

药剂学学科简介

我校药剂学学科始建于 1973 年，2003 年获药剂学硕士学位授予权，2006 年遴选为省级重点学科，2012 年药剂学教学团队遴选为省级教学团队，2017 年药剂学课程获得贵州省一流课程建设项目，2018 年药剂学教师团队获“贵州医科大学黄大年式教学团队”荣誉称号。

本学科涵盖药剂学教研室、药物制剂、制剂工程学、中药药剂学四个专业教研室，承担本专科及研究生《药剂学》、《工业药剂学》、《药用高分子材料学》、《制剂机械设备与车间工艺设计》、《化工原理》、《中药药剂学》等 7 门本科生专业主干课程、以及选修课《药物制剂设计与研发》、《中药药剂学进展》、《临床药剂学》等课程的教学工作，同时承担研究生《现代药剂学》、《高等生物药剂学与药物动力学》、《制剂新技术与新工艺实验技术》等专业课程的教学。

二、师资队伍

学科现有专任教师 25 人，其中高级职称教师 15 人，博士生导师 2 人，硕士、博士学位以上 23 人，占教师总数的 92%，平均年龄 36 岁。先后承担省级教改项目 2 项，校级教改项目 13 项。获省级教学成果二等奖 1 项，发表教改论文 4 篇。先后参编国家规划教材 7 部；教师获校级教学竞赛奖 2 项，团队教师指导的本科毕业论文共 20 人次获得校级优秀论文。

三、药剂学学科主要研究方向及特色

药剂学学科主要研究方向有药物制剂新技术、新剂型与新工艺研究；基础药物动力学研究；中药药代动力学研究、中药民族药药效物质基础研究；中药民族药新药研发等。2002 年以来主持和承担国家重大新药创制专项、国家自然科学基金、国家科技支撑计划和省重大专项及企业合作项目等 200 余项，获经费 6000 余万元。获得科研奖项共 21 项，包括“国家专利技术发明”二等奖、“贵州省科技进步奖”一等奖、“贵州省医学会科技奖”。获得或申请发明专利 30 余项，研究成果达国内领先水平。发表论文 400 余篇，其中 SCI 收录 33 篇，出版专著、教材 3 部。

四、教学建设与改革

先后承担省级、校级教改项目 13 项，现建设有《中药药剂学》、《中药制剂分析》网络精品课，正在建设《药剂学》校级网络课程、《药剂学实验》、《中

药药剂学》微课、《中药药剂学进展》网络精品课，发表教改论文 4 篇。2013 年“现代药物制剂 GMP 实验教学中心建设与培养创新型药学人才”获得省级教学成果二等奖，2017 年药剂学课程获得贵州省一流课程建设项目。

五、研究生培养

药剂学硕士点于 2003 年经国务院学位委员会批准设立，是药学院最早设置的专业硕士点，累计培养统招硕士研究生 300 余人。

药物化学学科介绍

药物化学学科由药物化学教研室、有机化学教研室及天然药物化学教研室构成。本学科明确提出了“紧紧抓住学科优势特色，努力探索新药创制规律，积极推动产学研合作”的教学与科研工作模式。

本学科现有在编教师 19 人，其中博士 12 人，教授 6 人，博士生导师 2 人，硕士生导师 17 人，教师中博士学位比率达到 63.1%，高级职称比率为 84.2%。本学科目前有教育部新世纪优秀人才 1 人，贵州省省管专家 1 人，贵州省高层次创新型人才（百层次）2 人，贵州省优秀青年科技人才 4 人，国家自然科学基金委项目函评专家 3 人，中组部“西部之光”访问学者 2 人，贵州省优秀教师 2 人，药学专业本科“十三五”规划教材副主编 2 人，贵州省科技厅项目评审专家 4 人。

本学科主要承担本科药学专业、药物制剂专业、中药学专业、药事管理专业、生物技术专业以及化学生物学专业的《药物化学》、《临床药物化学》、《天然药物化学》、《波谱解析》《有机化学》等课程教学工作。同时本学科还承担了药物化学专业硕士研究生《药物分子设计与合成》、《高等天然药物化学》课程的教学任务。承担过省级教改项目 2 项，校级教改项目 8 项。在《西部素质教育》、《药学教育》等发表多篇教学论文。近五年指导的本科生获得校级优秀论文奖 15 人次，研究生获得国家奖学金、省级与校级优秀毕业论文奖 12 人次。

药物化学学科组目前已形成了抗 2 型糖尿病活性化合物的结构优化；基于主动转运机理的黄酮类天然活性成分的结构修饰及其构效关系研究；活性天然产物、重要治疗靶点蛋白的化学生物学研究；UPLC- PDA-Q-TOF 等技术应用于天然化合物及其代谢产物、内源性代谢物的结构鉴定；以及天然活性有机分子不对称全合成研究等几个相对固定的研究领域。近年来药物化学学科组共主持或完成国家级项目十余项，省级项目 44 项，经费共 1000 余万元；教师发表 SCI 收录与核心期刊论文 180 余篇；为省内外企业完成了 20 余个化学药的技术开发工作，获得生产批件 2 个，受理通知书 1 个；以第一或第二发明人申请或获得发明专利 36 项，获得“贵州省科学技术进步奖”三等奖 3 项。

近年来，药物化学学科老师与美国康奈尔大学，美国加州大学戴维斯分校，美国明尼苏达大学，美国 Proteom Tech 公司以及法国巴黎 6 大等科研院所建立了学术交流与科研协作关系。学科组老师多次在国际和国内学术研讨会上做大会报告或提交研究工作摘要。

一、师资成员代表性照片

（药物化学教研室）



（有机化学教研室）



(天然药物化学教研室)



二、主持的代表性科研项目

序号	项目、课题名称 (下达编号)	项目来源	项目起讫时间 (年)	科研经费 (万元)	负责人 (姓名、专业技术职务)
1	基于黄嘌呤氧化酶抑制作用的金雀花根抗痛风物质基础及其构效关系研究 (81860690)	国家自然科学基金	2019-2022	34.0	廖尚高教授
2	木豆源芪类化合物及其衍生物靶向 PKM2 抗癌的作用和	国家自然科学基金	2019-2022	35.0	张嫩玲副教授

	机制研究 (20962004)				
3	基于代谢组学的海州常山抗人白血病 K562 细胞增殖的药效物质基础及机制研究 (81860689)	国家自然科学基金	2019-2022	35.0	李林珍副教授
4	基于群体行为控制的鲍曼不动杆菌生物膜抑制剂的设计、合成及其构效关系研究 (81660348)	国家自然科学基金	2017-2020	37.0	杨元勇副教授
5	3-氧吡啶酮类化合物参与的有机催化不对称串联反应研究 (21502030)	国家自然科学基金	2016-2018	25.2	赵永龙副教授
6	头花蓼内生菌抗尿路感染耐药菌化学小分子的发现及其作用机制研究 (81560570)	国家自然科学基金	2016-2019	35.0	徐国波副教授
7	基于肝靶向性转运机制的阿德福韦单 L-氨基酸酯, 单胆酸酯衍生物的设计、合成与生物活性研究 (81460523)	国家自然科学基金	2015-2018	45.0	傅晓钟教授

三、代表性获奖成果

序号	成果名称	项目完成人 (排名)	获奖时间	获奖等级和鉴定单位
1	莲菊感冒胶囊中药新药的研究开发	李勇军(1)	2015 年	贵州省科学技术进步三等奖
2	20 种贵州民族药地	李勇军(1)	2013 年	贵州省科学技术进步三

	标升国标的研究及应用			等奖
3	“参芎葡萄糖注射液”技术成果转化	李勇军(1)	2015年	贵州省科技转化一等奖
4	抗2型糖尿病胰岛素增敏剂的研究	汤磊(1)	2010年	贵州省科学技术进步三等奖
5	具胰岛素增敏活性的芳环羧酸类化合物的研究	汤磊(1)	2014年	贵州省高等学校科学研究优秀成果三等奖
6	阿德福韦双L-氨基酸酯与葫芦脲的主客体化学与抗乙型肝炎病毒活性研究	傅晓钟(1)	2011年	贵州省首届优秀医学学术论文三等奖
7	20种贵州民族药地标升国标的研究及应用	李勇军(1)	2012年	贵阳市科学技术二等奖

获奖证书照片





四、学术交流照片





药物分析学科简介

贵州医科大学药学院药物分析学科始建于1973年，是我省高校最早建立的培养高级药学人才的学科之一，学科现由药物分析教研室、分析化学教研室及无机化学与物理化学教研室组成。2008年，学科主干课程《分析化学》遴选为贵州省省级精品课程。2011年，药物分析学二级学科硕士点获批招生。2014年，药物分析学科被遴选为贵阳医学院第三批校级重点学科。

学科现有专职教师19人，其中教授2人，副教授10人，讲师7人，博士6人，硕士8人。高级职称教师比例为63%，硕博率74%，平均年龄42岁。教师兼任国家自然科学基金项目函评专家，教育部学位与研究生教育评估专家，贵州省药学高级职务评审专家、贵州省科技厅基金项目评审专家、《Journal of

Ethnopharmacology》、《中国药学杂志》等期刊的审稿专家。

学科带头人高秀丽，教授，博士研究生导师，国家化妆品评审专家、贵州省科技项目评审专家。任贵州省药学会和贵州省出国留学欧美联合会常务理事，贵州省药学会药物分析专业委员会主任委员。主要从事贵州特色资源的开发应用、中药民族药的开发、中药药效物质基础研究及药代动力学研究。主持完成国家级及省级科研课题 30 余项，获发明专利 8 项，发表论文 80 余篇。科研成果获贵州省首届科技论文二等奖、市科技进步二等奖，与多家生物医药企业及健康食品企业建立了“产-学-研”合作平台。所带领的科研团队曾获贵州省委组织部等联合颁发的“留学报国·服务贵州基地”授牌。

本学科主要承担本科各专业的《药物分析》、《分析化学》、《物理化学》、《无机化学》及硕士研究生的《药物分析选论》、《现代仪器分析》等 11 门专业主干课程的授课任务。获校级教学成果二等奖 1 项，三等奖 2 项。在《药学教育》等杂志发表教学研究论文 8 篇。获国家级教学质量工程项目 1 项，省级教学质量工程项目 3 项。参编国家级规划教材 14 部。学科教师获校级教学工作先进个人、优秀教师、本科优秀论文指导教师奖、讲课竞赛、多媒体课件评比等多项教学奖项和荣誉称号。

学科现已形成中药、民族药的有效成分研究与新药开发，体内药物分析与药物代谢动力学研究，中药药效物质基础及其质量控制技术研究等稳定的研究方向。近五年来，主持国家自然科学基金、贵州省中药现代化专项基金等科研项目 34 项。获贵州省科技进步奖三等奖 1 项、贵阳市科技进步奖二等奖 1 项，贵阳市优秀新产品一等奖 1 项，获发明专利 4 项。在国内外学术刊物发表论文 107 篇，被 SCI 收录 11 篇，出版学术专著 3 部。

教材与专著





教学论文



科研成果



教师荣誉



生药学/中药学学科简介

我校生药学/中药学学科前身为贵阳医学院中草药教研室,始建于1973年,经过四十五年的不懈努力与发展,于2011年获得生药学二级学科硕士点,2013年获得目录外二级学科博士点——民族药药理学博士点。目前学科点拥有专职人员15名,已经形成了一支年龄结构合理,研究方向稳定的教学、科研团队。本学科现由“三室一馆二重点实验室和一研究中心”组成,3室即药用植物学与生药学教研室、中药学教研室、中药药理学教研室,主要承担教学和科研工作。1馆即中药民族药标本馆,主要承担标本馆的管理、维护、教学、科研及对外交流与合作等。二重点实验室即天然药物药理及成药性评价重点实验室、天然药物资源优效利用重点实验室。一研究中心即贵州特色天然药物资源高效利用工程研究中心。

生药学/中药学学科承担本科、硕士、博士及博士后各级人才的培养任务,45年来立足地方培养医药卫生人才,服务地方经济方面,为贵州各级医药卫生人才的可持续发展做出应有的贡献。围绕贵州区域地产特色中药民族药资源,不断凝练学科内涵,形成4个明确的研究方向:方向一中药民族药效应机制及安全性评价、方向二天然药物质量控制技术及天然药物活性成分研究、方向三民族药资源品种品质研究、方向四中药民族药新剂型与新技术研究。主要研究运用现代天然药物分离制备技术、细胞生物学、分子生物学的技术,系统研究贵州地产民族药物资源道地性、药效物质基础、效应毒性机制及成药性研究,真正解决民族药资源合理配置及拥有自主知识产权创新药物的关键科学问题,提升我国新药创制的关键核心问题和能力,实现民族药产业结构优化升级和转变经济发展方式的战略任务,有利于助推我省中药民族药现代化产业经济的快速增长,助推我省经济、社会发展。

临床药学学科简介

临床药学是从药学中分离出来的科学分支，是以病人为对象，以提高临床用药质量为目的，以药物与机体相互作用为核心，研究和实践药物临床合理应用方法的综合性应用技术学科。临床药学处于创新药物研发链的末端，是评价新药临床安全性和有效性的重要阶段，符合目前精准医疗发展的需求。

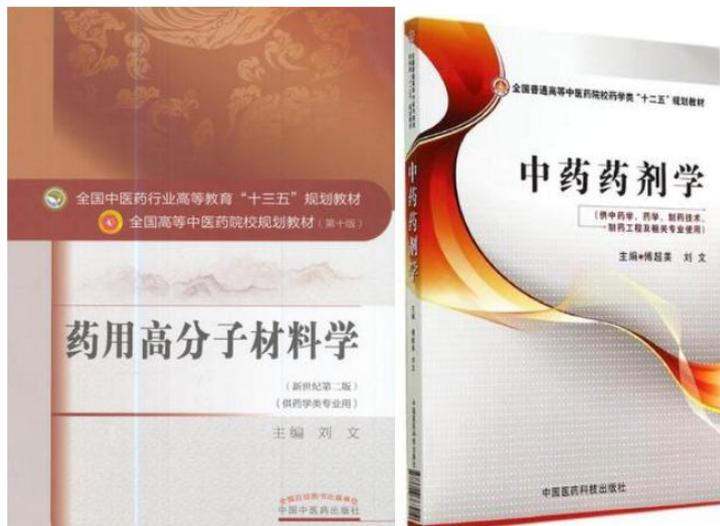
贵州医科大学药学院临床药学学科由临床药学教研室、微生物与生化药学教研室及生物药剂学与药物代谢动力学教研室组成。学科拥有临床药学和微生物与生化药学两个二级学科硕士授予点，目前已有硕士毕业生 6 人。本学科目前有专职教师 10 名，兼职教师 10 人，其中教授 2 名、副教授 9 人、助教 2 人，主任药师 5 人，副主任药师 2 人。具有博士学位 7 人，硕士学位 10 人。本学科承担的课程有《临床药学导论》、《临床药物治疗学》、《临床药理学》、《临床药物动力学》、《医院药事管理学》、《生物药剂学与药物动力学》、《生物技术制药》、《微生物制药》、《基因工程药物》、《药物制剂专业导论》等，授课对象包括硕士研究生及本科生两个层次。

学科带头人刘文，教授，博士研究生导师。现为省人民政府督学，省级教学名师，省教学指导委员会副主任委员，全国高等中医药院校规划教材委员会副主任委员，教育部审核评估专家。先后主持省级质量工程：《教学质量保障与监控体系的研究及实践》等项目的研究工作，主持参与省部级科研项目 20 余项，其中，主持国家自然科学基金 3 项，省创新群体重大项目、省优秀青年科技人才选拔项目各 1 项。发表教研教改论文 31 篇，行业规划教材主编 5 部、副主编 3 部。获得省教学成果一等奖 2 项，省教学成果二、三等奖各 1 项。编写论著 4 部，发表学术论文 150 余篇，获省科技进步三等奖 4 项、贵州省医学会医学科技二等奖 1 项，培养硕士研究生 32 人。

临床药学学科现已形成心血管药理学、临床药学、感染与免疫、中药民族药药理研究、生物药剂学与药物动力学等研究方向。近年来，学科教师主持国家自然科学基金数项、省市级科研项目十余项。获贵州省医学科技奖一等奖 1 项、贵阳市科技进步奖三等奖 2 项，贵州省高等学校科学研究优秀成果奖三等奖 1 项，获发明专利数项，在国内外学术刊物发表 SCI 及核心期刊论文数十篇。



参编教材



科研论文

Oxymatrine Inhibits Homocysteine-Mediated Autophagy via MIF/mTOR Signaling in Human Umbilical Vein Endothelial Cells

Yan-Yan Zhang¹, Yuan Zhang^{2*}, Jia-Yu Tang³, Shuang Zhu⁴, Chen Li⁵, Yongqian Huang⁶, Minhan Yin⁶

¹Key Laboratory of Optimal Medication of Natural Medicine Beiluo, Guizhou Medical University, Guizhou, China; ²Department of Neurology, Nanjing Hospital, Central South University, Changsha, Hunan, China; ³Department of Neurology, University of Michigan, Ann Arbor, MI, USA; ⁴Department of Neurology, Brain Hospital of Hunan Province, Chengde, Hunan, China; ⁵Department of Pharmacology, Institute of Chinese Medicine, Hunan Academy of Chinese Medicine, Changsha, Hunan, China; ⁶Department of Ecology and Evolutionary Biology, University of Michigan, Ann Arbor, MI, USA

Key Words: Oxymatrine • Homocysteine • Autophagy • Apoptosis • HUVEC

Abstract: Oxymatrine (Oxm) or natural alkaloids in homocysteine (Hcy)-induced endothelial cells, which is an independent risk factor for cardiovascular disease. Oxm has shown that Hcy leads to endothelial dysfunction, a hallmark of atherosclerosis, which may explain this fact. The precise mechanism remains unclear, but a growing possibility is excessive Hcy-induced autophagy. Autophagy has been better studied in ischemic reperfusion (IR) injury, and previous work showed that Oxymatrine (Oxm) is a protective agent against myocardial IR injury by inhibiting autophagy. The aim of this study was to determine whether Oxm inhibits autophagy in HUVEC. Autophagy in HUVEC cells treated with Hcy to the presence and absence of Oxm was visualized by immunofluorescence microscopy and the degree was determined by western blotting and GFP-LC3. Small interfering RNA (siRNA) was used to determine the efficiency of Oxymatrine (Oxm) inhibition. Cell apoptosis was detected by western blotting and flow cytometry analysis. Results: Oxm inhibited autophagy, and Oxm + Hcy cells showed the opposite. Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Keywords: Oxymatrine • Homocysteine • Autophagy • Apoptosis • HUVEC

Background/Aims: Oxymatrine (Oxm) or natural alkaloids in homocysteine (Hcy)-induced endothelial cells, which is an independent risk factor for cardiovascular disease. Oxm has shown that Hcy leads to endothelial dysfunction, a hallmark of atherosclerosis, which may explain this fact. The precise mechanism remains unclear, but a growing possibility is excessive Hcy-induced autophagy. Autophagy has been better studied in ischemic reperfusion (IR) injury, and previous work showed that Oxymatrine (Oxm) is a protective agent against myocardial IR injury by inhibiting autophagy. The aim of this study was to determine whether Oxm inhibits autophagy in HUVEC. Autophagy in HUVEC cells treated with Hcy to the presence and absence of Oxm was visualized by immunofluorescence microscopy and the degree was determined by western blotting and GFP-LC3. Small interfering RNA (siRNA) was used to determine the efficiency of Oxymatrine (Oxm) inhibition. Cell apoptosis was detected by western blotting and flow cytometry analysis. Results: Oxm inhibited autophagy, and Oxm + Hcy cells showed the opposite. Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Cellular Physiology and Biochemistry

DOI: 10.1159/000518101
Published online: September 21, 2017
Accepted: July 18, 2017

© 2017 The Author(s)
Published by S. Karger AG, Basel
www.karger.com/cpb

This article is intended solely for the personal use of the individual user and is not to be disseminated broadly.

Original Paper

Oxymatrine Ameliorates Doxorubicin-Induced Cardiotoxicity in Rats

Yan-Yan Zhang¹, Minhan Yin^{2*}, Yongqian Huang³

¹Department of Pharmacology, Guizhou Medical University, Guizhou, China; ²Information Security and Big Data Research Institute & School of Life Sciences, Central South University, Changsha, Hunan, China; ³Department of Ecology and Evolutionary Biology, University of Michigan, Ann Arbor, MI, USA

Key Words: Oxymatrine • Doxorubicin • Cardiotoxicity • Apoptosis • Oxidative stress

Abstract: Doxorubicin-induced cardiac toxicity has been a major concern of oncologists and is considered the main restriction on its clinical application. Oxymatrine has shown potent anti-cancer, anti-fibrosis, and anti-oxidative effects. Recently, it has been reported that oxymatrine is protective against some cardiovascular diseases. In this study, we aimed to

Background/Aims: Doxorubicin-induced cardiac toxicity has been a major concern of oncologists and is considered the main restriction on its clinical application. Oxymatrine has shown potent anti-cancer, anti-fibrosis, and anti-oxidative effects. Recently, it has been reported that oxymatrine is protective against some cardiovascular diseases. In this study, we aimed to

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Food and Chemical Toxicology

Contents lists available at ScienceDirect
journal homepage: www.elsevier.com/locate/foodchemtox

Food and Chemical Toxicology

Volume 118, 626–635 (2018)

Available online 14 May 2018

Article history:
Received 29 February 2018
Received in revised form 12 April 2018
Accepted 8 May 2018
Available online 14 May 2018

(-)-Epigallocatechin-3-gallate (EGCG) attenuates arsenic-induced cardiotoxicity in rats

Tao-Li Sun^{a,1}, Zhi Liu^{b,c,d,1}, Zheng-Jun Qi^a, Yong-Pan Huang^{b,h,i}, Xiao-Qin Gao^h, Yan-Yan Zhang^{h,i}

^aDepartment of Pharmacology, Shanghai College, Shanghai, Hunan, 431004, China; ^bDepartment of Pharmacology, Guizhou Medical University, Guiyang, Guizhou, 550004, China; ^cDepartment of Ecology and Evolutionary Biology, The Second Affiliated Hospital of Guizhou Medical University, Guiyang, Guizhou, 550004, China; ^dDepartment of Histology and Embryology, Guizhou Medical University, Guiyang, Guizhou, 550004, China; ^eDepartment of Pharmacology, Central South University, Changsha, Hunan, 410078, China

Abstract: Chronic arsenic exposure in drinking water is associated with the abnormalities of cardiac tissue. Excessive generation of ROS induced by arsenic has a central role in arsenic-induced cardiotoxicity. (-)-Epigallocatechin-3-gallate (EGCG), the most abundant polyphenol in green tea, possesses a potent antioxidant capacity and exhibits extensive pharmacological activities. This study was aimed to evaluate the effect of EGCG on arsenic-induced cardiotoxicity in vivo and in vitro. Treatment with NaAsO₂ seriously affected the morphology and ultrastructure of myocardial cells, induced cardiac injury, oxidative stress, intracellular calcium accumulation and apoptosis in rats. In consistent with in vivo study, the injuries, oxidative stress and apoptosis were also observed in NaAsO₂-treated H9c2 cells. All of these effects induced by NaAsO₂ were attenuated by EGCG. These results suggest EGCG could attenuate NaAsO₂-induced cardiotoxicity, and the mechanism may involve its potent antioxidant capacity.

Keywords: Arsenic; NaAsO₂; (-)-Epigallocatechin-3-gallate (EGCG); Cardiotoxicity; Oxidative stress

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

Conclusion: Oxymatrine (Oxm) inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells. In conclusion, Oxm inhibited autophagy and apoptosis in HUVEC cells.

教师荣誉

