

中药药对及其活性组分抗动脉粥样硬化药理作用研究进展

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摘要: 动脉粥样硬化 (AS) 在我国呈现高发病率与年轻化趋势, 是导致心脑血管疾病致残致死的主要病理基础。尽管化学药治疗有效, 但潜在的不良反应如肝肾损伤限制了其应用。《药治通义》强调“用药之妙, 莫如加减”, 药对以其精妙的配伍, 在 AS 防治中发挥关键作用。系统梳理文献, 将具有抗 AS 作用的中药药对依据功效归纳为活血化瘀、活血解毒、活血益气、祛痰化浊及其他五大类。其作用机制主要包括抗炎、调节脂蛋白代谢、保护血管内皮、抗氧化、抑制泡沫细胞形成、调控细胞凋亡与增殖、抑制斑块内新生血管、抑制平滑肌细胞异常增殖、抗血栓形成及调节肠道菌群结构等。旨在为抗 AS 新药研发及未来研究方向提供理论参考。

关键词: 中药药对; 活性组分; 动脉粥样硬化; 炎症反应; 细胞凋亡; 脂质代谢

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Research progress on pharmacological effects of traditional Chinese medicine pairs and their active components against atherosclerosis

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Abstract: Atherosclerosis (AS) exhibits a high prevalence and a trend towards affecting younger individuals in China, serving as the primary pathological basis for disability and mortality in cardiovascular and cerebrovascular diseases. While Western pharmacological treatments are effective, potential adverse effects, such as hepatorenal toxicity, constrain their application. The classic text "YaoZhiTongYi" emphasizes the precision of drug selection through addition and subtraction. Medicinal pairs (drug pairs), with their sophisticated compatibility, play a pivotal role in AS prevention and treatment. This review systematically categorizes AS-inhibiting TCM pairs based on their therapeutic effects into five major classes: blood-activating and stasis-resolving, blood-activating and detoxifying, blood-activating and qi-tonifying, phlegm-resolving and turbidity-eliminating, and others. Their mechanisms of action primarily encompass anti-inflammation, regulation of lipoprotein metabolism, vascular endothelial protection, anti-oxidation, inhibition of foam cell formation, modulation of apoptosis and proliferation, suppression of intraplaque neovascularization, inhibition of abnormal vascular smooth muscle cell proliferation, anti-thrombosis, and regulation of intestinal flora structure. This work aims to provide a theoretical foundation for the development of novel anti-AS drugs and future research directions.

Key words: traditional Chinese medicine pairs; active components; atherosclerosis; inflammation; apoptosis; lipid metabolism

动脉粥样硬化 (AS) 是一种复杂的血管病变, 病理过程涉及血管平滑肌细胞增殖、内皮损伤、血小板聚集与血栓形成、泡沫细胞产生及巨噬细胞介

导的炎症反应等^[1-3]。作为冠心病和慢性心力衰竭等多种心血管疾病的共同病理基础, AS 相关心脑血管疾病已成为全球主要的致死致残原因^[4]。当前西

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医治疗策略主要包括调脂、抗凝溶栓、扩血管、抗氧化及抗炎等^[3]，然而疗效有限且可能伴随细胞损伤、肝肾毒性及肌病等风险^[5]，亟需探寻更安全有效的防治手段。

相较于现代医学模式，中医药基于整体观念与辨证论治原则，具有独特优势。根据 AS 的临床表现，可将其归属于中医学“脉痹”“心脉癥积”“窠囊内结”等范畴^[6-7]。其斑块形成主要责之于正虚、痰浊、络瘀、毒郁等病理因素^[8]。近年来，从化痰、活血、补虚、清热、解毒等角度论治 AS 的基础与临床研究日益丰富，拓宽了中医防治思路^[9]。

药对（对药）是医家依据药物特性进行配伍，旨在增效减毒、促进溶出吸收、改善代谢，从而产生协同或综合效应^[10]。深入研究药对有助于解析复方的科学内涵^[11]。笔者通过中国学术期刊全文数据库（CNKI）以“动脉粥样硬化 OR AS”AND “对药 OR 药对”为检索式，筛选得到 63 篇相关文献，归纳整理出具有抗 AS 作用的药对 23 对，涵盖临床研究、网络药理学、细胞实验、分子对接、动物实验及代谢组学等多种研究方法。依据其核心功效，将其系统归类为活血化瘀、活血解毒、活血益气、祛痰化浊及其他 5 大类，分别对各类药对功效及作用机制进行分析总结，以期对 AS 防治及新药研发提供依据。

1 活血化瘀类药对

“瘀血”之名首载于《金匱要略》，张仲景详述其脉证并创立诸多活血化瘀方剂，奠定了血瘀证理法方药体系。血瘀既是 AS 的病理产物，也是致病因素，贯穿疾病始终^[12]。“血实宜决”理论指导下的活血化瘀法是治疗 AS 的重要原则^[13]。临床常用代表药对包括川芎-赤芍、川芎-丹参、川芎-当归、丹参-葛根、丹参-山楂、丹参-三七、桃仁-红花等。

1.1 川芎-赤芍

源自王清任《医林改错》诸逐瘀汤。川芎辛温行气活血，为“血中气药”；赤芍苦寒清热凉血止痛，可制川芎温燥之性，二者互补增强活血化瘀之效^[14]。该药对抗 AS 作用机制：（1）调脂稳斑：研究显示^[15-16]，该药对及其活性成分（川芎嗪、芍药苷）可降低 AS 模型动物三酰甘油（TG）、低密度脂蛋白-胆固醇（LDL-C）水平，上调微小 RNA126（miR-126）抑制血管内皮生长因子（VEGF）/血管内皮生长因子受体 2（VEGFR2）通路，抑制斑块内血管新生，稳定斑块。（2）多靶点干预：网络药理

学预测^[17-18]其可能通过芍药苷、黄芩素、 β -谷甾醇等成分干预调控核因子- κ B（NF- κ B）、脂质与 AS 信号通路等，发挥抗炎、抗氧化、抗血管生成作用，核心靶点涉及蛋白激酶 B（AKT1）、肿瘤坏死因子（TNF）、血管内皮生长因子 A（VEGFA）、TP53 等。

1.2 川芎-丹参

丹参长于养血活血（静），川芎偏于行气祛瘀（动），二者动静结合促进气血通畅^[19]。相关中药注射剂在保护内皮功能方面疗效确切^[20]。该药对抗 AS 作用机制：（1）抗炎：邓明玲等^[21]使用丹参川芎汤治疗 48 例老年 AS 患者后发现，AS 患者 C 反应蛋白（CRP）、白细胞介素 1 β （IL-1 β ）、白细胞介素 6（IL-6）、肿瘤坏死因子- α （TNF- α ）、单核细胞趋化蛋白（MCP-1）等促炎因子水平较治疗前有显著改善（ $P < 0.05$ ），且疗效优于常规治疗组。（2）多靶点干预：网络药理学及体外实验研究^[22]表明川芎-丹参药对能减少巨噬细胞泡沫化，抑制 AKT1、细胞外调节蛋白激酶（ERK）1/2 磷酸化及 VEGFA 蛋白表达进而发挥抗 AS 作用。

1.3 川芎-当归

首见于《仙授理伤续断秘方》当归散^[23]。二者配伍活血祛瘀、养血调经，主治血瘀证^[24]。动物实验揭示该药对多途径抗 AS 机制：（1）调脂稳斑：该药对可通过下调富含半胱氨酸 61 蛋白（Cyr61）、血清与糖皮质激素诱导的蛋白激酶 1（Sgk1）、胆固醇 25-羟基氢化酶（CH25H）基因表达继而发挥抗血栓、调节载脂蛋白代谢作用^[25]；通过降低促细胞外基质金属蛋白酶因子 CD147 表达减少细胞外基质降解，稳定斑块^[26]；（2）抑制细胞凋亡：调控 B 细胞白血病淋巴瘤-2（Bcl-2）/B 细胞白血病-淋巴瘤-2 相关蛋白基因（Bax）、血小板-白细胞 C 激酶底物（PLEK）、Polo 样激酶 3（PLK3）等基因表达抑制血管内皮细胞异常凋亡^[27-30]；相关研究同时发现其可抑制内膜增殖细胞核抗原（PCNA）、VEGF 信使核糖核酸（mRNA）、VEGFR-2 mRNA 表达以遏制细胞增殖，并通过抑制主动脉组织细胞色素 C（Cyt-C）表达及主动脉组织半胱氨酸天冬氨酸蛋白酶 3（Caspase-3）mRNA 水平抑制细胞凋亡^[31-33]。（3）抗炎：通过降低 IL-1 β ，促进细胞因子信号转导抑制因子 3（SOCS3）表达，抑制蛋白酪氨酸激酶 2/信号转导因子和转录激活因子 3（JAK2/STAT3）、Toll 样受体 4（TLR4）/NF- κ B 通路蛋白（TLR4、NF- κ B、MyD88）表达及 TNF- α 合成发挥抗炎作用^[34-35]；

(4) 抑制斑块内血管新生: 动物实验证实其可抑制 VEGF/VEGFR-2 表达进而抑制斑块内血管新生^[36], 且高剂量 ($10.4 \text{ g} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$) 效果优于低剂量 ($2.6 \text{ g} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$)^[24-27,36]。

1.4 丹参-葛根

出自《施今墨对药》, 丹参活血祛瘀凉血, 葛根解肌通络生津, 合用活血化瘀、祛瘀生新^[37-38], 为现代治疗心脑血管病常用中药药对。主要通过以下途径发挥作用: (1) 调脂稳斑: 动物实验^[39]表明其提取物可降低脂质过氧化, 抑制炎症, 减小斑块面积, 提高斑块稳定性。(2) 保护内皮细胞: 系列动物实验^[40-43]证实其能纠正血浆载脂蛋白 A1/载脂蛋白 B (ApoA1/ApoB) 异常, 升高血清一氧化氮 (NO) 含量及一氧化氮合酶 (NOS)、超氧化物歧化酶 (SOD) 活性, 恢复内毒素 (ET)/NO 平衡, 发挥抗脂质过氧化、增强抗氧化能力、调节脂代谢、抑制泡沫细胞形成及保护内皮细胞作用。

1.5 丹参-山楂

丹参“功同四物”补血调经, 山楂消食散瘀行气, 二者散收相济祛全身瘀血^[44]。主要通过以下途径发挥作用: (1) 抗氧化及抗炎: 动物模型^[45-46]证实其可显著降低总胆固醇 (TC)、TG、LDL-C、丙二醛 (MDA), 提高高密度脂蛋白-胆固醇 (HDL-C)、SOD、NO 水平, 降低血栓素 B2/6-酮-前列腺素 F1 α (TXB2/PGF1 α) 比值, 通过抗氧化及抑制炎症因子释放干预 AS 形成。(2) 多靶点干预: 网络药理学结合细胞实验^[47]预测并验证其有效组分配伍 (SC121) 可通过多靶点、多信号通路及多生物途径抑制内皮细胞损伤、抗炎及泡沫细胞的形成。

1.6 丹参-三七

三七散瘀止血定痛, 丹参活血化瘀凉血, 配伍增效, 活血通络止痛^[48], 为治疗心血管病中药常用组合。其主要通过下述机制发挥药效: (1) 抗 AS 血管钙化: 动物实验^[49]显示其可减轻小鼠主动脉钙化面积, 降低 Piezo1、Yes 相关蛋白 (YAP)、PDZ 结合基序转录共激活因子 (TAZ) mRNA 表达, 提示其通过抑制 Piezo1/YAP/TAZ 通路延缓 AS 进展。临床制剂丹七片^[50]被证实可通过调节脂蛋白代谢、降血脂、抗脂质过氧化、清除自由基、抑制血小板聚集、调节血管舒缩、保护内皮细胞及抗血栓形成预防 AS。

1.7 桃仁-红花

源自《医宗金鉴》桃红四物汤, 桃仁沉降破瘀力强, 红花升浮行血力胜, 相配破瘀行血之力

倍增^[51-52]。网络药理学、分子对接及动物实验^[53]表明其可通过调节磷脂酰肌醇 3-激酶 (PI3K)/Akt 等通路, 抑制 NF- κ B、Akt、TLR4 等炎症蛋白表达, 并调节血脂改善 AS。

综上, 活血化瘀类药对主要通过抑制经典炎症通路、降低炎症因子表达从而发挥抗炎作用, 同时通过调节脂质、保护内皮、抑制斑块新生血管等机制发挥抗 AS 作用。

2 活血解毒类药对

陈可冀院士团队提出 AS“瘀毒致病”学说, 认为瘀毒内蕴, 遇外因引动可骤发血脉痹阻危急重症^[54]。如《灵枢·痈疽》所述“热盛肉腐成脓”^[55], 瘀毒热结炽盛易致斑块溃破^[56]。活血解毒法在改善 AS 患者血脂及血流动力学方面疗效满意^[57]。常用药对包括川芎-栀子、川芎-黄连、丹参-黄连、黄芪-黄连、黄芩-黄连、人参-虎杖、三七-绞股蓝。

2.1 川芎-栀子

此药对基于《丹溪心法》越鞠丸。栀子泻火除烦凉血解毒^[58-59], 川芎活血行气祛风止痛^[60], 临床用于治疗 AS 疗效良好^[61]。其主要通过下述机制发挥抗 AS 作用: (1) 双向调控 DNA 异常甲基化, 影响下游基因的表达, 研究显示其可缩小兔 AS 斑块面积厚度, 调节异常基因甲基化水平^[62]。体外实验^[61]提示其可能通过提高血小板源性生长因子受体 α 多肽 (PDGFR α)、I 型胶原蛋白 $\alpha 2$ (COL1A2)、纤维连接蛋白 1 (FN1) 基因启动子区甲基化水平影响下游表达。(2) 调脂抗炎: 细胞实验研究^[63]表明其可减少泡沫细胞胆固醇含量, 降低 IL-6、细胞间黏附分子-1 (ICAM-1)、TNF- α 表达。动物实验^[64]证实其能减慢颈动脉脉搏波速度, 降低主动脉内中膜厚度, 减小斑块及坏死核心面积, 减少泡沫细胞, 增加胶原纤维, 调节血脂及炎症因子, 稳定易损斑块, 机制可能与提高 FGFR3 启动子甲基化介导丝裂原活化蛋白激酶/细胞外调节蛋白激酶 (MAPK/ERK) 通路核心蛋白下调, 降低凋亡指数, 减少 Bax、cleaved Caspase-3 表达, 增加 Bcl-2 表达相关。

2.2 川芎-黄连

二者协同增效^[65], 在冠心病治疗中具抗血小板活化、抗炎、稳定斑块作用^[66-67]。网络药理学^[68]提示其可能通过 MAPK 通路抗炎进而抗 AS, 并干预醛固酮调节的钠重吸收及分泌调控血压。

2.3 丹参-黄连

丹参活血凉血安神, 黄连清心除烦泻火, 合用

清心解毒安神^[69]。网络药理学^[69]预测其有效成分(如黄连素-丹参酮ⅡA)可能通过原癌基因酪氨酸蛋白激酶(SRC)、磷脂酰肌醇-3-激酶调节亚基 1(PIK3R1)、磷脂酰肌醇-4,5-二磷酸 3-激酶催化亚基 α (PIK3CA)、细胞分裂周期蛋白 42(CDC42)等多靶点,作用于脂质、PI3K/Akt、VEGF、MAPK、Ras 等多通路降低炎症因子。含该药对的丹黄通脉胶囊治疗毒瘀交结型颈 AS 的 50 例患者的随机对照试验中,其临床疗效确切,可显著改善中医症状积分、血脂,减小内、中膜厚度及斑块面积^[70]。

2.4 黄芪-黄连

黄芪补气升阳,黄连清热燥湿解毒,配伍符合益气活血解毒原则^[71]。丁书文教授创立的黄芪方含此药对,防治 AS 效果显著^[72-73]。动物实验研究^[74]表明其可通过激活 STAT5 信号通路、调节辅助性 T 细胞(Th)17/调节性 T 细胞(Treg)平衡抑制炎症反应,进而抗 AS。

2.5 黄芩-黄连

出自多首经方(如葛根芩连汤、黄连解毒汤)^[75]。黄芩清热燥湿泻火,黄连泻火力厚入血分,相须清利脉道热毒^[76]。系列动物实验^[76-78]证实其对高脂诱导的 AS 小鼠有治疗作用,机制与抑制 NOD 样受体热蛋白结构域相关蛋白 3(NLRP3)炎症小体激活、抑制 Toll 样受体 4(TLR4)-髓样分化因子 88(MyD88)-核转录因子 κ B(NF- κ B)通路,降低巨噬细胞焦亡水平、稳定易损斑块相关。

2.6 人参-虎杖

人参大补元气,虎杖祛风利湿散瘀解毒^[79-80]。该药对抗 AS 的主要机制:(1)抗炎,网络药理学研究^[81]提示人参虎杖复方可能主要作用于炎症靶点调节 IL-1 β 表达。动物实验^[82]证实人参-虎杖(人参 10 g、虎杖 12 g)可通过上调 NIMA 相关激酶(NEK7)DNA 甲基化,降低 NF- κ B 介导的 NEK7 表达,进而抑制 NLRP3 炎症小体活化,降低血清 IL-1 β ,抗炎、抗 AS。(2)调节肠道菌群:代谢组学与菌群研究^[83]提示人参-虎杖药对可能通过影响甘油磷脂代谢、烟酸/烟酰胺代谢通路及调节肠道菌群结构改善 AS。

2.7 三七-绞股蓝

三七散瘀止血消肿^[84],绞股蓝益气健脾清热解毒^[85]。实验证实三七-绞股蓝及其总皂苷配伍具有抗血管重构作用^[86]。家兔实验^[87-88]表明其配伍可调节血脂,改善主动脉病理形态,下调主动脉组织基质金属蛋白酶-9(MMP-9)mRNA 表达,上调基质

金属蛋白酶抑制剂-1(TIMP-1)mRNA 表达,抗 AS 血管重构。

活血解毒类药对抗 AS 机制突出表现在抗炎方面,尤其可抑制炎症小体,同时还可通过调节肠道菌群、调节脂质代谢等机制延缓 AS 进展。

3 活血益气类药对

《医林改错》指出“元气虚则血瘀”^[89]。气虚血弱,脉道不利滞涩成瘀;津血同源,血瘀津停生痰,痰瘀互结胶着脉管终成斑块^[90]。益气活血法治疗 AS 可有效减小内膜中层厚度、斑块面积,改善动脉狭窄及血流速度^[91]。代表药对包括黄芪-当归、人参-三七、人参-川芎、人参-丹参。

3.1 黄芪-当归

经典气血配伍,源自当归补血汤^[92]。黄芪为补气之最,当归养血活血,合用益气活血,治气虚血瘀^[93-94]。其主要通过下述机制发挥抗 AS 作用:(1)抑制细胞凋亡,动物实验^[95]显示其配伍能抑制血管壁细胞凋亡和焦亡相关因子激活,减轻炎症反应,延缓 AS 病变。(2)抗炎:研究^[96]证实其及补阳还五汤能抑制血管壁 TLR4/MyD88/NF- κ B 炎症通路,抑制巨噬细胞 M1 型极化,减轻血管炎症,且黄芪-当归是补阳还五汤抗 AS 血管炎症的主要药效物质。(3)调节脂质代谢,相关实验^[97]表明其有效成分可调控脂质代谢相关蛋白表达,干预脂质代谢保护 AS 小鼠,高剂量效果更优。

3.2 人参-三七

人参补气,三七补血,味同而功亦等,故称人参三七,为中药之最珍贵者^[98]。网络药理学研究^[99]提示人参-三七可能主要通过调节 AS 进程中的炎症反应环节发挥作用,同时调节脂质浸润、平滑肌细胞增殖等过程。

3.3 人参-川芎

人参大补元气,川芎行气活血祛风^[100-101],相配扶正活血祛瘀。其主要通过下述机制发挥抗 AS 作用:(1)调节脂质代谢,网络药理学研究^[102-103]预测其有效成分可能通过过氧化物酶体增殖物激活受体 γ (PPAR γ)/过氧化物酶体增殖物激活受体 γ 辅激活因子 1 α (PGC-1 α)/解偶联蛋白 1(UCP1)信号通路促进白色脂肪棕色化,改善 AS;并可能通过此机制降低慢性间歇性低氧复合肥胖胰岛素抵抗诱导的 AS 小鼠体重、血糖、LDL-C、TG 水平。

3.4 人参-丹参

人参甘缓入气分,丹参苦通入血分,相使益气

活血, 治气虚血瘀^[104]。网络药理学研究^[105]表明其有效组分配伍后主要通过调节脂质代谢、炎症反应、内皮功能、血小板聚集等发挥抗 AS 作用。

综上所述, 活血益气类药对侧重抑制细胞凋亡、改善免疫平衡等机制抗 AS。

4 祛痰化浊类药对

AS 本虚标实, 病机首重“脾虚”。脾虚气血生化乏源, 气虚血瘀; 脾失健运, “膏浊”内生, 痰浊互结壅塞脉道成斑块^[106]。《金匱要略浅注》言“痰浊宜荡涤”^[107], 祛痰化浊法切中病机。常用药对为瓜蒌-薤白、泽泻-白术。

4.1 瓜蒌-薤白

源自《金匱要略》治胸痹经方(瓜蒌薤白白酒汤等)^[108]。其主要通过下述机制发挥抗 AS 作用: (1) 调节脂质代谢, 网络药理学联合体外实验^[109]提示其可能通过抑制 MAPK 信号通路抗 AS。代谢组学与网络药理学^[110]证实配伍后能更强影响 AS 小鼠亚油酸代谢、甘油磷脂及花生四烯酸代谢, 协同改善脂质紊乱及炎症。动物实验^[111]表明其可调节 AS 小鼠主动脉不同阶段 NLRP3 炎症小体表达。肝脏代谢组学^[112]发现其可干预肝脏卵磷脂胆固醇酰基转移酶(LCAT)、分泌型磷脂酶 A2 (sPLA2)、乙醇胺磷酸转移酶 1 (EPT1)、碱性神经酰胺酶 1 (ACER1) 等关键酶表达, 调节甘油磷脂及鞘脂代谢紊乱。(2) 激活血管平滑肌细胞(VSMCs) 自噬, 研究^[113]还揭示其可能通过抑制上游 PI3K/Akt, 间接抑制下游哺乳动物雷帕霉素靶蛋白(mTOR) 活化, 激活 VSMCs 自噬, 抑制 AS 斑块发展。

4.2 泽泻-白术

出自《金匱要略》泽泻汤。泽泻利湿泄热化浊, 白术健脾运湿, 为健脾祛痰经典组合^[114]。其主要通过下述机制发挥抗 AS 作用: (1) 调节脂质代谢, 动物实验^[115]表明其配伍可能通过 B 族 I 型清道夫受体(SR-BI)-沉默信息调节因子 1 (SIRT1)-肝 X 受体 α (LXR α)-ATP 结合盒转运体 A1 (ABCA1)/SR-BI 通路促进胆固醇逆转运, 减轻肝脏沉积, 改善炎症, 抑制血管壁增厚和狭窄。(2) 抑制促 AS 菌群代谢产物生成: 另一研究^[116]显示含此药对的泽泻饮(泽泻 20 g、白术 20 g、鹿衔草 10 g) 可通过降低肝脏 FMO3 活性, 减少血浆三甲胺(TMA) 向促 AS 因子氧化三甲胺(TMAO) 的转化。

综上, 祛痰化浊类药对主要侧重于通过干预胆固醇逆转运调节脂质代谢、抑制 VSMCs 增殖等机

制改善 AS。

5 其他类药对

5.1 黄芪-党参

黄芪补肺脾升阳固表, 党参健脾益肺补血生津, 配伍补益不滋腻^[117]。药理学结合实验^[118]表明其可能通过调控 TP53 因子促进 AS 模型细胞增殖, 抑制其凋亡。

5.2 金银花-玄参

出自《石室秘录》^[119]四妙勇安汤。玄参泻火解毒滋阴凉血, 金银花清热解毒, 气血两清^[120]。网络药理学^[121]预测其有效成分可直接作用于 MAPK1、JUN、TP53、TNF 等靶点, 并可能通过 MAPK、TNF、Toll 样受体等信号通路抑制炎症、保护内皮、促进胆固醇逆转运及代谢。

5.3 三棱-莪术

治疗血瘀证常用组合。三棱“血中之气药”, 莪术“气中之血药”, 合用行气活血祛瘀^[122]。网络药理学及分子对接^[123]提示其可能主要通过 C 型凝集素受体信号通路、癌症通路等发挥抗炎、抗 AS 作用。相关研究^[26-27]显示其疗效亦具剂量依赖性, 且同等剂量下对某些指标改善优于川芎-当归组。

综上, 针对临床常见的 5 大类 23 种治疗 AS 药对进行分类, 其抗 AS 机制总结见图 1。

上述药对在临床 AS 治疗及相关研究的来源方剂及临床制剂汇总见表 1。

6 结语与展望

心血管疾病仍是全球主要死因, AS 是其核心病理环节^[156]。全球每年约 2 000 万人死于 AS 相关疾病^[157]。近年来, AS 在我国呈现高发病率与年轻化特点^[158]。研究发现多种中药成分及复方在肠道菌群、细胞程序性死亡、非编码 RNA(ncRNA) 等领域展现出治疗潜力^[159]。历代医家不仅善用专方, 更精于配伍药对, 使药精力专纠邪之偏^[160]。

AS 病因病机复杂, 现代医家普遍认为其核心在于虚、瘀、痰、毒, 呈进行性发展: 脾胃虚弱, 运化失司, 气血乏源; 过食肥甘, 碍脾生湿成痰; 痰瘀互结, 日久化毒, 终成斑块^[161]。因此, 临床多从虚、热、瘀、毒等角度施治。通过系统收集整理文献, 归纳出 5 类 23 对具有抗 AS 作用的中药药对, 其功效分布与 AS 病因病机高度契合。

中医药的精髓在于对于不同药物的配伍应用, 不同的配伍方式或比例可能会产生截然相反的药

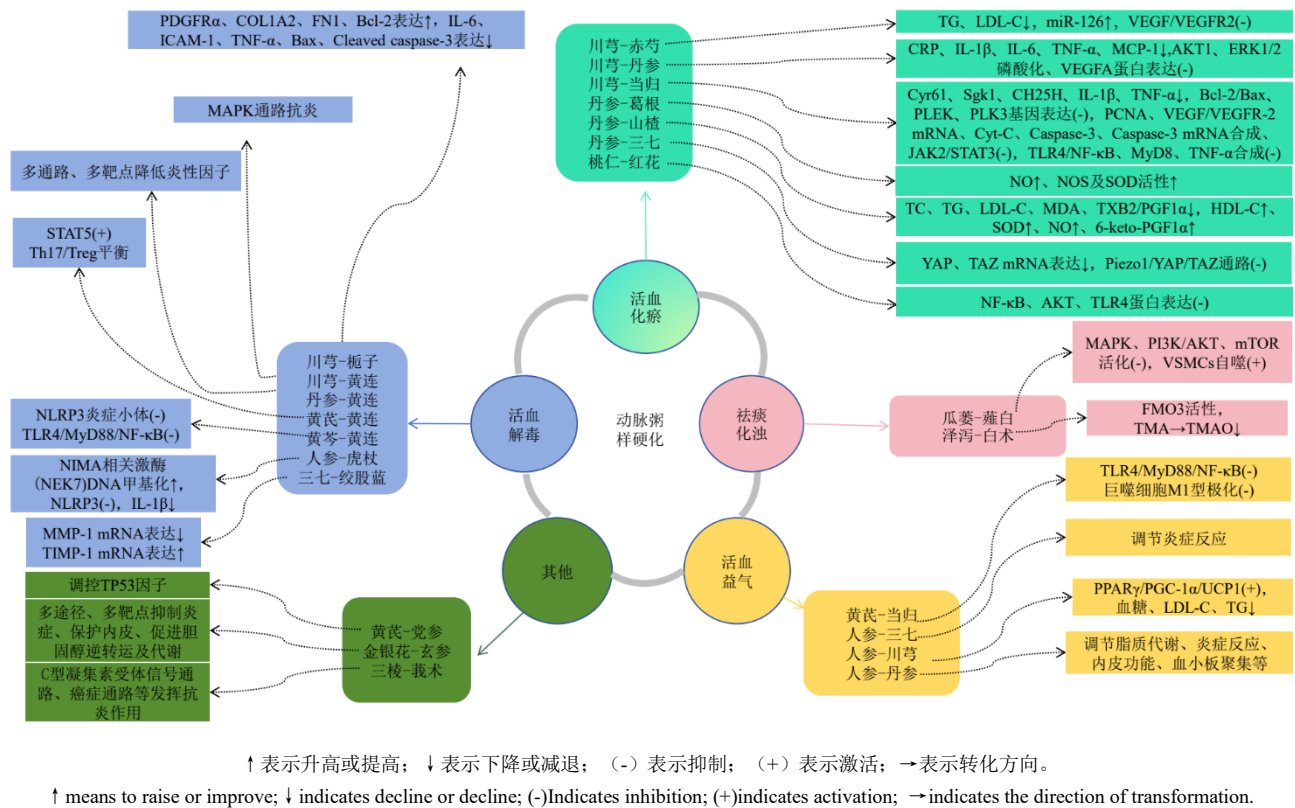


图 1 不同功效类别抗 AS 药对主要机制

Fig. 1 Main mechanism of anti-AS drug pairs with different efficacy classes

表 1 治疗 AS 中药药对的来源方剂与临床制剂

药对	方剂或临床制剂
川芎-赤芍	当归芍药散、芍药胶囊、调脂降浊方 ^[124] 、芩芍左归饮 ^[125] 、活血解毒I号方 ^[14] 、补阳还五汤、新血府逐瘀软胶囊、活血降脂灵 ^[126]
川芎-丹参	丹参通脉方 ^[127] 、护心康片 ^[128] 、复方丹参川芎中药配方颗粒 ^[119]
川芎-当归	当归芍药散、芩芍左归饮 ^[125] 、桃红四逆汤 ^[129]
丹参-葛根	心痛泰颗粒 ^[130] 、丹参通脉方 ^[128]
丹参-山楂	心痛泰颗粒 ^[130] 、复方三七颗粒 ^[131] 、丹参山楂降脂丸 ^[132] 、山楂降脂丸 ^[133] 、丹山合方 ^[134] 、血脉宁颗粒 ^[135]
丹参-三七	心痛泰颗粒 ^[130] 、复方三七颗粒 ^[131] 、五参脉脉汤 ^[136] 、护心康片 ^[128] 、复方丹参滴丸、星蛭软脉颗粒 ^[137] 、银丹心脑通软胶囊
桃仁-红花	补阳还五汤、桃仁红花煎、血府逐瘀汤
川芎-栀子	栀子川芎胶囊 ^[138]
川芎-黄连	清心解瘀颗粒 ^[139]
丹参-黄连	丹黄通脉胶囊 ^[70]
黄芪-黄连	—
黄芩-黄连	黄连解毒汤、葛根芩连汤、三黄片
人参-虎杖	人参虎杖复方 ^[81]
三七-绞股蓝	银丹心脑通软胶囊
黄芪-当归	当归补血汤、降脂消斑片 ^[140]
人参-三七	参七粉 ^[141]
人参-川芎	益气活血复方 ^[142]

表 1 (续)

药对	方剂或临床制剂
人参-丹参	参红通络颗粒 ^[143] 、养心通脉片 ^[144]
瓜蒌-薤白	瓜蒌薤白半夏汤、瓜蒌薤白白酒汤、参芪瓜蒌薤白半夏汤 ^[145] 、瓜蒌半夏中和汤 ^[146] 、护心康片 ^[128] 、化瘀消斑方 ^[147] 、丹蒌片、舒脉胶囊 ^[148]
泽泻-白术	当归芍药散、脂必泰胶囊、泽泻饮 ^[116]
黄芪-党参	健脾通脉方 ^[149] 、参七冠心方 ^[150] 、养心舒脉颗粒 ^[151]
金银花-玄参	四妙勇安汤、四妙通络颗粒 ^[152]
三棱-莪术	心脉康片 ^[153] 、芪棱汤 ^[154] 、调气参夏消积合剂 ^[155]

理作用。药对的配伍应用，具有化学成分复杂、成分与质量有一定模糊性等特点^[162]。在本文中，涉及的部分中药药对的具体用药剂量或使用比例并未明确予以说明，作为临床用药常用的配伍形式，研究药对配伍比例，明确不同疾病条件的最优配伍关系，是药对临床安全、合理用药的迫切需求，也应是药对未来研究的重点^[163]。伴随着中医药现代化研究的不断深入，王永炎和张伯礼院士提出了以组分配伍研制现代中药的有效组分配伍研究新策略^[164]。该策略兼顾了传统中医药配伍整体观念与辨证施治的精髓，又有精准医学体系下药效物质明确、靶点相对清楚的优势，其“配伍艺术”与“精准药物”相结合的巨大潜力，必将成为中医药现代化研究的关键点、突破点^[165]。

综上所述，中药药对主要通过多靶点、多途径（抗炎、调脂稳斑、保护内皮、抗氧化、抑制泡沫细胞、调控细胞凋亡/增殖、抑制斑块内血管新生及平滑肌增殖、抗血栓、调节肠道菌群等）发挥抗 AS 效应。深入探究中药药对的科学内涵，对于研发新型抗 AS 药物及指导临床精准用药具有重要意义。

利益冲突 所有作者均声明不存在利益冲突

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