# 南通大学高层次人才培育计划

# 培养对象申请表

| | |
|---|---|
| 申 请 人： | 毛苏苏 |
| 所在单位： | 神经再生重点实验室（与神经科学系、神经再生协同创新中心合署） |
| 申报类别： | B 计划 |
| 填表时间： | |

南通大学高层次人才工作办公室制

# 填写说明

一、填写本表前，请认真阅读《南通大学高层次人才支持计划实施办法》和相关通知。

二、申请表中应明确填写清楚申报类别、发展目标、目前已具备的基本条件、未来工作计划等各项内容。

三、表中所涉个人前期业绩成果均应做到真实完整、信息准确、无知识产权纠纷及其他异议。

四、本表请采用 A4 纸双面打印。

## 一、申请人基本情况

| 姓　　名 | 毛苏苏 | 性　　别 | 男 | 国　　籍 | 中华人民共和国 |
|---|---|---|---|---|---|
| 出生年月<br>年龄（周岁） | 1987.12<br>39 | 政治面貌 | 中国共产党党员 | 党政职务 | 无 |
| 最高学历 | 博士研究生毕业 | 最高学位 | 博士研究生毕业 | 联系电话 | 13913858501 |
| 现任专业技术职务及任职时间 | 副研究员（自然科学）<br>2020-07-01 | | | | |
| 所在学科一级学科及研究方向 | 基础医学<br>神经元轴突再生 | | | | |
| 最高学历毕业学校及毕业时间 | 南京大学<br>2015-06-01 | | | | |
| 海外学习及工作（含博后）经历 | 哈佛大学医学院波士顿儿童医院，2019/01-2020/01,访问学者，导师：神经再生领域世界顶级科学家 Zhigang He 院士。 | | | | |

注：海外经历请注明学习工作高校（科研院所）的世界排名、起止时间及个人职务等情况

## 二、培育目标及现有基础

| 申报类别 | B 计划 | |
|---|---|---|
| 培育目标 | A 计划第一档 | A 计划第二档 |
| | B 计划<br>江苏省"333 高层次人才培养工程” | |

| | |
|---|---|
| 现有基础 | （对照培育目标，简要列举申请人已具备的基础条件，着重标出已取得的标志性成果，500字内）<br>本人主要从事神经损伤与修复的机制研究，2026 年申请江苏省杰出青年项目，并出校。<br>现有基础如下：<br>一、科研项目：<br>1. 国家自然科学基金面上项目，卫星胶质细胞源性乳酸供给下调在老年周围神经再生中的作及机制研究，2025.01-2028.12，49 万，主持。<br>2. 国家重点研发计划子课题，精准电磁调控神经通路的生物物理机制，2021.12-2026.11，万，主持。<br>3. 国家自然科学基金青年项目，JAK2 可变剪接异构体调控周围神经再生的机制研究2018.01-2020.12，23 万，主持。<br>4. 江苏省高等学校自然科学重大项目，环状 RNA circ-Spidr 调控神经元轴突再生的功能和机研究，2021.08-2024.07，30 万，主持。<br>5. 江苏省自然科学基金青年项目，JAK2△14 调控周围神经再生的机制研究，2017.07-2020.020 万，主持。<br>6. 江苏省高校自然科学研究面上项目，FEZ1S/ Δ E4 调节周围神经轴突再生的研究2016.09-2018.08，3 万，主持。<br>7. 江苏省政府留学基金项目，JS-2018-189，2019/01-2020/01，12 万，主持。<br><br>二、论文：以第一作者或通讯作者（含共同）发表在 PLoS Biology、eLife、Science Signalin Advanced Science、JBC、Protein Cell、Journal of neuroinflammation 等期刊。其中 Nature Ind论文 2 篇，中科院一区论文 8 篇。<br>近 5 年内发表的 5 篇代表性论著:<br>1. Susu Mao（一作）, Yuanyuan Chen, Wei Feng, Songlin Zhou, Chunyi Jiang, Junjie Zhan Xiaohong Liu, Tianmei Qian, Kai Liu, Yaxian Wang, Chun Yao, Xiaosong Gu, Bin Yu; RSI |

## 三、近五年主要业绩

**（一）主要完成或在研的科研项目（作为负责人承担的国家级科研项目可全部列出，一般不超过 5 项）**

| 项目名称 | 开始时间/到账时间 | 项目分类 | 获得资助金额 | 本人作用 | 项目级别（纵向） |
|---|---|---|---|---|---|
| 卫星胶质细胞源性乳酸供给下调在老年周围神经再生中的作用及机制研究 | 2024-01 | 面上项目 | 49 | 主持 | 国家级 |
| 精准电磁调控神经通路的生物物理机制 | 2021-01 | 国家重点研发计划课题 | 60 | 主持 | 国家级 |

| | | | | | |
|---|---|---|---|---|---|
| 环状 RNAcirc-Spidr 调控神经元轴突再生的功能和机制研究 | 2021-01 | 重大项目 | 30 | 主持 | 市厅级 |

注：本人作用指负责主持；项目进展情况包括已完成、已完成且成果已转化应用或正在进行等。

**（二）近五年主要完成或在研的教学教改项目（作为负责人承担的教学项目可全部列出，一般不超过 5 项）**

| 项目名称 | 获奖、立项等级 | 本人排名 | 获奖、立项时间 |
|---|---|---|---|
| | | | |

**（三）近五年主要论文、著作情况**

**1.主要论文目录（按重要性排序，合计不超过 5 篇）**

| 期刊名称 | 论文题目 | 论文级别 | 发表时间 | 第一署名单位 |
|---|---|---|---|---|
| PLOS BIOLOGY | RSK1 promotes mammalian axon regeneration by inducing the synthesis of regeneration-related proteins | 三级：SCI（中科院一区） | 2022-06-01 | 第一单位 |

**2.主要著作目录（按重要性排序，合计不超过 5 本）**

| 书　名 | 出版社 | 出版日期 |
|---|---|---|
| | | |

**（四）获得主要学术奖励情况（包含科研、教学，按重要性排序，合计不超过 2 项）**

| 获奖项目名称 | 奖励名称 | 奖励等级 | 获奖时间 | 奖励单位 |
|---|---|---|---|---|
| | | | | |

**（五）近五年获得省部级以上荣誉称号情况（按重要性排序，合计不超过 2 项）**

| 荣誉称号 | 入选时间 | 颁发部门 |
| --- | --- | --- |
| 江苏省高校“青蓝工程”优秀青年骨干教师 | 2025-07 | 江苏省教育厅 |

**（六）授权国内与国际发明专利、成果转换情况（按重要性排序，合计不超过 5 项）**

**1.授权国内与国际发明专利情况**

| 专利名称 | 专利号 | 国别 | 授权时间 | 本人排名 |
| --- | --- | --- | --- | --- |
| 乌嘌呤和异乌嘌呤在制备治疗或修复周围神经损伤药物中的应用 | 202411570883X | 国内 | 2025-11-25 | 1 |

**2.成果转化情况**

| 成果名称 | 转化时间 | 首次受让企业 | 本人作用（主要负责或参与） |
| --- | --- | --- | --- |
| | | | |

**（七）其他成果**

**1.其他科研项目**

| 项目名称 | 开始时间/到账时间 | 项目分类 | 获得资助金额 | 项目状态 | 本人作用 | 项目级别（纵向） |
| --- | --- | --- | --- | --- | --- | --- |
| | | | | | | |

注：本人作用指负责主持；项目进展情况包括已完成、已完成且成果已转化应用或正在进行等。

**2.其他教学教改项目**

| 项目名称 | 获奖、立项等级 | 本人排名 | 获奖、立项时间 |
| --- | --- | --- | --- |
| | | | |

**3.其他论文**

| 期刊名称 | 论文题目 | 论文级别 | 发表时间 | 学校署名 |
|---|---|---|---|---|
| JBC/神经再生重点实验室 | G6PD facilitates axon regeneration via clathrin-mediated endocytosis | 三级 | 2026-04-01 | 第一单位 |
| Science signaling/神经再生重点实验室 | Metabolic alterations after traumatic neural injury: Mechanistic insights and potential translational targets for axon regeneration | 三级 | 2026-02-24 | 第一单位 |
| elife/神经再生重点实验室 | Promoting axon regeneration by inhibiting RNA N6-methyladenosine demethylase ALKBH5 | 三级 | 2023-08-03 | 第一单位 |
| Advanced science/神经再生重点实验室 | LINC MIR503HG Controls SC-β Cell Differentiation and Insulin Production by Targeting CDH1 and HES1 | 三级 | 2024-04-01 | 第一单位 |

**4.其他著作**

| 书　名 | 出版社 | 出版日期 |
|---|---|---|
| | | |

**5.其他学术奖励**

| 获奖项目名称 | 奖励名称 | 奖励等级 | 获奖时间 | 奖励单位 |
|---|---|---|---|---|
| | | | | |

**6.其他授权国内与国际发明专利情况**

| 专利名称 | 专利号 | 国别 | 授权时间 | 本人排名 |
|---|---|---|---|---|
| | | | | |

**8.其他成果转化情况**

| 成果名称 | 转化时间 | 首次受让企业 | 本人作用（主要负责或参与） |
|---|---|---|---|
| | | | |

**四、未来工作计划**

| | |
|---|---|
| 突破方向 | （对照培育目标及现有条件，分析目标达成中的现实差距及需要突破的方面，500 字内）<br>本人在神经损伤修复领域已积累较为扎实的学术基础，相关成果发表于 Plos biology、eLife、JBC、Advanced Science 等高水平期刊。其中，部分工作被遴选为 eLife digest 专题报道，美国康奈尔大学 Willis 教授专门就我们的研究在 Plos Biology 发表正面评述文章。近年来，本人围绕神经再生的代谢调控开展系列研究，逐步形成了自身的研究特色，在 Science 子刊 Science signaling 发表代谢调控神经再生的综述文章，该文被遴选为封面文章。此外，本人受邀担任 Science Translational Medicine、Advanced Science、Glia 等期刊审稿人，并任法国国家研究署研究基金评审专家，这些工作体现了国际同行的高度认可。<br>目前，本人正围绕衰老导致神经再生能力下降这一方向开展系统研究，相关课题已获国自然面上项目支持。为实现本阶段的培育目标，需在以下方面取得突破：<br>1.成果：发表 2-3 篇具有高影响力的论文，其中 1 篇发表于 cell metabolism 或 Nature aging 等大子刊，或两篇发表于 Nature communications、Science advances 等期刊。目前第一篇文章正处于数据整理和文稿撰写阶段。<br>2.项目：获得 1 项国家级项目（面上项目、专项项目或科技部重点研发计划课题）、1-2 项省部级以上项目；同时拓展项目评审的参与机会。今年已申请江苏省杰青项目和国自然面上项目。<br>3.学术交流：进一步加强与国内外著名专家的学术交流，目标成为神经损伤修复领域具有影响力的专家，推动本学科的持续发展。 |

| | |
|---|---|
| 未来工作计划 | （对照培育目标、现有条件及拟突破的方向，拟定未来的主要工作及安排，800 字内）<br>2026.08-2027.07：2026 年已申请江苏省杰出青年项目，并出校；如未获批，2027 年继续申请，并同时申请青拔项目。代谢与神经再生课题已基本完成，正撰写论文，计划 2026 年底投稿大子刊 cell metabolism；继续开展衰老导致神经再生能力下降的机制研究，申请基金委专项项目 1 项；联合清华大学饶子和院士团队开展的小核酸药物研究已进入投稿阶段，准备投稿 Nature 子刊；联合法国 Homaira Nawabi 教授，申请国家自然科学基金欧洲青年科研人员来华交流项目，进一步加强国际学术交流。<br>2027.08-2028.07：完善衰老导致神经再生能力下降的机制研究，总结数据，投稿子刊 nature aging；基于上述积累，申请省部级项目 1 项；继续深入开展小核酸药物的应用研究，联合清华大学饶子和院士团队申请科技部重点研发计划项目；在国际或国内重要学术会议上做报告。<br>2028.08-2029.07：进一步完善衰老影响神经再生能力的作用和机制研究，申请国自然面上项目 1 项，申请发明专利 1-2 项，总结数据，投稿；联合香港科技大学刘凯教授举办神经再生国际论坛，担任专业 SCI 期刊的青年编委；推进所获专利的成果转化，建设小核酸药物促神经再生研究平台。申报江苏省"333 高层次人才培养工程”。 |

**五、需要学校提供的支持**

| 是否实验类 | 是 | |
|---|---|---|
| 培育建设经费预算 | 工作计划 | 预算（万元） |
| | 实验材料费 | 15 |
| | 测试化验加工费 | 5 |
| | 劳务费、专家咨询费 | 4 |

| | | |
|---|---|---|
| | 出版费 | 3 |
| | 会议费 | 3 |
| | 合计 | 30 万元 |
| 其他支持需求清单 | 在中期完成情况良好的情况下，继续追加经费支持 | |
| | 在举办学术会议的场地、经费等方面给予支持 | |
| | 职称评审时，在同等条件下优先考虑 | |

**五、申请人承诺**

（对照未来拟开展的工作，列举需要学院及学校提供的其他支持事项的清单）

1. 以上所填信息均真实有效；
2. 如入选学校培育计划，按期完成培育任务，规范使用学校支持经费；
3. 培育目标达成后，本人志愿在本校服务 10 年以上。

申请人签字：_____

_____年_____月_____日

**六、二级单位审核推荐意见**

| | |
|---|---|
| 1. 申请人填写的内容是否属实？ | 是 |
| 2. 申请人发展目标是否符合学院发展规划？ | 是 |
| 3. 申请人是否具备培育目标的基础条件及培养潜力？ | 是 |
| 4. 申请人对目标达成的现实差距及突破点的分析是否准确？ | 是 |
| 5. 申请人制定的工作计划是否切实可行？ | 是 |
| 6. 申请人对培育建设经费的预算是否合理？ | 是 |
| 7. 学院是否同意推荐申请人申请相关计划？ | 是 |
| 培养期内，学院为申请人提供以下支持：<br><br>在研究生名额分配给予倾斜。<br><br>优先使用学院科研和教学平台。<br><br>提供优质的科研支撑团队。<br><br>党政负责人签字：<br><br>（单位公章）<br><br>_____年_____月_____日 | |

**七、学校人才工作领导小组意见**

经评审，同意_____同志入选_____培育计划。

组长（签章）：

_____年_____月_____日

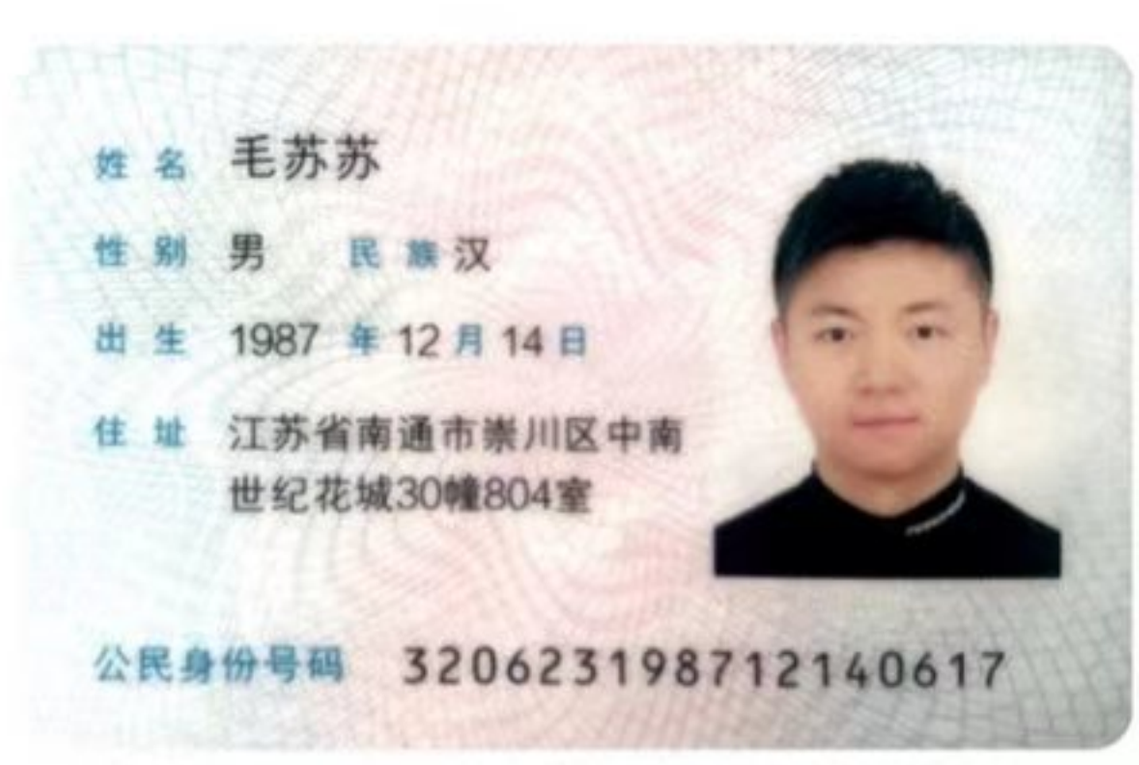
**八、学校意见**

学校公章：　　校长签字：

_____年_____月_____日

# 申报材料附件

毛苏苏


姓名 毛苏苏
性别 男 民族 汉
出生 1987 年 12 月 14 日
住址 江苏省南通市崇川区中南
世纪花城30幢804室
公民身份号码 320623198712140617


中华人民共和国
居民身份证
签发机关 南通市公安局崇川分局
有效期限 2021.01.09-2041.01.09

# 博士学位证书


毛苏苏，男，1987 年 12 月 14 日生。在 南京大学 生物学 学科（专业）已通过博士学位的课程考试和论文答辩，成绩合格。根据《中华人民共和国学位条例》的规定，授予 理学 博士学位。

南 京 大 学

校 长

学位评定委员会主席 陈骏

证书编号：1028422015300613

二〇一五 年 六 月 十五 日

姓　　名　毛苏苏

性　　别　男

身份证号　320623198712140617

工作单位　南通大学

编　　号　2020423G0101

经　江苏省南通大学教师高级专业技术资格评审委员会于2020年07月23日评审，毛苏苏已具备 副研究员 资格。

公布文号：　通大人〔2020〕10号

发证机关

（印章）

2020年07月23日

PLOS BIOLOGY

RESEARCH ARTICLE

# RSK1 promotes mammalian axon regeneration by inducing the synthesis of regeneration-related proteins

**Susu Mao[1☯], Yuanyuan Chen[1☯], Wei Feng[1☯], Songlin Zhou[1], Chunyi Jiang[1], Junjie Zhang[1], Xiaohong Liu[1], Tianmei Qian[1], Kai Liu[2], Yaxian Wang[1], Chun Yao[1], Xiaosong Gu[1,3], Bin Yu[1,3]***

**1** Key Laboratory of Neuroregeneration of Jiangsu and Ministry of Education, NMPA Key Laboratory for Research and Evaluation of Tissue Engineering Technology Products, Co-innovation Center of Neuroregeneration, Nantong University, Nantong, China, **2** Division of Life Science, State Key Laboratory of Molecular Neuroscience, The Hong Kong University of Science and Technology, Hong Kong, China, **3** Jiangsu Clinical Medicine Center of Tissue Engineering and Nerve Injury Repair, Affiliated Hospital of Nantong University, Nantong University, Nantong, China

☯ These authors contributed equally to this work.
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**Academic Editor:** Cody J. Smith, University of Notre Dame, Center for Stem Cells and Regenerative Medicine, UNITED STATES

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**Published:** June 1, 2022



**Data Availability Statement:** In addition to the data within the paper and its Supporting Information files, additional files are available at the SRA portal of NCBI under accession number SRP317959.

**Funding:** This work was supported by the National Key R&D Program of China (http://www.most.gov.cn; 2017YFA0104701 to BY, 2021YFA1201402 to SM, 2021YFA1201404 to BY); the National Natural Science Foundation of China (http://www.nsfc.gov.

## Abstract

In contrast to the adult mammalian central nervous system (CNS), the neurons in the peripheral nervous system (PNS) can regenerate their axons. However, the underlying mechanism dictating the regeneration program after PNS injuries remains poorly understood. Combining chemical inhibitor screening with gain- and loss-of-function analyses, we identified p90 ribosomal S6 kinase 1 (RSK1) as a crucial regulator of axon regeneration in dorsal root ganglion (DRG) neurons after sciatic nerve injury (SNI). Mechanistically, RSK1 was found to preferentially regulate the synthesis of regeneration-related proteins using ribosomal profiling. Interestingly, RSK1 expression was up-regulated in injured DRG neurons, but not retinal ganglion cells (RGCs). Additionally, RSK1 overexpression enhanced phosphatase and tensin homolog (PTEN) deletion-induced axon regeneration in RGCs in the adult CNS. Our findings reveal a critical mechanism in inducing protein synthesis that promotes axon regeneration and further suggest RSK1 as a possible therapeutic target for neuronal injury repair.

## Introduction

Successful axon regeneration will be of benefit in treating many human diseases involving axon damage, such as traumatic brain injury, stroke, spinal cord injury, sciatic nerve injury (SNI), and numerous neurodegenerative diseases, in both the central nervous system (CNS) and peripheral nervous system (PNS) [1–3]. In contrast to the CNS, injured neurons in the PNS have robust regenerative capability, which largely depends on the up-regulation of the expression or activity of key molecules that promote axon regeneration following injury [4–7]. Hence, uncovering the molecular mechanisms underpinning axon regrowth following neuronal injury in the PNS will greatly aid the understanding of the differential regenerative capacity between neurons in the PNS and CNS and contribute to identifying factors with the potential to facilitate nerve regeneration.

JBC RESEARCH ARTICLE

# G6PD facilitates axon regeneration *via* clathrin-mediated endocytosis

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**Metabolic reprogramming is a hallmark of neuronal repair, yet the roles of glucose metabolism-related enzymes remain poorly understood. To investigate their functions, we employed a sciatic nerve injury model, taking advantage the intrinsic regenerative capacity of peripheral neurons. After sciatic nerve crush injury, dorsal root ganglia exhibited sustained upregulation of several enzymes in the pentose phosphate pathway. Notably, glucose-6-phosphate dehydrogenase (G6PD), the rate-limiting enzyme of the pentose phosphate pathway, was markedly increased at both RNA and protein levels. Silencing G6PD impaired axon regeneration *in vitro* and *in vivo*, whereas its overexpression enhanced regrowth. Interestingly, G6PD overexpression did not alter the $NADP^+$/NADPH ratio, suggesting a nonmetabolic role. Using mass spectrometry, coimmunoprecipitation, and Duolink proximity ligation assays, we identified clathrin heavy chain as a specific binding partner of G6PD. Mechanistic analyses further showed that G6PD facilitated neuronal endocytosis through direct interaction with clathrin heavy chain, thereby promoting axon regeneration. These findings identify G6PD as a molecular link between metabolic reprogramming and membrane trafficking, revealing an unexpected nonmetabolic role in neural repair.**

Metabolism comprises a precisely coordinated network of chemical reactions that are fundamental to life. Beyond providing essential substrates and energy, it also serves as a key regulator of gene expression. Through metabolic enzymes and their products, metabolites, metabolism exerts profound influence over cell growth, survival, differentiation, and the maintenance of systemic homeostasis (1, 2). Metabolic reprogramming, the dynamic alteration of cellular metabolic pathways, enables cells to adapt to changing energy demands and biosynthetic needs under physiological or pathological conditions (3).

In recent years, increasing attention has been focused on the role of metabolic reprogramming in neural repair (4–6). Neural regeneration requires not only substantial energy but also an abundant supply of lipids, as well as the synthesis of proteins and nucleic acids, all of which depend on reprogrammed metabolic processes after injury and are driven by specific metabolic enzymes (7–10). Several of these enzymes have been identified as critical regulators of axonal regeneration. For instance, PTEN, a lipid phosphatase, dephosphorylates phosphatidylinositol (3,4,5)-trisphosphate (PIP3) into phosphatidylinositol (4,5)-bisphosphate, thereby negatively regulating the PI3K pathway. The PTEN–PI3K signaling axis has been well established as a key modulator of axonal regeneration, as deletion of PTEN significantly enhances regenerative capacity within the central nervous system (CNS) (11, 12). Similarly, Anja *et al.* identified plasticity-related gene 1, which encodes a membrane-associated lipid phosphate phosphatase expressed in neurons and localized to growing axonal membranes. Plasticity-related gene 1 acts as an ectoenzyme, counteracting phospholipid-induced axonal collapse and promoting hippocampal axon elongation (13). In addition, deletion of LPIN1, a phosphatidic acid phosphatase, enhances optic and sciatic nerve repair by lowering triglycerides, promoting phospholipid synthesis, and remodeling lipid metabolism (5). We further showed that LPIN2, another family member, supports regeneration in dorsal root ganglia (DRG) neurons by increasing triglyceride-enriched lipid droplets (14). Moreover, our previous work identified pyruvate dehydrogenase beta subunit, a core tricarboxylic acid (TCA) cycle enzyme, as a facilitator of axonal regeneration through coordinated regulation of energy metabolism and epigenetic reprogramming (15). Collectively, these findings highlight that targeting metabolic enzymes to induce metabolic reprogramming represents a promising therapeutic strategy for promoting axonal regeneration after neural injury.

Glucose metabolism is a fundamental pathway that supports cellular energy production and biosynthesis. Upon uptake through glucose transporters, glucose is catabolized *via* three major pathways, including glycolysis, the pentose phosphate pathway (PPP), and the TCA cycle. Although glucose metabolism is essential for cellular maintenance and repair, its specific role in axonal regeneration remain largely undefined. Recent studies highlighted injury-induced upregulation of glycolysis as a mechanism promoting axon regeneration (4), yet the contribution of individual metabolic enzymes requires further elucidation.

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NEUROSCIENCE



# Metabolic alterations after traumatic neural injury: Mechanistic insights and potential translational targets for axon regeneration

**Qianqian Cao[1]†, Wenwen Zhao[1]†, Junjie Yan[1], Stephane Belin[2], Bin Yu[1], Homaira Nawabi[2]*, Susu Mao[1]***

**Metabolism not only provides essential substances and energy for cells through catabolism and anabolism but also exerts broader regulatory roles through metabolic enzymes and products that influence gene expression, thereby maintaining homeostasis. Upon neuronal injury, metabolic changes in both neurons and supporting cells influence neuronal survival and regeneration by regulating energy supply, substrate availability, regeneration-related gene expression, and cell-cell metabolic interactions. Axon regeneration is a key process in neural repair after injury. Beyond the nervous system itself, systemic factors such as diet, exercise, circadian rhythms, and psychological stress also play crucial roles in axon regeneration through interorgan metabolic communication and microbiota-host metabolic cross-talk. In this Review, we summarize advances in understanding metabolic alterations during axon regeneration, with a focus on glycometabolism, lipid metabolism, protein degradation, mitochondrial activity, and systemic factor–driven metabolic cross-talk between nervous and non-nervous systems. We also highlight the therapeutic potential of metabolites themselves, analyze distinct metabolic responses after injury in the peripheral and central nervous systems, and discuss their spatiotemporal dynamics and cell type specificity. Last, we propose that successful neural repair requires the establishment of a systemic pro-regenerative state throughout the entire body.**

## INTRODUCTION

Axon regeneration is a critical process for restoring nervous system function after neural injury. This is a complex and coordinated process relying not only on the intrinsic regenerative capacity of neurons but also on the lesion microenvironment. In the central nervous system (CNS), limited intrinsic regenerative capacity and the presence of an inhibitory microenvironment pose challenges to axon regeneration after injury. However, in the peripheral nervous system (PNS), axon regeneration usually spontaneously initiates when neurons perceive injury, thereby activating a series of biological responses. Substantial progress has been made in elucidating the cellular and molecular mechanisms that govern axon regeneration. Nonetheless, the influence of metabolic alterations in supporting and modulating this process continues to be a subject of intense research (*1*–*3*). Metabolism is indispensable not only for maintaining cellular energy homeostasis but also for regulating signaling networks and the epigenetic modulation of gene expression, which in turn affects the pace and success of nerve regeneration (*4*–*7*).

Key metabolic pathways, such as glycometabolism, lipid metabolism, and protein degradation and synthesis, are recognized for their regulatory roles in axon regeneration. The basic function of glucose metabolism is to yield adenosine triphosphate (ATP), the energy currency underpinning diverse physiological activities and tissue repair processes. Lipid metabolism, particularly the role of fatty acids in membrane remodeling, is crucial for cellular structural adaptation (*8*). Furthermore, intermediate metabolites like pyruvate, lactate, phosphatidylinositol (3,4,5)-trisphosphate ($PIP_3$), phosphatidic acid (PA), lysophosphatidic acid (LPA), and arachidonic acid (AA), which are generated by glucose and lipid metabolism, are also signaling molecules that modulate intracellular neuronal pathways and epigenetic modifications, directly participating in neuronal restoration (*9*, *10*). The balance between protein degradation and synthesis is essential for neuronal recovery, providing necessary materials and ensuring the removal of damaged proteins (*11*, *12*). Moreover, mitochondria are instrumental in sustaining energy homeostasis and promoting axonal regrowth, and mitochondrial dysfunction is linked to various neurodegenerative conditions and hinders regeneration after nerve injury (*13*, *14*).

Although intrinsic neuronal metabolism is fundamental for axonal regeneration, other cell types, including glial cells, immune cells, and other supporting cells, also play essential roles (*15*–*19*). Metabolic cross-talk between the nervous system and non-nervous tissues contributes to axon regeneration (*20*–*23*). For example, the gut microbiota produces metabolites such as short-chain fatty acids (SCFAs) that can actively influence nerve regeneration (*24*, *25*). In addition, adipose tissue communicates with the nervous system through adipokines such as leptin, which supports nerve regeneration. Metabolic alterations in adipose tissue can affect neuronal energy supply, neuroprotection, and the repair mechanisms involved in axon regeneration (*26*). These findings suggest that cross-system metabolic cross-talk may provide new strategies for promoting neural repair.

In this Review, we provide an overview of metabolic changes during axon regeneration, focusing on glycometabolism, lipid metabolism, protein degradation, and mitochondrial dynamics. We examine how systemic factors such as diet, exercise, circadian rhythm, and psychological stress regulate regenerative processes, with particular

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RESEARCH ARTICLE

# Promoting axon regeneration by inhibiting RNA N6-methyladenosine demethylase ALKBH5

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**Abstract** A key limiting factor of successful axon regeneration is the intrinsic regenerative ability in both the peripheral nervous system (PNS) and central nervous system (CNS). Previous studies have identified intrinsic regenerative ability regulators that act on gene expression in injured neurons. However, it is less known whether RNA modifications play a role in this process. Here, we systematically screened the functions of all common m$^6$A modification-related enzymes in axon regeneration and report ALKBH5, an evolutionarily conserved RNA m$^6$A demethylase, as a regulator of axonal regeneration in rodents. In PNS, knockdown of ALKBH5 enhanced sensory axonal regeneration, whereas overexpressing ALKBH5 impaired axonal regeneration in an m$^6$A-dependent manner. Mechanistically, ALKBH5 increased the stability of *Lpin2* mRNA and thus limited regenerative growth associated lipid metabolism in dorsal root ganglion neurons. Moreover, in CNS, knockdown of ALKBH5 enhanced the survival and axonal regeneration of retinal ganglion cells after optic nerve injury. Together, our results suggest a novel mechanism regulating axon regeneration and point ALKBH5 as a potential target for promoting axon regeneration in both PNS and CNS.

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## Editor's evaluation

The work presents a valuable and significant advance in the genetic mechanisms of mRNA m$^6$A demethylation as a key regulator of axon regeneration. The fundamental work substantially advances our understanding of a major research question. Screening m$^6$A regulators during axon regeneration uncovered ALKBH5 as limiting regenerative dorsal root ganglia growth by enhancing the stability of *Lpin2* mRNA via erasing a single m$^6$A modification in the 3'UTR. The major strength of the manuscript is the convincing importance of ALKBH5 as a suppressor of axon regeneration in the CNS and PNS proven by in vivo model system. These findings further suggest the potential use of ALKBH5 inhibitors to enhance neural regeneration upon physical injury.

## Introduction

Triggering axon regeneration, accelerating nerve regeneration, and extending the regenerated nerve length are the most straightforward methods of nerve repair following injury (***Costigan et al., 2002***; ***Moore and Goldberg, 2011***; ***Palmisano et al., 2019***; ***Smith and Skene, 1997***). These processes are highly dependent on the intrinsic regeneration ability of neurons. However, diminished intrinsic

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# LINC MIR503HG Controls SC-$\beta$ Cell Differentiation and Insulin Production by Targeting CDH1 and HES1

*Yang Xu, Susu Mao, Haowen Fan, Jian Wan, Lin Wang, Mingyu Zhang, Shajun Zhu, Jin Yuan, Yuhua Lu, Zhiwei Wang, Bin Yu, Zhaoyan Jiang,* and Yan Huang**

**Stem cell-derived pancreatic progenitors (SC-PPs), as an unlimited source of SC-derived $\beta$ (SC-$\beta$) cells, offers a robust tool for diabetes treatment in stem cell-based transplantation, disease modeling, and drug screening. Whereas, PDX1$^+$/NKX6.1$^+$ PPs enhances the subsequent endocrine lineage specification and gives rise to glucose-responsive SC-$\beta$ cells in vivo and in vitro. To identify the regulators that promote induction efficiency and cellular function maturation, single-cell RNA-sequencing is performed to decipher the transcriptional landscape during PPs differentiation. The comprehensive evaluation of functionality demonstrated that manipulating LINC MIR503HG using CRISPR in PP cell fate decision can improve insulin synthesis and secretion in mature SC-$\beta$ cells, without effects on liver lineage specification. Importantly, transplantation of MIR503HG$^{-/-}$ SC-$\beta$ cells in recipients significantly restored blood glucose homeostasis, accompanied by serum C-peptide release and an increase in body weight. Mechanistically, by releasing CtBP1 occupying the CDH1 and HES1 promoters, the decrease in MIR503HG expression levels provided an excellent extracellular niche and appropriate Notch signaling activation for PPs following differentiation. Furthermore, this exhibited higher crucial transcription factors and mature epithelial markers in CDH1$^{High}$ expressed clusters. Altogether, these findings highlighted MIR503HG as an essential and exclusive PP cell fate specification regulator with promising therapeutic potential for patients with diabetes.**

## 1. Introduction

As a global health epidemic, diabetes afflicts an estimated 537 million people worldwide due to $\beta$ cell deficiency or dysfunction.[1] The generation of human pluripotent stem cell (hPSC)-derived functional pancreatic $\beta$ cells may solve the problem of cadaveric donor islet supply shortage for transplantation and it could also function as a model platform to shed light on the pathogenic mechanisms leading to various forms of diabetes[2] Encouraging progress has been achieved in prolonged in vitro hPSC differentiation toward mature and functional pancreatic $\beta$ cells by mimicking the in vivo normal developmental trajectory following less than 20 years of efforts.[3] However, imperfect differentiation efficiency, off-target and unexpected cell byproducts, multi-hormone cell production, and current cost- and time-consuming approaches limit the prospects of clinical applications and basic medical research.[3f,g,4] Currently, advanced in vitro differentiation protocols are based on at least six stages, initiating cells from the definitive endoderm (DE), which

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for the interplay between MIR503HG KO and the specific mechanism of action requires further investigation.

The Hippo/YAP pathway is another important signaling pathway, which participates in dominating the generation of PP-derived functional $\beta$ cells.[53] The Hippo pathway reportedly integrates the tissue architecture by balancing progenitor cell self-renewal and differentiation. Inhibition of Hippo signaling leads to nuclear translocation of the downstream effectors YAP and TAZ, which bind to TEAD coactivators and activate the transcription of genes controlling progenitor cell proliferation.[54] In contrast, activation of the pathway promotes terminal differentiation of mature cell types by inducing the phosphorylation, cytoplasmic retention, and subsequent degradation of YAP/TAZ, resulting in target gene downregulation.[55] Moreover, E-cad is reportedly involved in cell growth inhibition by inactivating the Hippo pathway.[56] In the present study, E-cad expression was enhanced by the alleviation from the repression of binding between MIR503HG and CtBP1 in MIR503HG$^{-/-}$ cells, resulting in PP proliferation constrained with increased phosphorylation and cytoplasmic YAP retention.

Taken together, our study demonstrates that MIR503HG is a promising target for SC-$\beta$ cell therapy. Accordingly, MIR503HG loss promotes PP differentiation, giving rise to higher-quality SC-$\beta$ cells more efficiently and safely. Therefore, harnessing MIR503HG regulation will provide strategies for promoting human pancreatic differentiation and developing effective approaches for generating significant amounts of functional SC-$\beta$ cells for the fundamental study of pancreatic biology and regenerative medicine for diabetes.

## Supporting Information

Supporting Information is available from the Wiley Online Library or from the author.

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## Conflict of Interest

The authors declare no conflict of interest.

## Author contributions

Y.X., S.M., H.F., and J.W. contributed equally to this work. Y.X., Z.J., and Y.H. conceived and designed the project. Y.X., H.F., S.M., and J.W. performed the experiments. Y.X., H.F., J.W., and Y.H. analyzed the sequencing data. M.Z., S.Z., Y.L. J.Y., and L.W. helped with the experiments. Y.X, J.W., S.M., and Y.H. analyzed the data. Z.W., Z.J., and Y.H. provided reagents. Y.X., H.F., J.W., B.Y., Z.W., Z.J., and Y.H. wrote and revised the manuscript.

## Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

## Keywords

differentiation, human pluripotent stem cells, lncRNA, pancreatic progenitors, scRNA-seq

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3. **2024年9月23日：**报送纸质版计划书（一式两份，其中一份包含申请书纸质签字盖章页）的截止时间。

4. **2024年10月8日：**报送修改后的申请书纸质签字盖章页的截止时间。

**请按照以上规定及时提交电子版计划书，并报送纸质版计划书和申请书纸质签字盖章页，逾期不报计划书或申请书纸质签字盖章页且未说明理由的，视为自动放弃接受资助；未按要求修改或逾期提交申请书纸质签字盖章页者，将视情况给予暂缓拨付经费等处理。**

附件：项目评审意见及修改意见表

国家自然科学基金委员会

2024年8月23日

# 国家重点研发计划课题组织实施协议

**甲方（课题牵头单位）：同济大学(统一社会信用代码 12100000425006125J)**

**乙方（课题参与单位）：南通大学(统一社会信用代码 123200004660129 19A)**

经甲乙双方协商一致，就合作研究 2021 年度国家重点研发计划“纳米前沿”重点专项（项目编号：2021YFA1201400，项目名称：基于磁性微纳器件的退行性骨科疾病电磁治疗技术及临床研究）的课题（课题编号：2021YFA1201402，课题名称：精准电磁调控神经通路的生物物理机制）（以下课题均为此申报课题）有关事宜达成如下协议：

**一、任务分工：**

**甲方：**

课题承担单位，课题负责人：成昱

负责针对精准电磁调控神经通路的生物物理机制研究的实验方案设计、分工、实施和成果汇总，并承担筛选神经细胞电磁生物效应感受器和研究电磁效应调控神经-骨微环境细胞因子释放的规律，以及组织协调课题 2 各参与单位共同完成课题研发任务。筛选出至少 1 类可用于临床退行性骨科疾病电磁刺激治疗的神经网络作用靶点，并且阐明电磁效应调控神经网络诱导骨再生和修复的作用机制。

**乙方：**

课题参与单位，子课题负责人：毛苏苏

1. 负责制定 1 套研究微纳尺度电磁效应调控神经-骨微环境细胞因子释放的方案。

2. 协助同济大学完成神经细胞电磁生物效应感受器的筛选。

3. 协助同济大学阐明电磁效应活化神经细胞的作用机制，并实现通过该响应信号通路调控神经-骨微环境生物因子的程序化释放。

4. 培养博士 1 名，硕士 1 名。

**双方具体任务分工以科技部批复的《国家重点研发计划课题任务书》为准。**

**二、经费分配**

**1. 甲方**作为课题承担单位，分配专项经费 270.00 万元，其中间接经费 61.90 万元。

**2. 乙方**作为课题参与单位，分配专项经费 60.00 万元，其中间接经费 13.76 万元。

**3.** 双方具体经费分配以科技部批复的《国家重点研发计划课题任务书》为准。

**三、知识产权约定**

本研究过程中，由甲方或乙方独立完成的知识产权归属于完成方。由甲乙双方共同完成的知识产权或知识产权中存在共同完成的部分，由甲乙双方商定；若无对方许可，任何一方不得擅自申请或使用共同完成的成果。但相应成果均应注明本项目作为第一资助。

**四、人员承诺**

甲乙双方要采取切实措施保证研究成果符合《中华人民共和国保守国家秘密法》和《科学技术保密规定》等相关法律法规，不得将研究过程中双方提供的技术资料用于本项目以外的其它任何用途。

**五、其他约定：**

**1、**如果在项目实施过程中需要调整研究骨干等事宜，双方应通报商议解决。

**2、**本协议一式 6 份，2 份作为课题任务书附件材料上报，双方各持 2 份。自甲方和乙方盖章之日起具有法律效力。

**3、**未尽事宜，由合作双方另行商议解决。

甲方：同济大学

法人代表签章：

课题负责人签字：

2021年 12月 10日

乙方：南通大学

法人代表签章：

子课题负责人签字：

2021年 12月 13日

项目编号：21KJA180002

# 江苏省高等学校 基础科学（自然科学）重大项目合同

## （2021 年度）

项 目 名 称 ： 环状 RNA circ-Spidr 调控神经元轴突再生的功能和机制研究

项 目 负 责人: 毛苏苏

项 目 联 系人: 仇群仁

联 系 电 话 ： 13912261457

联 系 地 址 ： 南通市啬园路 9 号

邮 政 编 码 ： 226019

起 止 年 限 ： 2021 年 10 月～2024 年 9 月

所 在 学 校 ： 南通大学

填 表 日 期 ： 2021-10-31



江苏省教育厅

二〇二一年

# 留 学 回 国 人 员 证 明

（2019 纽 教(文) 证字 09770号

兹证明 毛苏苏 （男☐、女☐，护照号码 E84943104 ）系我国
HARVARD MEDICAL SCHOOL/BOSTON CHILDREN'S HOSPITAL

在 美 国 哈佛医学院/波士顿儿童医院 学校（单位）

的高级研究学者☐、访问学者☑、博士后☐、博士研究生☐、硕士研究生☐、

本科生☐、大专生☐、其他留学人员☐

在我驻外使（领）馆报到日期 2019 年 11 月 01 日

注册入学日期 2019 年 01 月 15 日

毕（结）业日期 2020 年 01 月 16 日

拟回国日期 2020 年 01 月 16 日

毕（结）业证书名称 _____ 号码 _____

备注（留学经历描述） _____

留学回国人员签字：

经办人签字：

负责人签字：

教育（文化）处（组）公章

2019 年 12 月 16 日

第一联：交留学回国人员　　教育部国际合作与交流司 2012 年制表

注意事项

1、本证明只为学成回国工作的留学人员开具。
2、本证明由我驻外使（领）馆教育（文化）处（组）在留学人员回国时填写，不得涂改。
3、本证明经使（领）馆教育（文化）处（组）经办人、负责人签字并在第一、第二联加盖公章方为有效。
4、第一联由留学人员保存，其他单位可查验原件，收存复印件，不得收取原件。

# 特　許　証
(CERTIFICATE OF PATENT)

## 特許第７６９１１５１号
(PATENT NUMBER)

発明の名称
(TITLE OF THE INVENTION)
レンギョウ・オウギ復方漢方薬煎剤由来のマイクロリボ核酸、その製造方法、及び使用

特許権者
(PATENTEE)
中華人民共和国　２２６０１９　江蘇省南通市蔷園路９号

国籍・地域　中華人民共和国

**南通大学**

発明者
(INVENTOR)
**顧　暁松**

**毛　蘇蘇**

（その他別紙記載）

出願番号
(APPLICATION NUMBER)
特願２０２３－５５５３５９

出願日
(FILING DATE)
令和　４年　５月２５日 (May 25,2022)

登録日
(REGISTRATION DATE)
令和　７年　６月　３日 (June 3,2025)

この発明は、特許するものと確定し、特許原簿に登録されたことを証する。

(THIS IS TO CERTIFY THAT THE PATENT IS REGISTERED ON THE REGISTER OF THE JAPAN PATENT OFFICE.)

令和　７年　６月　３日 (June 3,2025)

特許庁長官
(COMMISSIONER, JAPAN PATENT OFFICE)

小野洋太

证书号第8510827号

专利公告信息

# 发明专利证书

发明名称：鸟嘌呤和异鸟嘌呤在制备治疗或修复周围神经损伤药物中的应用

专利权人：南通大学

地　　址：226000 江苏省南通市崇川区永福路79号1幢南通大学技术转移研究院

发 明 人：毛苏苏;于彬;李慧;张竣杰

专　利　号：ZL 2024 1 1570883.X　　授权公告号：CN 119424441 B

专利申请日：2024年11月06日　　授权公告日：2025年11月25日

申请日时申请人：南通大学

申请日时发明人：毛苏苏;于彬;李慧;张竣杰

国家知识产权局依照中华人民共和国专利法进行审查，决定授予专利权，并予以公告。

专利权自授权公告之日起生效。专利权有效性及专利权人变更等法律信息以专利登记簿记载为准。

局长
申长雨

国家知识产权局
2025年11月25日

证书号第6605477号

# 发明专利证书

发 明 名 称：一种翘芪复方中药汤剂来源的微小核糖核酸及其制备方法和应用

发　明　人：顾晓松;毛苏苏;龚蕾蕾;杨晓明;王星辉;孙华林;徐来

专　利　号：ZL 2022 1 0424718.8

专利申请日：2022年04月21日

专 利 权 人：南通大学

地　　址：226019 江苏省南通市崇川区啬园路9号

授权公告日：2024年01月05日　　授权公告号：CN 114606236 B

国家知识产权局依照中华人民共和国专利法进行审查，决定授予专利权，颁发发明专利证书并在专利登记簿上予以登记。专利权自授权公告之日起生效。专利权期限为二十年，自申请日起算。

专利证书记载专利权登记时的法律状况。专利权的转移、质押、无效、终止、恢复和专利权人的姓名或名称、国籍、地址变更等事项记载在专利登记簿上。

局长
申长雨

国家知识产权局
2024年01月05日



其他事项参见续页

证书号第6605477号

专利权人应当依照专利法及其实施细则规定缴纳年费。本专利的年费应当在每年04月21日前缴纳。未按照规定缴纳年费的，专利权自应当缴纳年费期满之日起终止。

申请日时本专利记载的申请人、发明人信息如下：

申请人：

南通大学

发明人：

顾晓松;毛苏苏;龚蕾蕾;杨晓明;王星辉;孙华林;徐来

证书号第5228465号

# 发明专利证书

发明名称：环状RNA circ-Ankib1在制备促进神经再生和修复神经损伤药物中的应用

发明人：于彬;姚淳;毛苏苏;张珊珊;顾晓松

专利号：ZL 2019 1 0496040.2

专利申请日：2019年06月10日

专利权人：南通大学

地址：226019 江苏省南通市崇川区啬园路9号

授权公告日：2022年06月14日　　授权公告号：CN 110141578 B

国家知识产权局依照中华人民共和国专利法进行审查，决定授予专利权，颁发发明专利证书并在专利登记簿上予以登记。专利权自授权公告之日起生效。专利权期限为二十年，自申请日起算。

专利证书记载专利权登记时的法律状况。专利权的转移、质押、无效、终止、恢复和专利权人的姓名或名称、国籍、地址变更等事项记载在专利登记簿上。

局长
申长雨

2022年06月14日



其他事项参见续页

证 书 号 第 5228465 号

专利权人应当依照专利法及其实施细则规定缴纳年费。本专利的年费应当在每年 06 月 10 日前缴纳。未按照规定缴纳年费的，专利权自应当缴纳年费期满之日起终止。

申请日时本专利记载的申请人、发明人信息如下：

申请人：

南通大学

发明人：

于彬；姚淳；毛苏苏；张珊珊；顾晓松

# 江 苏 省 教 育 厅

苏教师函〔2025〕16号

# 省教育厅关于公布2025年江苏高校“青蓝工程”培养对象的通知

各有关高校:

根据《省教育厅关于做好2025年高校“青蓝工程”培养对象选拔工作的通知》(苏教师函〔2025〕4号),经学校推荐、专家评审和公示等环节,遴选产生了2025年江苏高校“青蓝工程”优秀青年骨干教师培养对象500人、中青年学术带头人培养对象200人、优秀教学团队98个,现将名单予以公布(详见附件1、2、3),并就有关事项通知如下。

## 一、关于培养要求

培养对象的培养期为2025年7月至2028年7月。有关高校要按照《江苏高校“青蓝工程”管理办法》(苏教规〔2017〕2号)的规定,认真做好培养对象的培养工作,高标准明确学校、院系和培养对象各自的职责,抓好培养、管理和考核等环节。

## 二、关于资助经费

每位优秀青年骨干教师资助科研经费4万元,自然科学类、人文社会科学类中青年学术带头人分别资助科研经费10万元、8万元,每个优秀教学团队资助科研经费30万元,以上经费由省教

育厅和所在学校各承担50%。

资助经费主要用于开展科研和教学项目研究、参加学术会议和培训进修、出版学术专著等，不得用于发放生活津贴。所在学校应严格执行有关财务管理规定，对资助经费单独建账，专款专用。培养对象在“青蓝工程”资助下取得的成果，包括发表论著和成果鉴定等，须标注“江苏高校‘青蓝工程’资助”字样。

**三、关于培养计划**

各有关高校要组织培养对象研究制订3年培养计划，认真填写《2025年江苏高校“青蓝工程”培养对象目标责任书》（见附件4）。请各有关高校于2025年8月29日前将培养对象的目标责任书电子版报省教育厅教师工作处备案。联系人：杨菲菲，联系电话：025-83335619，邮箱：jsc2109@163.com。

附件：1. 2025年江苏高校“青蓝工程”优秀青年骨干教师培养对象名单

2. 2025年江苏高校“青蓝工程”中青年学术带头人培养对象名单

3. 2025年江苏高校“青蓝工程”优秀教学团队名单

4. 2025年江苏高校“青蓝工程”培养对象目标责任书

省教育厅

2025年7月17日

（此件不公开）

卢梦梦、袁颖、滕芸

**苏州托普信息职业技术学院（1 人）**

张天霞

**硅湖职业技术学院（1 人）**

闫琴

**昆山登云科技职业学院（1 人）**

陈施宇

**苏州高博职业学院（1 人）**

盛松梅

**南通大学（5 人）**

毛苏苏、卢焕俊、汤天培、张傲、张雷

**南通理工学院（2 人）**

冒兴峰、罗清

**南通大学杏林学院（1 人）**

顾锡娟

**江苏航运职业技术学院（4 人）**

黄志龙、陆嘉慧、吕文超、杨锡平

**江苏工程职业技术学院（4 人）**

崔灿、李军、卢欣欣、朱志杰

**江苏商贸职业学院（3 人）**

刘燕、朱丹璇、朱亚丽

**南通职业大学（3 人）**

李守振、李忠艳、邢雅

**南通科技职业学院（3 人）**