and the middle-inferior aspects tended to propagate anteriorly toward the frontal lobe, in a superior direction and to the adjacent insula, and in a bidirectional manner-that is, towards the middle-front and the posterior regions, respectively. **Conclusion:** Localizing semiology to central subregions and mapping clinical patterns to early spread networks allowed central epilepsy dynamics to be realized and helped define the range of epileptogenic anomalies preoperatively.

编号：YCQDZ-2026-1-9

引用格式：Wang Z, Li J, Li Y, Liu X, Lv S, Shu H, Guo Q, Yang X, Zhang C, Yang H, Liu S. Phenotypic individual clusters and metabolic tuber subtypes refine surgical strategy in tuberous sclerosis complex. Brain. 2025:awaf451. doi: 10.1093/brain/awaf451

第一作者：王中科

Abstract

We read with great interest the recent article by Dhawan et al., titled ‘Phenotypic clustering in tuberous sclerosis complex reveals four distinct disease trajectories’, published in Brain.1 In this study, the authors conducted an unsupervised clustering analysis of 29 clinical features in a large cohort of 947 individuals with tuberous sclerosis complex (TSC), identifying four distinct phenotypic subtypes: angiomyolipoma-predominant TSC (cluster 1), TSC with infantile spasms (cluster 2), neuropsychiatric TSC (cluster 3), and a milder phenotype of TSC (cluster 4). This phenotype-driven stratified approach revealed clinically meaningful subgroups that transcend traditional genotype analysis, and it advances clinical decision-making by facilitating precision medicine in TSC. Epilepsy affects 80%–90% of patients with TSC, with nearly two-thirds developing drug-resistant epilepsy.2 We reported the largest multicentre nationwide study on resective epilepsy surgery in patients with TSC and

long-term follow-up in Brain.3 Our findings demonstrated that resective surgery can significantly improve seizure control and quality of life, emphasizing the critical need for precise preoperative assessment tools to identify patients most likely to benefit from surgery. Current TSC management strategies, which primarily rely on genetic test results (TSC1/2 mutations) and clinical features, inadequately accounts for individual variability,4 potentially limiting the effectiveness of surgical interventions.

编号：YCQDZ-2026-1-10

引用格式：Li Y, Liu X, Li J, Xie M, Li S, Huang K, Zhao J, Chen X, He Z, He J, Sun L, Jiang R, Cui C, Wang L, Liu Z, Zhang S, Shu H, Lv S, Zhang C, Zhang D, Wang D, Yang H, Guo Q, Tan H, Yang X, Liu S, Wang Z. Development and validation of interpretable multimodal clinical-radiomics models for predicting epileptogenic foci and surgical outcomes in tuberous sclerosis complex: A multicenter study. PLOS Digit Health. 2026;5(2):e0001259. doi: 10.1371/journal.pdig.0001259

通讯作者：王中科

Abstract

Precise localization and resection of epileptogenic (epi) foci from multiple cortical foci determine surgical outcomes in the tuberous sclerosis complex (TSC). Although the use of intracranial electroencephalography (EEG) for detecting epileptic discharges remains the gold standard for identifying epi foci, its invasiveness and cost limit clinical application. We aimed to develop and validate a noninvasive, clinically applicable predictive model for epi foci identification and surgical outcome assessment in patients with TSC. This multicenter study focused on three retrospective cohorts and one prospective cohort from three comprehensive epilepsy centers from June 2013 to October 2024. Comprehensive clinical and imaging data (CT, MRI, and 18F-FDG PET) of cortical foci were collected. Nineteen individual machine learning (ML) models and three ensemble ML models (voting, averaging and super-learner [SL]) were developed on the basis of the clinical and radiomics features of cortical foci. Model performance

was evaluated by using the area under the curve (AUC), accuracy, precision, specificity, and sensitivity values, along with the F1 score, with additional validation being conducted via decision curve analysis (DCA) and calibration curves. Follow-up data were collected at 1, 3, and >5 years to validate the ability of the ML models to predict long-term postoperative outcomes. Non-epi foci were clustered by using the k-means algorithm to investigate the mechanisms underlying postoperative epileptogenic transformation. A web-based tool was developed to provide a user-friendly interface for clinical application. A total of 665 cortical foci (epi foci, n = 161; non-epi foci, n = 504) were included in this study. The model integrating multimodal clinical-radiomics features performed better than the individual models based only on single-modal clinical or radiomics features did. The ensemble SL model using clinical-radiomics features demonstrated the best stability and superior predictive performance compared to those of individual models and an additional two ensemble models in prospective (AUC: 0.92) and two retrospective cohorts (AUCs: 0.91 and 0.87); moreover, it outperformed previously reported prediction models. In addition, the SL model effectively predicted 1-, 3- and >5-year surgical outcomes (AUCs: 0.93, 0.91, and 0.92, respectively). K-means revealed two clusters of non-epi foci, including those foci with epileptogenic potential and those without, which were potentially confirmed by the follow-up data. The web-based tool significantly increased the accuracy of junior clinicians (from 0.61 to 0.78), which matched the accuracy of senior clinicians (0.80). The multimodal clinical-radiomics model represents a noninvasive tool for predicting epi foci, guiding preoperative evaluation, addressing diagnostic discrepancies and enabling personalized treatment strategies in patients with TSC. The clinical application of artificial intelligence (AI)-driven clinical-radiomics models provides a useful tool and auxiliary reference for clinicians in preoperative epileptogenic foci prediction.

编号：YCQDZ-2026-1-11

引用格式：Wang Z, Liu X, Huang K, Zhang C, Yang H, Yang X, Liu S.

Transmantle sign-like calcified radial lesion on CT serves as a potential imaging feature for epileptogenic foci in tuberous sclerosis complex: a Case Report. Front Radiol. 2026;6:1719889. doi: 10.3389/fradi.2026.1719889

第一作者：王中科

Abstract

The tuberous sclerosis complex (TSC) is an important cause of drug-resistant epilepsy (DRE) in children. According to international TSC diagnostic criteria, multiple cortical tubers are a key driver of DRE in these patients. Surgical resection of epileptogenic (epi) tubers remains an effective treatment for TSC-related DRE, and precise preoperative identification of these tubers is critical for favorable surgical outcomes. We report the case of a 2-year-old girl with TSC who presented for epilepsy surgery evaluation. She had a 1.5-year history of DRE and was unresponsive to multiple antiepileptic therapies. During preoperative assessment, conventional MRI failed to detect clear cortical tubers. However, CT imaging revealed rare bilateral hyperdense transmantle sign (TMS)-like lesions in central brain regions, which are usually associated with focal cortical dysplasia type IIb (FCD IIb) on MRI. Scalp electroencephalogram (EEG) and stereoelectroencephalogram (SEEG) monitoring confirmed that the seizures originated from the CT calcified radial lesions resembling TMS, which were subsequently resected. Neuropathological examination of the resected tissue revealed balloon cells and dysmorphic neurons, consistent with epi tubers. A 3-year postoperative follow-up confirmed that these CT calcified radial lesions resembling TMS were epi tubers. Notably, only 5%–10% of TSC cases show atypical cortical tubers on MRI and traditionally identified via metabolic abnormalities on magnetic resonance spectroscopy (MRS) or hypometabolic changes on positron emission tomography (PET). To our knowledge, no previous TSC case with atypical cortical tubers on MRI has been reported to exhibit CT calcified radial lesions resembling TMS. This case highlights the clinical value of CT-specific features in identifying epi tubers, especially when cortical tubers are atypical on conventional MRI.

编号：YCQDZ-2026-1-12

引用格式：Huang K, Li J, He Z, Li Y, Liu X, Wan Y, Yang X, Sang D, Li S, Wang Z, Liu S. G Protein-Coupled Receptor 32 Contributes to Inflammation Resolution and Neuronal Excitability Dysfunction in Patients With Focal Cortical Dysplasia IIb and Tuberous Sclerosis Complex. Neuropathol Appl Neurobiol. 2026;52(1):e70062. doi: 10.1111/nan.70062

通讯作者：王中科

Abstract

Background: Focal cortical dysplasia IIb (FCDIIb) and tuberous sclerosis complex (TSC) show persistent neuroinflammation that promotes epileptogenesis and epilepsy progression, suggesting that endogenous resolution of inflammation is inadequate to relieve neuronal network hyperexcitability. G- protein- coupled receptor 32 (GPR32) is a key regulator of inflammation resolution and we aimed to explore the roles of GPR32 in cortical lesions of patients with FCDIIb and TSC. **Method:** We examined the expression and distribution of GPR32 in patients with FCDIIb and TSC and its effects on human microglial cell activation, inflammation and the electrophysiological properties of neurons. **Results:** GPR32 and Resolvin D1 expression was significantly lower in cortical lesions of patients with FCDIIb and TSC than in controls and were negatively correlated with seizure frequency. GPR32 was widely distributed in neurons and microglia and was nearly absent in astrocytes. Furthermore, the Src homology region 2- containing protein tyrosine phosphatase 2 (SHP2) pathway was downregulated in patients with FCDIIb and TSC. The GPR32/SHP2/nuclear factor-kappa B pathway inhibited the M1 transformation of microglia to produce numerous pro- inflammatory mediators and promoted M2 polarisation. GPR32 also regulated neuronal excitability by reducing the amplitude and frequency of spontaneous excitatory postsynaptic currents. **Conclusion:** Our results suggest that GPR32 may help control epilepsy in patients with FCDIIb and TSC.

编号：YCQDZ-2026-1-13

引用格式：Wu Q, Barker-Haliski M. Benchmarks to Breakthroughs: How the 2007 Epilepsy Research Benchmarks Reframed Scientific and Clinical Trajectories of 21st-Century Epilepsy Care. Epilepsy Curr. 2026:15357597251412106. doi: 10.1177/15357597251412106

第一作者：吴倩

Abstract

The 2007 Epilepsy Research Benchmarks, developed through the National Institute of Neurological Disorders and Stroke (NINDS) Curing Epilepsy initiative, established a strategic framework that reshaped priorities across epilepsy research and clinical care. This commentary examines how these benchmarks guided advances in understanding epileptogenesis, therapeutic development, comorbidity management, and risk reduction for epilepsy-related mortality. Over the past two decades, benchmark-driven initiatives accelerated progress in molecular target discovery, improved animal models, neuroimaging technologies, and translational screening platforms such as the Epilepsy Therapy Screening Program. The benchmarks also broadened the field’s focus beyond seizure control to include cognitive, psychiatric, and behavioral comorbidities, as well as prevention strategies for sudden unexpected death in epilepsy (SUDEP). In parallel, standardization efforts through common data elements and global collaborative networks strengthened data harmonization and clinical research infrastructure. Collectively, the 2007 benchmarks helped shift epilepsy research toward disease-modifying and preventive strategies, shaping a coordinated roadmap that continues to influence international efforts to improve outcomes for people with epilepsy.

编号：YCQDZ-2026-1-14

引用格式：Wu Q, Guignet M, Vuong J, White HS, Kerr WT, Shih EK, Ngo LY,

Carrazana E, Rabinowicz AL. Preclinical signal for a disease-modifying effect on seizure cluster severity with intermittent diazepam treatment. Epilepsia. 2026;67(3):1497-1508. doi: 10.1002/epi.70051

第一作者：吴倩

Abstract

Objective: In epilepsy, daily treatment provides only symptomatic seizure control, leaving a significant unmet need for a treatment that affects the underlying predisposition to seizures. Here, in a first-of-its-kind study, we test the hypothesis that intermittent treatment of seizure clusters with diazepam in the kainic acid post-status epilepticus rat model of acquired epilepsy has an enduring effect on the seizure cluster phenotype, suggestive of potential disease modification. **Methods:** Following kainic acid-induced status epilepticus, rats with epilepsy were monitored for occurrence of seizure clusters (≥2 seizures in 24 h) for a 3-week baseline period before entering a 6-week treatment period using a previously established multidose regimen of diazepam (n = 7) or vehicle (n = 9) upon identification of a seizure cluster. In a subsequent 2-week outcome period during which no rats received diazepam, we evaluated changes in seizure cluster size, burden (cluster size × severity), duration, and other phenotype parameters. **Results:** A total of 3396 seizures and 216 seizure clusters were included for analysis. During the outcome period, time between seizures in a cluster (also interseizure interval [ISI]) was significantly longer in the diazepam group (log ISI = .25 longer, SE = .08, $p < .0001$), and the proportion of clustered seizures with an ISI of ≤30 min increased in the outcome period in the vehicle group ($p = .023$) but was stable in the diazepam group. Despite the occurrence of rebound seizures during the treatment period, improvement in several phenotypical parameters, including severity and proportion of seizures in a cluster, supported a positive impact of intermittent diazepam treatment on seizure cluster biology. **Significance:** Changes in several seizure cluster phenotypical parameters were suggestive of an enduring disease-modifying effect of diazepam, despite an apparent rebound effect of intermittent diazepam treatment on seizure frequency. Further study is warranted using a model incorporating a background

antiseizure medication regimen to potentially attenuate the unexpected rebound seizures.

编号：YCQDZ-2026-1-15

引用格式：Wu Q, Zhu L, Dai S, Xu J, Ge D. Intermittent immunoadsorption in critically ill patients with neuroimmunological disorders: a retrospective study. Front Neurol. 2025;16:1666042. doi: 10.3389/fneur.2025.1666042

通讯作者、第一作者：吴倩

Abstract

Objectives: This study aimed to evaluate the efficacy and safety of intermittent immunoadsorption (IA) in critically ill patients with refractory autoimmune neurological disorders. **Methods:** We retrospectively reviewed 13 patients admitted to the neurocritical care unit with severe autoimmune encephalitis, Guillain-Barré syndrome, neuromyelitis optica spectrum disorders, or chronic inflammatory demyelinating polyneuropathy, all of whom had failed first-line immunotherapy (intravenous methylprednisolone and/or intravenous immunoglobulin). IA was administered intermittently, with schedules individualized based on clinical status. **Results:** The modified Rankin Scale (mRS) improved significantly following IA ($p = 0.02$), while the Acute Physiology and Chronic Health Evaluation II scores (APACHE II) remained stable ($p = 0.95$). Serum IgG levels declined by a median of 55.6%. Pathogenic antibody negativity was achieved in 65% of plasma and 38% of cerebrospinal fluid samples. Although 92% experienced treatment interruptions (e.g., infection and hypotension), IA was generally well tolerated and not permanently discontinued. **Discussion:** This study supports the feasibility and clinical utility of intermittent IA in critically ill patients with treatment-refractory neuroimmunological disorders. Despite frequent complications, flexible scheduling allowed continued therapy with sustained benefit. These findings highlight a potentially adaptable treatment strategy in a population often excluded from therapeutic interventions and suggest that IA warrants further study in neurocritical care settings.

编号：YCQDZ-2026-1-16

引用格式：Su M, Zhang Y, Feng Z, Li W, Liu Y, Xu C, Zhao C, Kwan P, Tian X, Li L. Permethrin induces epileptic susceptibility via activation of Na+ channels and rise in glutamate. Commun Biol. 2025;8(1):1644. doi: 10.1038/s42003-025-09037-0

通讯作者：田鑫

Abstract

Permethrin, a common pyrethroid insecticide, is extensively used to control insect pests and has been widely detected in the environment. A growing body of epidemiological studies has revealed a substantial increase in the prevalence of epilepsy among individuals highly exposed to pesticides. However, the mechanism of action of permethrin in epilepsy remains elusive. In this study, we investigate the effects of permethrin on zebrafish and mice exposed to environmentally relevant concentrations. Acute exposure to permethrin induces dose-dependent epileptiform symptoms in zebrafish, including whirlpool-like movement, clonus-like convulsions, nystagmus and neuronal hyperactivity. Extended exposure results in neuronal hyperexcitation, impaired neurogenesis and enhanced glial cell hyperplasia in zebrafish. Additionally, pre-exposure to permethrin significantly increases susceptibility to seizures in mice. Mechanistically, the effects of permethrin are conserved across different species, promoting the activation of sodium ion channels, particularly Nav1.6. This activation increases glutamate levels, which play a critical role in epileptiform symptoms. Our findings suggest that permethrin exposure leads to the activation of sodium ion channels and disrupts the balance between excitatory versus inhibitory neurotransmitters, resulting in seizure-like symptoms and increased susceptibility to epilepsy. Sodium channels and neurotransmitter balance may be protective targets against permethrin exposure.

编号：YCQDZ-2026-1-17

引用格式：Chen B, Zhao Y, Shen N, Liu F, Jiang L, Ma Y, Tian X, Kwan P. Effects of general anesthetics on seizure termination, mortality, and neurological prognosis in a rat model of pediatric refractory status epilepticus: A comparative randomized controlled study. J Adv Res. 2025:S2090-1232(25)00872-0. doi: 10.1016/j.jare.2025.10.076

通讯作者：田鑫

Abstract

Introduction: Refractory status epilepticus (RSE) is a serious neurological emergency in children associated with significant mortality and morbidity. General anesthetics (GAs) are routinely used to treat RSE. However, the optimal selection of GAs for pediatric RSE remains unknown. **Objective:** We aimed to identify the optimal agent by comparing the efficacy and safety of different GAs in a rat model of RSE. **Methods:** Sprague-Dawley rats at postnatal day 20 were subjected to lithium-pilocarpine-induced SE. Animals that developed RSE (pharmacoresistant to the first- and second-line therapies) were randomized to receive one of five GAs (midazolam, propofol, dexmedetomidine, esketamine, or sevoflurane) as the third-line therapy or vehicles. The latency to SE termination (time from the initiation of GAs to SE termination) and 24-hour mortality were assessed. On Day 5 following SE, a series of behavioral tests were performed to evaluate the behavior and cognition. **Results:** All five GAs effectively terminated the RSE, with propofol and sevoflurane having the shortest latencies to SE termination. GAs significantly improved survival after the complete termination of SE. Additionally, GAs reduced the cognitive deficits caused by SE, especially episodic memory, without affecting the behavioral abnormalities. Finally, we proposed a multi-dimensional model that integrated the efficacy, safety, and effect of GAs on neurological prognosis, suggesting that sevoflurane might be the optimal treatment. **Conclusion:** Our study showed that treatment with GAs effectively terminated RSE and improved survival and short-term cognition. Our proposed model suggests that

volatile anesthetics may be the optimal choice for treating RSE. These results may be used to inform the design of comparative clinical trials of GAs for RSE in children.

编号：YCQDZ-2026-1-18

引用格式：Zhang N, Chen S, Jiang J, Jiang H, Wang Q, Raju S, Schumacher JG, Lu J, Lian Y, Zhang Y, Xu Y, Zhang L, Liu Y, Li J, Zhang Y, Wang Y, Gu Y, Wang T, Tian X. Multiomic insights into the MPO-mediated NET formation pathway in alcohol-induced epilepsy risk. Genes Dis. 2025;13(3):101917. doi: 10.1016/j.gendis.2025.101917

通讯作者：田鑫

Abstract

Epilepsy is a highly prevalent chronic central nervous system disorder that imposes substantial societal and economic burdens. Inconsistent associations of alcohol consumption, identified as a major global health risk factor, with epilepsy risk have been reported. The aim of the present study was to assess the relationship between alcohol use and epilepsy and to identify potential underlying mechanisms, with a particular focus on the role of neutrophil extracellular traps (NETs), using an integrated multiomic approach. We assessed the global risk of alcohol consumption for epilepsy using data from the Global Burden of Disease Study 2021, and we conducted a Mendelian randomization (MR) analysis to evaluate causality. Additionally, we employed machine learning algorithms and protein–protein interaction networks to identify key genes. Our results indicate that alcohol consumption significantly contributes to the risk of epilepsy, as confirmed by MR analysis (odds ratio = 1.30, 95% confidence interval 1.06–1.60; p = 0.011). Functional enrichment analysis revealed pathways related to NET formation, whereas machine learning identified key genes such as myeloperoxidase (MPO) and neutrophil elastase. Animal and molecular experiments confirmed that acute alcohol exposure increases the susceptibility to epileptic seizures, whereas the MPO inhibitor 4-aminobenzoic acid hydrazide showed

therapeutic potential for alcohol-induced epilepsy. This study provides novel insights into the role of NETs in alcohol-induced epilepsy and highlights potential therapeutic targets, thereby contributing to the development of innovative treatment strategies for epilepsy prevention and management.

编号：YCQDZ-2026-1-19

引用格式：Liu Y, Song F, Guo Y, Xu S, Hu L, Yuan Z, Duan R, Meng Y, Ke P, Tian X, Xiao F. Crotonylation of STXBP1 exacerbates seizure susceptibility by impairing GABAergic synaptic transmission. Cell Death Differ. 2026. doi: 10.1038/s41418-026-01688-8

通讯作者：田鑫

Abstract

Temporal lobe epilepsy (TLE) is the most common and severe form of epilepsy in adults; however, the underlying pathological mechanisms remain unclear. Post-translational modifications (PTMs) of proteins are increasingly recognized to contribute to the development and maintenance of epilepsy; however, the functional significance of lysine crotonylation (Kcr) in epilepsy formation is still unclear. Herein we found that high levels crotonylation promote seizure susceptibility. Through quantitative analysis of global crotonylome in the hippocampus of control and epileptic mice, we identified a significant decrease in K98 crotonylation (K98cr) of syntaxin-binding protein 1 (STXBP1) in epileptic mice hippocampus. In Stxbp1K98Q knock-in mice, the upregulation of STXBP1 K98cr reduces the binding with syntaxin-1B(STX1B), leading to a decreased assembly of soluble NSF attachment protein receptors (SNAREs) in presynaptic active zone and subsequent inhibition vesicle release, thereby promoting epilepsy formation. Additionally, we found that reduced E1A-binding protein p300 (p300)-mediated crotonylation is a major cause of the altered STXBP1 crotonylation in the hippocampus of epileptic mice. Furthermore, chemically inhibiting the generation of crotonyl-CoA alleviate the seizure susceptibility of mice. In summary, our results

indicate that crotonylation is an important regulatory factor in the process of epilepsy formation and modulating crotonylation may provide new insights for epilepsy treatment.

编号：YCQDZ-2026-1-20

引用格式：Zhang C, Hu L, Zhang H, Yang M, Zhang Y, Zhang N, Xu Y, Zhao Y, Ren L, Guo H, Li W, Wang X, Yang Y, Tian X. DIRAS2 modulates MAPK pathway-mediated ferroptosis to regulate excitation/inhibition balance and seizure susceptibility. Proc Natl Acad Sci U S A. 2026;123(13):e2516011123. doi: 10.1073/pnas.2516011123

通讯作者：田鑫

Abstract

Epilepsy is a common neurological disorder that is widely believed to be associated with an imbalance between neuronal excitation and inhibition (E/I). DIRAS2, a Ras-related GTPase, has not been well understood regarding its role and function within the nervous system. In this study, we found that DIRAS2 is downregulated in the hippocampus during the epileptogenesis phase in a kainic acid-induced epilepsy model, while it is upregulated during the chronic phase in this epilepsy model and in patients with temporal lobe epilepsy. Overexpression of DIRAS2 alleviates epileptic seizure susceptibility and activity, whereas knockdown of DIRAS2 has an opposite effect. Whole-cell patch-clamp recordings reveal that DIRAS2 reduces the neuronal E/I ratio and alleviates neuronal hyperexcitability. Mechanistically, quantitative proteomic analysis reveals that ferroptosis is involved in mediating the effects of DIRAS2. Knockdown of DIRAS2 can exacerbate ferroptosis, while overexpression protects against ferroptosis in both in vivo and in vitro studies. Ferrostatin-1, a ferroptosis inhibitor, can rescue the E/I imbalance and epileptic behavioral changes induced by DIRAS2 knockdown. Finally, we found that DIRAS2 regulates ferroptosis by inhibiting the extracellular signal-regulated kinase/p38 mitogen-activated protein kinase pathway

in epileptic mice. In summary, our study demonstrates the role of DIRAS2 in epilepsy and provides a potential target for epilepsy treatment.

编号：YCQDZ-2026-1-21

引用格式：Zhong S, Lian Y, Yi H, Jia K, Zhang N, Kwan P, Yang Y, Tian X. Global burden of epilepsy attributable to neonatal disorders in children from 1990 to 2021. iScience. 2026;29. doi: 10.1016/j.isci.2026.115296

通讯作者：田鑫

Summary

This study provides a comprehensive assessment of the global burden of childhood epilepsy attributable to neonatal disorders, using data from the Global Burden of Disease Study 2021. From 1990 to 2021, the age-standardized prevalence rate and number of years lived with disability increased globally, with higher burdens observed among boys and in regions with lower Sociodemographic Index levels. Preterm birth was identified as the most significant neonatal cause of childhood epilepsy. These findings depict the burden of epilepsy attributable to neonatal disorders in children and underscore the need for targeted perinatal care strategies, particularly in low-resource settings, to reduce the long-term neurological sequelae in children worldwide.

欢迎大家引用上述论文

编号：YGQG-2026-1-1

引用格式：Jia Y, Chen H, Zou Q, Chen S, Li J, Chen Y, Lu L, Hong F, Jia S, Jing X, Ren J, Muhammad F, Mi J, Duan J, Liao J, Liu Q, Xu F, Kenny PJ, Han MH, Wang L, Chen Z, Cao D, Liu XA. Gut-brain cholinergic signaling mediates the antiseizure effects of Bacteroides fragilis. Neuron. 2026;114(6):1021-1044.e9. doi: 10.1016/j.neuron.2025.11.029

通讯作者：操德智

Abstract

Gut dysbiosis has been implicated in epilepsy, yet probiotic efficacy and mechanisms remain unclear. Here, we identify that Bacteroides fragilis (B. fragilis) is markedly reduced in children with epilepsy and show that oral B. fragilis administration suppresses seizures in both pentylenetetrazole- and kainic-acid-induced mouse models. Mechanistically, B. fragilis activates colonic choline acetyltransferase-positive (ChAT+) cells and enhances gut-vagus-brain cholinergic signaling, as demonstrated by vagal recordings, pharmacological blockade, and chemogenetic manipulation, identifying a colonic ChAT+-nodose ganglion circuit mediating seizure suppression. Its antiseizure effects associate with enriched intestinal Lactobacillus colonization. A randomized clinical trial (CHiCTR2100042203) further confirms the therapeutic efficacy of B. fragilis in pediatric refractory epilepsy. These findings define a gut-brain cholinergic pathway through which B. fragilis exerts antiseizure effects and establish a mechanistic basis for microbiota-targeted therapies in epilepsy.

编号：YGQG-2026-1-2

引用格式：Wang RX, Afzal A, Jing XY, Zhou Y, Feng JX, Chen ZX, Cao DZ, Liu XA. Intergenerational effects of the microbiota on neurodevelopment: mechanisms and therapeutic perspectives. Acta Pharmacol Sin. 2026. doi: 10.1038/s41401-025-01693-6

通讯作者：操德智

Abstract

Neurodevelopment is governed by precisely timed biological processes that are sensitive to environmental influences across generations. Among these, the gut microbiota (GM) has emerged as a key regulator of neurodevelopmental trajectories, not only within individuals but also through intergenerational transmission. This review highlights the emerging significance of the GM in shaping offspring brain and behavior, emphasizing its capacity to mediate maternal influences across generations. We first summarize the temporal and intergenerational effects of GM on host physiology and neurobehavioral outcomes. We then explore the mechanistic basis of neuro-microbial-immunometabolic interactions including epigenetic regulation, neurotransmitter modulation, neuroinflammation and intestinal barrier function in the context of the microbiota-gut-brain axis. Particular attention is given to how these mechanisms mediate the long-term impact of maternal states-such as stress, diet and inflammation-on offspring neurodevelopment. We further highlight the translational gap from animal models to humans and propose integrating multi-omics, computational modeling, and clinical approaches to define developmental windows and guide precision microbiota-based interventions for neurodevelopmental disorders. By elucidating how microbiota influence neurodevelopment across generations, this review aims to inform the development of novel microbial and pharmacological therapies to promote brain health from the maternal period through early offspring life.

编号：YGQG-2026-1-3

引用格式：Wang T, Ouyang S, Zou D, Dong Z, Tan Z, Yang H, Liao J, Cao D, Zhang Y. Novel PAK1 variants related to a variable phenotypic spectrum ranging from mild developmental delay to infantile epileptic spasms syndrome. Seizure. 2025;133:88-95. doi: 10.1016/j.seizure.2025.10.010

通讯作者：操德智

Abstract

Objective: To identify the novel variants and explore the new phenotypes of patients with PAK1-related disorder. **Methods:** Five patients with PAK1 variants were identified by whole-exon sequencing. Damaging effects of variants were analyzed using protein modelling. **Results:** In this study, 5 patients were identified with 5 de novo PAK1 variants, including p.Ile312Ser, p.Asp407Asn, p.Met453Thr, p.Leu470Pro, and p.Ile476Thr. All variants were missense, and one of which was a mosaic variant (Leu470Pro), with a variant allele fraction of 13.4 % (13/97). Four of five patients with PAK1 variants had epilepsy, the seizure types included focal seizures, generalized tonic-clonic seizure and epileptic spasms. One patient diagnosed with infantile epileptic spasms syndrome (IESS). Four patients had macrocephaly. One patient only had mild developmental delay (DD) and normal head circumference. All missense variants identified in this study were predicted to be "damaging" by multiple in silico tools and to alter the hydrogen bonds with surrounding residues and/or protein stability. Notably, the variant Asp407Asn associated with a milder phenotype is predicted to have increased hydrogen bonds with ATP in contrast to our other reported variants. Spatial and temporal expression analysis showed that PAK1 had three peak expressions in infant, adolescent and early adult brain subregions. Collectively, in our study (n = 5) and published studies (n = 11), all variants were missense variants. PAK1-related disorders encompass a wide phenotypic spectrum, including macrocephaly, epilepsy and DD/intellectual disability (ID). Seizures were observed in 81.25 % (13/16) of patients, and 53.8 % (7/13) patients with epilepsy had febrile seizure. **Conclusions:** All variants of PAK1-related disorders were missense variants. In this study, five de novo variants were included, and Leu470Pro was the first reported mosaic variant in PAK1. PAK1-related disorders encompass a wide phenotypic spectrum, including macrocephaly, epilepsy and DD/ID. IESS is a rare newly recognized phenotype of PAK1-related epilepsy. More than half of patients with epilepsy had febrile seizure.

编号：YGQG-2026-1-4

引用格式：Chen J, Zhang Y, Zhang H, Zhang J, Wang N, Guo X, Liu Y, Liu Y, Zhang X, Chen Z, Ni G. Neuroinflammation in GAD65 Antibody-Associated Epilepsy Measured Using [18F]DPA-714 PET/MRI. Ann Clin Transl Neurol. 2026. doi: 10.1002/acn3.70324

通讯作者：陈子怡 倪冠中

Abstract

The timing for initiating immunotherapy in patients with glutamic acid decarboxylase 65 (GAD65) antibody-associated epilepsy is a challenge. We used the translocator protein radioligand [^{18}F]DPA-714 and PET to evaluate brain microglial activation. [^{18}F]DPA-714 PET brain images of 11 patients with GAD65 antibody-associated epilepsy and seven controls were analyzed. Patients with acute symptomatic seizures presented higher [^{18}F]DPA-714 SUVR in the hippocampus than did controls and patients with chronic temporal lobe epilepsy. Our findings highlight that [^{18}F]DPA-714 PET/MRI represents a useful tool to assess neuroinflammation in patients with GAD65 antibody-associated epilepsy.

编号：YGQG-2026-1-5

引用格式：Huang X, Wu WL, Song J, Tian Y, Zhou Y, Wei S, Yu B, Qin L, Yang S. NBEA gene variant in a child with developmental disorder and epilepsy: a case report. Front Neurosci. 2025;19:1662363. doi: 10.3389/fnins.2025.1662363

第一作者：黄晓利

Abstract

The *NBEA* gene encodes Neurobeachin, a brain-specific kinase-anchoring protein that plays a critical role in vesicle trafficking and synaptic regulation. Pathogenic variants in *NBEA* are definitively associated with neurodevelopmental disorders accompanied by epilepsy, including intellectual disability, autism spectrum disorder, and myoclonic-astatic epilepsy-like phenotypes. Most reported disease-causing variants are *de*

novo loss-of-function mutations, and although genotype-phenotype correlations remain limited, early-onset generalized seizures are frequently observed. Here, we describe a Chinese child presenting with global developmental delay and recurrent seizures with febrile sensitivity. Brain magnetic resonance imaging revealed no structural abnormalities, while electroencephalography showed epileptiform abnormalities. Genetic analysis identified a *de novo* nonsense variant in the *NBEA* gene: c.4715C > A [p.(Ser1572Ter)]. According to the American College of Medical Genetics and Genomics guidelines, the variant was classified as pathogenic. *NBEA* mutations are associated with neurodevelopmental disorders with or without early-onset epilepsy. Although no additional pathogenic variants were identified in the exome, the influence of other undetected genetic or epigenetic modifiers on the observed phenotype cannot be excluded. This case therefore refines the phenotypic spectrum of *NBEA*-related disorders, emphasizing that the c.4715C > A [p.(Ser1572Ter)] variant may be associated with developmental impairment and epilepsy with possible febrile sensitivity.

编号：YGQG-2026-1-6

引用格式：Song J, Wu W, Tian Y, Qin L, Wei S, Yu B, Su H, Huang L, Liu W, Huang X. Case Report: Compound heterozygous KCTD7 variants in two siblings presenting with myoclonic epilepsy and ataxia. Front Neurosci. 2025;19:1670008. doi: 10.3389/fnins.2025.1670008

通讯作者：黄晓利

Abstract

Objective: Biallelic variants in KCTD7 have been associated with progressive myoclonic epilepsy (PME), a rare autosomal recessive disorder characterized by early-onset epilepsy, cognitive decline, myoclonus, and ataxia. **Methods:** Whole-exome sequencing was first performed in the elder sister to identify candidate variants, followed by in silico pathogenicity prediction. Sanger sequencing was then used to validate the variants in both parents and the younger brother. **Results:** We report two

siblings with progressive myoclonic epilepsy (PME) carrying compound heterozygous KCTD7 variants: c.334C > T (p.Arg112Cys), a paternally inherited variant previously reported in homozygous form and currently classified as likely pathogenic, and c.640C > T (p.Arg214Trp), a novel maternally inherited variant currently classified as of uncertain significance. Both patients presented between 2 and 3 years of age with gait instability, myoclonic seizures, and developmental regression. EEG revealed background slowing, multifocal spike– slow wave discharges, and electrical status epilepticus during sleep. Brain MRI findings were initially unremarkable despite progressive neurological deterioration. Whole-exome sequencing and Sanger validation confirmed the variants and their segregation. In silico tools predicted both variants to be deleterious, and structural modeling using PyMOL and I-Mutant 3.0 demonstrated that both variants likely disrupt local residue interactions and reduce protein stability. Both patients received antiepileptic therapy and immunomodulatory treatment, including intravenous methylprednisolone and immunoglobulin. The proband achieved seizure control and improved gait following immunotherapy, though cognitive deficits persisted. The younger sibling exhibited a more severe disease course, with progressive cognitive decline, speech and visual impairment, and loss of independent ambulation, despite partial seizure control. These findings expand the genetic and phenotypic spectrum of KCTD7-related PME and suggest that immunotherapy may confer partial clinical benefit in selected cases. **Conclusion:** This case expands the variant spectrum of KCTD7-related disorders and emphasizes the utility of comprehensive genetic testing in early-onset neurodegenerative epileptic syndromes. Functional studies are needed to clarify the clinical significance of the novel KCTD7 variant.

编号：YGQG-2026-1-7

引用格式：Shen J, Tan H, Wu B, Luo J, Yang G, Weng X, Guo Q, Liang J. The SEEG brain network predicts epileptic surgical outcomes of radiofrequency

thermocoagulation. Epilepsy Res. 2026;219:107715. doi: 10.1016/j.eplepsyres.2025.107715

通讯作者：郭强

Abstract

Predicting the postoperative outcome of stereoelectroencephalography-guided radiofrequency thermocoagulation (SEEG-guided RF-TC) remains challenging despite its increasing use in epilepsy treatment. Although SEEG-guided RF-TC has attracted extensive clinical interest, reliable biomarkers for treatment efficacy are still lacking. This study aims to address this gap by analyzing the altered brain network to predict postoperative outcome. Thirty-one focal cortical dysplasia epileptic patients who underwent RF-TC based on SEEG were enrolled in this study. They were included in the favorable outcome and poor outcome groups according to the follow-up. Partial Directed Coherence and Directed Transfer Function were applied to construct SEEG brain networks, and then brain network features were extracted. Subsequently, the differences in the presurgical and postsurgical brain network features were compared using the Wilcoxon test in the favorable and poor outcome groups, respectively. Finally, four machine learning models were applied to predict the outcome of RF-TC. After RF-TC surgery, the Characteristic Path Length (L) and average Betweenness Centrality (BC) increased while the average Clustering Coefficient (C) and Assortativity Coefficient (R1, R2) decreased in the favorable outcomes group. In contrast, there were no significant changes in the patient group with poor outcomes. The Support Vector Machine (SVM) model achieved the highest performance, with accuracy, sensitivity, specificity, and ROC values of 0.887, 0.821, 0.920, and 0.879, respectively. This study sheds light on the mechanisms of epilepsy from the perspective of brain networks and introduces a novel therapeutic strategy by altering network features. These feature alterations can also support machine learning models in effectively distinguishing favorable from poor outcomes.

编号：ZWGM-2026-1-1

引用格式：Chen B, Guo J, Qiu Z, Shen B, Shi Y, Luo H, Jiang L, Wang Y, Chen L, Su P, Chen X, Fang J. Time-Dependent Effect of Anti-seizure Medications on Bone Metabolism in Patients with Epilepsy: A Cross-Sectional Study. Neurol Ther. 2026;15(1):93-112. doi: 10.1007/s40120-025-00853-4

通讯作者：方嘉佳

Abstract

Introduction: Patients with epilepsy (PWE) face an elevated risk of osteoporosis and bone fractures. This study aims to elucidate bone metabolic alterations in PWE and identify early detection biomarkers and contributing factors. **Methods:** This cross-sectional study analyzed PWE from the Epilepsy Clinical database stratified by anti seizure medication (ASM) exposure duration. We analyzed bone turnover markers (BTMs), including 25-hydroxy vitamin D, osteocalcin (OC), procollagen type 1 N-terminal propeptide (P1NP), β-crosslaps (β-CTX) and β-CTX/OC ratio. The effects of epilepsy and ASMs on bone metabolism were analyzed by XGBoost model and SHapley Additive exPlanations (SHAP). Finally, we analyzed the mediation analyses assessing inflammatory pathway contributions. **Results:** A total of 476 PWE were included in this study. Compared to ASM-naïve PWE, those receiving > 2 years of ASM therapy exhibited a reduced β-CTX/OC ratio ($p = 0.002$), while P1NP levels declined only after > 10 years of treatment ($p < 0.001$). Longitudinal data revealed a continued annual decline in the β-CTX/OC ratio during the 2-year follow-up period. After adjusting for confounders, longer ASM exposure duration was significantly correlated with decreased P1NP, β-CTX and β-CTX/OC ratio levels ($\beta = -1.74$, 95% CI - 2.56 to - 0.92; $p < 0.001$). XGBoost-SHAP analysis identified valproic acid (VPA), oxcarbazepine (OXC) and history of status epilepticus as key contributors to β-CTX/OC ratio variability. Polytherapy had a more pronounced effect than monotherapy, particularly when levetiracetam was combined with VPA or OXC. Mediation analysis demonstrated that platelet-to-lymphocyte ratio and neutrophil-to-lymphocyte ratio mediate epilepsy/ASM-related bone metabolic alterations. **Conclusion:** PWE exhibit

dynamic bone metabolic alterations. Following > 2 years of ASM therapy, osteoclast inhibition precedes the onset of osteoblast dysfunction. Prolonged ASM exposure eventually reduces bone formation markers, indicating progressive impairment of osteoblastic function and a concomitant decline in bone-forming capacity. Consequently, the β-CTX/OC ratio represents a pivotal early biomarker for monitoring bone health deterioration, demonstrating significant clinical utility.

编号：ZWGM-2026-1-2

引用格式：Shen B, Jiang L, Shi Y, Guo J, Chen B, Qiu Z, Wang S, Yuan Z, Fang J. Cognitive Impairment in Temporal Lobe Epilepsy: Alterations in the Basal Forebrain Cholinergic System. Brain Behav. 2025;15(11):e71090. doi: 10.1002/brb3.71090

通讯作者：方嘉佳

Abstract

Objective: Temporal Lobe Epilepsy (TLE) often leads to cognitive decline. The Basal Forebrain Cholinergic System (BFCS), essential for memory processes, may play a critical role. This study investigates BFCS alterations, associated white matter tracts, and cognitive correlations in TLE patients. **Methods:** We analyzed 100 unilateral TLE patients and 25 healthy controls. Neuropsychological assessments included the Wechsler Memory Scale-Revised and the Rey Complex Figure Test. Regions of interest (ROIs), such as the hippocampus, amygdala, and dorsolateral prefrontal cortex, were identified using the AAL3 atlas and SPM 8 Anatomy toolbox. BFCS tract integrity was assessed using FSL probabilistic tractography, focusing on FA and MD values. **Results:** Both left TLE (LTLE) and right TLE (RTLE) groups displayed significant deficits in memory quotient, verbal/nonverbal memory, and visuospatial working memory, with hippocampal sclerosis (HS) patients showing greater impairment. LTLE patients exhibited extensive BFCS impairment versus controls, with reduced FA (bilateral hippocampus/amygdala, all $p < 0.01$) and elevated MD (bilateral hippocampus $p < 0.05$; bilateral amygdala $p < 0.01$; DLPFC $p < 0.05$). RTLE showed

localized damage: decreased FA (left hippocampus/right amygdala, both $p < 0.05$) and increased DLPFC MD ($p < 0.05$). HS intensified BFCS-right hippocampal and bilateral amygdala damage in TLE. Correlation analyses indicated positive associations between BFCS-right hippocampal FA and verbal and nonverbal memory, whereas its MD negatively correlated with nonverbal memory decline. BFCS-right amygdala FA correlated with both memory domains, and BFCS-DLPFC MD inversely correlated with memory quotient. Elevated MD in the right Ch4 to ipsilateral hippocampal tracts served as a sensitive imaging biomarker for nonverbal memory impairment. **Conclusion:** TLE patients exhibit cognitive and visual-spatial memory deficits, with BFCS tract damage being more pronounced in LTLE and HS patients. The BFCS and its projection fibers demonstrate potential correlations with cognitive function and may be involved in the neural mechanisms of cognitive impairment in TLE.

编号：ZWGM-2026-1-3

引用格式：Song H, Du BQ, Ge Y, Chen YH, Geng SY, Yang YY, Wang ZJ, Long XP, Xu CA, Shao XT, He CM, Zhang YX, Chen C, Wang S, Hu Y, Xu S, Li R, Ding MP, Ding Y, Guo Y, Wang S, Li H, Shen CH. Epilepsia. 2026; doi: 10.1002/epi.70141

通讯作者：沈春红

Abstract

Objective: Patients with anti-leucine-rich glioma-inactivated protein 1 (LGI1) encephalitis frequently exhibit long-term cognitive impairment despite immunotherapy. In this study, we aimed to delineate hippocampal structural and functional alterations that underlie these deficits and examined their clinical correlates. **Methods:** We recruited 34 patients with anti-LGI1 encephalitis in the post-acute phase and 34 matched healthy controls. All participants underwent neuropsychological testing, high-resolution T1-weighted magnetic resonance imaging (MRI), and resting-state functional MRI. We assessed group differences in hippocampal volume and its whole-brain functional connectivity (FC) using a seed-based approach. Partial correlation,

multivariable linear regression, and mediation analyses were employed to relate imaging metrics to cognitive scores and clinical features. **Results:** Patients exhibited significant cognitive impairment, predominantly in verbal memory. This was paralleled by bilateral hippocampal atrophy, which strongly correlated with poorer performance across multiple cognitive domains. In contrast, patients demonstrated significantly increased FC between the left hippocampus and medial orbitofrontal cortex (mOFC). The enhanced connectivity was associated with better memory performance, suggesting a compensatory mechanism. Mediation analyses revealed that ipsilateral hippocampal volume mediated the relationship between acute medial temporal T2/fluid-attenuated inversion recovery (FLAIR) hyperintensity and memory scores. In addition, early immunotherapy was associated with an increase in left hippocampus-mOFC connectivity, contributing to improved cognitive performance. **Significance:** Our findings reveal a dual neural mechanism underlying cognitive outcome in anti-LGI1 encephalitis: the hippocampal atrophy correlates with cognitive deficits, whereas enhanced left hippocampal-mOFC connectivity represents a compensatory plastic response. Early immunotherapy may promote this beneficial plasticity, highlighting these structural and functional signatures as potential biomarkers for stratifying patients and monitoring therapeutic efficacy.

编号：ZWGM-2026-1-4

引用格式：Xi J, Deng F, Liang M, Ding Y, Li X, Gu Z, Lin Z, Liu Z, Li X. Global developmental delay and focal seizures in individuals with de novo truncating MACF1 variants. Hum Genomics. 2026;20(1):38. doi:10.1186/s40246-026-00917-y

通讯作者：李秀翠

Abstract

Objective: Microtubule and actin crosslinking factor 1 (MACF1) plays a critical role in cytoskeletal regulation. Pathogenic variants in MACF1 are associated with a heterogeneous range of phenotypes, including epilepsy, intellectual disability,

developmental delay, brain malformations, and hypotonia. This study aims to report two novel MACF1 variants and further explore the genotype-phenotype correlations and pathogenic mechanisms of MACF1-related disorders. **Results:** We identified two Chinese patients with de novo heterozygous MACF1 variants. Patient 1 carried a frameshift variant (c.18699_18700del p.V6234DTer2) in the spectrin repeats domain (SRD) and presented with global developmental delay. Patient 2 harbored a nonsense variant (c.19657C>T, p.Q6553*), also located in the SRD. This patient exhibited focal seizures that were readily controlled with valproic acid and perampanel, along with borderline intelligence, oppositional behavior, and hyperactivity. A review of the literature indicated that variants linked to neuropsychiatric disorders tend to cluster within the SRD, likely acting through a loss-of-function (LoF) mechanism and as risk factors for these type of disorders. Furthermore, the two truncating MACF1 variants we reported most likely act through haploinsufficiency. **Conclusions:** Our findings broaden the phenotypic spectrum of MACF1-associated disorders and provide additional evidence for genotype-phenotype correlations. The results also support the role of haploinsufficiency as a possible pathogenic mechanism in de novo MACF1 variants.

编号：ZWGM-2026-1-5

引用格式：Zhang XD, Tian Y, Ma J, Lei W, Yu WJ, Liu PH, Huang W, Huang W, Liu RF, Zhang XT, Wang F, Zhu Y, Lin YX. Iron single-atom nanozyme-mediated laser interstitial thermal therapy and anti-inflammatory effect for epilepsy. Mater Today Bio. 2026;37:102810. doi: 10.1016/j.mtbio.2026.102810.

通讯作者：王丰

Abstract

Developing a safe and effective treatment for epilepsy (EP) necessitates a minimally invasive system capable of precisely targeting and ablating the lesion area while effectively suppressing subsequent inflammation. In this study, we present an innovative therapeutic strategy for epilepsy, leveraging iron single-atom nanozymes

(Fe/SAN) in conjunction with laser interstitial thermal therapy (LITT) and their remarkable anti-inflammatory properties. This technology offers a significant advantage by enabling precise ablation of deep brain lesions and epileptic foci without the need for invasive craniotomy procedures. Notably, Fe/SAN exhibits exceptional near-infrared photothermal efficiency, which drives LITT to selectively eliminate diseased tissue. Moreover, near-infrared thermal imaging enables real-time monitoring of temperature changes in the epileptic lesion area, ensuring precise control of the therapeutic process. Additionally, Fe/SAN exhibits outstanding multienzyme-mimicking activities, which not only facilitate the precise ablation of epileptic foci but also scavenge reactive oxygen species and suppress inflammation. In kainic acid-induced epileptic mouse model, this combination effectively reduced seizure frequency and severity, improved spatial memory, and alleviated anxiety-like behaviors. This study introduces a novel "physical ablation-microenvironment regulation" strategy that achieves precise ablation of epileptic foci while modulating the local inflammatory microenvironment, thereby offering a highly promising minimally invasive treatment option for patients with drug-resistant epilepsy.

编号：ZWGM-2026-1-6

引用格式：Chen R, Hong ST, Wu SY, Jiang XQ, Su XJ, Zhang YQ, Mei Z, Lin H, Lin H, Hu XQ, Lin YX, Wang F. Postsurgical Seizure Outcome for Epilepsy Patients According to Histopathological Diagnosis: A Single-Center Experience. Neuropsychiatr Dis Treat. 2026;22:585977. doi: 10.2147/NDT.S585977

通讯作者：王丰

Abstract

Purpose: This study aimed to further explore the association between histopathology of the epileptogenic zone and postsurgical seizure outcome in epilepsy patients, with a specific focus on dual or multiple pathologies. **Patients and Methods:** In this single-center retrospective cohort study, 449 patients who underwent epilepsy surgery between 2017 and 2024 at The First Affiliated Hospital of Fujian Medical University

were included. Clinical data were collected, including histopathological diagnoses and seizure outcome at ≥12 months postoperatively. We investigated the association of histopathology, epilepsy duration, the number of preoperative antiseizure medications (ASMs), and MRI findings with seizure outcome, using logistic regression analysis. **Results:** Among 449 patients, the most common histopathological diagnoses were low-grade epilepsy associated neuroepithelial tumors (LEAT, 34.3%) and cerebral vascular malformations (24.5%). Of 382 patients with complete follow-up data, 76.4% achieved seizure freedom (Engel I), with a median follow-up of 47 months. Patients with LEAT had the highest seizure-free rate (88.4%), whereas worse seizure outcome was observed in patients with focal cortical dysplasia (FCD) and gliosis, with 59.2% and 58.8% remaining free from disabling seizure, respectively. Multivariate logistic regression analysis identified FCD (OR = 4.290), gliosis (OR = 4.359), and dual or multiple pathologies (OR = 3.558) as independent predictors of seizure recurrence. Longer epilepsy duration (OR = 1.005) and a greater number of preoperative ASMs (OR = 1.235) were associated with an unfavorable prognosis. Preoperative MRI results are not an independent predictor of postoperative seizure recurrence in epilepsy patients. **Conclusion:** Histopathological diagnosis, shorter epilepsy duration, and fewer preoperative ASMs predicted favorable surgical outcome in epilepsy patients. In contrast, dual/multiple pathologies, FCD, and gliosis carried an increased risk of postoperative seizure recurrence. These results highlight the importance of early surgical intervention, detailed histopathological assessment, and precise localization of the epileptogenic zone to improve outcome.

编号：ZWGM-2026-1-7

引用格式：Gong J, Lin M, Chen L, Xiong W, Zhang Y, Liu C, Chen S, Lin W, Zhu C, Huang H. Microbiota-sphingolipid pathway in generalized epilepsy: evidence from Mendelian randomization and clinical metabolomics. Front Microbiol. 2025;16:1662050. doi: 10.3389/fmicb.2025.1662050

通讯作者：林婉挥

Abstract

Objective: Epilepsy is a complex disorder with growing evidence linking gut microbiota and metabolism, though causal relationships unclear. This study investigated causal effects of gut microbiota on three epilepsy types via metabolic pathways, using Mediation Mendelian randomization (MR), evaluated directional consistency metabolomics of refractory epilepsy (RE) patients before and after medium-chain triglyceride (MCT) diet intervention. **Methods:** Two-step MR was applied to summary statistics for 207 species (Dutch Microbiome Project) and 196 species (MiBioGen consortium), evaluating 871 serum metabolites as mediators of three epilepsy types. For validation, directional consistency in metabolomics was conducted on serum samples from 9 RE patients before and after MCT diet intervention. **Results:** Only sphingomyelin (SM; d18:0/20:0, d16:0/22:0) and Glycocholate glucuronide (1) were the metabolites significantly associated with three epilepsy types. Mediation MR analysis revealed Mollicutes RF9 had a unidirectional effect via sphingomyelin (d18:1/22:1, d18:2/22:0, d16:1/24:1) modulation (P = 0.009). In contrast, Gamma-proteobacteria and Oxalobacter demonstrated bidirectional mediation: via glutamine conjugate of C6H10O2(2) and cerotoylcarnitine (C26) (P = 0.026 and P = 0.033, respectively); while these pathways were protective in mediation, higher abundances were associated with increased risk of generalized epilepsy. Notably, no significant mediators were identified for epilepsy or focal epilepsy. Metabolomics further confirmed MCT diet-induced elevations in 7 specific SM species. Among these, SM (d18:1/36:8) remained statistically significant after Benjamini-Hochberg false discovery rate (BH-FDR) correction. Notably, changes in SM (d18:1/36:8) and SM (d18:1/14:3) were positively correlated with seizure control rates. **Conclusion:** This study identifies both unidirectional and bidirectional microbiota-metabolite pathways modulating generalized epilepsy risk, with converging evidence pointing to sphingomyelin as a potential lipid biomarker and therapeutic target.

编号：ZWGM-2026-1-8

引用格式：Wan H, Zhao X, Hu W, Zhang C, Wang X, Zheng Z, Wang S, Liu D, Sang L, Meng X, Zhang K, Shao X. Anatomo-Electro-Clinical Features of Parietal Lobe Epilepsy: Insights From Scalp Video-Electroencephalography. CNS Neurosci Ther 2026;32(1):e70713. doi:10.1002/cns.70713

第一作者：万慧娟

Abstract

AIMS: To summarize the anatomo-electro-clinical characteristics of parietal lobe epilepsy (PLE) subgroups using unsupervised cluster analysis. **METHODS:** This retrospective cohort study included patients with drug-resistant PLE with seizure freedom after surgery and evaluated scalp video-electroencephalography (EEG) recordings from three epilepsy centers. Hierarchical cluster analysis associated interictal/ictal patterns and initial ictal semiology with anatomical subgroups. **RESULTS:** We analyzed 79 interictal EEG, 141 ictal EEG, and 141 semiological patterns in 47 patients. Cluster analysis associated interictal and ictal discharges from lateral superior parietal lobule epilepsy with centroparietal region distributions on scalp EEG, whereas discharges from other subgroups involved broader regions. Cluster heatmaps of the initial ictal semiology showed: Chapeau de gendarme, affective phenomena, and forced eye deviation in intraparietal sulcus; contralateral limb tonic/clonic or akinetic, affective phenomena, and visual illusions in SPL-lateral; Chapeau de gendarme, behavioral arrest, and vestibular in parieto-occipital sulcus; behavioral arrest in angular gyrus; distal gestural automatisms and cephalic sensations in posterior cingulate; body-perception illusion and contralateral versive in supramarginal gyrus; contralateral facial tonic/clonic in parietal operculum. **CONCLUSION:** PLE subgroups exhibited distinct scalp EEG features and ictal semiology, reflecting unique propagation networks and highlighting the importance of detailed video-EEG for identifying the epileptogenic zone and guiding intracranial electrode placement.

编号：SSL-2026-1-1

引用格式：Chen Y, Wang J, Xu S, Wang Q, Mei S, Lu J, Wang Y, Lin H, Wu D, Chen L, Zuo C, Guan Y, Ge J, Wu X. Exploring the Role of TSPO-PET Imaging Among MRI-Negative Patients with Temporal Lobe Epilepsy: From the Perspective of Heterogeneity. Brain Sci. 2026;16(2):246. doi: 10.3390/brainsci16020246

通讯作者：吴洵昳

Abstract

Background/Objectives: This study explored the heterogeneous distribution pattern of translocator protein 18kDa (TSPO)-PET/MRI using radioligand [18F] DPA-714 in temporal lobe epilepsy patients and identify clinical factors influencing imaging outcomes. **Methods:** The TSPO imaging in individ- ual patient were evaluated with both visual reading and quantitative assessment using asymmetry index based on cerebellum-normalized standardized uptake values. The association between clinical factors and TSPO imaging outcomes were assessed. Pathological evaluation was conducted in three patients. **Results:** Twenty-nine TLE patients and ten healthy controls were enrolled. Visual evaluation revealed increased [18F] DPA-714 uptake in twenty patients as compared to controls, pre-dominantly in a unilateral regional brain, while the remaining nine patients showed visually unde-tectable uptake of [18F] DPA-714. Consistently, quantitative analysis revealed that 69% (20/29) patients exhibited at least one brain area with significant asymmetry index, notably in the temporal lobe (85%, 17/20). A high asymmetry index could also be observed in the parietal (13.8%, 4/29) and occipital lobe (17.2%, 5/29). Significant associations were identified between asymmetry index and seizure frequency ($p = 0.045$, OR = 7.994), and the interval from last seizure to PET scan ($p = 0.033$, OR = 6.712). Moreover, we confirmed the pathology in three patients via immunohistochemistry, which underscored the potential of TSPO-PET in detecting minor lesion. **Conclusions:** The findings highlight the utility of TSPO imaging in localizing epileptic foci especially among MRI-negative patients. Some clinical factors such as seizure frequency and interval from last seizure to PET scan might attribute to heterogeneity of the distribution pattern varies among individual patient.

编号：SSL-2026-1-2

引用格式：Huang Y, Chen Y, Xu S, Wang Y, Wu D, Wu X. Self-supervised EEG transformer with adaptive multi-scale spectral attention and temporal modeling for seizure prediction. Biomedical Signal Processing and Control. 2026;119:109855. doi:10.1016/j.bspc.2026.109855

通讯作者：吴洵昳

Abstract

Reliable seizure prediction based on scalp electroencephalogram (EEG) signals is critical for improving the quality of life and safety of patients with epilepsy. Self-supervised learning (SSL) has shown strong potential for learning representations from unlabeled electroencephalogram recordings. However, many existing pretext tasks do not adequately account for non-stationarity and noise, and they do not explicitly model the joint spectral–temporal structure required for seizure prediction. To address these challenges, we propose a novel SSL framework that jointly models frequency- and time-domain features through two core components: the Multi Scale Adaptive Spectral Block (MSASB) and the Global and Local Temporal Module (GLTM). MSASB applies learnable multi-band filtering and frequency-domain attention to enhance spectral representations and suppress noise, while GLTM integrates multi-head self-attention and convolution to capture both global and local temporal dynamics. This dual-domain design significantly improves feature robustness and interpretability. Extensive experiments on three benchmark datasets demonstrate the effectiveness of our model. It achieves 98.14% accuracy on the Children's Hospital Boston–MIT (CHB-MIT) dataset and surpasses all existing SSL-based approaches. On the Temple University Hospital Abnormal EEG Corpus (TUAB) and the Temple University Hospital Seizure Corpus (TUSZ), the model attains state-of-the-art performance with a balanced accuracy of 0.814, AUROC of 0.897, and F1-score of 0.668, confirming its robustness and generalizability across diverse seizure prediction tasks. These findings demonstrate the potential of our framework as an effective foundation for future real-time seizure prediction and broader EEG-based clinical applications.

编号：SSL-2026-1-3

引用格式：Min X, Ying G, Wu X, Liu X, Yin J, Hu S, Fan Z, Ginowree NB, Ud Din MJ, Liu P, Zhang G. Analysis of Risk Factors for Relapse in Pediatric MOG Antibody-Associated Disease: A Single-Center Retrospective Cohort Study. Front. Neurol. 17:1790844. doi: 10.3389/fneur.2026.1790844

通讯作者：张刚

Abstract

Objective: Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an immune-mediated demyelinating disorder of the central nervous system. It has a higher incidence in children than in adults, carries a relatively high risk of relapse with unclear mechanisms, and significantly impacts patient prognosis. This study aimed to investigate the clinical characteristics of MOGAD and identify independent risk factors for relapse, to provide a basis for early intervention and individualized treatment. **Methods:** A total of 108 children diagnosed with MOGAD at the Children's Hospital of Nanjing Medical University between January 2020 and June 2024 were retrospectively enrolled. Clinical, laboratory, and radiological data were collected. Univariate analysis and multivariate logistic regression models were used to screen for risk factors associated with relapse. **Results:** The median follow-up time was 37 months, with a relapse rate of 30.6%. The proportion of patients presenting with seizures (54.5% vs. 20.0%, $p < 0.001$) and limb weakness (27.3% vs. 9.3%, $p = 0.018$) at onset was significantly higher in the relapse group compared to the monophasic group. The most common initial clinical phenotype was acute disseminated encephalomyelitis (ADEM) type (38.9%), while optic neuritis (ON) type became predominant at relapse (48.9%). Multivariate analysis identified seizures (OR = 7.155, 95% CI: 2.265–22.604, $p < 0.001$) and limb weakness (OR = 5.157, 95% CI: 1.322–20.117, $p = 0.018$) as independent risk factors for relapse, whereas a normal brain MRI (OR = 0.186, 95% CI: 0.035–0.985, $p = 0.048$) was a protective factor. The relapse group had a higher proportion of patients with high serum MOG antibody titers (≥1:100) (54.5% vs. 32.0%, $p = 0.027$) and elevated cerebrospinal fluid cell counts (>30 × 106/L) (66.7% vs. 44.0%, $p = 0.03$). Patients receiving

oral corticosteroids for ≥6 months during the remission phase had a significantly lower relapse rate (42.4% vs. 65.3%, p = 0.026). **Conclusion:** In pediatric MOGAD, the ADEM phenotype is most common at onset. Seizures and limb weakness at initial presentation are independent risk factors for relapse in children with MOGAD, while a normal brain MRI suggests a lower relapse risk. Prolonging the corticosteroid treatment course during remission (≥6 months) may help reduce the relapse risk. Enhanced follow-up and individualized treatment should be considered for children with high-risk factors.

编号：SSL-2026-1-4

引用格式：Zhang G, Lu Y, Xie L, Begemann A, Papuc SM, Zweier M, Steindl K, Rauch A, Mayr JA, Koch J, Feichtinger RG, Elmslie F, Kulosik L, Jamra RA, Harmsen S, Wang S, He M, Zhang L, Zhou W, Wang C, Liu X, Zhang A, Zheng B. De novo missense variants in BAIAP2 are associated with developmental and epileptic encephalopathies. Epilepsia. 2026;67(1):480-498. doi: 10.1111/epi.18661

第一作者：张刚

Abstract

Objective: Brain-specific angiogenesis inhibitor 1-associated protein 2 (*BAIAP2*) plays a crucial role in dendritic spine morphogenesis and excitatory synapse formation. We establish de novo variants in *BAIAP2* as a novel genetic cause for developmental and epileptic encephalopathies (DEEs). **Methods:** Using whole exome/genome sequencing, we identified de novo missense variants in *BAIAP2* in six patients with DEEs. Molecular docking was utilized to predict the effect of these variants on the protein structures. Functional assays were conducted by overexpressing wild-type and mutant *BAIAP2* in cultured cells, primary hippocampal neurons, and zebrafish. **Results:** All six patients exhibited severe infantile or early childhood onset epilepsy, with refractory seizures in four individuals. Language and motor development delays were prevalent, with varying degrees of intellectual disability observed. The missense variants were clustered within the multiple phosphorylation site region that is critical for the autoinhibited conformation of BAIAP2 through 14-3-

3 binding. In silico modeling and HeLa cell spreading assays demonstrated that *BAIAP2* mutants potentially disrupted its autoinhibited state and induced hybrid filopodia-lamellipodia protrusions in cells, phenocopying the effects of Rac1/Cdc42 overexpression. Electrophysiological recordings revealed that neurons expressing *BAIAP2* variants exhibited increased excitability, due to enhanced excitatory synaptogenesis. Additionally, transgenic overexpression of mutant BAIAP2 mRNA in zebrafish embryos led to developmental defects, neurite growth, and enhanced sensitivity to pentylenetetrazole-induced locomotor hyperactivity. **Significance:** De novo variants in *BAIAP2* represent a novel cause of DEEs. The functional consequences of these variants suggest that the gain of function of BAIAP2 can affect filopodia–lamellipodia formation, dendritic spine development, and synaptic transmission.

编号：SSL-2026-1-5

引用格式：Shen D, Wu W, Zhou J, Zhang X, Zhan L, Qian P, Qian J, Guo A, Li A, Zhang Q. Beyond Impaired GABAergic Signaling: Inflammation and Metabolic Dysfunction in Genetic Epilepsy Induced by GABRG2 Mutation. Mol Neurobiol. 2025;63(1):3. doi: 10.1007/s12035-025-05277-7

第一作者：沈丁玎

Abstract

Although the mechanisms underlying genetic epilepsies associated with mutations in the γ-aminobutyric acid type A receptor γ2 subunit gene (GABRG2) have been extensively investigated, prior studies primarily focused on the functional alterations of mutant ion channels. Emerging evidence has indicated that neuroinfammation and metabolic disturbances are involved in acquired epilepsies. To further investigate the potential involvement of these mechanisms in genetic epilepsies, we generated a transgenic zebrafsh line, Tg(hGABRG2I107T), harboring the GABRG2(I107T) mutation associated with developmental and epileptic encephalopathies. The Tg(hGABRG2I107T) zebrafsh exhibited spontaneous seizure-like behaviors and hyperexcitability as demonstrated by electrophysiological recordings and elevated c-

fos transcript levels. Total γ2 subunit expression increased and its cell membrane localization was reduced. These zebrafsh also displayed disrupted neuronal marker expression and abnormal synaptic ultrastructure, indicative of an excitatory/inhibitory imbalance. Transcriptome analysis revealed that diferentially expressed genes in the brain were enriched in the endoplasmic reticulum (ER) protein processing, metabolic pathway, and TGF-β signaling pathway. Pro-infammatory factors were upregulated, while genes involved in the tricarboxylic acid (TCA) cycle were downregulated. Similarly, HEK293T cells transfected with the mutant γ2(I107T) exhibited significant reductions in TCA-related gene expression, ATP levels, and mitochondrial density. In addition to impairing receptor trafcking, the γ2(I107T) mutation may induce ER stress, and disrupt infammatory and metabolic pathways, thereby leading to an imbalance of excitatory/inhibitory neurotransmission and potentially contributing to the pathogenesis of genetic epilepsy. Pharmacological intervention with dexamethasone and INCB3344, a C–C chemokine receptor type 2 antagonist, ameliorated seizure-like behavior in Tg(hGABRG2I107T) zebrafsh, further supporting the causal role of neuroinfammation in epileptogenesis in genetic epilepsy.

编号：SSL-2026-1-6

引用格式： Shen D, Zhao Z, Ji Y, Zhang J, Yao X, Qi L, Sun H. The role of ferroptosis in epilepsy: mechanisms and targeted therapies. Biochem Pharmacol. 2026;249:117912. doi: 10.1016/j.bcp.2026.117912

第一作者：沈丁玎

Abstract

Epilepsy is a common chronic neurological disorder. Approximately one-third of patients respond poorly to existing anti-seizure medications. There is an urgent need for novel therapeutic strategies targeting the fundamental disease processes. Ferroptosis is an iron-dependent form of regulated cell death characterized by the accumulation of lipid peroxides. In recent years, its role in the pathological mechanisms of epilepsy has gained increasing attention. This review systematically elaborates the core molecular

mechanisms of ferroptosis. These include dysregulated iron metabolism, failure of the glutathione peroxidase 4 (GPX4) antioxidant defense system, and excessive activation of lipid peroxidation. The article focuses on summarizing experimental and clinical evidence linking ferroptosis to the onset and progression of epilepsy. It reveals key alterations in ferroptosis markers in the brain tissues of epilepsy patients and model animals. Furthermore, it delves into the complex molecular regulatory networks involved. These networks encompass neuron-glia interactions (e.g., the C-X-C Motif Chemokine Ligand 10 (CXCL10)/C-X-C Motif Chemokine Receptor 3 (CXCR3) axis), MicroRNAs/noncoding RNAs, the nuclear factor erythroid 2-related factor 2 (Nrf2), mitochondrial dysfunction, and neuroinflammation. Based on this evidence, the article further evaluates the therapeutic potential of targeting ferroptosis. This covers emerging strategies such as direct inhibitors (e.g., Ferrostatin-1), natural compounds (e.g., quercetin, boswellic acid), drug repurposing (e.g., troglitazone, D-penicillamine), gene therapy and targeted delivery systems, as well as bioinformatics-guided target discovery. Finally, this review outlines future research directions and challenges. These include elucidating cell-specific mechanisms, developing non-invasive biomarkers, optimizing combination therapies, and promoting clinical translation. The aim is to provide new perspectives and a theoretical foundation for developing disease-modifying therapies for epilepsy.

编号：SSL-2026-1-7

引用格式：Wei L, Du X, Qiao Z, Yang B, Long S, Jiang Y, Wang Y, Wang H. Microstructural White Matter Alterations in Angelman Syndrome: A Fixel-Based Analysis. Autism Res. 2026;19(1):e70160. doi: 10.1002/aur.70160

第一作者：杜晓南

Abstract

Angelman syndrome (AS) is a neurodevelopmental disorder resulting from UBE3A gene mutations, characterized by intellectual disability, movement disorders, language difficulties, ataxia, microcephaly, and seizures. While previous studies have examined

brain connectivity in AS, the specifics of white matter structural changes have remained unclear. In this study, we utilized advanced diffusion MRI techniques to investigate the microstructural abnormalities of white matter for AS patients. A total of 30 AS patients and 19 age- and sex-matched healthy controls were included in the study. We used metrics derived from both fixel-based analysis (FBA) and diffusion tensor imaging to compare the white matter microstructure differences between AS patients and healthy controls. The results indicate that patients with AS have white matter microstructural differences throughout the whole brain, particularly in the corticospinal tract, arcuate fasciculate, and corpus callosum. FBA-derived metrics demonstrated greater specificity and sensitivity than tensor-based measures. Subsequently, we extracted six fiber tracts with significant differences from the FBA analysis and conducted tract-based statistics, including parieto-occipital pontine, anterior commissure, arcuate fasciculate, corticospinal tract, splenium of corpus callosum, and isthmus of corpus callosum. In all six fiber tracts, we found that AS patients with a higher frequency of seizures exhibited more white matter alterations. Overall, this study provides new insights into the structural differences in AS and their association with clinical symptoms, highlighting the extensive white matter differences and their potential impact on patient outcomes.
Keywords: Angelman syndrome; fixel-based analysis; magnetic resonance imaging; seizure.

欢迎大家引用上述论文

CAAE 青年委员会年度学术报告及交流大会

2026 年 1 月 23 日至 25 日，2025 年中国抗癫痫协会青年委员会年度学术报告与交流大会在广西壮族自治区南宁市成功举办。本次大会以“壮韵邕情、共谱痫章”为主题，汇聚多方力量搭建高端学术交流平台，近 500 名业界同仁齐聚邕城，共话癫痫诊疗领域的创新发展与未来方向。

本次会议规格颇高、阵容雄厚，中国抗癫痫协会领导周东会长、廖卫平监事长、周列民、邓艳春、林卫红、丁玎副会长、段立嵘秘书长兼支部书记、教育委员会王艺主任委员、江苏、陕西、河北、重庆、吉林、河北、湖南、广东、海南、甘肃、天津、内蒙、山西等长期支持青委会工作的省级抗癫痫协会负责人、特邀专家、青年委员及青年癫痫诊疗工作者等各界代表参会。值得关注的是，台北荣民总医院关尚勇教授、陈倩教授，香港威尔斯亲王医院梁浩云教授，澳门仁伯爵综合医院蔡翔教授等 12 名港澳台地区知名专家受邀莅临，进一步促进了我国癫痫领域的学术交融与经验共享，彰显了大会的开放性与影响力。

大会开幕式由中国抗癫痫协会第二届青年委员会委员、广西抗癫痫协会副会长兼秘书长余璐教授主持。中国抗癫痫协会会长周东教授发表讲话，围绕“回顾

奋斗历程、彰显发展成就、寄语青年力量”三大核心展开。他深情回顾了协会 21 年来的发展积淀，重点提及协会在去年实现的三大突破性进展——国际影响力显著提升、学术投稿量大幅激增、中国抗癫痫事业“一盘棋、一条魂”的发展格局正式确立。同时，周东教授高度肯定了青委会“活跃进取、刻苦钻研、外语功底扎实”的三大鲜明特质，并送上三重殷切祝愿：祝愿协会以“破”局之姿奋勇前行，凭借国际影响力攀升、学术成果爆发、体系建设完善的亮眼表现，筑牢中国抗癫痫事业根基；祝愿青年学者以“强”基之举赋能成长，形成 90 后挑大梁、80 后当骨干的蓬勃态势，依托扎实学识与外语优势，在国际学术舞台上响亮发出中国声音；祝愿本次大会以“聚”力之效绽放光彩，为行业交流、人才培育搭建优质平台，推动中国抗癫痫事业再攀新高峰。

台湾抗癫痫协会理事长陈倩教授在致辞中，深情回望了参与青委会活动的珍贵过往，细数中国抗癫痫协会 20 载发展的坚实足迹，更对此次与新老朋友重逢的暖心时刻倍感欣喜。作为台湾地区癫痫领域的领军代表，陈倩教授表达了与各位学者并肩深耕的坚定愿景，期盼两岸在癫痫诊疗与科研领域深化交流协作，携手为亚大地区癫痫事业的进步添砖加瓦。她特别引用梁启超先生的豪迈诗句为青年学者鼓劲加油，捎去马年顺遂、事业腾飞的新春祝福，更呼吁青年学者打破学术壁垒，让跨区域的学术合作与深厚情谊在更广阔的舞台上绽放光彩。

广西抗癫痫协会会长吴原教授代表广西抗癫痫协会及全体同仁，对莅临现场的各位专家同道、青年学者致以热烈欢迎。她详细介绍了本次大会的鲜明特色，指出会议学术内容覆盖基础研究、临床诊疗探讨等多个核心维度，更创新性融入广西民族特色医学人文活动，实现了学术深度与人文温度的双向融合。吴原教授勉励青年学者始终坚守“以患者为中心”的初心，将临床需求转化为科研创新动力，秉持开放进取、勇攀高峰的精神，如攀登青秀山般脚踏实地深耕学术领域。同时，她热忱邀请各位嘉宾在学术研讨之余，漫步南宁街头、感受城市独特文化，在收获知识与友谊的同时，爱上这座温情满溢的城市，并预祝大会取得圆满成功。

青年委员会主任委员梁树立教授致辞并作 2025 年度工作报告。他代表青委会，向广西抗癫痫协会、历届大会筹备团队表示诚挚感谢，为全体青委克服困难、如约相聚的热忱与强烈归属感倍感自豪，同时感恩中国抗癫痫协会、秘书处领导及各位专家对青委会的长期关怀与鼎力支持。报告中，梁树立教授全面回顾了

2025 年青委会的丰硕成果：顺利完成换届与区域布局调整，持续推进一系列特色品牌项目，在学术交流中兼顾深度与人文温度，青年学者在临床诊疗、科研创新领域纷纷崭露头角、斩获佳绩。此外，他明确了 2026 年青委会的核心工作方向，提出将优化现有项目开展形式，升级抗癫痫西部行、NEW 项目等重点工作，强化区域联动协作，推动 AI 技术与癫痫中心建设深度融合。

青委年度最佳研究评比：青年学者展风采，学术比拼显担当

“青委年度最佳研究评比”作为年会的经典传承环节，今年再度精彩上演，成为展现青年学者科研实力的核心舞台。来自全国各地的优秀青年学者们以全英文形式登台汇报，分享的科研成果均为 2025 年发表于国际知名期刊的高水平论文，内容涵盖致痫机制研究、新基因筛选、癫痫外科诊疗、脑网络解析等前沿领域。汇报中，选手们凭借扎实的学术积淀、清晰的逻辑思路，精准阐述研究核心内容，面对评审专家的提问从容应答、逻辑缜密，充分展现了中国癫痫领域青年研究者的创新视野、科研硬实力，以及接轨国际、顺畅跨文化沟通的卓越素养。期待这批青年才俊未来持续以过硬专业能力与国际化视野，在全球学术舞台上自信发声，向世界传递中国癫痫领域的前沿思考与学术力量。

本环节由关尚勇、周健、刘永红、陈倩、操德智、丁瑶六位教授分时段主持，保障评比活动有序开展。

参与汇报的青年学者及研究主题包括：

- 广州市第一人民医院张赛青委：《Exploratory analysis of a Novel RACK1 mutation and its potential role in epileptic seizures via Microglia activation》（新型 RACK1 突变的探索性分析及其通过小胶质细胞活化在癫痫发作中的潜在作用）
- 首都医科大学附属北京儿童医院刘婷红青委：《Resective Surgery for Drug-Resistant Epilepsy in Patients With Tuberous Sclerosis》（结节性硬化症耐药性癫痫的切除手术治疗）
- 中南大学湘雅医院冯莉主任：《Targeting ROCK2 to Restore Epileptic Synaptic Networks via Mitophagy Activation: Insights from Translational Imaging of SV2A In Vivo》（靶向 ROCK2 通过激活线粒体自噬修复癫痫突触网络：基于体内 SV2A 转化成像的研究）

- 重庆医科大学附属第一医院田鑫主任：《D2HGDHD efficiency Regulates Seizures through GSH/Prdx6/ROS-Mediated Excitatory Synaptic Activity》（D2HGDHD 活性通过 GSH/Prdx6/ROS 介导的兴奋性突触活动调控癫痫发作）
- 中南大学湘雅医院何芳主任：《The genetic spectrum features of 2261 Chinese children with epilepsy and intellectual disability》（2261 例中国癫痫合并智力障碍患儿的遗传谱特征分析）
- 浙江大学医学院附属邵逸夫医院金搏青委：《The response to anti-seizure medications and the development of pharmacoresistant epilepsy in malformations of cortical development》（皮质发育畸形患者对抗癫痫药物的反应及耐药性癫痫的发展机制）
- 天津市环湖医院金卫篷青委：《SEEG Emotion Recognition Based on Transformer Network With Channel Selection and Explainability》（基于通道选择与可解释性 Transformer 网络的立体脑电图情绪识别研究）
- 山东大学齐鲁医院徐硕青委：《Identification of plasma SEMA3E as the diagnostic biomarker for human epilepsy based on integrated bioinformatics analysis》（基于整合生物信息学分析筛选血浆 SEMA3E 作为人类癫痫诊断生物标志物）

经过评审专家严谨打分与现场观众投票，评比结果正式揭晓：刘婷红青委斩获一等奖；张赛、徐硕荣获二等奖；田鑫、冯莉、何芳、金搏、金卫篷 5 位医生获得三等奖；田鑫青委凭借超高人气摘得最佳人气奖。

2025 癫痫领域热点问题争鸣与研讨：聚焦前沿动态，碰撞学术火花

在“2025 癫痫领域热点问题争鸣与研讨”环节，5 位青年专家围绕癫痫共患病、基因治疗、迷走神经刺激（VNS）、外科手术等前沿话题发表精彩演讲，结合最新研究进展与临床实践经验展开深度分享，并与现场同道互动研讨、答疑解惑，充分碰撞学术思维火花。其中，癫痫相关基因治疗的突破性研究成果在本次青委会年会上首次亮相，引发全场广泛关注与热烈讨论。

本环节由李文玲、梁建民、陈子怡、彭镜、张春青 5 位教授分时段主持，保

障了研讨的专业性与高效性，具体演讲内容如下：

- 重庆医科大学儿童医院李听松青委，作《儿童癫痫共患病的系统化与规范化管理》主题演讲；
- 浙江大学医学院附属第二医院沈春红青委，作《新发局灶性癫痫的免疫性病因识别与临床管理进展》主题演讲；
- 广州医科大学附属第二医院何娜青委，作《基因的时空表达特征与癫痫临床特征的关系》主题演讲；
- 首都医科大学三博脑医院关宇光青委，作《从传统切除到 LITT：癫痫手术方式的更新与争议》主题演讲；
- 南京医科大学附属儿童医院张刚青委，作《儿童癫痫新基因发现与基因诊疗探索》主题演讲。

国产脑电图产研进展汇报会

“中国智造 2025—国产脑电图产研进展汇报会”以国产脑电设备研发应用为核心，搭建起产、学、研、用一体化交流平台，既展现了国产脑电图设备的技术突破与产业态势，又为推动我国抗癫痫事业高质量发展注入强劲动力。脑电图设备作为神经电生理核心诊断工具，其研发创新与产业化是践行“中国智造 2025”战略、构建医疗新质生产力的关键环节。随着战略深入实施，高端医疗器械迎来政策红利，临床急需装备优先审评、企业数智化转型等支持政策，为国产脑电设备发展保驾护航。当前，临床对脑电图设备的精准性、智能化需求持续攀升，行业正加速向多模态、智能化方向演进，国内企业凭借本土化创新优势与基层市场适配能力崭露头角，但仍需通过临床应用验证与市场认可进一步巩固竞争力。针对青年专家对国产脑电图产研现状认知不足的问题，中国抗癫痫协会青年委员会精心组织本次会议，为行业青年骨干搭建学习交流桥梁。

本次会议分为两大核心环节，兼顾实操验证与学术交流。1 月 23 日至 24 日，5 家参与企业的工程师在王礼、纪祥军、冷雪荣、陈蓓蓓、蒋铁甲、陈岩、刘宇、付萌萌、孔惠敏等 9 名青委代表的协助下，完成了脑电图设备 24 小时稳定性测

试，所有参与企业的产品均顺利通过考核，印证了国产脑电设备在核心性能上的可靠性。1 月 25 日，各企业依次开展产品特点与技术优势汇报，全面展示了国产设备在硬件性能、软件技术、功能拓展等方面的创新成果，呈现出行业内合作共赢与差异化发展的良好态势。王玉平教授在发表总结讲话，回顾了与国产脑电图设备相伴 40 年的历程，对国产设备的发展成就给予高度肯定。他指出，2010 年以来，国产脑电图设备在软硬件研发与生产领域实现跨越式突破，在与刺激设备兼容、脑机接口领域应用等方面展现出广阔前景，彰显了我国工业进步与医疗装备自主创新的硬实力。

专题学术会议

会议还同步举办了 7 场高水平专题学术会议，由梁树立、樊星、关宇光、吴原等专家主持，汇聚国内癫痫诊疗领域顶尖力量，聚焦临床热点与技术前沿展开深度研讨。浙江大学附属第二医院郭谊教授分享“局灶性癫痫的药物治疗进展与药物优化策略”，为临床用药提供精准指导；首都医科大学附属北京儿童医院梁

树立教授全面解读正在发表中的《弥漫性脑胶质瘤相关癫痫的诊断与治疗操作性指南》，助力指南落地应用；首都医科大学三博脑科医院关宇光教授汇报“VNS 治疗癫痫临床应用现状及新适应证拓展”，展现神经调控技术的临床价值；中山大学附属第一医院陈子怡教授深入解读“突触囊泡蛋白 2A 结合剂治疗癫痫的中国专家共识”，凝聚行业诊疗共识；四川大学华西医院周东教授聚焦“癫痫药物治疗临床管理新进展”，传递前沿诊疗理念；北京大学第一医院吴晔教授发布“基于 VNS 治疗癫痫的真实世界观察研究 CORE-VNS 数据”，并介绍智能程控技术，为精准治疗提供数据支撑；四川大学华西第二医院罗蓉教授分享“癫痫急症诊疗策略”，强化临床急症处置能力。系列专题报告为参会者带来全方位学术滋养，对指南共识推广、药物与 VNS 技术合理应用起到重要推动作用。

特色边会

大会期间，除主会场学术报告与交流外，还同步举办了多项特色边会活动，内容涵盖临床诊疗、科研创新、人才培养、产业合作等多个维度，亮点纷呈、干货满满。其中，“2025 年菁 YOUNG 计划总决赛”为青年癫痫工作者提供了展示才华、切磋技艺的舞台，助力青年人才成长；“LITT 多中心研究讨论会”聚焦激光间质热疗技术在癫痫治疗中的应用与突破，凝聚多中心研究共识；“癫痫外科新设备研究启动会”正式开启相关领域创新探索，为外科诊疗升级注入新动力；“抗癫痫发作药物产品交流会”搭建了产学研沟通桥梁，推动药物研发与临床应用深度结合；“我国难治性癫痫基线数据调查启动会”则为精准攻克难治性癫痫难题奠定数据基础，具有重要临床与科研价值。

闭幕式：

闭幕式上，中国抗癫痫协会青年委员会主任委员梁树立教授作总结发言。本次会议作为协会 2026 年开年首场大型线下会议，亦是农历乙巳蛇年的收官盛会，他首先向广西团队、秘书处全体人员及会务人员的精心筹备与辛勤付出表示感谢，向协会及秘书处领导的支持、各位专家同道、青年学者与参会企业的积极参与致以敬意。梁树立教授表示，本次会议不仅为国内外癫痫领域同仁搭建了高效的学术沟通与合作桥梁，助力青年一代癫痫工作者拓宽视野、提升能力，实现青年骨

干与权威专家双向赋能、深耕研讨，更聚焦癫痫诊疗技术突破与临床共识凝聚，在思想交融中汇聚行业合力，在经验传承中激发创新活力，为推动我国癫痫诊疗水平同质化提升、促进地区间学术交流融合、加快癫痫领域科研与临床创新发展注入了强劲动力，为我国抗癫痫事业提质增效、行稳致远筑牢根基。

本次大会圆满落下帷幕，与会嘉宾共同相约 2027 年相聚冰城哈尔滨，携手并肩再攀我国抗癫痫事业新高峰，共筑癫痫研究与诊疗水平提升的新未来。

感谢美丽绿城，难忘美好南宁！

冰天雪地的哈尔滨欢迎您！

2025 年青委年会医学人文主题活动

本次人文活动结合广西特色民族文化与鲜明地域特征，在青秀山展开了一场别开生面以“青秀登高凝初心 创新抗痫守安康”为主题的医学人文活动，让各位青年学者在紧张的学术交流之余，放松身心，开阔视野，增进友谊。

活动伊始，部分专家与青年委员会成员率先前往广西学生军抗日烈士纪念碑，开展庄重的献花仪式。在庄严肃穆的氛围中，青年学者们驻足凝视、鞠躬致敬，深切缅怀那些为国捐躯的英雄先烈，共同重温那段波澜壮阔、浴血奋战的抗战历史。先烈们的赤诚之心与报国之志，为在场每一位青年医者埋下了坚守初心、勇担使命的精神种子。

随后，全体人员齐聚风景秀丽的东盟友谊园大草坪，开启充满广西特色的民

族文化体验环节。活动启动前，梁树立主委发表致辞，他表示：“我们齐聚南宁青秀山学生军纪念碑前追思先烈、传承精神，倍感使命光荣、责任在肩。八十余年前，热血学生军舍书从戎，以青春生命扛起民族救亡重任，诠释了“少年强则国强”，他们的精神是不朽丰碑、前行动力。现在站在东盟友谊园大草坪，我们更当珍惜发展环境，深耕癫痫诊疗、勇攀科研高峰、做好科普消弭偏见，将青春理想与奉献担当融入事业，以精湛医术和温暖关怀守护患者，书写新时代青年医者的奋进篇章，不负先烈、不负时代！”

在民族文化体验环节，九大区域队伍轮番参与竹竿舞、背篓绣球、三人板鞋等极具广西特色的活动，沉浸式感受八桂大地多民族和谐共处的浓厚文化氛围。其中，背篓绣球活动尤为精彩，青年学者们充分发挥智慧与勇气，以攻坚克难、奋勇争先的劲头突破自我，各大区均圆满完成挑战，将“不可能”变为现实，展现出青年医者敢闯敢拼、团结协作的优良风貌。这场充满欢声笑语的文化互动，不仅拉近了各地学者的距离，更将民族文化中坚韧、协作的精神内核传递给每一个人。

青年癫痫学者联谊晚会

夜幕降临，以“山歌壮梦，医路同行”为主题的青年癫痫学者联谊晚会如期举行。晚会由李光健、张弼、徐馨三位青委与特邀主持人秦娴女士联袂主持，九大区域的青年学者轮番登场、各展所长，节目精彩纷呈、亮点纷呈，有奖竞答环节更是点燃全场热情，将晚会气氛一次次推向高潮，现场掌声、欢呼声此起彼伏，暖意融融。

晚会在广西抗癫痫协会带来的合唱《山歌载情，好客南宁》与歌舞《壮族敬酒舞》中温情启幕，嘹亮的山歌、灵动的舞姿尽显壮乡人民的热情好客，让在场嘉宾沉浸式沉醉于浓郁的民族风情之中。随后，各大区节目轮番上演，各有千秋、打动人心：浙皖赣闽大区的《星辰大海》以歌曲搭配手影舞，温柔诉说着青年医者对理想的坚守；京东津蒙大区合唱《大中国》、京西晋冀大区合唱《我会一直顺》，用激昂旋律点燃全场爱国情怀与奋进斗志；粤桂琼港大区的《一路生花》、豫鄂湘大区的舞蹈《库里之歌》，将对患者康复的祝福、对职业的敬畏融入表演，彰显青年医者的责任与担当。

黑吉辽大区的唱跳串烧《自由飞翔》《对面的女孩看过来》活力四射，甘宁青新大区的歌伴舞《西北节拍，一起抗痫》独具地域特色，两场表演将晚会气氛推向顶峰，点燃了全场的欢乐因子。最后，申苏鲁大区带来大合唱《明天会更好》，悠扬的歌声为当晚的相聚画上圆满句号，也承载着全体青年学者对癫痫诊疗行业未来发展的真挚期许与美好祝愿。

晚会期间还设置了四组知识问答环节，内容围绕广西壮族自治区概况与东盟国家相关知识展开，既增添了互动乐趣，又让大家在轻松氛围中加深了对地域文

化与国际交流常识的了解。此外，活动还对优秀论文及人文活动相关获奖集体与个人进行了表彰颁奖，以此激励青年学者深耕学术、践行人文，在专业道路上勇攀高峰。

此次系列活动将红色精神传承、民族文化体验与医学人文建设有机融合，不仅让青年癫痫学者在交流中增进了情谊、拓宽了视野，更凝聚起坚守医者初心、深耕抗痫事业的强大力量。未来，青年医者们将带着此次活动的收获与感悟，以更饱满的热情、更坚定的信念投身医学事业，用医术传递温暖，用担当书写华章。

2026 年 CAAE 青委会多中心研究项目汇总

多中心科研协作项目展示与招募环节作为 CAAE 青委会年度学术与交流大会的核心议程之一，为青年专家搭建了高质量的科研成果展示平台与跨界协作桥梁，赋能创新转化。该环节不仅为青年学者提供了充分彰显科研构想、释放创新潜力的契机，更有效凝聚了行业优质资源与科研合力，推动一批优质科研项目从理论蓝图迈向临床实践，为癫痫领域科研创新生态注入持续动能。青委会牵头开展的多中心研究成果丰硕，已在《Brain》《Neurology》等国际顶尖神经领域期刊发表多篇学术论文。本次会议共收录 13 项全国多中心项目报告，其中 10 项以口头汇报形式深度分享，3 项以海报汇报形式展示交流，研究内容全面覆盖神经调控治疗、药物治疗、自然病程研究三大核心领域。值得关注的是，自本年度起，青委会多中心研究相关产出成果将统一标注“CAAE-YC **** Group”署名，进一步强化学术品牌辨识度与团队凝聚力。

本环节由林一聪、闫志强两位教授联袂主持，多位青年医生带来了兼具创新性与临床价值的项目汇报，具体如下各项目介绍如下：

布立西坦预防溶栓相关卒中后癫痫安全性和有效性的前瞻性、随机、双盲、安慰剂对照、多中心临床研究方案

研究内容：

主要研究目的：评估布立西坦对溶栓相关卒中后癫痫一级预防的有效性。

次要研究目的：①评估布立西坦对溶栓后卒中后癫痫预防治疗的安全性；②评估布立西坦预防治疗溶栓相关卒中后癫痫后的卒中恢复情况的影响； ③评估布立西坦对卒中后抗板降脂药物血药浓度的影响；④比较布立西坦和安慰剂预防治疗溶栓相关急性缺血性卒中患者脑电变化情况。

纳入标准：

① 性别不限，年龄≥18 岁；

② 急性卒中患者 SeLECT-EEG≥7 分有影像学（CT/MRI）证据支持；

③ 急性缺血性卒中经溶栓治疗；

④ 本次卒中起病前 mRS 评分≤3；

⑤ 患者本人/法定监护人同意参加本研究并签署知情同意书。

备注：使用他汀药物治疗患者可以纳入研究

排除标准：

① 患者在本次卒中前 12 月内有缺血性或出血性卒中发作病史；

② 存在与卒中无关的导致癫痫显著危险因素，例如：既往诊断为癫痫、存在其他致痫性颅内病变或既往有颅内手术史；

③ 怀孕或哺乳期患者；

④ 对布立西坦或任何成分过敏或过敏体质者；

⑤ 吸毒、酗酒、其他物质滥用及精神疾病史；

⑥ 患有严重的肺、血液系统疾病，恶性肿瘤、免疫功能低下患者；

⑦ 筛选前 3 个月内参与其他临床研究者;

⑧ 偏头痛、三叉神经痛、糖尿病性周围神经痛、带状疱疹后神经痛、肌阵挛、不宁腿综合征、多发性硬化等入组时疾病需要服用抗发作药物卡马西平、奥卡西平、加巴喷丁、丙戊酸钠、左乙拉西坦、普瑞巴林等药物；

⑨ 研究者认为不适合参加本试验者。

退出标准:

① 病情恶化或出现严重不良反应，经研究者判定不宜继续参加研究者；

② 受试者依从性差，违背研究程序、用药方案或服用禁用药物，经研究者判定不宜继续参加研究者；

③ 受试者或其监护人撤回知情同意。

项目负责人：中山大学附属第一医院　　陈子怡 教授

靶向脑网络关键节点治疗药物难治性癫痫的临床多中心研究

研究目的

① 主要目的：基于个体化癫痫网络关键节点的神经调控，观察癫痫发作频率、脑电变化、认知评分等临床指标。

② 次要目的：根据药物难治性癫痫的发作 SEEG 信号，构建多种类型癫痫发作传播模式。

纳入标准：

①符合药物难治性癫痫的诊断标准；

②年龄在 12-65 岁之间，不限男女；

③已接受多学科系统术前评估，根据诊疗需求，进行 SEEG 监测；

④近 3 月发作频率≥1 次/周，伴/不伴继发性发作；

⑤在告知目前可选的治疗方案及潜在风险后，患者同意参加本研究，并签署知情同意书。

排除标准

①脑血管病、肿瘤、感染或自身免疫性疾病继发癫痫的患者；

②严重的心、肝、肾躯体疾病或严重的或进行性神经精神疾病严重心律失常、QTc＞450ms；转氨酶超过正常值（0-40U/L）上限 3 倍，总胆红素（3.4-17.1μmol/L）、肌酐（44-133umol/L）超过正常值上限 1.5 倍或肌酐清除率＜45ml/min）；

③韦氏智力量表评分＜50，存在精神、认知、心理障碍，无法配合完成测试者；

④既往有头部手术、脑外伤或其他原因所致脑内显著结构性改变；

负责人：中国人民解放军陆军军医大学　张春青 18983204130

新诊断成人局灶性癫痫患者长期预后：一项前瞻性、多中心临床研究

主要目的： 明确我国首次接受正规抗癫痫药物（ASM）治疗的新诊断成人局灶性癫痫患者发展为难治性癫痫（DRE）的发生率，并系统探索与 DRE 相关的早期预警因素，构建适合中国人群的多因素预后预测模型。

入组标准：

1）由神经科专科医生确定患者癫痫诊断，新诊断癫痫根据 2014 年 ILAE 推出新的癫痫临床实用性定义确定，局灶性癫痫符合 2017 年国际抗癫痫联盟（ILAE）颁布的癫痫分类标准；

2）既往未进行过任何抗癫痫治疗；

3）年龄≥18 岁；

4）患者签署知情同意书。

排除标准：

1）神经科病情进行性加重者；

2）存在严重心肝肾疾病者、严重高血压者、仅存在癫痫持续状态者、或者存在活动性精神障碍者（影响神经科检查）；

3）存在非癫痫发作（低钙抽搐，心因性发作等）

负责人：吉林大学第一医院 神经内科癫痫中心 李光健

邮箱：liguangjian555@jlu.edu.cn

联系电话：18343116616；微信号：guangjian0301

癫痫发作对难治性孤独症 VNS 治疗影响的前瞻性多中心对照研究

项目摘要：癫痫共患孤独症谱系障碍（ASD）率高，且癫痫发作常与症状加重及功能退化密切相关，尤其在难治性 ASD 中更为突出迷走神经刺激（VNS）已被证实可有效治疗药物难治性癫痫，研究表明其在癫痫共患 ASD 人群中可能产生附加的行为和情绪获益。此外，接受 VNS 治疗的癫痫合并 ASD 患者在癫痫发作得到控制的同时，情绪稳定性和适应性行为可出现不同程度改善。尽管已有研究提示 VNS 可能改善癫痫共患孤独症患者的部分行为和生活质量，但现有证据多为小样本、回顾性研究，且癫痫发作控制在其中的作用机制尚不明确。基于此，本项目拟系统评估癫痫发作控制程度在难治性孤独症患者接受 VNS 治疗过程中的作用，重点探讨癫痫发作对难治性孤独症相关行为改善的影响及其潜在调控机制，为 VNS 在癫痫共患难治性孤独症人群中的精准应用提供循证依据。

合作单位标准：

1.二级及以上癫痫中心或儿童癫痫中心

2.合作单位设置精神科或心理科，可以完成孤独症的诊断与量表评估

3.合作单位近 3 年，年均完成 VNS 手术大于 10 台

入组标准：

1. 年龄 3-18 岁，男性或女性不限；
2. 孤独症谱系障碍诊断明确并配合完成相关量表评估；
3. 孤独症谱系障碍伴或不伴癫痫，其中药物难治性癫痫定义为应用正确选择且能耐受的两种抗癫痫药物，仍未能达到持续无发作。
4. 家属同意入组并签署知情同意书。

排除标准：

1. 合并严重内科或外科禁忌如严重心肺功能不全、凝血功能障碍等不适合接受手术或神经调控治疗患者；
2. 既往接受其他神经调控手术治疗患者；
3. 严重精神共病无法完成行为学评估患者；
4. 家属拒绝同意入组患者。

负责人：首都医科大学附属北京儿童医院　邝苏慧 16321330862

电磁一体化刺激治疗心因性非痫性发作的疗效及机制研究

纳入标准：

1. 年龄 12-60 岁，右利手；
2. 符合 DSM-V 中功能性神经症状障碍（转换障碍）的诊断标准，以癫痫样发作或其它形式的发作性症状为主要临床表现；
3. 病程≥1 个月；
4. 签署知情同意书。

排除标准：

1. 存在神经系统器质性疾病（如脑卒中、肿瘤、脱髓鞘疾病等）或头部金属植入物；
2. 符合其他轴 I 精神障碍诊断（如精神分裂症、双相情感障碍、物质滥用）；
3. 严重躯体疾病或病情不稳定；
4. 存在 rTMS/tDCS 治疗禁忌症（如癫痫病史）；
5. 妊娠或哺乳期妇女；
6. 无法配合完成评估及治疗。

合作单位标准：

1. **资质与经验：**具备独立神经内科或神经心理科，年诊治 PNES 患者不少于 5-10 例，拥有丰富的临床研究经验，优先考虑已通过 GCP 认证的团队。
2. **设备要求：**具备并熟练使用经颅磁刺激（rTMS）和经颅直流电刺激（tDCS）治疗设备。同时，中心需具备 3.0T MRI、脑电图（EEG）设备，拥有近红外脑功能成像（fNIRS）设备者优先。
3. **人员配置：**拥有至少一名对 PNES 诊疗有经验的主治医师及以上职称的研究者，能负责患者的筛选、评估与治疗；配备一名及以上专职研究协调员（CRC），确保患者招募、随访与数据录入的规范性。
4. **质量承诺：**承诺严格遵守研究方案，参加研究者会议，统一执行标准操作流程（SOP），并接受中心启动、监查与质控。

项目负责及联系人：首都医科大学三博脑科医院 杨华俊

电话：13581770499（微信同号） 邮箱：doctor_yanghuajun@126.com

人工智能视频分析技术辅助癫痫性痉挛与不自主运动诊断的研究

研究对象：

婴儿癫痫性痉挛综合征患儿 150 例，不自主运动婴幼儿 150 例；每例病人需提供至少一次的经视频脑电图证实的发作性症状视频;视频数据质量符合分析标准；

合作单位要求：

1. 能够提供婴儿癫痫性痉挛综合征和婴幼儿不自主运动各 30 例以上的单位；
2. 每例病人需提供至少一次的经视频脑电图证实的发作性症状视频;
3. 视频数据质量符合分析标准；

招募患者要求：

1. 符合婴儿癫痫性痉挛综合征和婴幼儿不自主运动诊断的患儿；
2. 年龄 2 个月-2 岁；
3. 监护人签署知情同意书，授权同意使用患儿的发作性症状视频及脑电数据；

负责人：中国医科大学附属盛京医院小儿神经内科

范玉颖　fanyy@sj-hospital.org

司替戊醇治疗 Dravet 综合征的真实世界多中心研究

研究项目：

拟开展一项多中心、前瞻性、观察性的自身前后对照真实世界临床研究，观察和分析 STP 作为添加治疗 DS 患儿的有效性和安全性，探究其疗效相关的影响因素。入组接受 STP 添加治疗的 DS 患儿，与 4 周回顾性基线相比，观察 DS 患儿添加 STP 治疗 24 周时的 50%治疗应答率、药物保留率、维持剂量、不良反应；并探究 STP 疗效相关的影响因素；寻找适合中国 DS 患儿的理想目标剂量及药物滴定。

入组标准：

1. 年龄 > 6 月， < 18 岁，男女不限;
2. 符合 DS 的诊断标准；在 2023.07-2026.12 期间接受 STP 添加治疗;
3. 入组前正在接受 CLB 和/或 VPA 抗癫痫治疗，每月至少有一次发作;
4. 至少有条件随访 6 个月。

排除标准：

1. 患有精神疾病或具有冲动性行为的，或合并严重的其他器官功能障碍;
2. 在筛选前 60 天内参与了其他药物或医疗器械的临床研究

合作单位标准：年门诊新发 Dravet 综合征患者 30 例以上的癫痫中心

负责人：深圳市儿童医院　操德智教授　电话：18938691158

糖皮质激素冲击治疗伴睡眠中棘慢波激活的发育性和癫痫性脑病（D/EE-SWAS）的多中心随机对照研究

纳入标准：

1. EEG 异常：清醒为不连续的癫痫放电，NREM 睡眠期癫痫放电较清醒期明显增加，睡眠期 SWI 大于 50%，无睡眠期的广泛阵发性快速活动；
2. 2.2 岁-14 岁；
3. 均有认知行为评估（IQ 或 DQ）的结果。
4. 认知倒退明确（具有以下标准之一）
5. 经家长或医生评定在至少 1 个领域（运动、语言、社交、自理）丧失先前获得的技能。
6. 半年至 1 年内 IQ 或 DQ 下降超过基线的 20%
7. 既往未用过糖皮质激素。
8. 监护人签署知情同意书。

排除标准：

9. 诊断时具有强直发作或癫痫性痉挛发作的患儿
10. 脑炎、脑病等病因
11. Rett 综合征、进行性肌阵挛癫痫等特殊的会出现认知倒退的神经变性疾病。
12. 未完成 1 月激素治疗方案和随访脱落的患者
13. 无头颅 MRI 检查的患者
14. 明确为同年龄段易发的其它癫痫性脑病（如 Doose）的患儿

负责人：广州医科大学附属妇女儿童医疗中心 彭炳蔚

联系人：卓医生

VNS 治疗 FIRES 相关超难治性癫痫持续状态的多中心临床研究

研究目的：通过前瞻性对照研究观察 VNS 治疗 FIRES 相关超难治性癫痫持续状态的有效性和安全性

入组标准：

1. 年龄 2-30 岁，性别不限
2. 发热后 14 天内出现癫痫持续状态，且符合超难治性癫痫持续状态的诊断
3. 符合 FIRES 的诊断，排除病毒性和自身免疫性脑炎等
4. 呼吸机辅助治疗病史，气管切开或未切开
5. 发病前发育正常
6. 受试者或其监护人能够充分理解该疗法并且能签署知情同意书

排除标准：

1. 有癫痫病史或其它神经系统疾病病史
2. 发病后一周内死亡或者离院治疗
3. 短暂应用呼吸机后停用呼吸机
4. 手术前癫痫持续状态病程超过 60 天
5. 迷走神经病变、损伤或结构异常或疼痛
6. 特异性排异体质或全身一般情况差，不能耐受异物植入

负责人：首都医科大学附属北京儿童医院功能神经外科 刘婷红 18613318670

联系人：首都医科大学附属北京儿童医院功能神经外科 王雨欣 13466555066

伏隔核壳部脑深部电刺激(NAcS-DBS)治疗药物难治性部分性癫痫的疗效及安全性评价 ——前瞻性、三盲、随机交叉对照临床研究

研究目的：评估伏隔核壳部脑深部电刺激对药物难治性部分性癫痫患者的疗效及安全性

纳入标准：

1.年龄 18～65 岁；

2.规律服用两种或以上的抗癫痫药物达到最佳药物治疗剂量，但癫痫仍不缓解，符合药物难治性癫痫标准；

3.癫痫发作呈部分性发作的患者，包括继发强直阵挛性癫痫（sGTCS），基线阶段每月发作≥3 次；

4.经过严格术前评估，仍无法行切除手术或行切除性手术预期较差者；

5.患者或其家属能够充分理解该疗法并且签署知情同意书；

6.患者依从性良好，能够配合完成术后随访要求。

排除标准：

1.年龄小于 18 岁，或大于 65 岁；

2.未规律服用抗癫痫药物达到最佳药物治疗剂量者；

3.存在精神、认知、心理障碍，不能签署知情同意书或无法配合手术及随访；

4 伏隔核存在结构改变；

5.存在神经外科手术禁忌症；

6.存在其他伴随疾病，已经或需要植入心脏起搏器、除颤器、人工耳蜗等；

7.全面性或特殊类型的遗传性癫痫；

8.妊娠、哺乳期妇女或近期有生育计划者；

9.存在客观因素无法完成电刺激测试及磁共振扫描的患者；

10.筛选期实验室检查异常，或认为有临床上重要意义的其他实验室检查异常；

11.经研究者判断认为不适合参与本试验的患者。

负责人：南方医科大学南方医院神经外科　　龙浩　13725437221

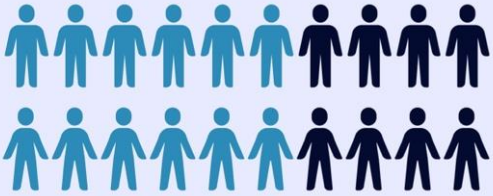
依达拉奉右莰醇舌下片预防卒中后癫痫的随机、双盲、安慰剂对照、多中心临床研究

负责人：王新施
神经内科副主任 主任医师 副教授
联系电话：13757897051

负责单位：温州医科大学附属第一医院

研究目的及意义

①评估依达拉奉右莰醇舌下片在卒中后癫痫高风险人群中预防迟发性癫痫发作的有效性和安全性
②探索依达拉奉右莰醇舌下片神经保护和疾病修饰作用机制。



研究类型

前瞻性、随机、双盲、安慰剂对照、多中心临床试验

招募患者入组标准

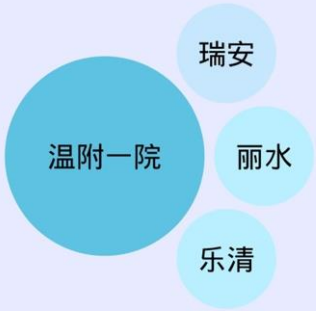
- 年龄18-80岁
- 急性缺血性脑卒中（MRI/CT确诊），发病≤48小时
- SeLECT评分≥6分
- 发病前无癫痫病史
- 可配合随访评估；⑥已签署知情同意书。

招募合伙单位及要求

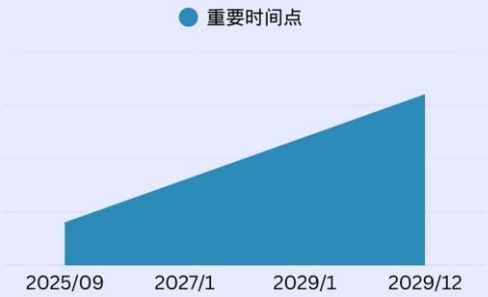
①具备卒中规范化诊疗能力

②具备临床研究和随访管理经验

③可按照统一方案完成病例筛选、入组和随访



现有研究中心及重要时间节点



预期进度和完成日期

2025.09——2026.01：完成人员培训与中心启动等工作。
2026.01——2027.01：完成患者入组与接受干预
2026.01——2029.01：完成患者随访观察，并进行中期总结
2029.01——2029.12：完成汇总分析全部疗效和安全性数据，并撰写和发表文章

癫痫术后初始抗癫痫药物治疗策略：一项比较单药、双药与标准多药治疗方案的前瞻性、多中心、随机、三臂平行对照研究

一、项目摘要

临床需求与争议：癫痫术后药物治疗策略缺乏高级别证据。Zeng 等（2012）回顾性研究发现术后双药治疗优于单药，但存在选择偏倚。Lamberink 等（2015）的系统综述指出，撤药风险与疾病本质相关，但未解答初始治疗强度是否影响早期控制、减药成功率和长期预后。Schmeiser 等（2018）则提出术后发作控制主要取决于手术本身，药物增减影响有限，但该研究未控制初始治疗方案。Eseonu 等（2018）在肿瘤手术中证实双药预防优于单药，但人群不同。目前，术后应起始单药、双药还是维持术前多药方案，尚无定论。现有证据均为回顾性观察，无法确定不同用药策略与结局的因果关系。

研究目的：主要目的：比较三种术后初始 AED 方案在术后 24 个月内维持无发作的有效性。**次要目的**：比较三组在术后早期（6 个月）无发作率和首次复发时间。

评估三组药物不良反应的发生率和严重程度。评估三组患者的生活质量和神经认知功能变化。探索不同病理类型（如海马硬化、局灶性皮质发育不良、肿瘤等）和手术部位对治疗反应的修饰效应。

研究干预措施：术后第 1 天起，根据服用 ASM 种类分为单药组、双药组、多药组。

随机化：采用中心分层、区组随机化。按病理（海马硬化/其他） 和手术部位（颞叶/颞叶外） 分层，确保组间基线平衡。出院后随访至术后 24 个月：每月电话随访，每 3 月门诊随访。目标为稳定控制，除非出现复发或不可耐受副作用，否则不调整治疗方案。

二、合作单位标准

综合（三级）癫痫中心 3-5 个

三、招募患者要求

1. 年龄、性别不限
2. 术前服用≥2 种 AEDs，确诊为药物难治性性癫痫，符合 ILAE 诊断标准
3. 接受首次根治性癫痫病灶切除术，切除脑区不限
4. 随访期：24 个月

四、其它信息

1. 项目负责单位：陆军军医大学新桥医院；负责人：张春青
2. 起止时间：2026 年 1 月至 2028 年 12 月
3. 联系人：孙晓琴 18883370911

北京儿童医院 BEIJING CHILDREN'S HOSPITAL

首都医科大学附属北京儿童医院(BCH-CCMU)
国家儿童医学中心(NCCH)
—— 功能神经外科 ——

儿童癫痫患者死亡原因多中心临床研究

项目信息

1.起止时间：2026年1月至2026年12月

2.研究目的：通过回顾性分析儿童癫痫患者死亡原因，有助于全面了解癫痫相关死亡的构成特征及其主要影响因素，为临床医师在癫痫患儿的风险评估、随访管理及个体化干预策略制定中提供循证依据，对降低儿童癫痫患者病死率、改善预后具有重要的临床与公共卫生意义。

3.项目进展：已完成研究方案设计，正在开展伦理审批及数据采集流程筹备工作

研究设计

研究方法

本研究为回顾性研究，收集儿童癫痫患者的临床及随访资料，记录患者人口学信息、癫痫诊疗情况、辅助检查结果，以及死亡年龄和死亡原因。死亡原因分为癫痫发作相关死亡、癫痫持续状态、发作或非发作相关意外、癫痫猝死、癫痫相关原发病死亡及其他原因死亡。将采用描述性和多因素回归分析等统计方法研究死亡原因构成，并探讨其与临床特征、社会因素等之间的关系。

纳入标准

①年龄1-18岁，性别不限。②癫痫诊断明确。③患者因癫痫发作、癫痫持续状态（含治疗过程中死亡）、SUDEP、意外伤害、肿瘤等各种原因（可能与癫痫无关）导致院内或院外死亡。④入组2020-2026年出现死亡的患者。

排除标准

①患者因颅脑、心脏、肾脏或者其它手术或手术并发症导致死亡。

招募合伙单位要求

需为CAAE认定的癫痫中心或三级医院的癫痫诊疗机构，有癫痫诊断能力。

负责人和负责单位：梁树立，首都医科大学附属北京儿童医院
联系人：郜雨辰 13522591413　韦志荣 17339801210

New 项目

NEW（Neuro updated research working forum）项目是中国抗癫痫协会（CAAE）青委会传统的学术活动，2025 年与神基公司合作的第 9 场 NEW 项目于 11 月 22 日在广州成功举办。陈子怡副主委担任大会主席。本次会议吸引了来自 20 多家医院约 40 位神经内科、神经外科、儿科癫痫工作者参会，共同见证了 2025 年 NEW 项目精品会议的热烈学术氛围。

会议特别邀请了余璐教授、何娜教授、操德智副主委等中青年专家参会，同时多名青年委员围绕自身免疫性脑炎、卒中后癫痫、癫痫的修饰治疗和功能 MRI 的应用等领域进行了新进展的解析和讨论。会议达到了追踪前沿，促进交流与合作，推动区域癫痫诊疗进展的目标。


2026 年 NEW 重新启航，今年共将开展 10 余场 NEW 项目，分别与神基公司、冀思公司联合开展。2026 年首场 NEW 项目 3 月 28 日在北京举办，会议由林一聪副主委和李光健副主委共同主持负责。周东会长专门进行了视频致辞，周会长指出“青年朋友们既要主动拥抱前沿，时刻保持知识的敏锐度，更要深耕指南共识，将最新的规范化作临床思维，以规范为基石，以创新为动力。愿你们做规范的践行者与传播者，推动中国癫痫事业再上新台阶。”梁树立主委在视频致辞中提到“新的 New 项目模式下，青年朋友们发扬十几年青委的优良传统，在辩论与争鸣之中体现个人综合能力、知识储备，体现我们作为新时代的癫痫青年工作者全方位的能力。”会议进行了局灶性癫痫药物治疗新进展和钠离子通道阻滞剂专家共识的讲座，随后京（东）津蒙大区和黑吉辽大区各四名青委围绕“既往使用钠离子通道阻滞剂治疗不佳的局灶性癫痫患者，后续添加是否考虑钠离子通道阻滞剂”开展了青年学术辩论，正反双方围绕疗效、安全性、个体化方案展开激烈辩论，青年医师充分展示了扎实的理论基础与卓越的临床思辨能力。卢强、王湘庆、杨志仙、陈述花等多位专家出席并进行专业点评，对辩论观点进行了系统梳理，指正逻辑漏洞，提出建设性意见，助力学术提升。50 余名癫痫专家和青年医师参会。

菁 Young 计划

2025 年 11 月 15 日，菁 YOUNG 计划·癫峰论道病例半决赛最后一场在济南举行。青委会副主任、浙江大学第二医院丁瑶教授、大区负责人、复旦大学附属华山医院刘晓英教授和大区负责人、中山大学附属第一医院倪冠中教授带队参会并担任评委。山东省立医院刘学伍教授和上海交通大学附属上海儿童医学中心叶晓莱教授担任主持。由申苏鲁、浙皖赣闽粤桂琼港三个大区各选派 2 名选手，本场 6 位选手参加了比赛。为位选手汇报难治性癫痫、自身免疫性脑炎等病例，专家点评诊疗亮点与不足，结合评分标准评选卓越及优秀选手。本次活动聚焦难治性病例诊疗难点，促进经验共享与技术提升，助力青年医生专业成长。本次活动聚焦难治性病例诊疗难点，促进经验共享与技术提升，助力青年医生专业成长，活跃了学术思想，激发了学习热情。参会人员数量 80 余人。

2026 年 1 月 23 日，CAAE 青委会菁 YOUG 计划巅峰病例决赛在广西南宁成功举办。近百名青委及青年癫痫诊疗工作者齐聚现场，共赴这场病例交流盛宴。本次决赛中，6 位在半决赛中表现优异的青年委员脱颖而出、登台分享，分别是浙江大学第二医院汪珊、空军军医大学第一附属医院陈蓓蓓、陆军军医大学第二附属医院孙晓琴、中国医科大学附属盛京医院范玉颖、临沂市人民医院赵娟、首都医科大学三博脑科医院杨华俊青委。

各位选手围绕所在单位的典型癫痫病例展开详细分享，病例涵盖遗传性疾病、脑皮质发育不良等多种病症，涉及内外科综合治疗场景。分享过程中，选手们不仅呈现了完整详实的病例资料、全面系统的检查报告，重点展示了遗传、代谢等相关专项检查结果，还深入分享了病例的精准诊疗方案制定过程及最新相关文献复习成果，充分展现了青年癫痫工作者的专业素养与临床实践能力。

本次决赛邀请到安徽省立医院杨斌主任、广州市妇女儿童医疗中心柳州医院黄晓利主任担任主持，山东省立医院刘学伍教授等专家组成评审团，全程对参赛病例的诊断思路、治疗方案及各位选手的临场表现进行了细致点评与全面总结，既肯定了亮点优势，也针对性提出了优化建议。此次巅峰病例决赛的成功举办，进一步搭建了癫痫领域多学科交流平台，有效推广了精准诊疗理念，为临床一线处理复杂癫痫病例提供了宝贵的实践参考与思路借鉴。

“菁 YOUNG 计划”旨在为中青年医师搭建一个展示、交流、成长的平台，推动癫痫诊疗理念的更新与临床实践的规范。他强调，青年医师是学科发展的未来，唯有在实践中磨砺、在交流中提升，才能真正实现“癫峰论践”的初心与使命。

AI 大模型在癫痫领域的应用研讨会

为顺应 AI 大模型广泛应用的时代趋势，帮助更多青年委员了解、合理运用 AI 技术，中国抗癫痫协会青年委员会于 2025 年积极探索创新，以“AI 赋能，智驱未来”为核心方向，通过医工融合等多渠道模式，将 AI 大模型逐步引入癫痫诊疗与研究领域。我们始终坚信，AI 大模型并非要替代医护工作者，而是将成为助力临床诊疗与科研创新的强大助手。借助医工结合、协同创新的力量，我们正稳步迈入一个由 AI 赋能，更精准、更智能、更具人性化的癫痫诊疗新时代。

2025 年 11 月 30 日上午，中国抗癫痫协会青年委员会第三场“AI 大模型在癫痫领域的应用研讨会”在北京隆重拉开帷幕。本次会议由中国抗癫痫协会青年委员会主任委员、首都医科大学附属北京儿童医院梁树立教授组织主持，来自京（西）冀晋、京（东）津蒙、黑吉辽、申苏鲁、陕甘青宁新五大区域的青年委员齐聚一堂，共话 AI 与癫痫领域的融合发展。

会议特别邀请到中国科学院自动化所、西北工业大学、广州实验室的三位科学家，以及小儿方健康科技有限公司、灵犀云医学科技（北京）有限公司的两位医学转化领域专家，围绕大语言模型的开发与临床应用开展专题讲座，分享前沿技术与实践经验。随后，邓劼、乔梁、刘强强、范玉颖、王雄飞等五位青年委员，就自身围绕大语言模型与癫痫诊疗工作深度结合开展的系列研究成果进行了专题分享。

本次会议由龙浩、赵瑞教授，以及关宇光、林一聪、李光健、闫志强、林楠等青年委员共同主持。研讨环节中，12 名青年委员围绕常规大语言模型，以及认知、儿科专家医生、癫痫脑电、癫痫外科等垂类大语言模型在癫痫领域的应用场景、实践难点与发展方向展开热烈讨论，现场交流氛围浓厚。会议伊始，中国抗癫痫协会段立嵘秘书长受邀参会并发表致辞，对本次研讨会的举办意义给予充分肯定，并对青年委员借助 AI 技术推动癫痫诊疗创新提出殷切期望。


AI助力医学发展
科技改变癫痫诊疗

AI大模型在癫痫领域的应用研讨会(北京站)

主办:中国抗癫痫协会青年委员会

2025年11月30日

时间	内容	主讲人/主持
08:30-08:50	开幕式 段立嵘秘书长致辞 梁树立主任委员致辞	关宇光 首都医科大学三博脑科医院
主持：林一聪 首都医科大学宣武医院、李光健 吉林大学第一医院、龙浩 南方医科大学南方医院		
08:50-09:10	面向脑认知的大语言模型及其应用	牛景昊 中科院自动化所
09:10-09:30	AI儿科医生的研发与应用	任 斌 小儿方健康科技有限公司
09:30-09:50	癫痫脑电基础模型的构建与应用	张 拓 西北工业大学
09:50-10:20	精准医疗新引擎-AI驱动下的个体化ASMs选择创新实践	邓 劼 首都医科大学附属北京儿童医院
10:20-10:40	讨论	谭泊静 首都儿科医学中心 王 秀 首都医科大学附属北京天坛医院 刘金之 山东第一医科大学第一附属医院 张 旭 中国人民解放军总医院
主持：闫志强 空军军医大学西京医院、林楠 北京协和医院、赵瑞 上海市儿童医院		
10:40-11:00	脑机大模型与精准诊疗技术	闫宇翔 灵犀云医学科技(北京)有限公司
11:00-11:18	AI人工智能助力相关科研	杨小枫 广州国家实验室
11:18-11:36	AI在癫痫外科的应用与展望	乔 梁 首都医科大学宣武医院
11:36-11:54	癫痫脑电大模型应用	刘强强 上海交通大学附属瑞金医院
11:54-12:14	讨论	徐成伟 内蒙古医科大学附属医院 黎 瀚 中国医科大学附属盛京医院 田小娟 首都医科大学附属北京儿童医院 谢 涛 河北医科大学第二附属医院
主持：樊星 首都医科大学附属北京天坛医院、杜晓萍 山西医科大学第一附属医院、刘亚青 兰州大学第二医院		
12:14-12:29	精准医疗赋能癫痫外科发展	李思阳 杭州佳量医疗科技有限公司
12:29-12:47	人工智能在 WEST 综合症中的应用	范玉颖 中国医科大学附属盛京医院
12:47-13:05	大语言模型下 Mapping 在癫痫中的应用	王雄飞 首都医科大学三博脑科医院
13:05-13:25	讨论	孙家行 哈尔滨医科大学第二附属医院 于 昊 北京大学第一医院 吕瑞娟 首都医科大学附属北京天坛医院 姚晓娟 天津医科大学总医院
13:25-13:30	总结	

统筹策划 张 慧 段立嵘 梁树立

刊头题字 李世绰

资料来源 全体青年委员 CAAE网站

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CHINA ASSOCIATION AGAINST EPILEPSY YOUTH COMMITTEE

THINKINGS AND ACTIONS