2026 肿瘤防治新模式国际会议

2026 International Conference on New Models for Cancer Prevention and Treatment

NMCPT 2026

会 议 日 程

Conference Program

2026 年 4 月 17-19 日 | 上海（青松城大酒店）

April 17-19, 2026 | Pine City Hotel, Shanghai, China

会议网站 | Website: https://www.nmcpt.com

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大会简介 | 2026 肿瘤防治新模式国际会议

2026 International Conference on New Models for Cancer Prevention and Treatment

2026 肿瘤防治新模式国际会议（NMCPT 2026）将于 2026 年 4 月 17 日至 19 日在上海青松城大酒店举办。

本次会议由复旦大学公共卫生学院、复旦大学营养研究院（筹）、新疆塔里木大学医学院、乐博医疗（LaboHeal）联合主办，智研医学承办，上海市肿瘤研究所、湖州师范大学协办，佰泰科技有限公司、赛默飞世尔科技（中国）有限公司赞助支持。会议以“肿瘤防治新模式”为议题，旨在打造一个汇聚全球临床医学专家、肿瘤学研究学者和医药科技代表的学术平台，深入探讨癌症精准诊疗、免疫治疗及生物标志物研究等领域的最新突破与发展趋势。

大会设置特邀报告、口头汇报、学术墙报展示等环节，与会者将有机会聆听来自世界各地专家的主题演讲，参与探讨，以及展示自己的研究成果和创新技术。

诚邀各位同仁齐聚上海，共话癌症防治新未来、共促医学领域新发展！

Introduction | 2026 肿瘤防治新模式国际会议

2026 International Conference on New Models for Cancer Prevention and Treatment

2026 International Conference on New Models for Cancer Prevention and Treatment (NMCPT 2026) will be held during April 17-19, 2026 at the Pine City Hotel in Shanghai, China.

This conference is jointly hosted by the School of Public Health, Fudan University; the Institute of Nutrition, Fudan University; the Medical College, Tarim University; LaboHeal. It is Organized by Zhiyan Med, supported by Shanghai Cancer Institute, Huzhou University, and sponsored by BIOTIMES, Thermo Fisher Scientific Inc.

Under the theme of "New Models for Cancer Prevention and Treatment", the conference will bring together leading experts and scholars from around the world for high-level academic exchanges and the presentation of the latest scientific research.

We look forward to meeting you in Shanghai to discuss the future of cancer prevention and treatment, and to jointly promote new developments in the medical field!

主办单位

复旦大学公共卫生学院

复旦大学营养研究院（筹）

新疆塔里木大学医学院

乐博医疗（LaboHeal）

承办单位

智研医学

协办单位

上海市肿瘤研究所

湖州师范大学

赞助单位

佰泰科技有限公司

赛默飞世尔科技（中国）有限公司

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大会主席（排名不分先后）

Nagy Habib，伦敦帝国理工学院肝胆外科教授、伦敦帝国理工学院商务事务副校长、法国国家外科医学院外籍院士

陈伟，西交利物浦大学芯片学院院长&教授、美国国家发明家科学院院士

Pier Paolo Piccaluga，意大利博洛尼亚大学副教授

组织委员会主席（排名不分先后）

何纳，复旦大学公共卫生学院院长&教授

高翔，复旦大学营养研究院（筹）院长&教授

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陶灵，复旦大学公共卫生学院和营养研究院（筹）青年研究员

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韩书文，新疆塔里木大学南疆中医药研究重点实验室主任

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倪以成，乐博医疗首席肿瘤专家、长江讲席教授、东南大学附属中大医院教授

符立梧，中山大学肿瘤防治中心职代会常设委员会主任&教授

董培新，日本北海道大学医学研究院首席研究员

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Organization | 2026 肿瘤防治新模式国际会议

2026 International Conference on New Models for Cancer Prevention and Treatment

Hosted by

School of Public Health, Fudan University

Institute of Nutrition, Fudan University

Medical College, Tarim University

LaboHeal

Organized by

Zhiyan Med

Supported by

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Huzhou University

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Zhiling Yu, Hong Kong Baptist University, China

Zulqarnain Baloch, Kunming University of Science and Technology, China

陈伟

西交利物浦大学芯片学院院长&教授、美国国家发明家科学院院士、英国皇家化学学会会士、美国 Sigma Xi 科学联合会国际专员、德州大学阿灵顿分校杰出学者学院院士。

研究领域：智能芯片、传感技术、新型半导体材料、可穿戴技术、糖尿病及早期癌症的检测与预防。

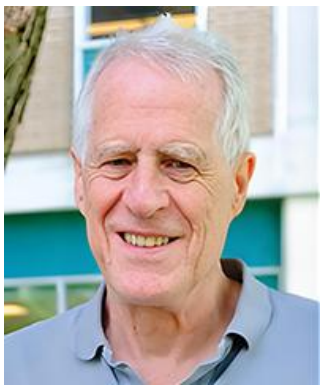
陈伟教授曾担任中国科学院半导体材料研发实验室副主任，荣获“中国科学院青年科学家奖”。他长期致力于纳米技术前沿研究，在《先进材料》、《纳米快报》国际知名学术期刊发表论文 355 余篇，论文总被引次数达 22372 次，h 指数为 76，持有 22 项美国专利。在 2024 年度“全球前 2%顶尖科学家”榜单中，他同时入选“年度科学影响力榜单”与“终身科学影响力榜单”。

倪以成

乐博医疗首席肿瘤专家、长江讲席教授、东南大学附属中大医院放射科教授。

研究领域：图像引导诊断与治疗整合、人工智能医学成像、肿瘤和癌症治疗、医疗机器人、靶向治疗、医学成像和癌症建模。

倪以成教授 1995 年在比利时鲁汶大学(全球 Top50 名校)获得博士学位后破格留校，并获聘为终身教授，创立并长期担任鲁汶大学“诊疗一体化研究室"主任，直至 2022 年荣誉退休。2023 年，倪教授入选教育部长江讲席教授计划，全职受聘于东南大学附属中大医院。曾主持欧盟科研项目及中国国家自然科学基金项目、发表 SCI 论文及专著数百篇，拥有十余项国际授权专利，累计获得数十项国内外奖项，是医学影像领域 Herbert M. Staufer Award 和造影剂领域 Elliott Laser Award 的全球唯一双奖均获者。入选全球前 2%顶尖科学家榜单，并被国际权威 KOL KevOoinipnLeaders)机构列为专项榜首。



Michael Thompson

加拿大多伦多大学化学系教授、多伦多大学生物芯片与生物工程中心主任、加拿大皇家学会院士。

研究领域：生物分析化学、临床诊断、药物发现、纳米技术、分子成像、系统生物学。

Michael Thompson 博士毕业于加拿大麦克马斯特大学，在加拿大多伦多大学担任生物分析化学教授前，曾任职于斯旺西大学和拉夫堡大学。多年来在新型生物传感器技术以及生化和生物实体表面化学研究领域的开创性工作得到了国际认可。为利用超高频声波物理学进行生物大分子相互作用和细胞表面行为的非标记检测做出了重大贡献。率先开发了防污表面改性，特别是抗血栓和抗菌粘附材料。1999 年被任命为加拿大皇家学会院士。其研究获得了许多著名的国际奖项，包括皇家化学学会的罗伯特·博伊尔金奖、加拿大化学学会的 E. W. R. Steacie 奖。

禹志领

香港浸会大学中医药学院教授、博士生导师。

研究领域：中药药理、抗癌、抗炎、饮片质量控制。

禹教授在香港科技大学获生物化学博士学位。在加入香港浸会大学之前，禹教授曾先后任职于中国药科大学、日本长崎大学、香港科技大学、厦门大学及澳门大学。禹教授以中药药理学与中药材炮制研究闻名，已发表 200 余篇经同行评审的研究论文。在香港浸会大学，康臣药业集团有限公司与其团队合作成立了"康臣中药肾脏病研究中心"，珍卡儿药妆有限公司与其团队共建"珍卡儿中药透皮治疗实验室"。禹教授担任《时珍国医国药》《BMC 补充医学与治疗》《当代营养保健品研究》《替代、补充与整合医学杂志》《药理学研究》等 30 余本期刊的编委/副主编/执行主编/顾问编辑。

Andrew Teschendorff

中国科学院上海营养与健康研究所教授。

研究领域：统计癌症表观遗传学、癌症系统生物学。

Teschendorff 于 2001 年至 2003 年加入华威大学数学生物学组担任研究助理。2003 年至 2008 年，他在剑桥大学乳腺癌功能基因组学实验室从事博士后研究，并担任计算生物学高级博士后研究员。2008 年，他加入伦敦大学学院，历任统计癌症基因组学首席研究助理（至 2013 年），并于 2015 年至 2018 年担任牛顿高级学者。2013 年至 2020 年，他在中国科学院-马克斯·普朗克学会计算生物学伙伴研究所（上海）担任计算系统表观基因组学教授。自 2020 年起，他任职于中国科学院计算生物学重点实验室（上海），担任教授及首席研究员，并于 2015 年获授伦敦大学学院荣誉研究员称号。

Ahmed G. Hegazi

埃及国家研究中心微生物学和免疫学教授、埃及科学研究技术院及国家微生物科学委员会主席。

研究领域：微生物学、免疫学、天然药物功效。

自 2018 年起担任埃及科学研究技术院及国家微生物科学委员会主席、埃及科学研究技术院专业委员会成员，并于 2023 年成为世界中医药学会联合会蜂疗专业委员会及中国中医药学会联合会成员。自 1977 年至今，他持续组织并参与国内外研究项目，并作为国家研究中心多个研究项目的首席研究员。于 1979 年在国家研究中心开设临床微生物学培训课程，奠定了教学基础。其学术成果丰硕，在国内外科学期刊发表 256 篇文章，担任 48 个期刊的编辑、98 个国际期刊的审稿人，并组织 196 次国际研讨会和会议，展现了卓越的学术影响力。

Dipayan Rudra

上海科技大学副教授。

研究领域：免疫耐受、自身免疫与炎症、调节性 T 细胞、肿瘤免疫学。

Dipayan Rudra 于 2005 年毕业于阿尔伯特·爱因斯坦医学院，获博士学位。2006 年至 2008 年，在华盛顿大学担任博士后研究员；现任上海科技大学生命科学与技术学院副教授、研究员、博士研究生导师。其主要研究方向包括免疫耐受、自身免疫与炎症、调节性 T 细胞及肿瘤免疫学。他在国际学术期刊上发表数十篇论文，并担任多家国际权威期刊审稿人。现任《科学报告》编委，曾担任《国际免疫学评论》特约编辑。

Saman Hosseinkhani

伊朗塔比阿特莫达雷斯大学教授。

研究领域：构建基于荧光素酶的新型生物传感器，以靶向多种癌症模型中的蛋白质复合物。

Saman Hosseinkhani 是伊朗塔比亚特莫达雷斯大学生物化学教授，同时也是伊朗科学院的成员。他的研究方向专注于萤火虫荧光素酶的蛋白质工程，将其开发为监测生物过程的报告基因。近期，他致力于构建基于荧光素酶的新型生物传感器，以靶向多种癌症模型中的蛋白质复合物。凭借其专业成就，他担任国际生物发光与化学发光学会科学顾问委员会委员。他的一项重要创新成果是设计了用于基因治疗的“肽模拟结构”（peptideticle），并通过荧光素酶检测技术验证其功能。该研究为基于肽的 DNA 癌症疫苗开发提供了新思路。Hosseinkhani 教授发表论文成果丰硕，累计发表国际同行评审论文 350 余篇（h 指数为 57）、综述文章 7 篇，并参与 6 部著作的撰写。

张凯

上海市肿瘤研究所副研究员。

研究领域：恶性进展型前列腺癌的细胞谱系可塑性调控、恶性进展型前列腺癌的免疫抑制性微环境、神经内分泌前列腺癌的远端转移机制。

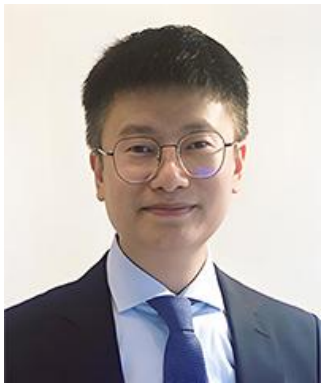
张凯博士，副研究员，硕士生导师。毕业于上海交通大学生物医学工程学院。先后在美国贝勒医学院（Baylor College of Medicine, Houston）、美国华盛顿大学（University of Washington, Seattle）医学院接受博士后训练。回国后在上海交通大学医学院附属仁济医院/上海市肿瘤研究所任副研究员，研究方向为：癌症的免疫抑制、治疗抵抗、远端转移的机制和靶点。

Christopher L. Antos

上海科技大学生命科学与技术学院副教授。

研究领域：组织再生、免疫学、发育生物学、再生疗法。

Christopher L. Antos 博士于 1993 年在印第安纳大学布卢明顿分校获得学士学位。随后他获得富布赖特奖学金前往德国弗莱堡，在马克斯·普朗克免疫学研究所 Rolf Kemler 博士的实验室进行研究。1996 年，Antos 博士在得克萨斯大学西南医学中心 Eric Olson 博士的实验室开始博士研究。完成博士学位后，他于 2003 年至 2007 年间在德国杜宾根的马克斯·普朗克发育生物学研究所 Christiane Nüsslein-Volhard 博士的实验室完成博士后培训。2008 年，Antos 博士在德累斯顿工业大学的德累斯顿再生治疗中心正式建立自己的独立实验室。2016 年 12 月，Antos 博士加入上海科技大学担任副教授职务。



苏华

复旦大学生物医学研究院研究员。

研究领域：肿瘤微环境与代谢、巨胞饮作用启动机制、胶原蛋白在肿瘤发展和肿瘤免疫中的作用、促纤维增生性肿瘤的肿瘤发生和肿瘤代谢。

浙江大学生物化学与分子生物学系博士，并曾在美国加州大学从事博士后研究。目前，他是复旦大学生物医学研究院的首席研究员，同时也是复旦大学附属中山徐汇医院的首席研究员。2022 年建立了专注于肿瘤微环境和肿瘤代谢的研究实验室。现任兽医传染病前沿的客座副主编，同时也是美国癌症研究协会、美国胰腺协会和每周华人生物科学学会的成员。曾荣获多项奖项，包括国家奖学金（2007 年、2015 年、2016 年）、罗氏教育卓越科研奖（2017 年）以及 2022 年上海市领军人才项目。曾获 2022 年国家自然科学基金优秀青年项目资助。

何锋

上海中医药大学中西医结合研究院研究员。

研究领域：细胞应急反应对机体免疫和代谢平衡的调节及其在肝炎和肝癌发生发展中的作用，以及寻找肝炎和肝癌预防，诊断和治疗的新方法。

何锋，博士毕业于美国俄克拉荷马大学健康科学中心，主要从事酒精性、非酒精性脂肪肝，毒物引发的脂肪肝、肝炎、肝硬化和肝癌的发病机制研究及肿瘤的免疫治疗研究。他曾获得 Eli Lilly 的科研创新奖以及 Joy Cappel 青年研究员奖，承担 Eli Lilly 的非酒精性脂肪肝炎和肝癌研究基金，还参与美国美国国立卫生研究院科学基金 5 项。

Wei Chen

Professor, XJTLU Entrepreneur College (Taicang), China; Fellow of the National Academy of Inventors in the United States.

Research Interests: Smart Chips, Sensing Technology, New Semiconductor Materials, Wearable Technology, Detection and Prevention of Diabetes and Early Cancer.

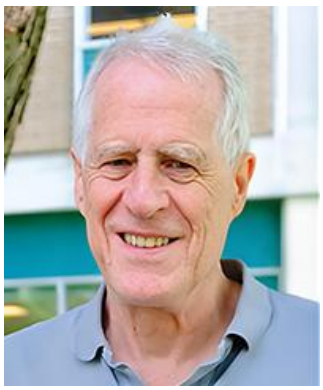
Dr. Wei Chen is previously served as the Deputy Director of the Semiconductor Materials Development Laboratory at the Chinese Academy of Sciences and received the CAS Young Scientist Award. He has long been engaged in cutting-edge nanotechnology research, and has published over 355 papers in internationally renowned academic journals. His papers have been cited 22372 times so far, with an h-index of 76, and he holds 22 US patents. He was selected for the "Annual Scientific Impact" and "Lifetime Scientific Impact" lists in the "2024 Global Top 2% Scientists" ranking.

Yicheng Ni

LaboHeal Chief Oncology Expert; Professor, Zhongda Hospital Affiliated to Southeast University, China.

Research Interests: Image-guided Diagnosis and Treatment Integration, Artificial Intelligence Medical Imaging, Tumor and Cancer Treatment, Medical Robots, Targeted Therapy, Medical Imaging and Cancer Modeling.

Professor Yicheng Ni is a Changjiang Scholar Professor and Professor of Radiology at Zhongda Hospital Affiliated to Southeast University. He has led EU research projects and National Natural Science Foundation of China projects, published hundreds of SCI papers and monographs, holds more than ten international patents, and has received dozens of domestic and international awards.



Michael Thompson

Professor, University of Toronto, Canada.

Research Interests: Bioanalytical Chemistry, Clinical Diagnostics, Drug Discovery, Nanotechnology, Molecular Imaging, Systems Biology.

Michael Thompson is a Professor of Bioanalytical Chemistry with the University of Toronto. He has held a number of distinguished research posts, including the Leverhulme Fellowship at Durham University and the Science Foundation Ireland E. T. S. Walton Research Fellowship at the Tyndall National Institute, Cork City. He has been awarded many prestigious international prizes for his research, including the Robert Boyle Gold Medal of the Royal Society of Chemistry, the E. W. R. Steacie Award of the Chemical Society of Canada.

Zhiling Yu

Professor, Hong Kong Baptist University, China.

Research Interests: Pharmacology of Chinese Medicines, Anti-Cancer, Anti-Inflammation, Quality Control of Decoction Pieces.

Prof. Yu obtained his PhD degree in biochemistry from the Hong Kong University of Science and Technology. Before joining the Hong Kong Baptist University, Prof. Yu once worked at China Pharmaceutical University, Nagasaki University, Hong Kong University of Science and Technology, Xiamen University and University of Macau. Prof. Yu is known for his research in herbal pharmacology, and in processing of Chinese medicinal materials. He has published more than 200 peer-reviewed research papers.

Andrew Teschendorff

Professor, Chinese Academy of Sciences, China.

Research Interests: Statistical Cancer Epigenomics, Cancer Systems Biology.

Teschendorff was a senior post-doctoral fellow in Computational Biology at the University of Cambridge. He then joined the CAS Max-Planck Partner Institute of Computational Biology in Shanghai as a professor from 2013 to 2020. Since 2020, he has been a professor and principal investigator at CAS Key Lab of Computational Biology in Shanghai. He has been a recipient of the Heller Research Fellowship, Cambridge-MIT fellowship, and an Advanced International Newton Fellowship from the Royal Society. In 2023, he was a recipient of a Highly Cited Researcher award from Clarivate.

Ahmed G. Hegazi

Professor, National Research Center, Egypt.

Research Interests: Microbiology, Immunology, Natural Medicine Efficacy.

Ahmed Hegazi is currently a Professor of Microbiology and Immunology in the National Research Center, Egypt. Dr. Hegazi contributed Chairman of the National Committee for Microbiological Sciences (NCMS) in Egypt, Academy of Science and Technology, Egypt, from 2018, Member of specialized Scientific Councils, Academy of Scientific Research & Technology; Being a member of the Specialty Committee of Apitherapy of the World Federation of Chinese Medicine Societies (2023) and Membership of the Federation of Chinese Medicine Societies (2023). He has 256 articles in national and international scientific journals.

Dipayan Rudra

Associate Professor, ShanghaiTech University, China.

Research Interests: Immune Tolerance, Autoimmunity and Inflammation, Regulatory T Cells, Tumor Immunology.

Dipayan Rudra has been worked as Principal Investigator in Academy of Immunology and Microbiology, Institute for Basic Science from 2013-2019, Research Associate Professor in Integrative Biosciences and Biotechnology (IBB), Pohang University of Science and Technology from 2019-2020, and Senior Scientist in Immunobiome Inc. from 2020-2021. Now he is the Associate Professor, Researcher, and Doctoral Supervisor in School of Life Science and Technology, ShanghaiTech University.

Saman Hosseinkhani

Professor, Tarbiat Modares University, Iran.

Research Interests: Creating Novel Luciferase-based Biosensor to Target Protein Complexes in Various Modes Cancer Models.

Saman Hosseinkhani is a Professor of Biochemistry at Tarbiat Modares University and associated member of Iran Academy of Sciences. His research specializes in the protein engineering of firefly luciferase, developing it as a reporter gene for monitoring biological processes. Recently, he has focused on creating novel luciferase-based biosensor to target protein complexes in various modes cancer models. In recognition of his expertise, he serves on the Scientific Advisory Board of the International Society for Bioluminescence and Chemiluminescence.

Kai Zhang

Associate Researcher, Shanghai Cancer Institute, China.

Research Interests: Mechanisms and Therapeutic Targets Underlying Cancer Immunosuppression, Treatment Resistance, and Distal Metastasis.

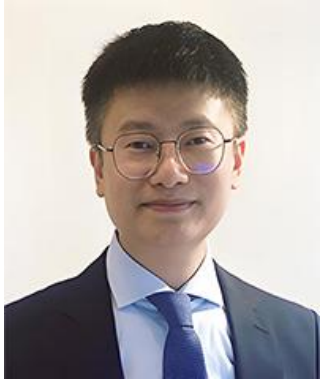
Dr. Kai Zhang subsequently conducted postdoctoral training at Baylor College of Medicine, USA and the University of Washington School of Medicine, USA. After returning to China, he has been serving as an Associate Researcher at Renji Hospital, Shanghai Jiao Tong University School of Medicine/Shanghai Cancer Institute. His research focuses on the mechanisms and therapeutic targets underlying cancer immunosuppression, treatment resistance, and distal metastasis.

Christopher L. Antos

Associate Professor, ShanghaiTech University, China.

Research Interests: Tissue Regeneration, Immunology, Developmental Biology, Regenerative Therapy.

Dr. Christopher L. Antos started his Ph.D. work in the laboratory of Dr. Eric Olson at UT Southwestern Medical Center at Dallas in 1996. After finishing his Ph.D., he then completed his post-doctoral training in the laboratory of Dr. Christiane Nüsslein-Volhard at the Max-Planck Institute for Developmental Biology in Tübingen, Germany from 2003-2007. In 2008, Dr. Antos established his own laboratory as faculty at the DFG-Center for Regenerative Therapies Dresden of the Technische Universität Dresden. Dr. Antos joined ShanghaiTech University as an Associate Professor in Dec. 2016.



Hua Su

Researcher, Fudan University, China.

Research Interests: Tumor Microenviroment and Metabolism, Mechanism of Macropinocytosis Initiation, Functions of Collagen in Tumor Development and Tumor Immunity, Tumorigenesis and Tumor Metabolism of Desmoplastic Cancer.

Dr. Hua Su conducted his Ph.D. training at Zhejiang University. He worked as the Postdoctoral Scholar in University of California on 2018-2022. Now, he is the Principal Investigator at Institutes of Biomedical Sciences, Fudan University, and he also is the Principal Investigator at Zhongshan-Xuhui Hospital, Fudan University. In 2022, Dr. Su established a research laboratory focus on tumor microenvironment and tumor metabolism.

Feng He

Researcher, Shanghai University of Traditional Chinese Medicine, China.

Research Interests: Stress Response Signaling in the Regulation of Inflammation and Metabolism, and their roles in Pathogenesis of Cancer, Exploring the New Methods for Cancer Prevention, Diagnosis, and Treatment.

Dr. He has received Lilly Innovative Fellowship Award from Eli Lilly and Joy Cappel Young Investigator Award. As a principle investigator, he undertook Eli Lilly's non-alcoholic steatohepatitis and liver cancer research fund and also participated in 5 research funds from the National Institutes of Health. Dr. He has published more than 20 research papers in journals such as Cancer Cell, J Hepatol, Proc Natl Acad Sci USA, EMBO J, etc., with an average of more than 70 citations per paper.

会议日程 | 2026 肿瘤防治新模式国际会议

2026 International Conference on New Models for Cancer Prevention and Treatment

日期	时间	内容
上海青松城大酒店 - 大厅（上海市徐汇区东安路 8 号）		
2026 年 4 月 17 日	14:00-18:00	会议报到
上海青松城大酒店黄山厅（上海市徐汇区东安路 8 号三楼） **腾讯会议室链接：**https://meeting.tencent.com/dm/1NWX1rr1cyPh **腾讯会议室 ID：943-186-323**		
2026 年 4 月 18 日	08:00-08:40	会议报到
	09:00-12:00	开幕式 & 主讲报告 **主持人：**卢国栋，复旦大学教授
	09:05-09:15	致辞嘉宾：高翔，复旦大学营养研究院（筹）院长&教授
	09:15-09:20	合影
	09:20-12:00	主讲报告
	09:20-09:55	特邀报告一：陈伟，西交利物浦大学芯片学院院长&教授、美国国家发明家科学院院士 题目：癌症治疗中的光动力疗法新进展
	09:55-10:30	特邀报告二：倪以成，乐博医疗首席肿瘤专家、长江讲席教授、东南大学附属中大医院放射科教授 题目：梯次双靶广谱诊疗一体化（OncoCiDia）：一种实践第一性原理思维的抗癌新模式？
	10:30-10:50	茶歇 & 墙报
	10:50-11:25	特邀报告三：Michael Thompson，加拿大多伦多大学教授、多伦多大学生物芯片与生物工程中心主任、加拿大皇家学会院士 题目：用于早期卵巢癌筛查的生物标志物的多通道生物传感器检测
	11:25-11:40	特邀学术出版报告：沈嘵嘵，Wiley 生命科学期刊内容发展编辑经理 题目：Wiley 生命科学期刊和特刊助力学术影响力提升
	11:40-11:50	主题分享：智研医学 2026 旗舰国际会议亮点前瞻
	12:00-14:00	午餐 & 午休

<table>
<tr><td colspan="3">分会场 1：多组学、免疫学与肿瘤转化创新
上海青松城大酒店香山厅（上海市徐汇区东安路 8 号四楼）
腾讯会议室链接：https://meeting.tencent.com/dm/1NWX1rr1cyPh
腾讯会议室 ID：943-186-323
主持人：Pier Paolo Piccaluga，意大利博洛尼亚大学副教授</td></tr>
<tr><td rowspan="9">2026 年 4 月 18 日</td><td>14:00-14:30</td><td>特邀报告一：禹志领，香港浸会大学教授
题目：同时抑制 EGFR 与 TLR4-MD2 作为肿瘤治疗的新策略</td></tr>
<tr><td>14:30-15:00</td><td>特邀报告二：Andrew Teschendorff，中国科学院上海营养与健康研究所教授
题目：基于单细胞组学探索癌症风险预测策略的开发</td></tr>
<tr><td>15:00-15:30</td><td>特邀报告三：Ahmed G. Hegazi，埃及国家研究中心微生物学和免疫学教授
题目：癌症预防与治疗的新模式：新兴策略与创新举措</td></tr>
<tr><td>15:30-15:40</td><td>休息</td></tr>
<tr><td>15:40-16:10</td><td>特邀报告四：Dipayan Rudra，上海科技大学副教授
题目：关于调节性 T 细胞介导的肿瘤免疫耐受的新见解</td></tr>
<tr><td>16:10-16:40</td><td>特邀报告五：Saman Hosseinkhani，伊朗塔比阿特莫达雷斯大学教授
题目：MiRGD@NTA@Au-Fe_3O_4 纳米颗粒：一种用于循环肿瘤细胞（CTCs）分离与检测的平台</td></tr>
<tr><td>16:40-16:55</td><td>口头汇报一：钱波涛，南充市中心医院
题目：胃肠道吻合口并发症的诊断与治疗新进展</td></tr>
<tr><td>16:55-17:10</td><td>口头汇报二：刘欣然，北京大学
题目：智能化与个体化时代的胃癌外科治疗</td></tr>
<tr><td>17:10</td><td>会议结束</td></tr>
<tr><td colspan="3">分会场 2：肿瘤微环境与靶向、免疫治疗新浪潮
上海青松城大酒店华山厅（上海市徐汇区东安路 8 号四楼）
腾讯会议室链接：https://meeting.tencent.com/dm/aQCGJQuRKWeR
腾讯会议室 ID：954-715-937
主持人：王海波，中国科学院杭州医学研究所博士生导师，课题组组长，智能分子诊断研究中心副主任</td></tr>
<tr><td rowspan="2">2026 年 4 月 18 日</td><td>14:00-14:30</td><td>特邀报告一：张凯，上海市肿瘤研究所副研究员
题目：神经内分泌前列腺癌的肝转移机制</td></tr>
<tr><td>14:30-15:00</td><td>特邀报告二：Christopher L. Antos，上海科技大学副教授
题目：电生理信号如何在再生与发育过程中通过生物机制整合以调节附肢的生长发育</td></tr>
</table>

	15:00-15:30	特邀报告三：苏华，复旦大学研究员 题目：胶原蛋白水解信号决定胰腺癌的发展
	15:30-15:40	休息
	15:40-16:10	特邀报告四：何锋，上海中医药大学研究员 题目：应激反应蛋白 ATF4 在肝损伤、肝再生及肝癌发生中的作用
	16:10-16:25	口头汇报一：闫加艳，复旦大学附属中山医院 题目：外科手术诱导肝再生研究体系模式摸索
	16:25-16:40	口头汇报二：崔萌，内蒙古医科大学讲师 题目：宫颈阴道菌群失调和微环境破坏与宫颈癌变相关
	16:40-16:55	口头汇报三：李晓，上海中医药大学 题目：大柴胡汤通过维持肠道血管屏障抑制营养过剩诱导的结直肠癌肝转移
	16:55	会议结束

Date	Time	Content
Pine City Hotel - Lobby (No. 8 Dong'an Road, Xuhui District, Shanghai)		
April 17, 2026	14:00-18:00	Registration
Huangshan Hall, Pine City Hotel (3rd Floor, No. 8 Dong'an Road, Xuhui District, Shanghai) **VooV Meeting Link: https://meeting.tencent.com/dm/1NWX1rr1cyPh** **VooV Meeting ID: 943-186-323**		
April 18, 2026	08:00-08:40	Registration
	09:00-12:00	Opening Ceremony & Keynote Speeches **Host:** Prof. Guodong Lu, Fudan University, China
	09:05-09:15	Opening Speech: Xiang Gao, Dean & Professor, Institute of Nutrition, Fudan University, China
	09:15-09:20	Group Photo
	09:20-12:00	Keynote Speeches
	09:20-09:55	Keynote Speech 1: Wei Chen, Professor, XJTLU Entrepreneur College (Taicang), China; Academician of the National Academy of Inventors Title: New Development on Photodynamic Therapy for Cancer Treatment
	09:55-10:30	Keynote Speech 2: Yicheng Ni, LaboHeal Chief Oncology Expert; Professor, Zhongda Hospital Affiliated to Southeast University, China Title: Dual-Targeting Pan-anticancer Theragnostics (OncoCiDia): A New Model Derived from First-principles Thinking?
	10:30-10:50	Tea Break & Poster Display
	10:50-11:25	Keynote Speech 3: Michael Thompson, Professor, University of Toronto, Canada; Fellow of the Royal Society of Canada. Title: Multiplexed biosensor detection of biomarkers for screening of early-stage ovarian cancer
	11:25-11:40	Invited Academic Publishing Speech: Liangliang Shen, Manager, Strategic Content Acquisition, Life

		Sciences, Wiley Life Sciences Journals Title: Unleash your research impact with Wiley life sciences journals and special issue program
	11:40-11:50	Theme Presentation: Highlights of the 2026 Flagship International Conference of Zhiyan Med
	12:00-14:00	Lunch & Break
Session 1: Multi-Omics, Immunology & Translational Innovations in Cancer **Xiangshan Hall, Pine City Hotel (4th Floor, No. 8 Dong'an Road, Xuhui District, Shanghai)** **VooV Meeting Link: https://meeting.tencent.com/dm/1NWX1rr1cyPh** **VooV Meeting ID: 943-186-323** **Session Chair：** Prof. Pier Paolo Piccaluga, University of Bologna, Italy		
April 18, 2026	14:00-14:30	Keynote Speech 1: Zhiling Yu, Professor, Hong Kong Baptist University, China Title: Dual Inhibition of EGFR and TLR4-MD2 as a Novel Strategy for Cancer Treatment
	14:30-15:00	Keynote Speech 2: Andrew Teschendorff, Professor, Chinese Academy of Sciences, China Title: Towards the development of cancer risk prediction strategies from single-cell omics
	15:00-15:30	Keynote Speech 3: Ahmed G. Hegazi, Professor, National Research Center, Egypt Title: New Models for Cancer Prevention and Treatment: Emerging Strategies and Innovations
	15:30-15:40	Break
	15:40-16:10	Keynote Speech 4: Dipayan Rudra, Associate Professor, ShanghaiTech University, China Title: Novel Insights on Regulatory T cell Mediated Tumor Immune Tolerance
	16:10-16:40	Keynote Speech 5: Saman Hosseinkhani, Tarbiat Modares University, Iran Title: MiRGD@NTA@Au-Fe3O4 nanoparticles: a platform for circulating tumor cells (CTCs) isolation and detection
	16:40-16:55	Oral Presentation 1: Botao Qian, Nanchong Central Hospital, China Title: New Advances in the Diagnosis and Treatment of Gastrointestinal Anastomotic Complications
	16:55-17:10	Oral Presentation 2: Xinran Liu, Peking University, China Title: Gastric Cancer Surgery in the Era of

		Intelligence and Individualization
	17:10	Closing
	Session 2: Tumor Microenvironment & the New Wave of Targeted/Immunotherapies **Huashan Hall, Pine City Hotel (4th Floor, No. 8 Dong'an Road, Xuhui District, Shanghai)** **VooV Meeting Link:** **https://meeting.tencent.com/dm/aQCGJQuRKWeR** **VooV Meeting ID: 954-715-937** **Session Chair：** Dr. Haibo Wang, Hangzhou Institute of Medicine Chinese Academy of Sciences, China	
April 18, 2026	14:00-14:30	Keynote Speech 1: Kai Zhang, Associate Researcher, Shanghai Cancer Institute, China Title: The Molecular Basis of Neuroendocrine Prostate Cancer Liver Metastasis
	14:30-15:00	Keynote Speech 2: Christopher L. Antos, Associate Professor, ShanghaiTech University, China Title: How An Electrophysiological Signal is Biologically Integrated to Scale Appendages During Regeneration and Development
	15:00-15:30	Keynote Speech 3: Hua Su, Researcher, Fudan University, China Title：Collagen Cleavage Signaling Dictates Pancreatic Cancer Development
	15:30-15:40	Break
	15:40-16:10	Keynote Speech 4: Feng He, Researcher, Shanghai University of Traditional Chinese Medicine, China Title: The Role of Stress Response Protein ATF4 in Liver Injury, Regeneration, and Hepatocarcinogenesis
	16:10-16:25	Oral Presentation 1: Jiayan Yan, Zhongshan Hospital, Fudan University, China Title: Exploration of the Research System Model for Surgical Induction of Liver Regeneration
	16:25-16:40	Oral Presentation 2: Meng Cui, Lecture, Inner Mongolia Medical University, China Title: Cervicovaginal Dysbiosis and Microenvironment Disruption are Associated with Cervical Carcinogenesis

	16:40-16:55	Oral Presentation 3: Xiao Li, Shanghai University of Traditional Chinese Medicine, China Title: Dachaihu Decoction Inhibits Hypernutrition-induced Liver Metastasis from Colorectal Cancer by Maintaining the Gut Vascular Barrier
	16:55	Closing

会议地点：青松城大酒店

地址：上海市徐汇区东安路 8 号

交通：上海青松城大酒店交通便利，距地铁 7、9 号线肇嘉浜路站仅 140 米，距上海南站约 6.7 公里，距上海虹桥国际机场约 11.6 公里。

Venue | 2026 肿瘤防治新模式国际会议

2026 International Conference on New Models for Cancer Prevention and Treatment

Venue: Pine City Hotel

Address: No. 8 Dong'an Road, Xuhui District, Shanghai

Traffic: Pine City Hotel enjoys a convenient location, just 140 meters from Zhaojiabang Road Station on Metro Lines 7 and 9; 6.7 km from Shanghai South Railway Station; 11.6 km from Shanghai Hongqiao International Airport.

No.	Paper ID	Title
1.	N20260408003	Dachaihu decoction inhibits hypernutrition-induced liver metastasis from colorectal cancer by maintaining the gut vascular barrier

Xiao Li, The Center for Cancer Research, School of Integrative Medicine, Shanghai University of Traditional Chinese Medicine, Shanghai, China
Ruolei Wang, The Center for Cancer Research, School of Integrative Medicine, Shanghai University of Traditional Chinese Medicine, Shanghai, China
Feng He, The Center for Cancer Research, School of Integrative Medicine, Shanghai University of Traditional Chinese Medicine, Shanghai, China

Abstract—Background: Colorectal cancer (CRC) is the third most common malignancy and the second deadliest cancer worldwide. Metastasis to the liver, the most common metastatic site in CRC, is the leading cause of death in patients with CRC. Hyperlipidemia, which is common in patients with CRC, promotes CRC progression and metastasis. Hyperlipidemia is commonly observed in obese patients and is often induced by hypernutrition. The underlying mechanism of hypernutrition-induced hyperlipidemia in promoting CRC liver metastasis remains unclear, and there is an unmet need for effective and low-cost treatments for patients with CRC.

Methods: A mouse cecum orthotopic CRC model combined with high-fat diet (HFD) feeding, was established to mimic liver metastasis in CRC in obese patients. The effects of Dachaihu decoction (DCHD), a traditional herbal medicine used to treat inflammation and nonalcoholic fatty liver disease, and of the conventional prescription medicine obeticholic acid (OCA) were evaluated. HFD-induced obesity, hyperlipidemia, and CRC liver metastasis were assessed, along with the histology and pathology of the liver and intestine and the expression of metabolic genes in these tissues. The effects of DCHD and OCA on HFD-induced outcomes were evaluated, and human umbilical vein endothelial cells (HUVECs) treated with bile acids (BAs) and DCHD were used to study the underlying echanisms in vitro.

Results: HFD-mediated obesity and hyperlipidemia promoted CRC metastasis, accompanied by disruption of the gut vascular barrier (GVB) and altered bile acid (BA) metabolism. DCHD decreased HFD-induced hyperlipidemia and liver metastasis in CRC, improving overall survival. Those effects of DCHD were equivalent to or better than those of OCA. DCHD regulated the expression of genes of BA metabolism and tight junctions (TJ) to prevent HFD induced disruption of the GVB. In HUVECs, DCHD prevented the increases in intracellular Ca2+ and accumulation of reactive oxygen species induced by primary conjugated BAs, assisting in the maintenance of redox homeostasis and preventing the downregulation of TJ proteins, thereby maintaining the integrity of the endothelial barrier.

Conclusions: The data provide a link between hypernutrition and GVB disruption, which contributes to high liver metastasis in patients with CRC. DCHD may represent a novel therapy in CRC, and targeting abnormal lipid metabolism could be a promising therapeutic strategy for avoiding hypernutrition-associated CRC

metastasis.

No.	Paper ID	Title
2.	N20260319003	ATF4 suppresses hepatocarcinogenesis by inducing SLC7A11 (xCT) to block stress-related ferroptosis

Lianheng Lu, The Center for Cancer Research, School of Integrative Medicine, Shanghai University of Traditional Chinese Medicine, Shanghai, China

Chunyan Wang, The Center for Cancer Research, School of Integrative Medicine, Shanghai University of Traditional Chinese Medicine, Shanghai, China

Feng He, The Center for Cancer Research, School of Integrative Medicine, Shanghai University of Traditional Chinese Medicine, Shanghai, China

Abstract—Background: Hepatocellular carcinoma (HCC), a leading cause of cancer-related death, is associated with viral hepatitis, non-alcoholic steatohepatitis (NASH), and alcohol-related steatohepatitis, all of which trigger endoplasmic reticulum (ER) stress, hepatocyte death, inflammation, and compensatory proliferation. Using ER stress-prone MUP-uPA mice, we established that ER stress and hypernutrition cooperate to cause NASH and HCC, but the contribution of individual stress effectors, such as activating transcription factor 4 (ATF4), to HCC and their underlying mechanisms of action remained unknown.

Methods: Hepatocyte-specific ATF4-deficient MUP-uPA mice (MUP-uPA/Atf4Δhep) and control MUP-uPA/Atf4F/F mice were fed a high-fat diet to induce NASH-related HCC, and Atf4F/F and Atf4Δhep mice were injected with diethylnitrosamine to model carcinogen-induced HCC. Histological, biochemical, and RNA-sequencing analyses were performed to identify and define the role of ATF4-induced solute carrier family 7a member 11 (SLC7A11) expression in hepatocarcinogenesis. Reconstitution of SLC7A11 in ATF4-deficient primary hepatocytes and mouse livers was used to study its effects on ferroptosis and HCC development.

Results: Hepatocyte ATF4 ablation inhibited hepatic steatosis, but increased susceptibility to ferroptosis, resulting in accelerated HCC development. Although ATF4 activates numerous genes, ferroptosis susceptibility and hepatocarcinogenesis were reversed by ectopic expression of a single ATF4 target, Slc7a11, coding for a subunit of the cystine/glutamate antiporter xCT, which is needed for glutathione synthesis. A ferroptosis inhibitor also reduced liver damage and inflammation. ATF4 and SLC7A11 amounts were positively correlated in human HCC and livers of patients with NASH.

Conclusions: Despite ATF4 being upregulated in established HCC, it serves an important protective function in normal hepatocytes. By maintaining glutathione production, ATF4 inhibits ferroptosis-dependent inflammatory cell death, which is known to promote compensatory proliferation and hepatocarcinogenesis. Ferroptosis inhibitors or ATF4 activators may also blunt HCC onset.

No.	Paper ID	Title
3.	/	Anti-renal cell carcinoma effects and mechanisms of deoxyelephantopin

Jin-jin He, School of Chinese Medicine, Hong Kong Baptist University, China

Xiu-qiong Fu, School of Chinese Medicine, Hong Kong Baptist University, China

Zhi-Ling Yu, School of Chinese Medicine, Hong Kong Baptist University, China

Abstract—Background: Renal cell carcinoma (RCC) is the most prevalent form of kidney cancer, and current

treatments are often limited by adverse effects, drug resistance, and low response rates. The poor 5-year survival of advanced RCC underscores the need for new therapeutic strategies. Deoxyelephantopin (DEO), a bioactive sesquiterpene lactone from Elephantopi Herba, has shown anti-RCC effects in our previous work, but its molecular targets in RCC remain largely undefined. This study aimed to identify key mediators of DEO's anti-RCC activity through integrated bioinformatics, pan-cancer analyses, and experimental validation, with a focus on the candidate gene HIBCH.

Methods: Eighteen RCC gene expression datasets from the GEO database were analyzed to define tumor-associated differentially expressed genes (DEGs). RNA sequencing of DEO-treated 786-O cells identified DEO-responsive DEGs. Overlapping genes dysregulated in RCC that were reversed by DEO were evaluated by survival analysis and cross-validated using The Cancer Genome Atlas (TCGA). HIBCH expression was assessed in 235 paired RCC tumor–adjacent normal samples from GEO, and its association with tumor stage was analyzed in TCGA cohorts. Pan-cancer expression profiles of HIBCH were examined using TIMER and the Human Protein Atlas, followed by receiver operating characteristic (ROC) analysis and survival assessment via the Kaplan-Meier Plotter. RT-qPCR and Western blotting determined HIBCH mRNA and protein levels in DEO-treated 786-O cells. Weighted gene co-expression network analysis (WGCNA) and KEGG enrichment were used to predict HIBCH-associated pathways in RCC.

Results: Integration of GEO and RNA-seq data identified 12 RCC-dysregulated genes reversed by DEO treatment. Survival analysis highlighted five genes (GABARAPL1, ABCB1, HIBCH, GAL3ST1, IGFBP3) associated with RCC prognosis, and TCGA cross-validation showed that only HIBCH was consistently downregulated across the three major RCC subtypes (KIRC, KIRP, KICH). Analysis of 235 paired RCC samples confirmed significant HIBCH downregulation in tumors versus adjacent normal tissues. HIBCH expression decreased progressively with advancing tumor stage ($F = 20.6$, $P = 6.65E-13$). Pan-cancer analyses revealed widespread dysregulation of HIBCH, with the most pronounced alterations in RCC. At the mRNA level, HIBCH was significantly downregulated in 12 cancer types, with the greatest reductions in kidney cancers (KICH, KIRC, KIRP). Protein-level analyses showed downregulation in six cancers, again most substantial in KIRC. ROC analysis demonstrated excellent diagnostic performance across multiple cancers, with the highest accuracy in KIRC (AUC = 0.976, 95% CI 0.954–0.997). Survival analyses indicated tumor-specific roles of HIBCH, with the strongest protective association in KIRC (HR = 0.35, $P = 5.9E-10$). RT-qPCR and Western blotting confirmed that DEO upregulated HIBCH at both mRNA and protein levels in 786-O cells. WGCNA identified 76 HIBCH co-expressed genes in RCC, which were significantly enriched in the valine, leucine, and isoleucine degradation pathway.

Conclusions: These findings indicate that HIBCH upregulation is at least partly involved in the anti-RCC effects of DEO. The consistent downregulation of HIBCH in paired RCC tumors, its stage-dependent decline, and its superior diagnostic and prognostic performance in RCC relative to other cancers support HIBCH as a key regulator of RCC progression, potentially acting through branched-chain amino acid degradation. Collectively, this study identifies HIBCH as a potential molecular target of DEO and a promising biomarker and therapeutic target for RCC.

No.	Paper ID	Title

4.	/	Quantifying Epigenetic Reprogramming and Cellular Plasticity in Preneoplastic Lesions Reveals Loss of a Fetal Gene Expression Program as a Key Precancer Hallmark

Jason Tham Han Kiat, Shanghai Institute of Nutrition and Health, University of Chinese Academy of Sciences, Chinese Academy of Sciences, China

Andrew E.Teschendorff, Shanghai Institute of Nutrition and Health, University of Chinese Academy of Sciences, Chinese Academy of Sciences, China

Abstract—Background: Epigenetic reprogramming and cellular plasticity are two fundamental cancer hallmarks that increase the risk of carcinogenic transformation. However, quantifying these properties at the single-cell level remains a significant challenge in the field of cancer biology.

Methods: We developed and validated PLASTIC, a general pan-tissue computational framework designed to quantify cellular plasticity and epigenetic reprogramming from single-cell RNA-seq (scRNA-seq) data. This framework allows for the construction of Waddington-like landscapes, which are utilized to identify high cancer-risk states across various preneoplastic tissue settings.

Results: When applied to a pan-preneoplastic tissue setting, PLASTIC revealed a direct correlation between the level of epigenetic reprogramming and both cellular plasticity and cancer risk. A key finding is that high-risk states are characterized by increased dedifferentiation, which often exceeds the levels observed in corresponding fetal cell counterparts. Additionally, PLASTIC successfully predicted the emergence of metaplasia in the normal lung cells of smokers, identifying a high-risk signature that was subsequently validated in independent datasets.

Conclusions: PLASTIC provides a robust method to quantify cellular plasticity and dedifferentiation in single cells across any tissue type. By pinpointing aberrant dedifferentiation states, this framework opens up new strategies for early cancer risk prediction and the development of targeted prevention measures.

No.	Paper ID	Title
5.	/	Inhibition of CBX4 Signaling Contributes to the Molecular Mechanisms of Ginsenoside Rg3 Combined with Artesunate in Overcoming Sorafenib Resistance in Hepatocellular Carcinoma

Xiaofei Yu, School of Chinese Medicine, Hong Kong Baptist University, Hong Kong, China

Xiaoqi Wang, School of Chinese Medicine, Hong Kong Baptist University, Hong Kong, China

Xiuqiong Fu, School of Chinese Medicine, Hong Kong Baptist University, Hong Kong, China

Zhiling Yu, School of Chinese Medicine, Hong Kong Baptist University, Hong Kong, China

Abstract—Background: Background: Hepatocellular carcinoma (HCC) is the most common primary liver cancer and a leading cause of cancer-related mortality worldwide. Although sorafenib delays HCC progression by targeting multiple tyrosine kinases, acquired resistance limits its benefit. Ginsenoside Rg3 (Rg3) and artesunate (ART) exhibit anticancer activity and can synergize with sorafenib, and Rg3 plus ART has inhibited sarcoma growth. Importantly, both agents are marketed: Rg3 is the active component of the anticancer adjuvant Shenyi Capsule used clinically in China, and ART is an approved antimalarial,

highlighting their repurposing potential. Our prior work showed that Rg3 plus ART overcomes sorafenib resistance in HepG2-resistant models by inhibiting Src/STAT3 signaling. Here, we further define the pharmacological effects and mechanisms of the Rg3–ART combination in additional sorafenib-resistant HCC models.

Methods: Sorafenib-resistant Huh7 (Huh7-SR) and SK-HEP1 (SK-HEP1-SR) cells were treated with Rg3 plus ART. Cell viability, apoptosis, cell cycle progression, migration, and invasion were assessed. Mechanistic studies employed RNA sequencing, RT-qPCR, and Western blotting. Functional validation used rescue assays, including CBX4 overexpression and activation of TEAD-dependent transcription.

Results: Rg3 plus ART synergistically reduced cell viability, induced apoptosis, caused S-phase arrest, and suppressed migration and invasion in Huh7-SR and SK-HEP1-SR cells. Transcriptomic and protein analyses revealed significant downregulation of CBX4 and its downstream targets (including BCL2, MYC, CDK1, POLA1, CDC6, CDC7, BIRC2, BIRC5, MCL1, and CTGF). Given that CBX4 promotes oncogenic transcription by cooperating with YAP1 and TEADs, we found that CBX4 overexpression attenuated the combination's suppression of CBX4-pathway genes at both mRNA and protein levels and weakened its ability to overcome sorafenib resistance. Conversely, activating TEAD-dependent transcription increased expression of CBX4/YAP1/TEAD-regulated genes and diminished the anti-resistance effects of Rg3 plus ART.

Conclusions: Rg3 and ART act synergistically to overcome sorafenib resistance in HCC, at least in part by inhibiting CBX4 signaling and downstream YAP/TEAD-mediated transcription. These findings support the repurposing of Rg3 plus ART as a potential adjuvant strategy for sorafenib-resistant HCC.

No.	Paper ID	Title
6.	/	Cervicovaginal dysbiosis and microenvironment disruption are associated with cervical carcinogenesis

Meng Cui, Inner Mongolia Medical University, China

Abstract—Cervical cancer poses a serious threat to women's health. Emerging evidence indicates that changes in the cervicovaginal microbiota (CVM) and vaginal microenvironment may play a crucial role in cervical carcinogenesis. In this study, we examined CVM and vaginal microenvironment profiles in 510 participants with varying stages of cervical lesions using 16S rRNA sequencing, vaginal pH and H2O2 measurements, and cleanness assessment. A co-occurrence network and generalized multifactor dimensionality reduction (GMDR) model were employed to analyze the correlation and interaction between microbial taxa and microenvironment profile. Our results demonstrated that CVM disorder and abnormal vaginal microenvironment were associated with cervical lesions. As cervical cancerization progressed, the abundance of Lactobacillus gradually decreased with thriving anaerobe levels in the deteriorating vaginal microenvironment. This interaction was characterized by high vaginal pH, a non-Lactobacillus-dominant microbiota, and high CVM diversity (Shannon index ≥ 0.81). Co-occurrence network analysis revealed that abnormal pH and H2O2 levels correlated with Lactobacillus depletion and had a positively co-occurring relationship with anaerobes in precancerous cervical lesions. CVM function significantly varied with changes in the vaginal microenvironment across different stages of cervical lesions. Our findings suggest that CVM dysbiosis, particularly when combined with an

abnormal vaginal microenvironment, may promote the progression of cervical lesions. This highlights the importance of addressing vaginal microecology to warn against and prevent cervical carcinogenesis.

No.	Paper ID	Title
7.	N20260319004	Paris Polyphylla var. yunnanensis Saponins Inhibit Inflammation-Driven Hepatocarcinogenesis via AKT/PERK Pathway Crosstalk and ER Stress Apoptosis

Junbin Wu, Yunnan University of Chinese Medicine, China

Abstract—Background: Hepatocyte injury and death are the initiating factors of hepatic inflammation. Endoplasmic reticulum stress (ERS) exerts bidirectional regulatory effects on liver diseases: mild to moderate ERS can restore endoplasmic reticulum homeostasis, whereas severe or persistent ERS activates the protein kinase RNA-like endoplasmic reticulum kinase (PERK)/activating transcription factor 4 (ATF4)/C/EBP homologous protein (CHOP) pathway, leading to unfolded protein response (UPR) overload, exacerbating non-alcoholic steatohepatitis (NASH)-induced liver injury, and accelerating the development of hepatocellular carcinoma (HCC). The crosstalk between the protein kinase B (AKT) pathway and the PERK/ATF4/CHOP pathway can regulate cell survival and death. Previous studies have confirmed that Paris Polyphylla var. yunnanensis saponins (RPS) can inhibit the transformation from non-alcoholic fatty liver disease (NAFLD) to HCC in mice and regulate the aforementioned pathways, demonstrating significant research value.

Methods: This study established a STAM mouse NAFLD-HCC model to observe the effects of RPS on tumor formation rate and liver pathology. Flow cytometry was used to detect the distribution of immune cells in the STAM mouse model; HE staining, oil red staining, and Masson's trichrome staining were employed to detect pathological changes in the livers of model animals; immunofluorescence and Western blot methods were used to detect the expression and localization of AKT/PERK-related pathway proteins and their dynamic changes during liver disease progression. For cellular experiments, hepatocellular carcinoma cell lines (such as HepG2 and Huh-7) were used as research subjects. Colony formation assay, scratch wound healing assay, and Transwell assay were performed to detect cell proliferation and migration capabilities; flow cytometry and fluorescence methods were used to detect cell apoptosis rate and reactive oxygen species (ROS) levels; qRT-PCR and Western Blot were employed to detect the expression of related proteins.

Results: RPS inhibited the tumor transformation rate in STAM mice in a concentration-dependent manner, suppressed liver fibrosis progression, alleviated inflammatory responses, and relieved insulin resistance and oxidative stress. During weeks 0-8, low-level ERS-mediated PERK pathway inhibited the AKT pathway, promoting autophagy feedback to suppress ERS. As the disease progressed continuously, inflammation and fibrosis became evident in STAM mice, with massive production of inflammatory cytokines. ATF4 translocation promoted substantial CHOP expression, leading to persistent cell apoptosis and exacerbated hepatocyte injury. Meanwhile, myeloid-derived suppressor cells (MDSCs) and regulatory T cells (Tregs) accumulated extensively to form the tumor microenvironment. RPS could inhibit the crosstalk between the AKT-PERK pathway and prevent the formation of the tumor microenvironment.

Conclusions: The PERK-ATF4-CHOP pathway plays a dual role in "moderate" and "excessive" ERS, and regulating this pathway may serve as a "switch" that determines hepatocyte survival or death. RPS exerts

anti-inflammatory and anti-cancer transformation effects in mice by regulating the phosphoinositide 3-Kinase (PI3K)/AKT pathway and the PERK-ATF4-CHOP pathway. It can target key proteins in the PERK pathway to regulate cell survival and death, effectively inhibiting the progression of non-alcoholic fatty liver disease-hepatocellular carcinoma (NAFLD-HCC), providing new insights for traditional Chinese medicine intervention in the treatment of liver diseases.

No.	Paper ID	Title
8.	N20260408002	Comparison of Short-Term Outcomes Among Hand-Sewn, Stapled, and two-stage Turnbull-Cutait Pull-Through Anastomoses in TaTME for Low Rectal Cancer

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Guobiao Chen, epartment of Gastrointestinal Surgery,Beijing Anzhen Nanchong Hospital of Capital Medical University & Nanchong Central Hospital, Nanchong, China

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Yunhong Tian, epartment of Gastrointestinal Surgery,Beijing Anzhen Nanchong Hospital of Capital Medical University & Nanchong Central Hospital, Nanchong, China

Abstract—Background: The transanal total mesorectal excision (TaTME) technique has been recognised as an important surgical method for low rectal cancer, yet optimal anastomotic techniques remain debated. This study compared short-term outcomes of hand-sewn coloanal anastomosis (CAA), single-stapled anastomosis (SSA), and two-stage Turnbull-Cutait pull-through coloanal anastomosis (TCA).

Methods: A retrospective analysis of 147 patients undergoing TaTME for low rectal cancer (tumors ≤5 cm from anal verge) was conducted between July 2020 and July 2023. The patients were categorized into CAA, SSA and TCA groups based on the anastomosis. The primary endpoint was anastomotic related complications that included anastomotic leakage, anastomotic bleeding, and anastomotic stricture. The secondary endpoints included neorectal prolapse, pathological outcomes, perioperative outcomes, and functional outcomes assessed through the LARS scoring system.

Results: Among 147 patients with low rectal cancer, 42 patients underwent hand-sewn coloanal anastomosis (CAA), 69 patients underwent single-stapler anastomosis (SSA), and 36 patients underwent two-stage Turnbull-Cutait pull-through coloanal anastomosis (TCA). Operative time was shortest in the SSA group and

longest in the TCA group (p<0.001). The TCA group demonstrated a lower protective stoma rate (p=0.005), higher splenic flexure mobilization rate (p=0.014), and extended postoperative hospital stay (p<0.001) compared to CAA and SSA. Pathologically, TCA resulted in longer bowel resection length (p<0.001), with similar distal resection margins and lymph node harvest. Complication rates for anastomotic leakage, bleeding, stricture, and intraoperative bleeding were comparable, but TCA showed a higher neorectal prolapse incidence (p<0.001). Low anterior resection syndrome scores gradually improved postoperatively among the three groups, with no intergroup differences among the three groups (p≥0.305).

Conclusion: Although TCA avoids the need for protective stoma creation, it is not recommended as a routine anastomotic approach for TaTME due to higher rates of neorectal prolapse, prolonged postoperative hospital stay, extended operative time, and the necessity for longer bowel resection.

No.	Paper ID	Title
9.	N20260401002	Bridging the Evaluation Gap: The Transformative Shift in Medical AI Assessment From Benchmark Testing to Real-World Clinical Decision-Making

Mengchao Yue, Intelligent Medicine Insititute, Fudan University, China

Abstract—Background: Large Language Models (LLMs) have been rapidly adopted in the medical field, yet their performance evaluation still primarily relies on static question-and-answer datasets and standardized benchmark testing. Such an evaluation paradigm fails to recapitulate the dynamic evolution, multi-stage progression, and multi-agent interaction characteristics of real-world clinical decision-making, which easily leads to a significant disconnect between models' benchmark performance and their actual clinical value. Reconstructing the evaluation framework for medical artificial intelligence from the perspectives of clinical epidemiology and evidence-based medicine has become a core methodological challenge for its clinical translation.

Objective: To clarify the key methodological flaws of the existing medical AI evaluation system within the clinical decision chain, construct a multi-dimensional evaluation framework integrating clinical, public health, and humanistic dimensions, explore evaluation pathways adapted to real clinical scenarios, and provide methodological support for evidence-based medical practice and the clinical translation of medical AI.

Methods: A research design combining methodological analysis and scenario simulation was adopted. We systematically sorted out mainstream static evaluation methods and analyzed their limitations from clinical epidemiological dimensions such as external validity, outcome indicator adaptability, and completeness of the decision chain. We constructed a digital clinical environment to simulate disease progression, stepwise information disclosure, resource constraints, and clinical workflows, and carried out dynamic decision-making evaluation. A multi-dimensional outcome indicator system covering clinical outcomes, system efficiency, and humanistic care was established to compare the performance differences and decision path characteristics of 6 mainstream large models between traditional benchmark testing and real-scenario evaluation.

Results: The performance of medical AI in static benchmark testing cannot stably predict its real clinical decision-making quality, and there is systematic overestimation. After incorporating temporal evolution, resource constraints, and multi-role interaction, the models exhibited more prominent problems such as diagnostic delay, overtesting, and insufficient decision consistency. Different models presented differentiated

trade-off relationships between clinical accuracy and system efficiency. In the humanistic dimension, the models showed significant differences in patient communication, value trade-offs, and patient-centeredness, with limited correlation with traditional accuracy indicators.

Conclusions: The existing medical AI evaluation system has a clear evaluation gap, whose core crux is that the static evaluation paradigm fails to fully cover the dynamic, systematic, and humanistic attributes of real clinical decision-making. The multi-dimensional evaluation framework based on the digital clinical environment can more comprehensively identify the real clinical value and potential risks of medical AI, providing methodological support for its evidence-based evaluation, regulatory review, guideline development, and clinical application. In the future, we should promote the in-depth transformation of medical AI evaluation from a static accuracy-oriented paradigm to an evidence-based clinical value-oriented one.

No.	Paper ID	Title
10.	N20260331002	Engineered Nanovesicles in Cancer Prevention and Therapy: Early Interception, Tumor Microenvironment Reprogramming, and Clinical Translation

Wangshu Li, Women and Children's Hospital Affiliated to Dalian University of Technology, China

Abstract—Cancer control is shifting from a treatment-only paradigm toward a prevention-to-treatment continuum that emphasizes risk stratification, ultra-early detection, immune interception, and personalized therapeutic delivery. Engineered nanovesicles, including extracellular vesicles (EVs), exosomes, microvesicles, and cell-membrane-derived vesicle mimetics, occupy a uniquely strategic position in this transition because they can function both as disease-informative biomarkers and as biologically compatible delivery vehicles. Their lipid bilayers protect nucleic acids and proteins, their surface ligands support tissue tropism, and their cargo can be reprogrammed to modulate tumor cells, stromal compartments, and immune effectors. This review reframes engineered nanovesicles not only as therapeutic carriers, but also as an interface technology linking multi-omics prevention, liquid-biopsy screening, immunoprevention, microenvironment remodeling, and resistance-overcoming precision oncology. We summarize their roles in biomarker-driven early interception and recurrence surveillance; engineering strategies including donor-cell genetic programming, bio-orthogonal surface chemistry, membrane fusion, and active or passive cargo loading; and therapeutic applications spanning RNA interference, mRNA delivery, CRISPR systems, chemotherapeutic co-delivery, CAR-exosome platforms, and myeloid or stromal reprogramming. We also discuss translational bottlenecks, including scalable GMP manufacturing, standardized potency assays, source selection, and regulatory classification. Overall, engineered nanovesicles are emerging as one of the few nanotechnology platforms able to bridge early cancer interception with treatment escalation and post-treatment recurrence prevention.

No.	Paper ID	Title
11.	N20260330002	Conceptual Framework for AI-Based Prediction of Breast Cancer Recurrence Risk and Personalised Management Plans

Menglu Chang, Shaanxi University of Chinese Medicine, China

Abstract—Objective

This study aims to develop an artificial intelligence-based model for predicting the risk of breast cancer

recurrence and to formulate personalised management plans. There are significant individual variations in the risk of breast cancer recurrence; traditional predictive tools rely on limited clinical and pathological parameters, struggle to fully integrate multimodal data, and lack dynamic, personalised intervention strategies. The objective of this study is to utilise deep learning and multi-source data fusion techniques to enhance the accuracy and interpretability of recurrence risk prediction, whilst establishing risk-stratified, individualised management plans. This will facilitate a shift from population-based average risk assessment to precise individualised management, thereby optimising clinical decision-making, prolonging patient survival and improving quality of life.

Methods

A combined retrospective and prospective study design was adopted. Data on clinical and pathological characteristics, genomic data, radiomic features, and treatment history were collected from breast cancer patients across multiple centres. Following data standardisation, cleaning, missing value imputation, and feature selection, a multimodal fusion deep neural network model was constructed. Incorporating an attention mechanism and a survival analysis loss function, the model predicted the probability of recurrence at three and five years post-surgery. Model performance was evaluated using five-fold cross-validation and an independent external validation set, and compared with traditional logistic regression, random forest, XGBoost, and Cox proportional hazards models. Based on the prediction results, patients were classified into three groups: low, medium, and high recurrence risk. Utilising a reinforcement learning framework, personalised recommendations were generated for each group regarding follow-up frequency, adjuvant therapy intensification strategies, lifestyle interventions, and psychological support plans.

Results

The model achieved an area under the receiver operating characteristic curve (AUC) of 0.92 on the validation set, significantly outperforming the highest value of 0.83 achieved by traditional models. The attention mechanism revealed that lymph node ratio, Ki-67 and specific image texture features contributed most significantly to the prediction. The low-risk group accounted for 42 per cent, with an actual five-year recurrence rate of 4.7 per cent; the medium-risk group accounted for 38 per cent, with a recurrence rate of 18.3 per cent; the high-risk group accounted for 20%, with a recurrence rate of 44.6%. Recommendations based on reinforcement learning increased treatment adherence by 26% in the medium- and high-risk groups, and reduced recurrence events by 13 per 100 patients in the high-risk group. Unnecessary follow-up examinations were reduced by 31% in the medium-risk group, whilst early intervention was brought forward by approximately four months in the high-risk group.

Conclusion

Artificial intelligence models based on multimodal data and deep learning can significantly improve the accuracy of breast cancer recurrence risk prediction, outperforming traditional clinical tools. The model's interpretability results align with existing biological understanding, enhancing clinical confidence. Individualised treatment plans generated by combining risk stratification with reinforcement learning can optimise the allocation of medical resources, bring forward the treatment window and enable personalised patient management, thereby helping to reduce recurrence rates and minimise overtreatment. Although

validation via prospective randomised controlled trials is still required, this framework provides an efficient and feasible technical pathway for precision treatment of breast cancer and may serve as a model for risk management in other solid tumours. Future research could integrate liquid biopsy and real-time health data to enable dynamic risk monitoring and adaptive plan updates.

No.	Paper ID	Title
12.	N20260327002	Chronic stress promotes breast cancer progression via MTA2 lactylation-mediated destabilization of the NuRD complex

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Abstract—Chronic stress is associated with increased risk of breast cancer metastasis and poor clinical outcomes; however, the underlying mechanisms remain incompletely understood. Here, we demonstrate that chronic stress elevates lactate levels in the tumor microenvironment, thereby promoting lactylation of metastasis associated 1 family member 2 (MTA2) at lysine 460. Mechanistically, chronic stress not only enhances lactate metabolism within the tumor microenvironment, providing sufficient substrate for lactylation, but also activates the β2-adrenergic receptor (β2-AR)/PKA signaling axis, leading to P300 phosphorylation and cooperatively facilitating MTA2 lactylation. This modification weakens the interaction between MTA2 and RBBP7, resulting in destabilization of the NuRD complex and impaired transcriptional repression. This is accompanied by increased H3K9 acetylation and upregulation of genes associated with both tumor progression and metastasis, including MMP16, NRP1, and VCAM1. Importantly, a cell-penetrating peptide specifically targeting MTA2 lactylation effectively inhibits breast tumor growth and reduces lung metastatic burden in chronic stress models. Together, our findings identify protein lactylation especially MTA2 as a key regulatory event in chronic stress-induced tumor progression, providing new insights into the link between tumor epigenetics and chronic stress, and suggesting a potential biomarker and therapeutic target for prognostic assessment and clinical intervention in breast cancer patients under chronic stress.

No.	Paper ID	Title
13.	N20260320004	LCNet: A Compact 3-D Residual Convolutional Network for Pulmonary Nodule Candidate Classication in Chest CT

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Abstract—Early identication of malignant pulmonary nodules remains a central challenge in chest computed tomog-raphy (CT) screening. We present LCNet, a com-pact three-dimensional residual convolutional neu-ral network for candidate-level nodule classication, trained and evaluated on the LUNA16 benchmark de-rived from the LIDC-IDRI collection. The proposed pipeline resamples CT volumes to isotropic spacing, clips Hounseld units to a lung-tissue window, nor-malises intensities to [0, 1], extracts centred 32×32×32 voxel patches, and applies geometric and inten-sity augmentation during training. On the validation split the model achieved 97.53% accuracy, 70.23%precision, 96.88% recall, an F1-score of 81.43 %, and an AUC-ROC of 0.9958. These gures demonstrate that a lightweight 3-D network with roughly 4.8×10^5parameters can produce strong candidate-level dis-crimination when coupled with disciplined prepro-cessing, balanced sampling, and weighted loss opti-misation.

No.	Paper ID	Title
14.	N20260320003	The TIL-TLS Axis in Tumor Immunity: Functional Mechanisms and Clinical Implications

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Abstract—Background: The "TIL-TLS axis" composed of tumor-infiltrating lymphocytes (TIL) and tertiary lymphoid structures (TLS) constitutes a core functional unit in the tumor immune microenvironment. This article aims to systematically elaborate on the immunobiological characteristics of the TIL-TLS axis, explore the development mechanism, structural heterogeneity of TLS, and its clinical value as an immunotherapy target, providing a theoretical basis for optimizing tumor immunotherapy strategies.

Methods: By systematically reviewing relevant domestic and international literature, we integrate and analyze the distribution characteristics, cellular composition of TIL and TLS in various solid tumors, as well as their correlations with patient prognosis and immune therapy response. We focus on sorting out the bidirectional immunoregulatory role of TIL-B cells in TLS, as well as the latest research progress of spatial immunomics technology in revealing the spatial heterogeneity of the TIL-TLS axis.

Results: As ectopically formed lymph node-like structures, TLS regulate local adaptive immune responses by organizing lymphocyte subsets in an orderly manner and promoting B cell maturation. The maturity of TLS (from immature to mature structures with germinal centers) is positively correlated with patient prognosis and closely related to the therapeutic response to immune checkpoint inhibitors. TIL-B cells play a dual role in TLS: effector B cells exert anti-tumor effects by producing high-affinity antibodies and presenting antigens, while regulatory B cells promote immune escape by secreting inhibitory factors such as IL-10 and IL-35. Spatial immunomics technology has revealed the distribution differences of TIL and TLS in the tumor core and invasion front, and this spatial heterogeneity directly affects the evaluation of immunotherapy efficacy.

Conclusions: The TIL-TLS axis is a key regulatory hub of the tumor immune microenvironment, and its structural integrity and functional maturity are crucial for the initiation and maintenance of local anti-tumor immune responses. A deeper understanding of the cellular composition, spatial distribution, and regulatory mechanisms of the TIL-TLS axis can provide an important basis for the development of novel immunotherapy strategies and biomarkers. Future research should integrate multi-omics technologies to further clarify the origin and dynamic changes of TIL B cells, and promote the development of individualized immunotherapy based on the TIL-TLS axis.

No.	Paper ID	Title
15.	N20260320002	Spatial Heterogeneity of Tumor Lactate Metabolism and Metabolic Niches: A New Perspective for Precision Immunotherapy

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Abstract—Background:

To investigate the role of lactate metabolism in tumor immune microenvironment remodeling from the perspective of spatial heterogeneity and metabolic niches, and to summarize how lactate generation, transport, sensing, lactylation, and interactions with other metabolic pathways influence immune suppression, immune exclusion, and therapeutic heterogeneity in cancer.

Methods:

This manuscript is a review article. Based on the literature incorporated in the manuscript, the mechanisms of lactate metabolism in tumor immunity were systematically summarized, including lactate production, transport, sensing, lactylation, and spatial metabolic organization. Differences and common features across multiple tumor types were further compared, and potential intervention strategies targeting LDH, MCT1/4, GPR81, and the acidic microenvironment, as well as their combination with ICIs, CAR-T, oncolytic viruses, and metabolic interventions, were reviewed. The review included 106 references.

Results:

Current evidence indicates that lactate is not merely a glycolytic end product, but a central hub linking metabolic reprogramming, acidic microenvironment, intercellular signaling, and epigenetic regulation in tumors. Lactate is unevenly distributed within tumors and forms spatially heterogeneous metabolic gradients under the combined influence of hypoxia, abnormal perfusion, stromal barriers, and transporter expression differences. These gradients shape lactate metabolic niches composed of tumor cells, cancer-associated fibroblasts, myeloid cells, and lymphocytes in different functional states, thereby affecting CD8+ T-cell infiltration, Treg enrichment, TAM polarization, MDSC expansion, and responsiveness to immune checkpoint therapy. Across tumor types, high-lactate regions are generally associated with low effector immune infiltration, enhanced myeloid suppression, immune exclusion, immune tolerance, and therapeutic resistance, although the dominant cellular sources and molecular axes may differ among cancers. Targeting lactate generation, transport, sensing, and acidic microenvironment has shown potential to remodel local immune niches and improve the efficacy of immunotherapy.

Conclusions:

Lactate metabolism is a key bridge connecting tumor cell state, immune niche organization, and precision immunotherapy stratification. The biological and clinical significance of lactate depends not only on its overall abundance, but also on where it accumulates, which cells produce and utilize it, and whether it spatially

overlaps with hypoxia, low pH, myeloid suppression, and checkpoint signaling. Therefore, lactate gradients and metabolic niches provide an important framework for understanding tumor immune heterogeneity and represent promising targets for precision immunotherapy and combination treatment strategies.

No.	Paper ID	Title
16.	N20260319008	Extrachromosomal Circular DNA (eccDNA) in Aging and Age-Related Diseases: Mechanisms, Clinical Significance, and Future Perspectives

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Abstract—Background: Extrachromosomal circular DNA (eccDNA) is a special form of genetic material. It exists in eukaryotic cells. It is separate from linear chromosomes. Recently, scientists have paid attention to eccDNA. It plays a key role in aging. It is also important for age-related diseases. We need to understand its specific functions.

Methods: We reviewed current research studies. First, we looked at the classification of eccDNA. Then we studied how it forms. We explored the molecular basis of eccDNA accumulation. We focused on the aging process. We analyzed how cellular stressors cause genomic instability. This instability drives the production of eccDNA. We investigated the link between eccDNA and different diseases.

Results: eccDNA has many functions in diseases. First, it promotes the growth of tumors. It amplifies oncogenes. It causes genomic instability. Second, it contributes to neurodegenerative disorders. It accumulates in neurons. This disrupts the balance of cells. Third, it exacerbates metabolic diseases. It regulates the expression of metabolic genes. Fourth, it impairs immune function. It changes immune cell activation. It affects cytokine production. The review highlights the clinical significance. eccDNA is a potential biomarker. It is

useful for early diagnosis. It helps in the prognosis of diseases.

Conclusions: eccDNA is a new mediator. It is critical for aging. It plays diverse roles in pathological processes. Its unique characteristics are promising. It has potential for clinical applications. It helps in disease diagnosis. It is useful for targeted therapy. Future research should focus on specific tissues. We need to develop sensitive detection methods. We must explore therapeutic strategies. These strategies can modulate eccDNA levels. This will improve the prevention of diseases. It will also improve treatment for age-related diseases.

No.	Paper ID	Title
17.	N20260318004	Spatio-Temporal Distribution and Determinants of Lung Cancer Incidence in Guangzhou, 2010–2020

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Abstract—Background: This study investigated the spatiotemporal patterns and localized determinants of lung cancer incidence in Guangzhou at the street/town scale, which remain poorly understood.
Methods: We analyzed 55,698 lung cancer cases (2010–2020) using spatial statistics (Moran's I, spatiotemporal scanning, standard deviation ellipse) and Geographically Weighted Regression (GWR) to explore associations with hospital, factory, and community hospital densities.
Results:Lung cancer presented an upward trend with significant spatial clustering (Moran's I = 0.50, $p < 0.001$). The incidence centroid migrated northeastward, coinciding with industrial relocation. GWR outperformed OLS (ΔAICc = –22.5). Hospital density showed positive associations in the urban core (43.1%), while factory density exhibited positive effects in industrial belts and suburbs (23.8%). Community hospitals displayed limited protective effects (7.5%). Poor model fit in peripheral high-incidence clusters revealed unaccounted local risks.
Conclusion: Medical and industrial impacts on lung cancer are highly spatially stratified. Geographically targeted interventions are urgently needed, especially in historical pollution hotspots and suburban areas with excess risk, to improve precision in cancer prevention and control.
Keywords: Lung Cancer, Incidence, Spatial Aggregation, Spatial Distribution, Environmental Exposure, Geographically Weighted Regression, Kernel Density Estimation

No.	Paper ID	Title
18.	N20260317002	Spatial and temporal distribution of nasopharyngeal carcinoma incidence in Guangzhou from 2011 to 2020

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Abstract—Objective To investigate the spatial and temporal distribution of NPC (nasopharyngeal cancer) incidence in Guangzhou between 2011 and 2020, and develop effective strategies to control NPC in different areas.

Methods NPC incidence data were obtained from the Guangzhou Cancer Registry System. Joinpoint software (version 5.2.0) was used to calculate the total and regional ASIRs and time trends in Guangzhou. Spatial autocorrelation analysis and spatio-temporal scan were used to assess the spatio-temporal cluster distribution of NPC cases. The spatiotemporal evolution of NPC incidence was analyzed by standard deviation ellipse.

Result Guangzhou recorded 10,413 instances of NPC between 2011 and 2020. The age-standardized incidence rate (ASIR) fell from 10.21/105 to 8.43/105. While the incidence of NPC was declining annually on average in urban areas (AAPC=-1.69%) and suburban areas (AAPC=-1.84%), and rising annually in country areas (AAPC=2.80%). Eighteen streets in the southern and western parts of the city, including urban areas and some suburban areas, were the primary hotspots of high NPC incidence. From 2011 to 2020, reported NPC cases showed significant spatial clustering patterns (Moran's $I > 0$ for all, $P<0.05$). The regional and temporal evolution trend indicates that Guangzhou's NPC incidence center is progressively moving northeast.

Conclusions The NPC incidence in Guangzhou from 2011 to 2020 shows an overall decreasing trend and the regional gap was narrowing. The spatial distribution of NPV incidence is aggregated, with the high-risk areas mainly located in the western and southern regions of Guangzhou. Comprehensive prevention and control measures should be taken in high-incidence areas.

No.	Paper ID	Title
19.	N20260316002	Research on Health Management Strategies for Female College Students in Shanxi Province Based on Profile Characteristic Analysis

Abstract—Objective: Taking female college students in Shanxi Province as the study population, this study aimed to identify different health characteristic groups and their differences based on health profiling analysis, explore related influencing factors, and evaluate the effects and stratified response characteristics of health education combined with positive emotion intervention, so as to provide a scientific basis for stratified and classified health management of female college students.

Methods: A convenience sample of female students from 10 universities in Shanxi Province was surveyed from September 2022 to December 2025 using a self-designed online questionnaire. A total of 1,140 valid responses were analyzed. Health profile characteristics were examined to identify typologies. Based on the Health Belief Model, a one-week intervention was implemented among 118 participants, followed by a one-month follow-up. Multiple linear regression and mixed-effects models were used to evaluate changes in outcomes.

Results: Participants were classified into three health types: Health Literacy–Favorable (42.4%), Psychological Distress–Dominant (23.5%), and Severe Health Knowledge Deficiency (34.1%). The latter two groups showed poorer mental health, lower health cognition, and higher behavioral risks. Post-intervention, significant

improvements were observed in lifestyle habits (time effect: $F=21.11$, $P<0.001$) and health cognition indicators ($P<0.05$). Significant time × group interactions were found for breast cancer knowledge, preventive attitudes, and cervical cancer knowledge ($P<0.05$). Anxiety also showed a significant interaction effect ($F=5.63$, $P<0.001$). Improvements were more pronounced in medium- and high-risk groups.

Conclusions: Female college students exhibit distinct health profiles. Internet-based health education combined with positive emotion intervention yields differential benefits, particularly among psychologically distressed and low-cognition groups. Stratified interventions are recommended to optimize health resource allocation and improve outcomes.

No.	Paper ID	Title
20.	N20260415003	Mechanism of Changium smyrnioides in disease prevention and treatment supported by network pharmacology, molecular docking, and experimental validation

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Abstract—Background: Changium smyrnioides is a monospecies of the Umbelliferae family and a second-class protected plant commonly distributed in East and South of China. This study primarily investigates the multi-target mechanisms of its bioactive compounds in disease prevention and treatment, including binding affinity, molecular dynamics, and signal pathway regulation.

Methods: This study uses network pharmacology, molecular docking, dynamic simulation, and bioflim methods to screen for the interactions and signaling pathways of effective complexes.

Results: The results showed the main eight chemical components in Changium smyrnioides have significant

interactions with six different genes, MP1, CA13, APP, LMNA, ERBB2, and MET by validation of biofilm experiment. It can be used to prevent and treat the main seven diseases of tuberculosis, hepatitis, gastritis, coronary heart disease, angina pectoris, scabies, and COVID-19. All the active complex systems exhibited good structural stability with Root Mean Square Deviation (RMSD) values. The therapeutic effects of Changium smyrnioides are mainly associated with HIF-1 and tumor necrosis factor (TNF) signaling pathways, as well as, related to the differentiation of T helper cell 17 (Th17) and the balance of regulatory T cells.

Conclusions: The results indicated that the main six targets with 8 chemicals within Changium smyrnioides are mainly effective against the seven diseases. We will further explore how the chemical components exhibit their efficacy through multiple targets and pathways, providing a scientific basis for further studies and clinical applications.

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No.	Paper ID	Title
21.	N20260310002	Development of a risk prediction model for CIN1 persistence/progression by integrating biomarkers and behavioral factors: a prospective cohort study

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Abstract—Background: Cervical cancer ranks as the fourth most common tumor among women, and its cervical intraepithelial neoplasia（CIN）stage represents a critical target for secondary prevention. Current risk prediction systems for CIN1 progression exhibit significant shortcomings, failing to integrate biological factors with modifiable behavioral factors. This study aims to establish a predictive model for the CIN1 persistence/progression, providing crucial evidence for precision risk stratification.

Methods: A total of 479 patients with histologically confirmed CIN1 were recruited from a married women's community cohort in Jiexiu, Shanxi Province (June-December 2022). All participants underwent repeat pathological examination 12 months later to assess regression, persistence, or progression of CIN1. Participants were randomly divided into a training set (70%, n=335) and a validation set (30%, n=144). Then, clinical and full-factor logistic regression models were constructed respectively based on univariate analysis and multivariate logistic regression analysis. The optimal model was selected according to the AUC value, and the

C-index, Hosmer-Lemeshow test, calibration curve, and decision curve analysis curve were used to evaluate the optimal model.

Results: Univariate analysis and multivariate logistic regression analysis showed that serum folate level, HR-HPV infection, genital cleaning frequency, underwear change frequency, and gynecological history were influencing factors for the CIN1 persistence/progression. The AUC value of the full-factor model (0.815) was higher than that of the validation set of the clinical model (0.696) ($P<0.05$); therefore, the full-factor model was selected as the optimal model. Internal validation of the logistic regression model yielded a concordance index of 0.767, indicating good predictive ability. The Hosmer-Lemeshow test showed good model fit ($\chi^2=1.449$, $P=0.485$), and calibration curves for both training and validation sets closely aligned with the ideal diagonal. The DCA curve showed that the model exhibits a significant improvement in clinical net benefit when the threshold probability ranges from 10% to 60%.

Conclusion: A logistic regression-based model using serum folate, HR-HPV status, genital cleaning frequency, underwear change frequency, and gynecological history effectively predicts CIN1 persistence/progression risk. The developed nomogram enables intuitive visualization of individual risk, while potentially enhancing the accuracy of risk stratification and optimizing management strategies for screen-detected CIN1 patients. It holds particular significance for CIN1 patients, laying a theoretical foundation for the early prevention and personalized management of cervical lesions.