编号：

湖北医药学院高层次人才聘期考核表

（聘期：2021 年 10 月 11 日－ 2024 年 10 月 11 日）

所在单位 第三临床学院

姓　　名 万国兴

专业技术职务 讲师

联系电话 13593761715

填表日期：2024 年 11 月 28 日

湖北医药学院人事处制

姓名	万国兴	性别	男	出生年月	1988.07
所在学院	第三临床学院			来校时间	2019.04
学历学位	博士研究生	毕业时间	2018.12	毕业院校	汕头大学
完成聘期目标任务概述	聘期目标任务： 1. 完成本科生、留学生、研究生教学任务，年均课时量大于 30 学时。 2. 积极申报国家及省部级各类项目，主持完成国家级项目 1 项或省部、厅局级、横向课题到账经费 80 万元以上 3. 发表中科院 2 区以上论文 2 篇，单篇影响因子>5 或者累积影响因子>10. 4. 履职尽责，完成岗位工作及第三临床学院及附属人民医院交办的其他工作。 5. 恪守教师行为准则及新时代教师行为规范。 实际完成情况： 聘期讲授留学生《诊断学》，本科生《内科学》、《缓和医疗与安宁疗护实践》、《成人健康护理学》、《护理科研》，研究生规培带教等课程，圆满完成教学任务，年均课时量 56.4。作为本科生导师带教本科生，两次获评优秀本科生导师，指导获批大学生创新创业训练项目国家级 2 项、校级 1 项，教学期间未发生任何教学事件及失德失范行为。积极按要求申报各类科研课题，获批主持国家自然科学基金 1 项、省自然科学基金 1 项、省教育厅科研项目 1 项、教育部重点实验室开放基金 1 项，作为主要参与者承担省自然基金联合基金 1 项（3/7）。发表 SCI 论文 10 篇，其中包括中科院一区 2 篇、二区 1 篇、三区 2 篇、四区 5 篇，累积影响因子。在附属人民医院肿瘤中心工作岗位上积极履职尽责，完满完成了医院及第三临床学院交办的各项工作。				

	年度	课程名称	授课对象	当量课时	承担角色	备注
教学情况	2024	《内科学》	本科生	8	教师	
	2024	《护理科研》	本科生	4	教师	
	2024	《缓和医疗与安宁疗护实践》	本科生	7	教师	
	2023	《缓和医疗与安宁疗护实践》	本科生	7	教师	
	2023	《成人健康护理学》	本科生	4	教师	
	2022	《成人健康护理学》	本科生	4	教师	
	2020	《诊断学》	留学生	96	教师	
	2022	规培带教	研究生	36	教师	
	2023	规培带教	研究生	36	教师	
	2024	规培带教	研究生	36	教师	
	2022	优秀本科生导师	本科生	30	指导教师	
	2023	优秀本科生导师	本科生	30	指导教师	

	2022	国家级大创项目	本科生		指导教师			
	2022	校级大创项目	本科生		指导教师			
	2024	国家级大创项目	本科生		指导教师			
论文论著专利	论文（论著）题目/专利名称		刊物（出版社）、卷（期）号/专利号	级别/专利类型	发表时间/授权公告时间	收录情况	本人排名	湖医药是否第一署名单位
	Valid Analysis of Brain-Specific Progression-Free Survival		JAMA Oncol,2024 Aug 1;10(8):1135	中科院 1 区	2024	SCI	通讯作者	是
	Prognostic value of the immune infiltration score in early breast cancer patients receiving dual HER2 blockade with trastuzumab and pertuzumab: An exploratory analysis of a randomized clinical trial		Ann Oncol, 2020, 31(S2): S18	中科院 1 区	2020	SCI	第一作者	是
	Network Pharmacology Along with Molecular Docking to Explore the Mechanism of Danshen Injection against Anthracycline-induced Cardiotoxicity and Transcriptome Validation		Curr Pharm Des. 2024;30:952-967	中科院 4 区	2024	SCI	通讯作者	是
	Mechanism Exploration of Astaxanthin in the Treatment of Adriamycin-induced Cardiotoxicity Based on Network Pharmacology and Experimental Validation		Curr Med Chem,2024 Oct 28. doi: 10.2174/0109298673329567241014071914	中科院 4 区	2024	SCI	通讯作者	是
	Exploring the molecular mechanism of ginseng against anthracycline-induced cardiotoxicity based on network pharmacology, molecular docking and molecular dynamics simulation		Hereditas,2024 Sep 6;161(1):31.	中科院 3 区	2024	SCI	通讯作者	是
	Elevated Preoperative NMPR Predicts an Unfavorable Chance of Survival in Resectable Esophageal Squamous Cell Carcinoma		Medicina (Kaunas),2022 Dec 8;58(12):1808	中科院 4 区	2024	SCI	通讯作者	是
	Weighted gene co-expression network-based approach to identify key genes associated with anthracycline-induced cardiotoxicity and construction of miRNA-transcription factor-gene		Mol Med,2021 Nov 3;27(1):142.	中科院 2 区	2021	SCI	第一作者	是

	regulatory network						
	Identification of shared mechanisms and targets between immune checkpoint inhibitor-associated myocarditis and autoimmune myocarditis.	European Journal of Inflammation. 2024;22. doi:10.1177/1721727X231223578	中科院 4 区	2024	SCI	通讯作者	是
	High C-Reactive Protein to Albumin Ratio Predicts Inferior Clinical Outcomes in Extranodal Natural Killer T-Cell Lymphoma	Dose Response. 2020;18(2):1559325820917824	中科院 4 区	2020	SCI	通讯作者	是
	Bevacizumab added to neoadjuvant chemotherapy in HER2-negative non-metastatic breast cancer	J. Cancer 2019; 10(2): 416-417	中科院 3 区	2019	SCI	第一作者	是

科研教研项目	项目名称	项目来源	立项时间	金额（万）	项目级别	本人排名	湖医药是否第一署名单位
	Asb2/Srf/Jph2 信号下调介导的细胞钙超载在阿霉素心脏毒性中的作用及机制研究	国家自然科学基金	2022	30	国家级	第一	是
	基于 PPAR α /miR-133b-5p/Lox-1 信号探讨紫檀芪对阿霉素心脏毒性的心肌保护作用及机制	湖北省自然科学基金	2020	5	省部级	第一	是
	阿霉素通过抑制Pirin基因诱导心肌细胞铁死亡的机制研究	湖北省教育厅	2022	1	厅局级	第一	是
	SIRT1单核苷酸多态性对Notch1酰基化修饰与新疆不同民族乳腺癌易感性和进展的研究	教育部重点实验室开放基金	2021	5	厅局级	第一	是
	SERPINB6 抑制自噬促进肝癌细胞生长增殖的机制研究	湖北省自然科学基金	2024	10	省部级	第三	是

其他	本人在此郑重声明：本表填写的信息材料真实可靠。若提供的材料弄虚作假，视作聘期考核不合格，并终止相应的聘用合同。 签　　名： 年　　月　　日
审核人意见	学院审核人签字：　　年　　月　　日

学院党政联席会议意见 ☐完成首聘期任务，考核为合格 ☐完成首聘期部分任务，考核为基本合格，但同意延期两年 ☐未完成首聘期任务，考核不合格，但同意延期两年 负责人签字:　　学院盖章:　　年　月　日
学校意见 ☐完成首聘期任务，考核为合格 ☐完成首聘期部分任务，考核为基本合格，但同意延期两年 ☐未完成首聘期任务，考核不合格，但同意延期两年 人事处盖章:　　年　月　日

国家自然科学基金委员会

项 目 批 准 通 知

国科金计项〔2022〕45号

关于批准资助2022年度国家自然科学基金第二批项目的通知

湖北医药学院（单号：2022-45-0425）：

根据《国家自然科学基金条例》有关规定和专家评审意见，国家自然科学基金委员会（以下简称自然科学基金委）决定批准资助你单位国家自然科学基金项目 18 项，直接费用 665 万元。上述资助项目清单详见附件。

依托单位和项目负责人须按要求完成电子及纸质《国家自然科学基金资助项目计划书》（以下简称《计划书》）填写、提交与报送工作。项目负责人登录科学基金网络信息系统（https://grants.nsfc.gov.cn）先行填报《计划书》电子版并提交至依托单位，由依托单位审核确认后提交至自然科学基金委。《计划书》电子版经自然科学基金委审核通过后，项目负责人再行打印《计划书》纸质版（一式两份，双面打印），依托单位审核并加盖单位公章，将申请书纸质签字盖章页订在其中一份《计划书》之后，一并报送至自然科学基金委项目材料

接收工作组。电子版和纸质版《计划书》内容应当保持一致。逾期不报《计划书》或申请书纸质签字盖章页且未说明理由，视为自动放弃接受资助；未按要求修改电子版《计划书》和申请书纸质签字盖章页，或逾期提交纸质版《计划书》和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。

邮寄地址：北京市海淀区双清路83号项目材料接收工作组

邮编：100085

联系电话：010-62328591

附件：2022年度国家自然科学基金资助项目清单

国家自然科学基金委员会

2022年10月20日

2022年度国家自然科学基金资助项目清单（湖北医药学院）

单号：2022-45-0425　　直接费用单位：万元

序号	项目批准号	负责人	申请代码	项目名称	直接费用	起止日期	资助类别/亚类说明/附注说明
12	82204540	万国兴	H3512	Asb2/Srf/Jph2信号下调介导的细胞钙超载在阿霉素心脏毒性中的作用及机制研究	30	2023.01.01-2025.12.31	青年科学基金项目
13	82204602	覃陈虎	H3203	基于中药材全蝎毒腺多肽组学的抗癫痫多肽发现及其功能机制研究	30	2023.01.01-2025.12.31	青年科学基金项目
14	82270299	唐俊明	H0202	RGC-32与NFATs交互反馈调节心肌重构影响缺血性心衰进程的作用及机制	52	2023.01.01-2026.12.31	面上项目
15	82270488	付坚	H0214	EGLN3通过调节NLRP3泛素化调控MerTK介导的胞葬作用及动脉粥样硬化斑块稳定性的研究	52	2023.01.01-2026.12.31	面上项目
16	82270530	丁妍	H0219	NSUN2通过m5C　RNA修饰调控Nrf2缓解阿霉素心肌毒性的作用机制研究	52	2023.01.01-2026.12.31	面上项目
17	82273451	钦闪闪	H1809	胃癌上皮间充质转化的异质性和可塑性及其精细调控分子机制	52	2023.01.01-2026.12.31	面上项目
18	82274155	汪选斌	H3210	预知子活性成分α-常春藤皂苷促进ACOX2介导的脂肪酸β-氧化抑制肝癌脂代谢重编程	52	2023.01.01-2026.12.31	面上项目

共18项，665.0000万元

湖北省科技计划项目

验 收 证 书

鄂科验〔2022〕BF686 号

湖北医药学院：

你单位承担的湖北省科技计划项目，已完成预期指标任务，通过结题，特此证明。

计划类别：自然科学基金面上类项目

项目编号：2020CFB158

项目名称：基于PPARα/miR-133b-5p/Lox-1信号探讨紫檀芪对阿霉素心脏毒性的心肌保护作用

项目负责人：万国兴

批准机关：湖北省科技厅

颁证日期：二〇二二年十月十七日

湖北省科学技术厅 科技计划验收专用章

湖北省教育厅科学研究计划项目

结项证书

项　目　名　称　：　阿霉素通过抑制Pirin基因诱导心肌细胞铁死亡的机制研究

立　项　年　度　：　2022

项　目　编　号　：　Q20222111

承　担　单　位　：　湖北医药学院

项 目 负 责 人　：　万国兴

项 目 参 加 者　：　曹风军,佐志刚,王一西,邓楚晴

该项目提交的研究资料完整，结项报告系统详实，经审查符合结项要求，准予结项。

湖北省教育厅

2024 年 08 月 15 日

石河子大学医学院文件

院发〔2020〕18号

关于公布新疆地方与民族高发病教育部重点实验室2020年开放课题立项项目的通知

各有关部门：

新疆地方与民族高发病教育部重点实验室2020年 开放课题立项工作已圆满结束，共有5项课题获得资助。现将获资助科研课题名称、主要完成单位及项目主持人予以公布（见附件）。

各获资助课题在项目主持人与实验室签定合同书后正式启动，各项目主持人要严格按照合同书的计划内容如期完成研究任务。项目实施过程中如有问题，请及时与新疆地方与民族高发病教育部重点实验室管理委员会联系，以保证立项课题的顺利实施。

附件：新疆地方与民族高发病教育部重点实验室2020年

开放课题立项清单

石河子大学医学院

2020 年 11 月 16 日

新疆地方与民族高发病教育部

重点实验室

2020 年 11 月 16 日

附件：

新疆地方与民族高发病教育部重点实验室2020年开放课题立项清单

项目编号	主持人	课 题 名 称	单 位	研究年限	经 费（万元）
KF2021-1	陈香梅	NFATc3通过转录激活III型干扰素发挥抗病毒和抗肿瘤的作用及机制研究	北京大学	2021.01-2022.12	5
KF2021-2	左后娟	花生四烯酸表氧化酶代谢产物活性小分子在新疆维吾尔族心力衰竭患者发病机制中的研究	华中科技大学	2021.01-2022.12	5
KF2021-3	姚 平	AS斑块铁沉积空间病理生理学机制初探	华中科技大学	2021.01-2022.12	5
KF2021-4	万国兴	SIRT1单核苷酸多态性对Notch1酰基化修饰与新疆不同民族乳腺癌易感性和进展的研究	湖北医药学院	2021.01-2022.12	5
KF2021-5	孙 湛	肾结核发病过程中巨噬细胞极化对肾小管上皮细胞损伤的机制研究	新疆医科大学	2021.01-2022.12	5

湖北省科学技术厅文件

鄂科技发资〔2024〕8号

省科技厅关于下达2024年省自然科学基金创新发展联合基金项目的通知

各有关单位:

根据年度工作安排，我厅组织完成了2024年度省自然科学基金创新发展联合基金项目的申报和立项工作，经省人民政府批准，本次省自然科学基金创新发展联合基金项目立项456项，其中：黄石创新发展联合基金项目30项；襄阳创新发展联合基金项目30项；恩施创新发展联合基金项目30项；十堰创新发展联

合基金项目 30 项；宜昌创新发展联合基金项目 83 项；气象创新发展联合基金项目 17 项；中医药创新发展联合基金项目 128 项；三峡创新发展联合基金项目 23 项；地质创新发展联合基金项目 31 项；智慧交通创新发展联合基金项目 16 项；恒瑞医药创新发展联合基金项目 38 项。现将立项项目下达你们，并就有关事项通知如下：

1. 各联合基金项目承担单位、推荐单位应于 2024 年 5 月 30 日前与省科技厅签订项目任务书，明确项目的研究内容、考核指标、绩效目标、经费安排、实施期限以及实施各方的权利和义务，研究内容和考核指标应尽可能细化、量化。

2. 各联合基金项目承担单位应在立项文件下达后 7 个工作日内提供相应额度的票据（行政事业单位提供行政事业单位资金往来票据、企业提供盖有财务专用章的收据），并注明“省创新发展联合基金项目经费”。省科技厅将在收到票据后拨付项目经费。

3. 各联合基金项目承担单位、推荐单位要按照《湖北省科技计划项目管理办法》（鄂科技规〔2021〕2 号）和省级财政科研项目经费管理等相关规定，加强科技计划项目和经费管理。要按任务书确定的研究内容推进项目实施，认真配合开展项目检查、评估、验收、绩效管理，做好项目档案管理。

附件：1.2024年黄石创新发展联合基金项目立项清单

2.2024年襄阳创新发展联合基金项目立项清单

3.2024年恩施创新发展联合基金项目立项清单

4.2024年十堰创新发展联合基金项目立项清单

5.2024年宜昌创新发展联合基金项目立项清单

6.2024年气象创新发展联合基金项目立项清单

7.2024年中医药创新发展联合基金项目立项清单

8.2024年三峡创新发展联合基金项目立项清单

9.2024年地质创新发展联合基金项目立项清单

10.2024年智慧交通创新发展联合基金项目立项清单

11.2024年恒瑞医药创新发展联合基金项目立项清单

湖北省科技厅

2024年5月14日

13	2024AFD431	肿瘤相关巨噬细胞 APOC1 通过 TGF-β/Smad 通路促进胶质母细胞瘤恶性进展和放射抵抗的机制研究	华中科技大学	20	前资助
14	2024AFD432	体细胞拷贝数变异和克隆演化在复发转移宫颈癌免疫逃逸中的作用及机制研究	襄阳市中心医院	20	前资助
15	2024AFD433	ER/PR 单阳性乳腺癌 ER 及相关信号通路异常的机制研究	华中科技大学	20	前资助
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28	2024AFD446	SERPINB6 抑制自噬促进肝细胞癌生长增殖的机制研究	十堰市人民医院	10	前资助
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30	2024AFD448	去泛素化酶 USP39 通过铁死亡关键蛋白 SLC7A11 介导喉癌顺铂耐药的机制研究	武汉大学	10	前资助

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Letter to the Editor

Bevacizumab Added to Neoadjuvant Chemotherapy in HER2-Negative Non-Metastatic Breast Cancer

Guoxing Wan✉, Fengjun Cao, Xuanbin Wang, Xue Sun

Department of Oncology, Renmin Hospital, Hubei University of Medicine, Shiyan 442000, China.

✉ Corresponding author: Guoxing Wan, MD, PhD, Department of Oncology, Renmin Hospital, Hubei University of Medicine, 39 Chaoyang Road, 442000 Shiyan, China. Phone: +86-719-8637385; Fax: +86-719-8637385; Email: 15gxwan@stu.edu.cn

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To the Editor,

Recombinant humanized monoclonal antibody bevacizumab binding and inactivating VEGF-A has recently been a particularly attractive focus of research in anti-angiogenic treatment strategy for breast cancer especially the HER-2 negative subtype [1]. Three recently published phase III randomized controlled trials-ARTemis [2], GBG 44- GeparQuinto [3] and SWOG S0800 trial [4]-have showed that adding bevacizumab to neoadjuvant chemotherapy (NAC) increased the pathological complete response (pCR) but did not favor the survival of HER-2 negative non-metastatic breast cancer (NMBC), while NSABP B-40 trial [5] contradicted the findings by demonstrating an improved survival particularly disease-free survival (DFS). To settle the disputes, we therefore performed a meta-analysis to evaluate the survival benefit and risk of this treatment strategy.

Involving 4122 participants with a median follow-up from 3 to 4.7 year, the pooled data suggested no significant effect of bevacizumab on either DFS or overall survival (OS). Similar results on DFS were also found according to the hormone receptor (HR) status. Unexpectedly, a significantly reduced DFS was indicated in patients achieving a pCR (hazard ratio, 2.36; 95%CI, 1.33-4.19) while not in patients without a pCR. The stratification analyses by HR status regarding OS showed no significant effect as well (Figure 1). Involving two trials with a total of 1897 participants, the result showed significantly increased risk of any surgical complications (risk ratio, 1.39; 95%CI, 1.20-1.62) in patients receiving NAC and neoadjuvant bevacizumab (Figure 1).

Although an individual patient-level meta-analysis would be ideal, our results highlighted that adding bevacizumab to NAC for HER-2 negative NMBC did not provide survival benefit but significantly increased the risk of surgical complications. Also, the results did not support the role of HR status in discriminating the effect of bevacizumab, which contradicted the finding from NSABP-B40 that addition of bevacizumab resulted in improved survival especially in HR-positive patients. In NSABP B40 but not in other trials, patients received bevacizumab not just preoperatively but also postoperatively, such substantial differences may have contributed to the discordant results because bevacizumab was able to affect not only the primary tumor but also dormant micrometastases. Although most previous trials incorporating the currently included trials showed consistently an increased pCR rate with neoadjuvant bevacizumab, reduced DFS was revealed in patients achieving pCR in the present study, suggesting that the pCR advantage seemed not always to be translated into a survival advantage. Our analysis failed to demonstrate this benefit while confirmed increased toxicity, supporting utmost cautions against the adoption of neoadjuvant bevacizumab in this setting.

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GXW contributed to the initial concept, protocol writing, data interpretation and manuscript writing; FJC contributed to data collection and interpretation, manuscript review; XBW and XS helped with protocol writing, data collection and interpretation.

Competing Interests

The authors have declared that no competing interest exists.

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Identification of shared mechanisms and targets between immune checkpoint inhibitor-associated myocarditis and autoimmune myocarditis

Kai Yang[1,*], Min Zhang[2,*], Dong Li[3,*], Yuandong Yu[2], Fengjun Cao[2] and Guoxing Wan[2,4]

Abstract

Objective: This study aimed to explore the shared mechanisms and targets between immune checkpoint inhibitor-associated myocarditis (ICIM) and autoimmune myocarditis.

Methods: Relevant data were retrieved from public datasets and Gene Expression Omnibus (GEO) database. Gene set enrichment analysis (GSEA) of differentially expressed genes (DEGs) was used to identify significant shared signaling pathways between ICIM and non-ICI associated autoimmune myocarditis (NICIAM) represented by ICIM model and experimental autoimmune myocarditis (EAM) model, respectively. Cell type enrichment analysis and immune infiltration analysis by clusterProfiler and ImmuCellAI were performed to identify critical immune cell component involved in ICIM and NICIAM. Additionally, core shared genes across ICIM and NICIAM were identified and validated by various models and methods.

Results: Interferon-γ response, inflammatory response and allograft rejection signaling were identified as the shared signaling pathways associated with ICIM and NICIAM. Enrichment analysis of cell type supported an important role of increased infiltration of T cells and macrophages in both ICIM and NICIAM. However, the predominant increase of infiltrated T cells was CD4+ T cells in NICIAM, while that were CD8+ T cells in ICIM. Core shared genes Lck and Cd3d expression were found increased in both ICIM and NICIAM, and Lck inhibition was further identified and validated as potential therapeutic approach.

Conclusions: Our study initially established a comorbidity model to identify potential molecular mechanism including interferon-γ response, inflammatory response and allograft rejection signaling accounting for the concerns of myocarditis risk in patients with preexisting autoimmune disease (PAD) receiving ICI treatment, and supported the therapeutic potential of targeting Lck or Cd3d.

[1]Department of Emergency, Renmin Hospital, Hubei University of Medicine, Shiyan, China
[2]Department of Oncology, Renmin Hospital, Hubei University of Medicine, Shiyan, China
[3]Department of Cardiology, Renmin Hospital, Hubei University of Medicine, Shiyan, China
[4]Institute of Cancer, Renmin Hospital, Hubei University of Medicine, Shiyan, China

*These authors contributed equally to this work.

Corresponding author:
Guoxing Wan, Department of Oncology, Renmin Hospital Hubei University of Medicine 39# Chaoyang Road Shiyan, Hubei 442000, China.
Email: 15gxwan@stu.edu.cn

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MDPI

Article

Elevated Preoperative NMPR Predicts an Unfavorable Chance of Survival in Resectable Esophageal Squamous Cell Carcinoma

Meng-Ying Peng [1,2,†], Zhi-Gang Zuo [2,†], Feng-Jun Cao [2], Yuan-Dong Yu [2], Xiao-Jun Cai [2,*] and Guo-Xing Wan [2,3,*]

[1] Graduate Student Training Base, Graduate School of Jinzhou Medical University, Jinzhou 121004, China
[2] Department of Oncology, Renmin Hospital, Hubei University of Medicine, Shiyan 442000, China
[3] Institute of Cancer, Renmin Hospital, Hubei University of Medicine, Shiyan 442000, China
* Correspondence: xiaojuncaishiyan@126.com (X.-J.C.); 15gxwan@stu.edu.cn (G.-X.W.)
† These authors contributed equally to this work.

Abstract: *Background and objectives*: Combined peripheral neutrophil–platelet indexes reflecting the systemic inflammatory status have been reported to predict the clinical outcome in patients with various types of cancer. However, the prognostic value of combined neutrophil–platelet indexes in operable esophageal squamous cell carcinoma (ESCC) remains unclear. The study introduced a novel combined neutrophil–meanplateletvolume–platelet ratio (NMPR) index and investigated its clinical and prognostic value in patients with operable ESCC receiving curative surgery. *Materials and Methods*: A retrospective analysis of the clinicopathologic data of 277 consecutive ESCC patients who received curative resection at Zhejiang Cancer Hospital in China between January 2007 and December 2010 was conducted (the training cohort). In addition, the clinicopathologic data of 101 resectable ESCC patients at Renmin Hospital of Hubei University of Medicine between December 2018 and June 2021 were collected (the external validation cohort). The optimal cutoff value of NMPR concerning overall survival (OS) in the training cohort was determined by X-tile software. Univariate and multivariate Cox regression analyses were used to evaluate the prognostic value of NMPR along with other variables in the training cohort, which was further validated with the same cutoff value in the external validation cohort. Significant predictors of OS were used to construct the nomogram, of which the discrimination and calibration was evaluated by concordance index (C-index) and calibration plots. *Results*: With a cutoff value of 16.62, the results from both the training and external validation cohorts supported the association of high NMPR (>16.62) with increased tumor length and advanced T stage but not with other variables. In the training cohort, a significant association between shorter OS and high NMPR ($p = 0.04$) as well as high CRP ($p < 0.001$), poor tumor differentiation ($p = 0.008$), advanced T stage ($p = 0.006$), advanced N stage ($p < 0.001$) and high CEA ($p = 0.007$) was revealed. Additionally, the high NMPR was verified to independently predict unfavorable OS ($p = 0.049$) in the external validation cohort. The C-index of the OS nomogram cooperating significant predictors in the training cohort was 0.71 and the calibration plots of the OS nomogram fitted well. *Conclusions*: The present study demonstrates that high NMPR is an independent predictor of unfavorable OS in resectable ESCC patients without neoadjuvant therapy.

Keywords: esophageal squamous cell carcinoma; neutrophil–mean-platelet-volume–platelet ratio; prognosis; biomarker

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1. Introduction

Esophageal cancer (EC) ranks sixth in annual incidence and fourth in mortality in China [1]. Unlike in Western countries, esophageal squamous cell carcinoma (ESCC) is the predominant histopathological type accounting for approximately 90% of the EC cases in Asian populations [2]. Surgical resection with or without chemoradiotherapy remains the mainstay of curative treatment for operable ESCC [3]. Although great progress in perioperative techniques, staging methods, surgical and oncological management has been

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High C-Reactive Protein to Albumin Ratio Predicts Inferior Clinical Outcomes in Extranodal Natural Killer T-Cell Lymphoma

Quan-shu Di[1], Tao Xu[1], Ying Song[1], Zhi-gang Zuo[1], Feng-jun Cao[1], Xiong-jie Yu[1], Ji-ying Tang[1], Wei Zhang[1], Chen Li[2,3], Guo-xing Wan[1], and Xiao-jun Cai[1]

Abstract

Objective: The prognostic value of C-reactive protein to albumin ratio (CAR) has been identified in several cancers but not in extranodal natural killer T-cell lymphoma (ENKTL) as yet. We aimed to evaluate the prognostic value of CAR in ENKTL.

Methods: A retrospective study with 246 patients with ENKTL was performed to determine the prognostic value of pretreatment CAR and examine the prognostic performance of CAR incorporating with International Prognostic Index (IPI) or natural killer/T-cell lymphoma prognostic index (NKPI) by nomogram.

Results: The Cox regression analyses showed that high CAR (>0.3) independently predicted unfavorable progression-free survival (PFS, $P = .011$) and overall survival (OS, $P = .012$). In the stratification analysis, the CAR was able to separate patients into different prognoses regarding both OS and PFS in Ann Arbor stage I+II as well as III+IV, IPI score 0 to 1, and NKPI score 1 to 2 subgroups (all $P < .05$). Additionally, the predictive accuracy of the IPI-based nomogram incorporating CAR, albumin to globulin ratio (AGR), and IPI for OS and PFS appeared to be lower than the NKPI-based nomogram incorporating CAR, age, AGR, extranodal site, and NKPI.

Conclusion: Pretreatment CAR is a simple and easily accessible parameter for independently predicting OS and PFS in patients with ENKTL.

Keywords

extranodal natural killer T-cell lymphoma (ENKTL), C-reactive protein to albumin ratio (CAR), prognosis, nutrition, inflammation

Introduction

Extranodal natural killer T-cell lymphoma (ENKTL) is a globally rare entity of non-Hodgkin lymphoma (NHL) with high aggressiveness,[1] accounting for less than 1% of NHL in the Western population and 7% to 10% of NHL in Asian and Latin American population.[2] Generally, ENKTL is a heterogeneous disease with poor prognosis, and most large cohort studies demonstrate a 5-year overall survival (OS) rate less than 50%.[1] Although upfront use of radiotherapy incorporating nonanthracycline-based chemotherapy regimens (eg, etoposide, methotrexate, ifosfamide, platinum, and L-asparaginase) has achieved improved outcome in recent decades, optimal treatment strategy and prognosis of ENKTL remain inadequately defined.[3] Considering the unsatisfactory prognosis in a substantial proportion of patients, prognostic stratification according to individual risk is very important and instrumental in facilitating decision-making and treatment modification for physicians.

Several prognostic factors for ENKTL have been widely adopted in recent years, such as age at diagnosis, regional lymph node involvement, Ann Arbor stage, performance

[1] Department of Oncology, Renmin Hospital, Hubei, China
[2] Laboratory of Medicinal Plant, Institute of Basic Medical Sciences, School of Basic Medicine, Hubei University of Medicine, Shiyan, Hubei, China
[3] Hubei Key Laboratory of Wudang Local Chinese Medicine Research, Biomedical Research Institute, Hubei University of Medicine, Shiyan, Hubei, China

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Corresponding Authors:
Guo-xing Wan and Xiao-jun Cai, Department of Oncology, Renmin Hospital, Hubei University of Medicine, 39 Chaoyang Road, Shiyan, Hubei 442000, China.
Emails: 15gxwan@stu.edu.cn; xiaojuncaishiyan@126.com

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RESEARCH ARTICLE

Network Pharmacology Along with Molecular Docking to Explore the Mechanism of Danshen Injection against Anthracycline-induced Cardiotoxicity and Transcriptome Validation

Quankai Dai[1,#], Yijun Pan[1,#], Xiwen Zhu[1], Mengyao Chen[2], Lin Xie[2], Yu Zhu[3] and Guoxing Wan[1,*]

[1]*Department of Oncology, Renmin Hospital, Hubei University of Medicine, Shiyan 442000, Hubei, China;* [2]*Department of Oncology, Renmin Hospital, Institute of Medicine and Nursing, Hubei University of Medicine, Shiyan 442000, Hubei, China;* [3]*Department of Research and Teaching, Renmin Hospital, Hubei University of Medicine, Shiyan 442000, Hubei, China*

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Abstract: ***Introduction*:** Although anthracyclines have demonstrated efficacy in cancer therapy, their utilization is constrained by cardiotoxicity. In contrast, Danshen injection (DSI), derived from *Salvia miltiorrhiza*, has a longstanding tradition of being employed to ameliorate cardiovascular ailments, including anthracycline-induced cardiotoxicity (AIC). Nonetheless, there is a notable dearth of comprehensive systematic investigation into the molecular mechanisms underlying DSI's effects on AIC. Consequently, this study was undertaken to explore the underlying mechanism by which DSI acted against AIC.

***Methods*:** Employing network pharmacology approach, the current investigation undertook a comprehensive analysis of the impact of DSI on AIC, which was further validated by transcriptome sequencing with *in vitro* AIC model. Additionally, molecular docking was conducted to evaluate the binding of active ingredients to core targets. A total of 3,404 AIC-related targets and 12 active ingredients in DSI, including chrysophanol, luteolin, tanshinone IIA, isoimperatorin, among others, were collected by differentially expressed analysis and database search, respectively.

***Results*:** The network pharmacology and enrichment analysis suggested 102 potential targets and 29 signaling pathways associated with the protective effect of DSI on AIC. Three core targets (CA12, NOS3, and POLH) and calcium signaling pathways were further validated by transcriptomic analysis of the *in-vitro* model. The high affinity of the active ingredients binding to corresponding targets was confirmed by molecular docking.

***Conclusion*:** The present study suggested that DSI might exert a cardioprotective effect on AIC *via* the inhibition of CA12, NOS3, and POLH, as well as the modulation of calcium signaling. Further experiments are warranted to verify the findings.

Keywords: Danshen injection, anthracycline-induced cardiotoxicity, network pharmacology, transcriptome sequencing, molecular docking, cardioprotection.

1. INTRODUCTION

Doxorubicin (DOX), commonly referred to as adriamycin, stands as the predominant anthracycline chemotherapeutic agent for the treatment of malignant neoplasms afflicting both adults and pediatric patients in clinical practice, encompassing conditions like lymphoma, sarcomas, and a diverse spectrum of malignancies [1]. Notably, notwithstanding its substantial clinical efficacy, a noteworthy proportion of patients, approximately a quarter, encounter cardiotoxicity. The etiology of AIC conventionally ascribes its origin to mitochondrial malfunction, precipitating the disproportionate accrual of reactive oxygen species (ROS) [2]. Furthermore, various contributory elements, encompassing oxidative stress, perturbations in iron metabolism, release of nitric oxide, induction of inflammatory mediators, disturbances in calcium regulation, and aberrations in autophagic processes, are collectively regarded as pivotal factors conferring susceptibility to AIC [3, 4].

In clinical practice, the adverse manifestations of AIC predominantly encompass heart failure and structural impairment, thereby imposing a sustained harmful impact on the quality of life experienced by affected patients. In severe instances, it has the potential to precipitate sudden cardiac demise [5]. Despite the extensive duration of research efforts spanning several decades, therapeutic modalities targeting AIC remain shockingly scarce. At present, dexrazoxane (DEX) is the unique pharmacological agent endorsed by the United States Food and Drug Administration (FDA) for the treatment of AIC. Recent clinical investigations have advanced the notion that optimal cardiac safeguarding may be attained by maintaining a DEX to DOX dosage ratio of 10:1, administered across a span exceeding three treatment cycles, equivalent to nine weeks. Nonetheless, the inclusion of DEX within the therapeutic arsenal for AIC elicits concerns due to potential sequelae involving the development of secondary malignancies, coupled with the observed variability in therapeutic efficacy among distinct patient cohorts, thus substantially curbing its widespread adoption [6, 7]. Intriguingly, while some agents recognized for their cardioprotective attributes, such as angiotensin-converting enzyme inhibitors, beta-blockers [8], moderate aerobic exercise [9], and adherence to appropriately tailored dosing regimens have garnered empirical support in mitigating AIC [10], it is imperative to acknowledge the existence of incongruities within the extant research findings, often underpinned by a relative paucity of robust evidence. Consequent-

*Address correspondence to this author at the Department of Oncology, Renmin Hospital, Hubei University of Medicine, Shiyan 442000, Hubei, China; Tel: +86-719-8637385; Fax: +86-719-8637385; E-mail: 15gxwan@stu.edu.cn
[#]*These authors contributed equally to this work.*

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Letters

COMMENT & RESPONSE

Valid Analysis of Brain-Specific Progression-Free Survival

To the Editor In the recent phase 2 randomized clinical trial published in *JAMA Oncology*, Chen et al[1] reported that the induction treatment of bevacizumab, etoposide, and cisplatin (BEEP) followed by whole-brain radiotherapy (WBRT) significantly prolonged brain-specific progression-free survival (BPFS) compared with WBRT alone in patients with untreated brain metastases from breast cancer (BCBM). Nevertheless, the results of this trial should be generalized with caution due to the ambiguity in clarification of some key baseline characteristics.

The absence of extracranial metastases and control of primary tumor both favoring the outcome were the main components of Radiation Therapy Oncology Group (RTOG) Recursive Partitioning Analysis (RPA), and Basic Score for Brain Metastases (BSBM) indices for patients with BCBM receiving WBRT.[2,3] However, the design choices regarding extracranial metastases and control of primary tumor may have resulted in imbalances in baseline risk in this study.[1] First, the number of extracranial metastasis was summarized along with intracranial metastasis although they were both considered as significant independent predictors of survival outcome.[3] Are the proportions of extracranial and intracranial metastases balanced in the experimental and control arms? Then, systemic treatments that may affect BPFS through the control of primary tumor were allowed during BEEP treatment. Whereas, no information was given about the effect of systemic treatments on the control of primary tumor in both arms, which may lead to inappropriate interpretation of the primary outcome. Furthermore, 2 multiinstitutional studies[4,5] with large sample sizes have reported the BPFS benefit of a longer time to brain metastases. We noted that the median time to relapse (15.7 vs 6.8 months) and brain metastases (39.4 vs 34.8 months) seemed longer in the experimental arm compared with the control arm in this study,[1] which may bias the effect of BEEP plus WBRT vs WBRT. Finally, no advantage of brain-specific objective response rate (64.9% vs 76.3%) was observed for BEEP plus WBRT vs WBRT alone. Therefore, we believe that an in-depth analysis is urgently needed to provide more accurate results after eliminating confounding effects of benefit-related characteristics.

Given high interest in cognitive dysfunction in patients receiving WBRT, hippocampal avoidance (HA) and memantine coupled with WBRT was recommended for patients with favorable prognosis and brain metastases ineligible for surgery and/or stereotactic radiosurgery by international consensus guidelines. However, cognitive function was not reported in this study.[1] Was HA or memantine used during WBRT treatment? Did the BEEP induction affect cognitive function? These concerns are supposed to be clarified.

Yuandong Yu, MD
Fengjun Cao, MD
Guoxing Wan, MD

Author Affiliations: Department of Oncology, Renmin Hospital, Hubei University of Medicine, Shiyan, China.

Corresponding Author: Guoxing Wan, MD, Department of Oncology, Renmin Hospital, Hubei University of Medicine, 39# Chaoyang Rd, Shiyan, Hubei 442000, China (15gxwan@stu.edu.cn).

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Molecular Medicine

RESEARCH ARTICLE

Open Access

Weighted gene co-expression network-based approach to identify key genes associated with anthracycline-induced cardiotoxicity and construction of miRNA-transcription factor-gene regulatory network

Guoxing Wan[1], Peinan Chen[2], Xue Sun[1], Xiaojun Cai[1], Xiongjie Yu[1], Xianhe Wang[1*] and Fengjun Cao[1,1*]

Abstract

Background: Cardiotoxicity is a common complication following anthracycline chemotherapy and represents one of the serious adverse reactions affecting life, which severely limits the effective use of anthracyclines in cancer therapy. Although some genes have been investigated by individual studies, the comprehensive analysis of key genes and molecular regulatory network in anthracyclines-induced cardiotoxicity (AIC) is lacking but urgently needed.

Methods: The present study integrating several transcription profiling datasets aimed to identify key genes associated with AIC by weighted correlation network analysis (WGCNA) and differentially expressed analysis (DEA) and also constructed miRNA-transcription factor-gene regulatory network. A total of three transcription profiling datasets involving 47 samples comprising 41 rat heart tissues and 6 human induced pluripotent stem cell-derived cardiomyocytes (hiPSCMs) samples were enrolled.

Results: The WGCNA and DEA with E-MTAB-1168 identified 14 common genes affected by doxorubicin administrated by 4 weeks or 6 weeks. Functional and signal enrichment analyses revealed that these genes were mainly enriched in the regulation of heart contraction, muscle contraction, heart process, and oxytocin signaling pathway. Ten (Ryr2, Casq1, Fcgr2b, Postn, Tceal5, Ccn2, Tnfrsf12a, Mybpc2, Ankrd23, Scn3b) of the 14 genes were verified by another gene expression profile GSE154603. Importantly, three key genes (Ryr2, Tnfrsf12a, Scn3b) were further validated in a hiPSCMs-based in-vitro model. Additionally, the miRNA-transcription factor-gene regulatory revealed several top-ranked transcription factors including Tcf12, Ctcf, Spdef, Ebf1, Sp1, Rcor1 and miRNAs including miR-124-3p, miR-195-5p, miR-146a-5p, miR-17-5p, miR-15b-5p, miR-424-5p which may be involved in the regulation of genes associated with AIC.

*Correspondence: wangxianhe@shiyanhospital.com; fengjuncaoshiyan@126.com
[1] Department of Oncology, Renmin Hospital, Hubei University of Medicine, 39 Chaoyang Road, Shiyan 442000, Hubei, China
Full list of author information is available at the end of the article

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Identification of shared mechanisms and targets between immune checkpoint inhibitor-associated myocarditis and autoimmune myocarditis

Kai Yang[1,*], Min Zhang[2,*], Dong Li[3,*], Yuandong Yu[2], Fengjun Cao[2] and Guoxing Wan[2,4]

Abstract

Objective: This study aimed to explore the shared mechanisms and targets between immune checkpoint inhibitor-associated myocarditis (ICIM) and autoimmune myocarditis.

Methods: Relevant data were retrieved from public datasets and Gene Expression Omnibus (GEO) database. Gene set enrichment analysis (GSEA) of differentially expressed genes (DEGs) was used to identify significant shared signaling pathways between ICIM and non-ICI associated autoimmune myocarditis (NICIAM) represented by ICIM model and experimental autoimmune myocarditis (EAM) model, respectively. Cell type enrichment analysis and immune infiltration analysis by clusterProfiler and ImmuCellAI were performed to identify critical immune cell component involved in ICIM and NICIAM. Additionally, core shared genes across ICIM and NICIAM were identified and validated by various models and methods.

Results: Interferon-γ response, inflammatory response and allograft rejection signaling were identified as the shared signaling pathways associated with ICIM and NICIAM. Enrichment analysis of cell type supported an important role of increased infiltration of T cells and macrophages in both ICIM and NICIAM. However, the predominant increase of infiltrated T cells was CD4+ T cells in NICIAM, while that were CD8+ T cells in ICIM. Core shared genes Lck and Cd3d expression were found increased in both ICIM and NICIAM, and Lck inhibition was further identified and validated as potential therapeutic approach.

Conclusions: Our study initially established a comorbidity model to identify potential molecular mechanism including interferon-γ response, inflammatory response and allograft rejection signaling accounting for the concerns of myocarditis risk in patients with preexisting autoimmune disease (PAD) receiving ICI treatment, and supported the therapeutic potential of targeting Lck or Cd3d.

[1]Department of Emergency, Renmin Hospital, Hubei University of Medicine, Shiyan, China
[2]Department of Oncology, Renmin Hospital, Hubei University of Medicine, Shiyan, China
[3]Department of Cardiology, Renmin Hospital, Hubei University of Medicine, Shiyan, China
[4]Institute of Cancer, Renmin Hospital, Hubei University of Medicine, Shiyan, China

*These authors contributed equally to this work.

Corresponding author:
Guoxing Wan, Department of Oncology, Renmin Hospital Hubei University of Medicine 39# Chaoyang Road Shiyan, Hubei 442000, China.
Email: 15gxwan@stu.edu.cn

6P Circulating tumour cells (CTCs) as biomarkers of resistance to the CDK4/6 inhibitor (CDK4/6i) palbociclib (P) in patients (pts) with ER+/HER2-negative advanced breast cancer (ABC)

F. Galardi[1], C. Biagioni[2], F. De Luca[1], G. Curigliano[3], A.M. Minisini[4], M. Bonechi[1], E. Moretti[5], E. Risi[6], I. Migliaccio[7], A. McCartney[5], M. Benelli[8], D. Romagnoli[8], V. Conti[9], L. Biganzoli[5], A. Di Leo[10], L. Malorni[2]

[1]Sandro Pitigliani Translational Research Unit, Nuovo Ospedale di Prato Santo Stefano - Azienda Usl Toscana Centro, Prato, Italy; [2]"Sandro Pitigliani" Medical Oncology Department, Bioinformatics Unit, Nuovo Ospedale di Prato Santo Stefano - Azienda Usl Toscana Centro, Prato, Italy; [3]Early Drug Development for Innovative Therapies Division, University of Milan, Istituto Europeo di Oncologia, Milan, Italy; [4]Oncology Department, "Azienda Sanitaria Universitaria del Friuli Centrale", Udine, Italy; [5]"Sandro Pitigliani" Medical Oncology Department, Nuovo Ospedale di Prato Santo Stefano - Azienda Usl Toscana Centro, Prato, Italy; [6]"Sandro Pitigliani" Medical Oncology Department, Nuovo Ospedale di Prato, Prato, Italy; [7]"Sandro Pitigliani" Translational Research Unit, Ospedale di Prato Sandro Pitigliani Med. Oncology Unit, Prato, Italy; [8]Bioinformatics Unit, Nuovo Ospedale di Prato Santo Stefano - Azienda Usl Toscana Centro, Prato, Italy; [9]Laboratory of Neurobiology Child Neurology Unit, Children's Hospital A. Meyer, Florence, Italy; [10]Medical Oncology Department, Nuovo Ospedale di Prato, Prato, Italy

Background: Resistance to CDK4/6i is inevitable. CTC count is prognostic in ABC, but its role in pts treated with CDK4/6i is not well defined. Genetic loss of RB1 is a known yet infrequent marker of CDK4/6i resistance. We assessed the prognostic role of CTC count and gene-expression (GE) levels of RB1 in CTCs in pts receiving P.

Methods: The TREnd trial (NCT02549430) randomized pts with endocrine resistant ABC to either P alone or P plus the endocrine therapy received in the prior line of treatment. In TREnd, blood samples were prospectively collected in CellSave® tubes before starting P (T0), after the first cycle (T1) and at disease progression (T2). CTCs were isolated and counted by CellSearch® System (CS) using CellSearch™ Epithelial Cell kit. Samples with ≥5CTCs were sorted by DEPArray system® (DA). RNA extraction and retro-transcription for GE experiments were performed by Cell Lysis Two-Step RT-qPCR. RB1 and GAPDH GE levels were measured by ddPCR, with a multiplex assay with a sensitivity of 30-10 pg of cDNA, set up on three different cell lines sensitive and resistant to P.

Results: 46 pts were suitable for CTC analysis. CTC count at T0 did not show significant prognostic value in terms of progression free survival (PFS). However, pts with at least 1 detectable CTC at T1 (n=26) had a worse PFS than those with 0 CTCs (n=16) (p=0.02). Similar results were observed with a cut-off of 5 CTCs (p=0.04). At T1, 7 out of 39 pts had an increase of at least 3 CTCs which proved prognostic (p=0.01). Pts with ≥5CTCs at T2 (n=6/23) who received chemotherapy as post-study treatment had a shorter time to treatment failure (p=0.02). DA sorting was conducted on 20/46 pts and GE data for RB1 were obtained from 19 pts. CTCs showed heterogeneous RB1 expression. Pts with detectable expression of RB1 in at least one time-point had better, but not significant, outcomes than those with undetectable levels.

Conclusions: Persistence or an increase in CTCs after one cycle of P may identify pts with worse outcome. High CTC counts at disease progression on P may indicate poor post-treatment prognosis. Measuring RB1 GE levels on CTCs by ddPCR is feasible, but its clinical significance is yet unclear.

Clinical trial identification: NCT02549430.

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7P Prognostic value of the immune infiltration score in early breast cancer patients receiving dual HER2 blockade with trastuzumab and pertuzumab: An exploratory analysis of a randomized clinical trial

G. Wan, F. Cao, X. Cai, X. Yu, Z. Zuo, Y. Song, T. Xu, Y. Li, Y. Yu, X. Wang, X. Wang

Oncology, Hubei University of Medicine, Shiyan, China

Background: Although the survival benefit of dual epidermal growth factor receptor 2 (HER2) blockade with trastuzumab and pertuzumab was definitely demonstrated in HER2-amplified early breast cancer, sufficient biomarkers are urgently required to explain the heterogeneous response to dual HER-2 blockade therapy. The prognostic significance of immune infiltration in TRYPHAENA trial was investigated to tailor treatment in current analysis.

Methods: Among the 225 HER2-amplified early breast cancer patients randomly assigned to trastuzumab/pertuzumab concurrently or sequentially with standard chemotherapy as neoadjuvant therapy in TRYPHAENA trial, 162 patients with available gene expression profile and complete follow-up data were enrolled. The normalized gene expression matrix (GSE109710) based on the NanoString nCounter array was downloaded from Gene Expression Omnibus database and further used to estimate the immune infiltration score (IIS) for each patient by the Immune Cell Abundance Identifier tool. A cut-off of IIS to stratify patients was determined by the R-based survminer package. Multivariable Cox proportional event-free survival (EFS) hazard ratios were preformed.

Results: Among the 162 women included in the analysis (median [range] age, 49.0 [27-81] years), the pathologic complete response (pCR) rate was 50.0% (21/42) in patients with a high IIS (>0.628) and 66.7% (80/120) in patients with a low SII (≤0.628). At a median follow-up of 4.7 years, the multivariable-adjusted hazard ratio for EFS was 2.933 (95%CI, 1.223-7.033) for the high IIS and 0.356 (95%CI, 0.127-0.999) in patients who achieved pCR, respectively.

Table 7P: Cox regression for EFS

Variable	Univariate analysis		Multivariable analysis	
	Hazard ratio (95%CI)	P-value	Hazard ratio (95%CI)	P-value
Age (≥50 vs <50 y)	1.628(0.747-3.545)	0.220	1.779(0.760-4.165)	0.184
Histology grade (G3 vs G1/G2)	0.855(0.563-1.300)	0.464	1.019(0.633-1.641)	0.938
Hormone receptor (positive vs negative)	0.918(0.426-1.982)	0.828	0.920(0.369-2.296)	0.859
Clinical stage (III vs II)	2.207(0.975-4.995)	0.058	1.278(0.820-1.991)	0.279
pCR (yes vs no)	0.408(0.187-0.889)	0.024	0.356(0.127-0.999)	0.050
IIS (high vs low)	2.812(1.300-6.084)	0.009	2.933(1.223-7.033)	0.016

Conclusions: Our analysis demonstrates an independent prognostic value of IIS in patients receiving trastuzumab/pertuzumab-based neoadjuvant chemotherapy.

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RESEARCH ARTICLE

Mechanism Exploration of Astaxanthin in the Treatment of Adriamycin-induced Cardiotoxicity Based on Network Pharmacology and Experimental Validation

Yu Zhu[1,#], Mengyao Chen[2,#], Lin Xie[2], Yijun Pan[3], Yuntian Yang[3] and Guoxing Wan[3,*]

[1]*Department of Research and Teaching, Renmin Hospital, Hubei University of Medicine, Shiyan, 442000, Hubei, China;* [2]*Department of Oncology, Renmin Hospital, Institute of Medicine and Nursing, Hubei University of Medicine, Shiyan, 442000, Hubei, China;* [3]*Department of Oncology, Renmin Hospital, Hubei University of Medicine, Shiyan, 442000, Hubei, China*

Abstract: ***Introduction*:** Astaxanthin (AXT), a natural antioxidant recognized for its therapeutic potential in cancer and cardiovascular diseases, holds promise in mitigating adriamycin-induced cardiotoxicity (AIC). Nevertheless, the underlying mechanisms of AXT in AIC mitigation remain to be elucidated. Consequently, this study endeavors to elucidate the mechanism of AXT against AIC, employing an integrated approach.

***Methods*:** Network pharmacology, molecular docking, and molecular dynamics simulations were harnessed to explore the molecular mechanism underlying AXT's action against AIC. Furthermore, the *in-vitro* AIC model was established with the H9c2 cell to generate transcriptome data for validation.

***Results*:** A total of 533 putative AXT targets and 1478 AIC-related genes were initially screened by database retrieval and bioinformatics analysis. A total of 248 potential targets of AXT against AIC and several signaling pathways were identified by network pharmacology and enrichment analysis. Two core genes (CCL2 and NOS3) and the AGE-RAGE signaling pathway in diabetic complications were further highlighted by transcriptome validation based on the AIC *in-vitro* model. Additionally, molecular docking and dynamics analyses supported the robust binding affinity of AXT with the core targets.

***Conclusion*:** The study suggested that AXT might ameliorate AIC through the inhibition of CCL2 and NOS3 as well as AGE-RAGE signaling, which provide a theoretical basis for the development of a strategy against AIC.

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1. INTRODUCTION

Doxorubicin (DOX), belonging to the anthracycline class of anti-tumor chemotherapy drugs, has been demonstrated with widespread application in the clinical management of diverse cancer types [1], encompassing hematological malignancies and solid tumors, owing to its remarkable efficacy and comprehensive therapeutic effects [2]. However, its clinical utility is significantly curtailed by the cumulative and dose-dependent cardiotoxic side effects [3]. Despite notable efforts, the precise molecular mechanisms underlying DOX-induced cardiac injury remain elusive. The genesis of adriamycin-induced cardiotoxicity (AIC) entails a complex interplay of various mechanisms, including oxidative stress, myocardial cell apoptosis, mitochondrial dysfunction, autophagy, DNA damage, and inflammatory responses, among others [4]. Among these, the induc-

*Address correspondence to this author at the Department of Oncology, Renmin Hospital, Hubei University of Medicine, Shiyan 442000, Hubei, China, Tel: +86-719-8637385, Fax: +86-719-8637385, E-mail: 15gxwan@stu.edu.cn

[#] *These authors contributed equally to this work.*

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第 2 条，共 2 条

标题: Network Pharmacology Along with Molecular Docking to Explore the Mechanism of Danshen Injection against Anthracycline-induced Cardiotoxicity and Transcriptome Validation

作者: Dai, QK (Dai, Quankai); Pan, YJ (Pan, Yijun); Zhu, XW (Zhu, Xiwen); Chen, MY (Chen, Mengyao); Xie, L (Xie, Lin); Zhu, Y (Zhu, Yu); Wan, GX (Wan, Guoxing)

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地址: [Dai, Quankai; Pan, Yijun; Zhu, Xiwen; Wan, Guoxing] Hubei Univ Med, Renmin Hosp, Dept Oncol, Shiyan 442000, Hubei, Peoples R China.
[Chen, Mengyao; Xie, Lin] Hubei Univ Med, Renmin Hosp, Inst Med & Nursing, Dept Oncol, Shiyan 442000, Hubei, Peoples R China.
[Zhu, Yu] Hubei Univ Med, Renmin Hosp, Dept Res & Teaching, Shiyan 442000, Hubei, Peoples R China.
通讯作者地址: Dai, QK; Pan, YJ; Wan, GX (通讯作者)，Hubei Univ Med, Renmin Hosp, Dept Oncol, Shiyan 442000, Hubei, Peoples R China.
电子邮件地址: 15gxwan@stu.edu.cn
Affiliations: Hubei University of Medicine; Hubei University of Medicine; Hubei University of Medicine

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第 1 条，共 2 条

标题: Identification of shared mechanisms and targets between immune checkpoint inhibitor-associated myocarditis and autoimmune myocarditis

作者: Yang, K (Yang, Kai); Zhang, M (Zhang, Min); Li, D (Li, Dong); Yu, YD (Yu, Yuandong); Cao, FJ (Cao, Fengjun); Wan, GX (Wan, Guoxing)

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地址: [Yang, Kai] Hubei Univ Med, Renmin Hosp, Dept Emergency, Shiyan, Peoples R China.
[Zhang, Min; Yu, Yuandong; Cao, Fengjun; Wan, Guoxing] Hubei Univ Med, Renmin Hosp, Dept Oncol, Shiyan, Peoples R China.
[Li, Dong] Hubei Univ Med, Renmin Hosp, Dept Cardiol, Shiyan, Peoples R China.
[Wan, Guoxing] Hubei Univ Med, Renmin Hosp, Inst Canc, Shiyan, Peoples R China.
[Wan, Guoxing] Hubei Univ Med, Renmin Hosp, Dept Oncol, 39 Chaoyang Rd, Shiyan 442000, Hubei, Peoples R China.

通讯作者地址: Wan, GX (通讯作者)，Hubei Univ Med, Renmin Hosp, Dept Oncol, 39 Chaoyang Rd, Shiyan 442000, Hubei, Peoples R China.

电子邮件地址: 15gxwan@stu.edu.cn

Affiliations: Hubei University of Medicine; Hubei University of Medicine; Hubei University of Medicine; Hubei University of Medicine; Hubei University of Medicine

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地址: [Yu, Yuandong; Cao, Fengjun; Wan, Guoxing] Hubei Univ Med, Renmin Hosp, Dept Oncol, 39 Chaoyang Rd, Shiyan 442000, Hubei, Peoples R China.

通讯作者地址: Wan, GX (通讯作者)，Hubei Univ Med, Renmin Hosp, Dept Oncol, 39 Chaoyang Rd, Shiyan 442000, Hubei, Peoples R China.

电子邮件地址: 15gxwan@stu.edu.cn

Affiliations: Hubei University of Medicine

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第 1 条，共 1 条

标题: Mechanism Exploration of Astaxanthin in the Treatment of Adriamycin-induced Cardiotoxicity Based on Network Pharmacology and Experimental Validation

作者: Zhu, Y (Zhu, Yu); Chen, MY (Chen, Mengyao); Xie, L (Xie, Lin); Pan, YJ (Pan, Yijun); Yang, YT (Yang, Yuntian); Wan, GX (Wan, Guoxing)

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地址: [Zhu, Yu] Hubei Univ Med, Renmin Hosp, Dept Res & Teaching, Shiyan 442000, Hubei, Peoples R China.

[Chen, Mengyao; Xie, Lin] Hubei Univ Med, Renmin Hosp, Inst Med & Nursing, Dept Oncol, Shiyan 442000, Hubei, Peoples R China.

[Pan, Yijun; Yang, Yuntian; Wan, Guoxing] Hubei Univ Med, Renmin Hosp, Dept Oncol, Shiyan 442000, Hubei, Peoples R China.

通讯作者地址: Wan, GX (通讯作者)，Hubei Univ Med, Renmin Hosp, Dept Oncol, Shiyan 442000, Hubei, Peoples R China.

电子邮件地址: 15gxwan@stu.edu.cn

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第 3 条，共 3 条

标题: Prognostic value of the immune infiltration score in early breast cancer patients receiving dual HER2 blockade with trastuzumab and pertuzumab: An exploratory analysis of a randomized clinical trial

作者: Wan, G (Wan, G.); Cao, F (Cao, F.); Cai, X (Cai, X.); Yu, X (Yu, X.); Zuo, Z (Zuo, Z.); Song, Y (Song, Y.); Xu, T (Xu, T.); Li, Y (Li, Y.); Yu, Y (Yu, Y.); Wang, X (Wang, X.)

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标题: Exploring the molecular mechanism of ginseng against anthracycline-induced cardiotoxicity based on network pharmacology, molecular docking and molecular dynamics simulation

作者: Xie, L (Xie, Lin); Liu, HZ (Liu, Hanze); Zhang, K (Zhang, Ke); Pan, YJ (Pan, Yijun); Chen, MY (Chen, Mengyao); Xue, XY (Xue, Xiangyue); Wan, GX (Wan, Guoxing)

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地址: [Xie, Lin; Chen, Mengyao; Xue, Xiangyue] Hubei Univ Med, Renmin Hosp, Inst Med & Nursing, Dept Oncol, Shiyan 442000, Hubei, Peoples R China.

[Liu, Hanze; Zhang, Ke; Pan, Yijun; Wan, Guoxing] Hubei Univ Med, Renmin Hosp, Dept Oncol, 39 Chaoyang Rd, Shiyan 442000, Hubei, Peoples R China.

通讯作者地址: Wan, GX (通讯作者)，Hubei Univ Med, Renmin Hosp, Dept Oncol, 39 Chaoyang Rd, Shiyan 442000, Hubei, Peoples R China.

电子邮件地址: 15gxwan@stu.edu.cn

Affiliations: Hubei University of Medicine; Hubei University of Medicine

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地址: [Wan, Guoxing; Cao, Fengjun; Wang, Xuanbin; Sun, Xue] Hubei Univ Med, Renmin Hosp, Dept Oncol, 39 Chaoyang Rd, Shiyan 442000, Peoples R China.

通讯作者地址: Wan, GX (通讯作者)，Hubei Univ Med, Renmin Hosp, Dept Oncol, 39 Chaoyang Rd, Shiyan 442000, Peoples R China.

电子邮件地址: 15gxwan@stu.edu.cn

Affiliations: Hubei University of Medicine

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5 年影响因子（2021 年）：	**4.505**	
2021 年影响因子：	**4.478**	
2022 年中科院分区（升级版）	**大类分区：**	医学 **3** 区
	小类分区：	肿瘤学 **3** 区

第 3 条，共 4 条

标题: High C-Reactive Protein to Albumin Ratio Predicts Inferior Clinical Outcomes in Extranodal Natural Killer T-Cell Lymphoma

作者: Di, QS (Di, Quan-shu); Xu, T (Xu, Tao); Song, Y (Song, Ying); Zuo, ZG (Zuo, Zhi-gang); Cao, FJ (Cao, Feng-jun); Yu, XJ (Yu, Xiong-jie); Tang, JY (Tang, Ji-ying); Zhang, W (Zhang, Wei); Li, C (Li, Chen); Wan, GX (Wan, Guo-xing); Cai, XJ (Cai, Xiao-jun)

来源出版物: DOSE-RESPONSE 卷: 18 期: 2 文献号: 1559325820917824
DOI: 10.1177/1559325820917824 出版年: APR 2020
Web of Science 核心合集中的 "被引频次": 4
被引频次合计: 4
入藏号: WOS:000525194100001
文献类型: Article

地址: [Di, Quan-shu; Xu, Tao; Song, Ying; Zuo, Zhi-gang; Cao, Feng-jun; Yu, Xiong-jie; Tang, Ji-ying; Zhang, Wei; Wan, Guo-xing; Cai, Xiao-jun] Renmin Hosp, Dept Oncol, Shiyan, Hubei, Peoples R China.
[Li, Chen] Hubei Univ Med, Sch Basic Med, Inst Basic Med Sci, Lab Med Plant, Shiyan, Hubei, Peoples R China.
[Li, Chen] Hubei Univ Med, Biomed Res Inst, Hubei Key Lab Wudang Local Chinese Med Res, Shiyan, Hubei, Peoples R China.
通讯作者地址: Wan, GX; Cai, XJ (通讯作者)，Hubei Univ Med, Renmin Hosp, Dept Oncol, 39 Chaoyang Rd, Shiyan 442000, Hubei, Peoples R China.
电子邮件地址: 15gxwan@stu.edu.cn; xiaojuncaishiyan@126.com
Affiliations: Hubei University of Medicine; Hubei University of Medicine

ISSN: 1559-3258
输出日期: 2023-06-05

5 年影响因子（2021 年）：	**3.01**	
2021 年影响因子：	**2.623**	
2022 年中科院分区（升级版）	**大类分区：**	医学 **4 区**
	小类分区：	药学 **4 区**
		核医学 **4 区**

湖北医药学院图书馆检索报告附件

第 2 条，共 4 条

标题: Weighted gene co-expression network-based approach to identify key genes associated with anthracycline-induced cardiotoxicity and construction of miRNA-transcription factor-gene regulatory network

作者: Wan, GX (Wan, Guoxing); Chen, PN (Chen, Peinan); Sun, X (Sun, Xue); Cai, XJ (Cai, Xiaojun); Yu, XJ (Yu, Xiongjie); Wang, XH (Wang, Xianhe); Cao, FJ (Cao, Fengjun)

来源出版物: MOLECULAR MEDICINE 卷: 27 期: 1 文献号: 142

DOI: 10.1186/s10020-021-00399-9 出版年: DEC 2021

Web of Science 核心合集中的 "被引频次": 2

被引频次合计: 2

入藏号: WOS:000714371300001

文献类型: Article

地址: [Wan, Guoxing; Sun, Xue; Cai, Xiaojun; Yu, Xiongjie; Wang, Xianhe; Cao, Fengjun] Hubei Univ Med, Renmin Hosp, Dept Oncol, 39 Chaoyang Rd, Shiyan 442000, Hubei, Peoples R China.

[Chen, Peinan] Shantou Univ, Affiliated Hosp 2, Med Coll, Dept Cardiol, Shantou 515000, Guangdong, Peoples R China.

通讯作者地址: Wang, XH; Cao, FJ (通讯作者)，Hubei Univ Med, Renmin Hosp, Dept Oncol, 39 Chaoyang Rd, Shiyan 442000, Hubei, Peoples R China.

电子邮件地址: wangxianhe@shiyanhospital.com; fengjuncaoshiyan@126.com

Affiliations: Hubei University of Medicine; Shantou University

ISSN: 1076-1551

eISSN: 1528-3658

输出日期: 2023-06-05

5 年影响因子（2021 年）：	**6.15**	
2021 年影响因子：	**6.382**	
2022 年中科院分区（升级版）	大类分区：	医学 **2** 区
	小类分区：	生化与分子生物学 **3** 区
		细胞生物学 **3** 区
		医学：研究与实验 **3** 区

湖北医药学院图书馆检索报告附件

第 1 条，共 4 条

标题: Elevated Preoperative NMPR Predicts an Unfavorable Chance of Survival in Resectable Esophageal Squamous Cell Carcinoma

作者: Peng, MY (Peng, Meng-Ying); Zuo, ZG (Zuo, Zhi-Gang); Cao, FJ (Cao, Feng-Jun); Yu, YD (Yu, Yuan-Dong); Cai, XJ (Cai, Xiao-Jun); Wan, GX (Wan, Guo-Xing)

来源出版物: MEDICINA-LITHUANIA 卷: 58 期: 12 文献号: 1808

DOI: 10.3390/medicina58121808 出版年: DEC 2022

Web of Science 核心合集中的 "被引频次": 0

被引频次合计: 0

入藏号: WOS:000902873000001

文献类型: Article

地址: [Peng, Meng-Ying] Jinzhou Med Univ, Grad Student Training Base, Grad Sch, Jinzhou 121004, Peoples R China.

[Peng, Meng-Ying; Zuo, Zhi-Gang; Cao, Feng-Jun; Yu, Yuan-Dong; Cai, Xiao-Jun; Wan, Guo-Xing] Hubei Univ Med, Renmin Hosp, Dept Oncol, Shiyan 442000, Peoples R China.

[Wan, Guo-Xing] Hubei Univ Med, Renmin Hosp, Inst Canc, Shiyan 442000, Peoples R China.

通讯作者地址: Cai, XJ; Wan, GX (通讯作者)，Hubei Univ Med, Renmin Hosp, Dept Oncol, Shiyan 442000, Peoples R China.

Wan, GX (通讯作者)，Hubei Univ Med, Renmin Hosp, Inst Canc, Shiyan 442000, Peoples R China.

电子邮件地址: xiaojuncaishiyan@126.com; 15gxwan@stu.edu.cn

Affiliations: Jinzhou Medical University; Hubei University of Medicine; Hubei University of Medicine

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输出日期: 2023-06-05

5 年影响因子（2021 年）：	**2.985**	
2021 年影响因子：	**2.948**	
2022 年中科院分区（升级版）	**大类分区：**	**医学 4 区**
	小类分区：	**医学：内科 3 区**

第三临床学院2022年本科生导师制聘期考核结果公示

发布科室： 第三临床学院 发布时间： 2023-03-20 17:46

按照《湖北医药学院第三临床学院临床本科生导师制实施办法（三院字【2021】7号）》文件精神，第三临床学院对2022年遴选的本科生导师进行聘期考核。经个人材料填报自评、学生评分及第三临床学院导师制工作领导小组审核认定，现将考核结果公示如下（排名不分先后）：

优秀：

万国兴、杜 俊、贾 佳、孟祖东

合格：

汤继英、阿彩岭、赵沛誉、杨 芳、孙志丰、徐 莲、叶婷婷

范 丽、李慧卉、王 恒、陈 涛、彭先兵、陆 雪、张 毅、

龚宝兰、周发明、邓艳青、李德坤、李小丽

根据考核结果，对本科生导师进行工作量核算，该类工作量在教学职称晋升时予以认定，不计入年度课时费核算：考核为优秀的导师，按每名导师30学时/年计算教学工作量；考核为合格的导师，按每名导师20学时/年计算教学工作量。

公示时间：2023年3月20日-23日，如有异议，请以书面形式或者电话形式致电第三临床学院教学科研办公室,联系电话：0719-8637859。

湖北医药学院第三临床学院

2023年3月20日

第三临床学院2023年本科生导师制聘期考核结果公示

发布科室： 第三临床学院　　发布时间： 2024-01-20 16:53

按照《湖北医药学院第三临床学院本科生导师制实施办法》及本科生导师工作量化考核细则（2023年3月修订）》文件精神，对学院2023年选聘的本科生导师进行考核。经导师自评、学生评分及工作领导小组对导师提交的全部培养工作支撑材料复核评分，报请学院党政联席会讨论通过，现将考核结果公示如下（排名不分先后）：

优秀（12人）：

杜俊、余邦伟、贾佳、孟祖东、许雪、阿彩岭、汤继英、梅虹、杨威、付宇、杨建权、万国兴

合格（43人）：

杨芳、李德坤、孙志丰、赵沛誉、陈世满、孙婷、周发明、尚瑞、
王全兵、曲新国、金灵莉、刘经星、李著艳、闫益芬、周春芳、栾兰、叶青（风湿）、吴雪、廖应英、邓娜、刘芳芳、闫洪伟、王国兵、王德利、陈涛、吴三五、何淼、李小丽、解翠、李慧卉、张赛圣、范丽、刘南暖、向阳、苟志斌、胡俊华、熊艳林、刘丹荣、王伟（肝胆）、王伟（肾内）、邓毛、王琰、王强

根据考核结果，对本科生导师进行工作量核算，该类工作量在教学职称晋升时予以认定，不计入年度课时费核算：考核为优秀的导师，按每名导师30学时/年计算教学工作量；考核为合格的导师，按每名导师20学时/年计算教学工作量。

公示时间：2024年月1月20日-1月23日，如有异议，请以书面形式或者电话形式致电第三临床学院教学办公室，联系人：柳金金　电话：0719-8637859　　手机：18086250773

第三临床学院教学办公室

2024年1月20日

湖北医药学院教务处
湖北医药学院药护学院教学管理部

湖北医药学院、湖北医药学院药护学院关于2024年大学生创新创业训练计划拟立项项目通知

各学院：

根据《省教育厅办公室关于做好2024年大学生创新创业训练计划项目立项和结题验收工作的通知》和学校有关要求，各学院积极组织学生进行项目申报，校本部共收到207项申报书,药护共收到63项申报书。公示已结束，现对本部“DMF 琥珀酸化HSP90的表达抑制HIF-1α促进Treg细胞分化缓解溃疡性结肠炎的机制研究”等93个项目，药护“健康中国视域下助产士替代性创伤后成长的现状及影响因素的路径研究”等35个项目予以立项，并择优分别推荐申报国家级、省级大学生创新创业训练计划项目。

附件：湖北医药学院2024年大学生创新创业训练计划项目一览表

湖北医药学院药护学院2024年大学生创新创业训练计划项目一览表

湖北医药学院教务处

湖北医药学院药护学院教学管理部

2024年7月4日

湖北医药学院药护学院2024年大学生创新创业训练计划项目一览表（拟立项35项）

年份	学院	项目编号	项目 级别	项目名称	项目类型	项目负责人姓名	项目负责人学号	项目其他成员信息	指导教师姓名	指导教师职称	支持经费(元)	项目所属专业	项目简介(500字以内)
2024	第三临床学院	202413249001	国家级	“健康中国”视域下助产士替代性创伤后成长的现状及影响因素的路径研究	创新训练项目	吴汶彦	202110032046	王希婷/202110012256,王江兰/202110012276,欧昕荣/202110012277	陈久丽	实验师	10000	1011	采用质性和量性研究相结合的方法对助产士替代性创伤后成长现状调研，完善我市助产士心理干预的提升策略；再采用演绎归纳法提出助产士替代性创伤后成长优化对策，提升我市助产士积极心理体验，更好地推动“健康中国”战略的落实。
2024	第三临床学院	202413249002	国家级	香叶木素改善阿霉素心脏毒性的机制研究	创新训练项目	上官宵悦	202110012266	郭靖/202110012246,廖悦/202110012267,赵欣/202110012262,胡裕/2021103012056	万国兴	讲师	10000	1002	随着阿霉素的广泛使用，其副作用逐渐不可忽视。本项目拟在DIC模型上，探讨香叶木素对DIC的心肌保护作用及机制。本项目若实施，不仅有助于揭示香叶木素拮抗DIC的作用机制，而且为防治阿霉素致心脏损伤提供新的策略和实验依据。
2024	第三临床学院	202413249003X	国家级	月贴悦暖——贴心呵护每一步	创业训练项目	武佳音	202210012221	李琳玲/202203082018,欧阳慧/202313052142,刘金容/202203012022,彭昕玮/202301022096	许毛	讲师	10000	1007	据报道，经期问题仍然困扰许多中国女性，特别是异常严重所占比例仍然很大。痛经贴发展逐年向好，市场渗透率及集中度也在进一步提升。在中国人口基数大的背景下，痛经贴作为一次性用品，每年的消费量十分可观。目前市面上痛经便携产品基本功能单一，也不能根据每个人的具体情况进行协调变动，会让痛经群体在选择的时候产生不同的疑问，进而选择一些不适合自己的产品，进入产品误区。本产品旨在生产更智能化、贴心化，功能强大且价格实惠的痛经便携产品，为消费者带来便利。

湖北医药学院、湖北医药学院药护学院
关于公布2022年大学生创新创业训练计划项目的通知

各学院：

根据《教育部高等教育司关于公布2022年国家级大学生创新创业训练计划项目和重点支持领域项目名单的通知》（教高司函〔2022〕10号）《省教育厅办公室关于做好2022年大学生创新创业训练计划项目申报工作的通知》（鄂教高办函〔2022〕18号）要求，学校组织开展2022年大学生创新创业训练计划项目遴选推荐工作。经各学院推荐、学校评审公示、并报教育部和省教育厅审核通过，我校本部2022年大学生创新创业训练计划立项共162项，其中国家级20项、省级62项、校级80项；药护2022年大学生创新创业训练计划立项共40项,其中国家级5项、省级15项、校级20项。

现将2022年大学生创新创业训练计划立项项目予以公布，请各学院高度重视项目管理，积极为项目提供条件支持，加强过程管理指导，确保项目在规定期限内（自2022年12月12日起至2023年12月11日止）顺利结题，产出成果。同时，要求对2020、2021年大学生创新创业训练计划立项项目进行清理，限期结题（2020年项目务必于2022年12月16日前结题，否则中止该项目）；结题报告分别送学校教务处和提交至国家大学生创新创业训练计划平台（http://gjcxcy.bjtu.edu.cn/）。

专此通知。

附件：

1. 湖北医药学院2022年大学生创新创业训练计划项目

2. 湖北医药学院药护学院2022年大学生创新创业训练计划项目

3. 湖北医药学院国家级大学生创新创业训练计划项目管理办法（修订版）

4. 湖北医药学院大学生创新创业训练计划项目经费使用说明

湖北医药学院教务处

湖北医药学院药护学院教学管理部

2022年12月12日

102	X202210929020	校级	第三临床学院	经腋窝路径腔镜辅助下甲状腺手术	创新训练项目	王涵玉	202003012045	傅楚云/202010012339, 郑常乐/202003012004, 马亦飞/202003012056 毛芳/202003012050	师贞宗	副教授、副主任医师	3000
103	X202210929021	校级	第三临床学院	真实世界胱抑素C/低密度脂蛋白对青年脑卒中的预测价值	创新训练项目	陈致轩	202003012007	潘奕君/202003012036, 李卓旺/202003012015, 王庭龙/202003012002, 唐百川/201901012018	鲍毅	副主任医师	3000
104	X202210929022	校级	第三临床学院	构建贝叶斯网状模型评估不同COVID-19疫苗对正常成年人群有效性及安全性的决策评价	创新训练项目	鲜于晨阳	202003012047	邓念嘉/202003012044, 高腾宇/202003012001, 李慧君/202003012032, 张智煞/202010012242	张超	中级（副主任）	3000
105	X202210929023	校级	第一临床学院	阿司匹林通过PI3K/Akt通路增强去势抵抗性前列腺癌对多西紫杉醇化疗敏感性研究	创新训练项目	刘杨端	202005032012	郑安康/202001082008, 杨轩/202001012033, 刘志一/202005202009, 席林烨/202001012008	凌生涛	副主任医师	3000
106	X202210929024	校级	第三临床学院	反复种植失败患者经基因芯片检测子宫内膜容受性进行个体化移植的临床研究	创新训练项目	杨梦	201903082054	刘玉芳/201903082042, 王泽瑞/201903082001, 李昊耘/201903082009	孙志丰	副主任医师/副教授	3000
107	X202210929025	校级	第三临床学院	低危型妊娠滋养细胞肿瘤的耐药情况及其影响因素探究	创新训练项目	陈芳雨	201803012042	李昊阳/201803012013, 薛莹莹/201913052078	赵晓燕\汪龙	中级	3000
108	X202210929026	校级	生物医学工程学院	基于影像组学模型探讨冠脉周围脂肪衰减系数在冠脉CTA检查阴性病人中预测冠心病的价值	创新训练项目	王若欣	201913352022	唐晓雪/201913052049，覃沛银/202013052050，吴	李鑫玉	助教	3000
109	X202210929027	校级	第一临床学院	植物激素ABA对失重性骨质疏松发生发展的作用及机制研究	创新训练项目	张君怡	202001022035	王田美子/202001022080，郭旭/202001022018，格桑周毛/202103082066,	赵小英	讲师	3000
110	X202210929028X	校级	第一临床学院	运动康复对帕金森病人安装脑起搏器术后状态支持	创业训练项目	王盈	201901022130	吴若彬/201910022086, 马琳欣/202001082032, 李函璐/202010022046, 吴昊/202010022055, 刘帅桐/202010022082	孙强	副主任医师	3000
111	X202210929029	校级	第四临床学院	无创左室压力应变环技术评估高血压患者心肌功能的应用价值研究	创新训练项目	文惠燕	201913052129	黄克鹏/201913052107, 王腾飞/201913052103	谢满英	主治医师	3000
112	X202210929030	校级	第三临床学院	基于ACOG临床指南：构建网状模型系统评估抗高血压药物在妊娠高血压人群中的临床效应	创新训练项目	邓念嘉	202003012044	鲜于晨阳/202003012047, 李慧君/202003012032, 高腾宇/202003012001,	郭冲	副主任\副主任医师	3000
113	X202210929031	校级	口腔医学院	结直肠癌的最佳综合治疗：网状meta分析和证据评价	创新训练项目	王颜	201911072055	付顺康/202010012027, 曹哲豪/202011072020, 陈虹宇/202011072074	孙敏	副主任医师	3000
114	X202210929032	校级	第一临床学院	褪黑素对神经病理性疼痛作用机制探索	创新训练项目	吴语桐	202001022098	黄丽偌/202002022095, 胡俊杰/202001022051	李清	教授	3000
115	X202210929033	校级	第三临床学院	不同免疫联合化疗方案一线治疗晚期食管鳞癌的疗效及安全性比较分析	创新训练项目	杨云天	201903082019	熊英祺/201903082016, 刘亦君/201903082044, 杨桃桃/201903082055	万国兴	讲师	3000

湖北医药学院药护学院2022年大学生创新创业训练计划项目一览表（立项40项）

立项年份	项目编号	项目级别	学院	项目名称	项目类型	项目负责人姓名	项目负责人学号	项目其他成员信息	指导教师姓名	指导教师职称	经费支持（元）
1	202213249001	国家级	第一临床学院	双氢青蒿素通过阻断NRAS通路驱动DNA损伤选择性抗肺癌的研究	创新训练项目	杨子怡	201910022177	李祺睿/201810022113,徐祥/201901022053,　刘	李童斐	副教授	10000
2	202213249002	国家级	第三临床学院	基于网络药理学探讨活心丸干预阿霉素心脏毒性的基础研究	创新训练项目	王广立	202010012257	潘奕君/202003012036,薛翔月/202010012301,	万国兴	讲师	10000
3	202213249003	国家级	第一临床学院	新冠疫情下医学院校大学生在线学习投入影响因素及其对策	创新训练项目	程思锐	202010022113	顾兴萍/202105382050,李诗韵/202110022116,唐俪丹/202110022118,汪	高斌	讲师	10000
4	202213249004	国家级	第一临床学院	标准化病人对医学生人文素养的评价质量研究	创新训练项目	黄焦博坤	202010022008	李孙娜/202010022133,苏雅欣/202010022081,刘	陈森	副教授	10000
5	202213249005	国家级	第四临床学院	机械手套-手功能康复训练器	创新训练项目	苏亚亚	202010012412	吴若彬/201910022086,王良芳/202010012427,郭艺/202010012415,陈平芬	冀丽丽	主治医师	10000
6	S202213249001	省级	第一临床学院	ZNF580抑制原型泡沫病毒双启动子活性的机制研究	创新训练项目	张书欣	201910012036	聂曼,焦怡霖	袁佩佩	讲师	5000
7	S202213249002	省级	第一临床学院	泛耐药解鸟氨酸拉乌尔菌基因组结构分析及 Zn（ Il ）离子对 NDM -1酶分泌影响研究	创新训练项目	严廷苇	201910022130	妥娟/202001082073,刘星宇/202001082050,王沛/202001082051,　周	余春芳/金志雄	讲师/教授	5000
8	S202213249003	省级	第三临床学院	H2O2经FTO/NLRP3/GSDMD-N轴诱导人脑微血管内皮细胞焦亡的机制研究	创新训练项目	张淼	202010012297	雷敏/201910012364,黄腾/202001082081,崔菁菁/202007172034,	刘睿	副教授	5000
9	S202213249004	省级	第一临床学院	叶酸受体（FRα）调控Wnt/β-catenin信号促进喉癌发生发展的机制研究	创新训练项目	刘任浩	201910012016	刘向鹏/201910012014,屈林	赵虎子	讲师	5000
10	S202213249005	省级	第五临床学院	围绝经期综合征中西医治疗的对比研究	创新训练项目	陈丽羊	201910012565	李耀科/201910012539,刘家惠/201910012564,	周权	副主任医师	5000