同行专家业内评价意见书编号：20240860015

附件1

浙江工程师学院（浙江大学工程师学院）
同行专家业内评价意见书

姓名： 邓敏捷

学号： 22160373

申报工程师职称专业类别（领域）： 生物与医药

浙江工程师学院（浙江大学工程师学院）制

2024年03月21日

一、个人申报

（一）基本情况【围绕《浙江工程师学院（浙江大学工程师学院）工程类专业学位研究生工程师职称评审参考指标》，结合该专业类别(领域)工程师职称评审相关标准，举例说明】

1.对本专业基础理论知识和专业技术知识掌握情况

在奥翔药业实习实践期间，我充分学习了药物设计及开发方面的基础理论知识和专业技术知识。学会了CADD技术、药物分子改造策略、药物筛选评价策略与技术、原料药工艺开发与工艺优化、原料药盐型与晶型筛选、稳定性评价、原料药CMC阶段基础知识、专利及文章撰写等内容。

2.工程实践的经历

在奥翔药业实习实践期间，针对前期发现的一款CHK1小分子抑制剂存在hERG离子通道抑制率较高，容易诱发心脏毒性的风险。首先对该分子展开了系统的结构改造，主要从降低分子脂溶性、增加亲水性、减少碱性中心的三个策略着手结构优化，并结合分子-细胞-动物层面的药物评价手段，在企业原料药研发部门老师帮助与指导下，获得了一款具备更优成药性的候选分子。并对其合成工艺及原料药稳定性进行初步考察。合成工艺优化，采用逆合成分析，对注册工艺路线进行优化，成功打通关键中间体合成路线，并对纯化工艺进行优化，整体产率提升约30%。对原料药进行了初步的稳定性考察，考察了高温、高湿及强光照下，原料药稳定性。此外，由于化合物存在伯胺类结构，也对其考察了强酸、强碱、强氧化剂条件下的破坏性试验。

3.在实际工作中综合运用所学知识解决复杂工程问题的案例

由于部分化疗药物在临床阶段或上市后存在心脏毒性（抑制hERG离子通道，导致QT间期延长，心动过速），不得不遗憾退市。目前，化药开发领域着重关注分子毒性问题，hERG离子通道抑制已经成为IND申请前的关键一环，时刻影响药物研发与工艺开发进程。项目组前期发现具备2-氨基嘧啶类骨架的优选分子具备良好的CHK1抑制活性、成药性及抑瘤活性。在药学研究阶段发现，优选分子hERG离子通道抑制率较高，可能会诱发心脏毒性。因此，我们针对上述问题，采用CADD技术，分析分子与蛋白结合规律，采用减低亲脂性、增加亲水性、减少碱性中心，针对其嘧啶4-位及吡啶2-位进行结构优化，共设计60个目标分子。其中优选分子II-12具备良好的激酶、细胞活性及成药性，相较于先导分子hERG抑制率从41%降至9%，明显改善其潜在的心脏毒性。考虑到其结构中存在伯胺，为评估合适的存放条件，结合药典要求，进一步考察II-12在强酸、强碱、强氧化剂（室温或高温80℃）条件下的稳定性。发现其在酸性条件下，稳定成盐，且与温度无关。而在强碱、强氧化剂条件下不够稳定。因此，判断考虑进一步考察II-12的盐型与晶型，以判断其后续制剂所采取的原料药形态。

由于II-12初步合成工艺存在产率较低、纯化工艺冗杂、关键中间体价格昂贵且缺乏市售公斤级供应商，因此结合有机化学与工艺化学知识，进一步对关键中间体及原料药合成工艺进行优化。基于文献及专利调研结果，成功打通关键中间体合成路线，产率达44%。采用逆合成分析手段，分析发现原料药合成总共四步：氨化、亲核取代、偶联、酸性脱保护与中和。氨化反应存在同分异构，原路线产率仅40%，产率较低，产物溶解度较差不易纯化，原路线采用柱层析纯化，不符合生产工艺要求，成本较高。因此考察了不同碱、溶剂及温度对产率影响，同步筛选了不同纯化工艺，最终确定氨水、乙腈、冰浴为反应条件，重结晶为纯化工艺，相较于原路线产率不变，但纯化时间与成本明显改进。偶联反应原路线采用金属钯配体作为反应催化剂，钯属于管制杂质，对于开发为口服制剂的原料药而言，药典与ICH中有明确规定其杂质限量。因为，我们考察了不同碱、溶剂、配体比例以及Lewis酸催化的取代反应对产率及钯残留量的影响。最终确定，ZnCl2作为Lewis酸催化可以有效改善上述问题。

脱保护反应中，原路线采用三氟乙酸，成本较高。优化后选择甲醇为溶剂，盐酸为酸化试剂成本明显降低。合成工艺总计提升约30%，已初步完成合成及分析部分的注册文件。结合药典与ICH-CTD要求，需要对原料药开展稳定性实验，我们初步考察了30天内，原料药在高温、高湿、强光照条件下存放的稳定性。II-12具备良好的稳定性，短期内可以室温储存，长期建议在低温条件下保存。目前，II-12在进行正式毒理学研究，预计24-25年申请pre-IND。

该项目也取得了两篇SCI文章成果。

（二）取得的业绩（代表作）【限填3项，须提交证明原件（包括发表的论文、出版的著作、专利证书、获奖证书、科技项目立项文件或合同、企业证明等）供核实，并提供复印件一份】					
1. 公开成果代表作【论文发表、专利成果、软件著作权、标准规范与行业工法制定、著作编写、科技成果获奖、学位论文等】					
成果名称	成果类别 [含论文、授权专利（含发明专利申请）、软件著作权、标准、工法、著作、获奖、学位论文等]	发表时间/授权或申请时间等	刊物名称/专利授权或申请号等	本人排名/总人数	备注
Design, Synthesis, and Biological Evaluation of 2-Aminothiazole Derivatives as Novel Checkpoint Kinase 1 (CHK1) Inhibitors	权威期刊	2023年02月02日	ChemMedChem	1/8	SCI期刊收录
Discovery of 5-trifluoromethyl-2-aminopyrimidine derivatives as potent dual inhibitors of FLT3 and CHK1	权威期刊	2023年12月04日	RSC Medicinal Chemistry	1/8	SCI期刊收录

2.其他代表作【主持或参与的课题研究项目、科技成果应用转化推广、企业技术难题解决方案、自主研发设计的产品或样机、技术报告、设计图纸、软课题研究报告、可行性研究报告、规划设计方案、施工或调试报告、工程实验、技术培训教材、推动行业发展中发挥的作用及取得的经济社会效益等】

（三）在校期间课程、专业实践训练及学位论文相关情况	
课程成绩情况	按课程学分核算的平均成绩： 83 分
专业实践训练时间及考核情况（具有三年及以上工作经历的不作要求）	累计时间： 1 年（要求1年及以上） 考核成绩： 80 分（要求80分及以上）

本人承诺

个人声明：本人上述所填资料均为真实有效，如有虚假，愿承担一切责任，特此声明！

申报人签名：邓敏瑾

2216037

二、日常表现考核评价及申报材料审核公示结果

日常表现考核评价	非定向生由德育导师考核评价、定向生由所在工作单位考核评价： ☑优秀 ☐良好 ☐合格 ☐不合格 德育导师/定向生所在工作单位分管领导签字（公章）：2022年3月26日
申报材料审核公示	根据评审条件，工程师学院已对申报人员进行材料审核（学位课程成绩、专业实践训练时间及考核、学位论文、代表作等情况），并将符合要求的申报材料在学院网站公示不少于5个工作日，具体公示结果如下： ☐通过 ☐不通过（具体原因： ） 工程师学院教学管理办公室审核签字（公章）： 年 月 日

浙 江 大 学 研 究 生 院

攻读硕士学位研究生成绩表

学号：22160373	姓名：邓敏捷	性别：女	学院：工程师学院	专业：生物与医药	学制：2.5年
毕业时最低应获：24.0学分	已获得：25.0学分			入学年月：2021-09	毕业年月：2024-03
学位证书号：1033532024602245			毕业证书号：103351202402600471		授予学位：生物与医药硕士

学习时间	课程名称	备注	学分	成绩	课程性质	学习时间	课程名称	备注	学分	成绩	课程性质
2021-2022学年秋季学期	高等药物化学		2.0	93	专业选修课	2021-2022学年冬季学期	工程伦理		2.0	91	公共学位课
2021-2022学年秋季学期	数值计算方法		2.0	82	专业选修课	2021-2022学年秋冬季学期	研究生论文写作指导		1.0	88	专业学位课
2021-2022学年秋季学期	微生物药物合成生物学		2.0	86	专业选修课	2021-2022学年春季学期	科技创新案例探讨与实战		2.0	89	专业选修课
2021-2022学年秋季学期	药物波谱解析		2.0	71	专业选修课	2021-2022学年春季学期	自然辩证法概论		1.0	75	公共学位课
2021-2022学年冬季学期	新药发现理论与实践		2.0	90	专业学位课	2021-2022学年夏季学期	研究生英语基础技能		1.0	免修	公共学位课
2021-2022学年冬季学期	现代药剂学研究方法		2.0	85	专业学位课	2021-2022学年夏季学期	研究生英语		2.0	免修	公共学位课
2021-2022学年秋冬季学期	中国特色社会主义理论与实践研究		2.0	87	公共学位课	2021-2022学年夏季学期	药品创制工程实例		2.0	86	专业学位课

说明：1.研究生课程按三种方法计分：百分制，两级制（通过、不通过），五级制（优、良、中、及格、不及格）。

2.备注中“*”表示重修课程。

学院成绩校核章：

成绩校核人：张梦依

打印日期：2024-04-02

ChemMedChem

Research Article
doi.org/10.1002/cmdc.202200664

Chemistry Europe
European Chemical Societies Publishing

www.chemmedchem.org

Design, Synthesis, and Biological Evaluation of 2-Aminothiazole Derivatives as Novel Checkpoint Kinase 1 (CHK1) Inhibitors

Minjie Deng[+],[a] Peipei Wang[+],[b, e] Xiubing Long[+],[c] Gaoya Xu,[b, e] Chang Wang,[e] Jia Li,*[b, d, e, f] Yubo Zhou,*[b, d, e] and Tao Liu*[a, g]

A series of 2-aminothiazole derivatives were designed, synthesized on the basis of bioisosterism strategy and evaluated for their CHK1 inhibitory activity. Most of them exhibited potent CHK1 inhibition, and excellent antiproliferative activity against MV-4-11 and Z-138 cell lines. Systematic structure-activity relationship (SAR) efforts led to the discovery of a promising compound **8n**, which showed potent CHK1 inhibitory activity with IC_{50} value of 4.25 ± 0.10 nM, excellent antiproliferative activity against MV-4-11 and Z-138 cells with IC_{50} value of 42.10 ± 5.77 nM and 24.16 ± 6.67 nM, respectively, as well as moderate oral exposure ($AUC_{(0-t)} = 1076.25$ h·ng/mL) in mice. Additionally, treatment of MV-4-11 cells with compound **8n** for 2 h led to robust inhibition of CHK1 autophosphorylation on serine 296. Furthermore, kinase selectivity assay revealed that **8n** displayed acceptable selectivity toward 15 kinases. These results demonstrated that compound **8n** may be a promising potential anticancer agent for further development.

Introduction

The serine/threonine kinase Checkpoint kinase 1 (CHK1) plays an important role in the process of DNA damage response. When DNA damage occurs, especially single-stranded DNA breaks arising during replication or caused by genotoxic agents and ionizing radiation, CHK1 is mainly activated by phosphorylation of Ser317 and Ser345 by upstream kinase ataxia telangiectasia and Rad3-related (ATR), followed by autophosphorylation of Ser296.[1–3] A number of downstream proteins including cell division cycle 25 (Cdc25) family are phosphorylated by the activated CHK1 and finally lead to cell cycle arrest in S- or G2/M phase.[4] The cell cycle checkpoints controlled by CHK1 therefore provide an opportunity for the repair of the damaged DNA before the replicating cell enters mitosis. Therefore, CHK1 has become an important target for tumor treatment.

CHK1 inhibitors have been widely studied and a number of CHK1 inhibitors have been reported and evaluated in clinical trials,[5] but most of them were terminated for unfavorable toxicity profile or side effects,[6] only LY2606368, CCT245737, LY2880070, and GDC-0575 are still active in the clinical (Figure 1).[7–10] Most CHK1 inhibitors in clinical trails could only be administered intravenously, only three CHK1 inhibitors CCT245737, LY2880070, and GDC-0575 were oral administration. It was universally known that orally bioavailable compounds would offer advantages of ease of administration and flexibility of scheduling.[8] Thus, there still remains a strong need for novel CHK1 inhibitors with oral bioavailability, potent inhibitory activity as well as low toxicity.

In our previous study, compound MCL1020 was obtained as a CHK1 inhibitor with an IC_{50} value of 1.61 μM through virtual screening in-house compound library.[11] With the aim of discovering novel CHK1 inhibitors with suitable pharmacological profiles, compound **A** was synthesized through a bioisosterism strategy on MCL1020. Whereas, the CHK1 inhibitory activity was loss ($IC_{50} > 10$ μM). To achieve further insights, the molecular docking analysis of compound **A** was conducted to elucidate its possible binding mode with the CHK1. (PDB:2YM8).

[a] *M. Deng,[+] Prof. T. Liu*
College of Pharmaceutical Sciences
ZJU-ENS Joint Laboratory of Medicinal Chemistry
Zhejiang University, 310058 Hangzhou (P. R. China)
E-mail: lt601@zju.edu.cn
[b] *P. Wang,[+] G. Xu, Dr. J. Li, Dr. Y. Zhou*
School of Chinese Materia Medica
Nanjing University of Chinese Medicine
210023 Nanjing (P. R. China)
E-mail: jli@simm.ac.cn
ybzhou@simm.ac.cn
[c] *X. Long[+]*
Wuxi Apptec Co., Ltd.
288 Fute Zhong Road, 200131 Shanghai (P. R. China)
[d] *Dr. J. Li, Dr. Y. Zhou*
Zhongshan Institute for Drug Discovery
Shanghai Institute of Materia Medica
Chinese Academy of Sciences
528400 Zhongshan (P. R. China)
[e] *P. Wang,[+] G. Xu, C. Wang, Dr. J. Li, Dr. Y. Zhou*
State Key Laboratory of Drug Research
Shanghai Institute of Materia Medica
Chinese Academy of Sciences
201203 Shanghai (P. R. China)
[f] *Dr. J. Li*
Shandong Laboratory of Yantai Drug Discovery
Bohai Rim Advanced Research Institute for Drug Discovery
264117 Yantai (P. R. China)
[g] *Prof. T. Liu*
Hangzhou Institute of Innovative Medicine
Institute of Drug Discovery and Design, Zhejiang University
310058 Hangzhou (P. R. China)
[+] *These authors contributed equally to this work.*

Supporting information for this article is available on the WWW under https://doi.org/10.1002/cmdc.202200664

《SCI-EXPANDED》收录、《JCR》期刊影响因子、分区及中科院期刊分区证明

经检索《Web of Science》、《Journal Citation Reports（JCR）》及《中国科学院文献情报中心期刊分区表》数据库，《Science Citation Index Expanded (SCI-EXPANDED)》收录论文及其期刊影响因子、分区情况如下。（检索时间：2024 年 3 月 19 日）

第 1 条，共 2 条

标题:Design, Synthesis, and Biological Evaluation of 2-Aminothiazole Derivatives as Novel Checkpoint Kinase 1 (CHK1) Inhibitors

作者:Deng, MJ(Deng, Minjie);Wang, PP(Wang, Peipei);Long, XB(Long, Xiubing);Xu, GY(Xu, Gaoya);Wang, C(Wang, Chang);Li, J(Li, Jia);Zhou, YB(Zhou, Yubo);Liu, T(Liu, Tao);

来源出版物:CHEMMEDCHEM 卷:18 期:7 文献号:e202200664 提前访问日期:FEB 2023 DOI:10.1002/cmdc.202200664 出版年:APR 3 2023

入藏号:WOS:000922553100001

文献类型:Article

地址:

[Deng, Minjie; Liu, Tao] Zhejiang Univ, Coll Pharmaceut Sci, ENS Joint Lab Med Chem, ZJU, Hangzhou 310058, Peoples R China.

[Wang, Peipei; Xu, Gaoya; Li, Jia; Zhou, Yubo] Nanjing Univ Chinese Med, Sch Chinese Mat Med, Nanjing 210023, Peoples R China.

[Long, Xiubing] Wuxi Apptec Co Ltd, 288 Fute Zhong Rd, Shanghai 200131, Peoples R China.

[Zhou, Yubo] Chinese Acad Sci, Zhongshan Inst Drug Discovery, Shanghai Inst Mat Med, Zhongshan 528400, Peoples R China.

[Wang, Chang; Li, Jia; Zhou, Yubo] Chinese Acad Sci, Shanghai Inst Mat Med, State Key Lab Drug Res, Shanghai 201203, Peoples R China.

[Li, Jia] Bohai Rim Adv Res Inst Drug Discovery, Shandong Lab Yantai Drug Discovery, Yantai 264117, Peoples R China.

[Liu, Tao] Zhejiang Univ, Hangzhou Inst Innovat Med, Inst Drug Discovery & Design, Hangzhou 310058, Peoples R China.

通讯作者地址:

Liu, T (corresponding author), Zhejiang Univ, Coll Pharmaceut Sci, ENS Joint Lab Med Chem, ZJU, Hangzhou 310058, Peoples R China.; Li, J; Zhou, YB (corresponding author), Nanjing Univ Chinese Med, Sch Chinese Mat Med, Nanjing 210023, Peoples R China.; Zhou, YB (corresponding author), Chinese Acad Sci, Zhongshan Inst Drug Discovery, Shanghai Inst Mat Med, Zhongshan 528400, Peoples R China.; Li, J; Zhou, YB (corresponding author), Chinese Acad Sci, Shanghai Inst Mat Med, State Key Lab Drug Res, Shanghai 201203, Peoples R China.

电子邮件地址:jli@simm.ac.cn; ybzhou@simm.ac.cn; lt601@zju.edu.cn

IDS 号:C9BE3

ISSN:1860-7179

eISSN:1860-7187

期刊《ChemMedChem》2022 年的影响因子为 3.4，五年影响因子为 3.3。期刊《ChemMedChem》2022 年的 JCR 分区情况为：

Edition	JCR® 类别	类别中的排序	JCR 分区
SCIE	CHEMISTRY, MEDICINAL	33/60	Q3
SCIE	PHARMACOLOGY & PHARMACY	125/278	Q2

期刊《ChemMedChem》2023 年升级版的中科院期刊分区情况为：

刊名	ChemMedChem		
年份	2023		
ISSN	1860-7179		
	学科	分区	Top 期刊
大类	医学	4	否
小类	CHEMISTRY, MEDICINAL 药物化学	4	-
小类	PHARMACOLOGY & PHARMACY 药学	4	-

第 2 条，共 2 条

标题:Discovery of 5-trifluoromethyl-2-aminopyrimidine derivatives as potent dual inhibitors of FLT3 and CHK1

作者:Deng, MJ(Deng, Minjie);Gao, Y(Gao, Yue);Wang, PP(Wang, Peipei);Du, WJ(Du, Wenjing);Xu, GY(Xu, Gaoya);Li, J(Li, Jia);Zhou, YB(Zhou, Yubo);Liu, T(Liu, Tao);

RSC Medicinal Chemistry

ROYAL SOCIETY OF CHEMISTRY

RESEARCH ARTICLE

View Article Online
View Journal | View Issue

Cite this: *RSC Med. Chem.*, 2024, **15**, 539

Discovery of 5-trifluoromethyl-2-aminopyrimidine derivatives as potent dual inhibitors of FLT3 and CHK1†

Minjie Deng,‡[a] Yue Gao,‡[bc] Peipei Wang,[bc] Wenjing Du,[a] Gaoya Xu,[bc] Jia Li,[bcde] Yubo Zhou*[ce] and Tao Liu *[afg]

Received 25th October 2023,
Accepted 4th December 2023

DOI: 10.1039/d3md00597f

rsc.li/medchem

Here, we discover an FLT3/CHK1 dual inhibitor (**30**) that exhibits excellent kinase potency and antiproliferative activity against MV4-11 cells. Simultaneously, **30** possesses high selectivity over c-Kit enzyme and low hERG inhibitory ability. Compound **30**, meanwhile, overcomes varied resistance in BaF3 cell lines carrying FLT3-TKD and FLT3-ITD mutations. Moreover, **30** demonstrates favorable oral PK properties and kinase selectivity. These conclusions support that compound **30** may be a promising potential FLT3/CHK1 dual agent for further development.

Introduction

Acute myeloid leukemia (AML) is a malignant cancer with an abnormal hematopoietic system, characterized by impaired differentiation and indefinite proliferation of immature cells.[1,2] At present, the "7 + 3" regimen is mainly recognized as the standard treatment method for AML patients, which contains seven continuous days of cytarabine infusion and daily boluses of anthracycline on the first three days. Unfortunately, although this treatment regimen has significant early efficacy, the prognosis of AML patients in the later stage is not satisfactory, with primary refractory diseases and recurrence.[3,4] The five-year survival period for AML patients receiving treatment is roughly 35% in middle aged and young people (age < 60) and less than 15% in elderly people (age > 60).[4–6] Hence, it is still necessary to develop AML treatment strategies and drugs with higher efficacy through molecular and genetic research.

Fms-like tyrosine kinase 3 (FLT3) belongs to the type III receptor tyrosine kinase family (RTK), which plays a notable role in regulating the propagation and hemopoiesis of progenitor cells in medulla ossium.[7,8] FLT3 gene mutation is one of the most common genetic abnormalities in AML, including internal tandem duplication (ITD) as well as missense mutations of the tyrosine kinase domain (TKD), which is closely related to poor prognosis and high relapse risk in patients.[9–11] Subsequently, a large number of FLT3 inhibitors (FLT3i) have been exploited for the treatment of AML, in response to the high correlation between FLT3 and AML.[12] The first-generation FLT3i were multitarget and nonselective inhibitors, including midostaurin,[9] which possibly caused off-target side effects. The second-generation FLT3i had better specificity and less toxicity, such as gilteritinib[13] and quizartinib[14] (Fig. 1). Regretfully, acquired target mutations (acquired resistance) and abnormal activation of off-target pathways (adaptive resistance) pose substantial limitations to the use of FLT3 inhibitors in AML control.[15] As a result, targeting FLT3 and related drug resistance mechanisms simultaneously may be a more effective treatment measure for AML.

Checkpoint kinase 1 (CHK1) is a principal regulator of the DNA damage response pathway (DDR).[16] The inhibition of CHK1 is going to force tumor cells into mitosis, carrying the wrong genetic material, and thereby induce cell apoptosis.[17] Our earlier discovery identified that inhibiting FLT3 and CHK1 together is able to restore the p53 pathway activity, which is inactivated by FLT3i, and then overcome FLT3i resistance.[18] Therefore, the concomitant targeting of FLT3

[a] *ZJU-ENS Joint Laboratory of Medicinal Chemistry, College of Pharmaceutical Sciences, Zhejiang University, Zijingang Campus, Hangzhou 310058, P.R. China. E-mail: lt601@zju.edu.cn*
[b] *School of Chinese Materia Medica, Nanjing University of Chinese Medicine, Nanjing, 210023, P.R. China*
[c] *State Key Laboratory of Chemical Biology, Shanghai Institute of Materia Medica, Chinese Academy of Sciences, Shanghai 201203, P.R. China. E-mail: ybzhou@simm.ac.cn*
[d] *Shandong Laboratory of Yantai Drug Discovery, Bohai Rim Advanced Research Institute for Drug Discovery, Yantai 264117, P.R. China*
[e] *Zhongshan Institute for Drug Discovery, Shanghai Institute of Materia Medica, Chinese Academy of Sciences, Zhongshan 528400, P.R. China*
[f] *Hangzhou Institute of Innovative Medicine, Institute of Drug Discovery and Design, Zhejiang University, Hangzhou 310058, P.R. China*
[g] *National Key Laboratory of Advanced Drug Delivery and Release Systems, Zhejiang University, Hangzhou 310058, P.R. China*

† Electronic supplementary information (ESI) available. See DOI: https://doi.org/10.1039/d3md00597f

‡ These authors contributed equally to this work.

《SCI-EXPANDED》收录、《JCR》期刊影响因子、分区及中科院期刊分区证明

来源出版物:RSC MEDICINAL CHEMISTRY 卷:15 期:2 页:539-552 提前访问日期:DEC 2023 DOI:10.1039/d3md00597f 出版年:FEB 21 2024

入藏号:WOS:001135690400001

文献类型:Article

地址:

[Deng, Minjie; Du, Wenjing; Liu, Tao] Zhejiang Univ, Coll Pharmaceut Sci, ZJU ENS Joint Lab Med Chem, Zijingang Campus, Hangzhou 310058, Peoples R China.

[Gao, Yue; Wang, Peipei; Xu, Gaoya; Li, Jia] Nanjing Univ Chinese Med, Sch Chinese Mat Med, Nanjing 210023, Peoples R China.

[Gao, Yue; Wang, Peipei; Xu, Gaoya; Li, Jia; Zhou, Yubo] Chinese Acad Sci, Shanghai Inst Mat Med, State Key Lab Chem Biol, Shanghai 201203, Peoples R China.

[Li, Jia] Bohai Rim Adv Res Inst Drug Discovery, Shandong Lab Yantai Drug Discovery, Yantai 264117, Peoples R China.

[Li, Jia; Zhou, Yubo] Chinese Acad Sci, Zhongshan Inst Drug Discovery, Shanghai Inst Mat Med, Zhongshan 528400, Peoples R China.

[Liu, Tao] Zhejiang Univ, Hangzhou Inst Innovat Med, Inst Drug Discovery & Design, Hangzhou 310058, Peoples R China.

[Liu, Tao] Zhejiang Univ, Natl Key Lab Adv Drug Delivery & Release Syst, Hangzhou 310058, Peoples R China.

通讯作者地址:

Liu, T (corresponding author), Zhejiang Univ, Coll Pharmaceut Sci, ZJU ENS Joint Lab Med Chem, Zijingang Campus, Hangzhou 310058, Peoples R China.; Zhou, YB (corresponding author), Chinese Acad Sci, Shanghai Inst Mat Med, State Key Lab Chem Biol, Shanghai 201203, Peoples R China.; Zhou, YB (corresponding author), Chinese Acad Sci, Zhongshan Inst Drug Discovery, Shanghai Inst Mat Med, Zhongshan 528400, Peoples R China.; Liu, T (corresponding author), Zhejiang Univ, Hangzhou Inst Innovat Med, Inst Drug Discovery & Design, Hangzhou 310058, Peoples R China.; Liu, T (corresponding author), Zhejiang Univ, Natl Key Lab Adv Drug Delivery & Release Syst, Hangzhou 310058, Peoples R China.

电子邮件地址:ybzhou@simm.ac.cn; lt601@zju.edu.cn

IDS 号:IQ7C2

eISSN:2632-8682

期刊《RSC Medicinal Chemistry》2022 年的影响因子为 4.1，五年影响因子为 4.1。期刊《RSC Medicinal Chemistry》2022 年的 JCR 分区情况为：

Edition	*JCR® 类别*	*类别中的排序*	*JCR 分区*
SCIE	BIOCHEMISTRY & MOLECULAR BIOLOGY	**112/285**	**Q2**
SCIE	CHEMISTRY, MEDICINAL	**23/60**	**Q2**

期刊《RSC Medicinal Chemistry》2023 年升级版的中科院期刊分区情况为：

刊名	RSC Medicinal Chemistry
年份	2023
ISSN	2632-8682

	学科	分区	Top 期刊
大类	医学	4	否
小类	CHEMISTRY, MEDICINAL 药物化学	3	-
小类	BIOCHEMISTRY & MOLECULAR BIOLOGY 生化与分子生物学	4	-

注：

1. 期刊影响因子及分区情况最新数据以 JCR 数据库、《中国科学院文献情报中心期刊分区表》最新数据为准。
2. 以上检索结果来自 CALIS 查收查引系统。
3. 以上检索结果均得到委托人及被检索作者的确认。

教育部科技查新工作站（3209）

检索人（签章）：[illegible]

2024 年 3 月 19 日