民族药治疗动脉粥样硬化的研究进展

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摘要: 动脉粥样硬化 (atherosclerosis, AS) 是众多心脑血管疾病的共同病理基础, 民族医药凭借其独特的理论体系和丰富的药物资源, 在AS治疗中展现出独特优势。本综述系统梳理了不同民族医学对AS的病机认识及常用药物, 揭示“生理失衡”为其共性病机; 并重点从改善肠道菌群、调控脂质代谢、拮抗氧化应激、抑制炎症反应、调节血管稳态等多维度, 阐释了民族药的多靶点作用机制。此外, 本文进一步总结了现有临床证据, 剖析了当前研究在理论现代化、机制系统解析与临床证据升级方面的核心问题, 并对未来研究方向作出展望。

关键词: 动脉粥样硬化; 民族药; 动态平衡; 多靶点; 作用机制

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Advances in Chinese ethnomedicine for the treatment of atherosclerosis

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Abstract: Atherosclerosis (AS) is a common pathological basis for cardiovascular and cerebrovascular diseases. Chinese ethnomedicine, with its distinct theoretical frameworks and abundant resources, shows unique potential in managing AS. This review systematically synthesizes the pathogenesis of AS and corresponding therapeutic strategies from various Chinese ethnomedical systems, revealing that "physiological imbalance" serves as a common underlying mechanism. We detail the mechanisms of these ethnomedicines, including improving the composition of gut microbiota, regulating lipid metabolism, mitigating oxidative stress, suppressing inflammation and modulating vascular homeostasis. Available clinical evidence is critically evaluated. Key challenges are discussed, such as the need for theoretical modernization, deeper mechanistic studies, and more rigorous clinical validation. Promising future research directions are also outlined.

Key words: atherosclerosis; Chinese ethnomedicine; dynamic balance; multi-target; mechanism of action

动脉粥样硬化 (atherosclerosis, AS) 是一种多因素介导的慢性动脉炎症性疾病, 以动脉壁纤维脂肪性斑块形成为主要特征, 随着病变进展可引发心肌梗死、缺血性卒中及外周动脉疾病等严重临床后果, 这些病症也是全球范围内导致死亡的主要原因[1]。虽然现代医学在AS发病机制研究及靶向药物研发方面已取得显著进展, 但现有疗法如药物治疗与支架辅助介入仍面临一定不良反应与术后风险[2]。在此背景下, 强调整体观念与辨证论治的传统医学备受关注[3]。民族药作为我国各少数民族在长期历史实践中形成的传统医药体系, 融合了各民族的文化传统、地域特色与临床医疗经验, 在防治慢性复杂性疾病方面展现出独特价值[4]。近年来, 随着现代研究方法的不断引入, 民族药在抗AS领域已积累了一系列研究进展[5-7], 其多成分、多靶点、多环节的整体干预特点逐渐受到认可。然而, 该领域的研究较为零散, 尚未形成系统化的理论框架与完整的证据链体系。为此, 本文系统梳理我国主要民族

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医学对AS的理论认识, 整合其药物作用机制与临床实践方面的进展, 分析当前存在的关键问题, 并对未来研究方向提出展望, 以期为民族药的深度挖掘与现代化发展提供学术参考。

1 民族医学治疗AS的基础理论

尽管传统民族医学体系中并无“动脉粥样硬化”这一现代医学病名的直接对应概念, 但根据其临床表现, 各民族医学可将其纳入自身的疾病分类体系, 具有独具特色的病因病机理论与药物。

1.1 蒙医学 蒙医根据AS的临床表现与并发症将其主要归入“黑脉病”的范畴。《蒙医内科学》认为该病因以三根理论中赫依 (气)、希拉 (火)、巴达干 (土和水) 之间的动态平衡为核心, 当三根失调, 尤以“普行赫依”功能受损为关键环节, 可导致“黑脉病”发生[8]。在此病理基础上, 人体胃火衰败可引发“糟粕不消化症”, 进而演变为“精华不消化症”。未被正常吸收的精华物质与异常增生的巴达干黏液相结合, 渗入“黑脉” (血管) 并沉积于管壁, 最终形成脂质斑块[9]。此外, 饮食不节及劳逸失度等外在因素, 亦可导致恶血希拉偏盛损伤脉络、巴达干黏液阻滞气血运行, 或赫依引发血脉僵化, 从而成为黑脉病的重要诱因[10]。蒙医在黑脉病的治疗上以调理三根、促进清浊分化、改善气血运行作为核心治则, 进而排除“沃赫”脂质构成的消化代谢障碍, 保利尔胶囊[11]、五味沙棘散[12]、通拉嘎-5[13]、清血八味片[14]、朱日亨滴丸[15]、三子散[16]等多种有效方药在AS治疗中展现出一定潜力。

1.2 维吾尔医学 维医学根据AS的临床表现, 将其归属于“心压感怔忡”“心绞痛”等范畴[7]。维吾尔医学体液学说认为, 人体内的血液质、胆液质、黏液质及黑胆质是肝脏在自然四元素 (火、气、水、土) 与个体气质的共同作用下, 对营养物质进行转化而产生的[17]。当各种内外不良因素影响人体, 导致体液 (多为胆液质和血液质偏盛) 失衡时, 便会引发心脏气质失调。异常体液随之沉淀于血管壁, 导致心血管硬化、阻滞, 进而引发心肌缺血、缺氧, 最终影响心脏的正常生理功能。治疗上遵循四大物质理论, 以“成熟与清除异常体液、调节体液平衡”为总纲, 具体通过芳香开窍、活血通络、解淤散结、清血凉血以排除腐败血液等治法, 达到降血脂、降血黏度、改善周围循环的目的。常见的维成药有益心巴迪然吉布亚颗粒、养心达瓦依米西克蜜膏、爱维心口服液等[18], 此外, 香青兰[19]、新塔花[20]等单味药材亦被证明具有确切的药理疗效。

1.3 藏医学 藏医学将AS相关症状归于“匝滞布” (脉衰)、“萨滞布” (脑卒中)、“匝那乃” (血管疾病) 等范畴[21]。藏医认为所有疾病的内因在于三因理论中隆 (气性)、赤巴 (火性)、培根 (水土性) 的失衡[22,23]。对于“匝那乃”, 其病因是隆、赤巴、培根、坏血、黄水等紊乱侵入脉道, 或各种热邪 (伤热、紊乱热、疬疫等) 犯脉。脉被视为“机体之根”, 分为黑脉 (血管) 和白脉 (神经)。动脉被称为“红命脉”, 是生命之隆 (气) 与血混合运行的通道。由于脉中血液属热, 故脉病多属热病。在治疗上, 藏医强调整体观念与辨证论治, 遵循“顺隆才能脉通, 脉通才能血行”的理念。其治疗原则以调和气血、通经活络、平衡三因、清热凉血化瘀为主[21], 常用方剂与药材包括三味檀香胶囊[24]、然那桑培丸[25]、藏红花[26]、滇结香[27]、余甘子[28]等。

1.4 傣医学 在傣医学中, 关于动脉粥样硬化的研究相对匮乏, 有学者将其归属于“心慌、心悸、胸痛”范畴[29]。“四塔”“五蕴”理论是傣医学的核心理论, “四塔”是构成人体和自然界的4种重要元素, 即风、火、水、土。疾病的发生是因为内在的“四塔”功能失调, 加之感受外在的各种帕雅 (病邪)[30]。其AS的核心病因是“食物毒”, 即过食导致“四塔”失衡, 毒邪内蕴而发病。治疗遵循“先解后治”原则, 先以“雅解”方药清除病根, 理顺病理之道, 再给服治病之药, 恢复机体平衡。基于此理论, 代表性方剂“雅解益栽 (心) 方”在初步研究中已展现出明确的抗动脉粥样硬化活性[29]。

1.5 壮医学 在壮医学理论中, AS为“龙路病”范畴。所谓龙路病, 是指因各种病因造成龙路不通, 气血运行受阻, 进而使内脏、筋骨及肌肉失去濡养; 或因龙路功能失常, 血行偏离常道, 出现孔窍出血等症候的一类疾病[31]。针对该病的治疗, 需根据“毒”与“虚”之病机本质, 辨别“堵”和“损”的具体病位, 运用药物或非药物疗法, 以解毒补虚、疏通龙路、控制损伤为核心治法, 从而消除龙路壅滞, 恢复气血通畅[32]。另有学者将AS归属于“瘀毒病”范畴[33]。瘀毒多为内生之邪, 常因饮食失宜、作息紊乱、情志不畅、过劳过逸, 或久病正虚、年老脏腑功能衰退等, 导致瘀毒蕴结体内, 阻滞龙路与火路, 造成气血失衡, 天、地、人三气失于协调而发病。基于瘀毒致病的认识, 临床治疗强调以药物为主, 借助“三道” (谷道、水道、气道) 途径清除瘀毒。邪毒祛除, 则正气得以恢复, 机体逐步康复。研究显示, 部分壮药如壮通饮[34]、绞股蓝[35]、鸡血藤[36]、水蛭[37]等, 均对动脉粥样硬化具有改善作用。

1.6 苗医学 Liu等[5]基于苗医学的“交环学说”, 将AS归为“交环上亢下亏症”范畴。该理论认为, 人体上下脏腑之间通过“交环”相互联系、滋养与制约。若因营养不足或主司精神调节的“脑架”功能失调, 可致下交环 (主司消化吸收) 功能亏损, 难以充分供养上交环 (主司心肺功能)。上交环因而出现功能偏亢, 表现为

多饥、多食、多饮等代偿性反应, 而下交环消化吸收负荷过重, 形成恶性循环, 整体失衡最终导致疾病发生。治疗上应以“帮交环”为本, 恢复其协调功能; 以“赶毒、排毒、攻毒”为标, 清除脉道中的积毒; 并配合“通气散血”以疏通筋脉壅阻[38]。灯盏生脉胶囊[39]、银丹心脑通软胶囊[40]等苗药在改善AS方面均显示出良好疗效。

1.7 其他 相较于藏、蒙、维等医学体系, 其他民族医学对AS的系统性药理阐述相对有限, 仍有研究表明部分方药具有潜在价值。如朝医方麻黄定痛汤[41]和太阴调胃汤[42]可调节AS大鼠的脂代谢与氧化应激; 彝药塔若散[43]具有调节血脂、炎症因子及协调基质金属蛋白酶9 (matrix metalloproteinase-9, MMP-9)/金属蛋白酶组织抑制因子1 (tissue inhibitor of metalloproteinase-1, TIMP-1) 的作用。此外, 佤药聂良给在临床中亦显示出良好的AS症状改善潜力[44]。

综上, 虽然各民族医药基于其独特的理论体系对AS的认识表述各异, 但其核心理念存在共通之处, 即普遍秉持着一种追求人体内在动态平衡的哲学思想, 与中医“阴阳平衡”理论在哲学层面上高度契合[45]。尤其蒙医的“三根”、藏医的“三因”、维医的“体液”与傣医的“四塔”这四大民族医学体系, 皆以自然元素 (气、木、水、火、土) 为理论构建基础, 具有明显的理论同源性 (图1)。这些医学体系均认为, 核心系统的失衡是产生“糟粕”“异常体液”“毒邪”等病理产物的根源, 进而导致脉络阻滞。据此, 在治疗上各民族医学均以恢复机体平衡为核心, 一方面调理本体以恢复机体平衡 (如调理三根、平衡体液), 另一方面清除病邪以疏通脉道 (如化痰祛瘀、清热解毒、清除异常体液、疏通龙路)。这种跨文化的共通之处, 既源于各民族在长期应对疾病过程中对人体生理病理现象的客观观察与朴素总结, 也反映了人类生命活动与疾病演变规律的普遍性。此外, 地理邻近与文化交融、相似的疾病谱系以及天然药物资源的类同性等因素, 也促进了不同民族医学在认知框架与应对策略上的趋近。其中所蕴含的“整体观”与“辨证论治”思想, 不仅彰显了各民族医药之间的理论共鸣, 也为AS的治疗体系贡献了独特且丰富的思路与药物资源。

2 民族药治疗AS的核心作用机制

为系统阐明民族药治疗AS的药理学基础, 本文通过梳理各民族医学体系中的相关药物及其具体作用途径, 在此基础上归纳出六大核心作用机制, 如图2所示, 即改善肠道菌群、调节脂质代谢、拮抗氧化应激、抑制炎症反应、调节血管稳态及稳定斑块。这些机制相互关联, 构成一个多维协同的作用网络, 揭示了民族药多靶点、多途径的作用特点。

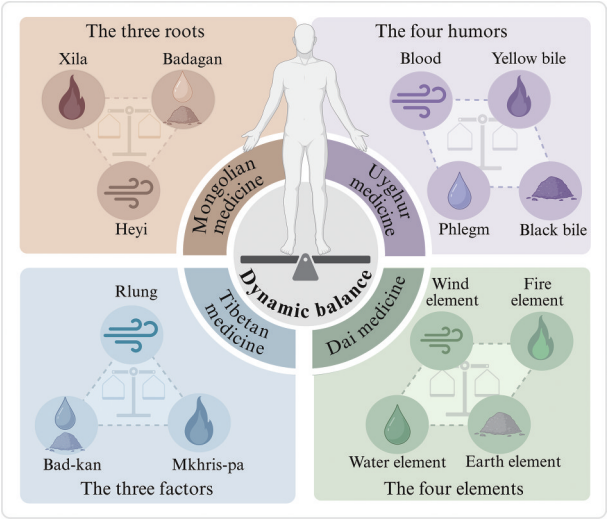


Figure 1 A shared foundation across four Chinese ethnic medical systems: Natural elements and the pursuit of physiological dynamic balance

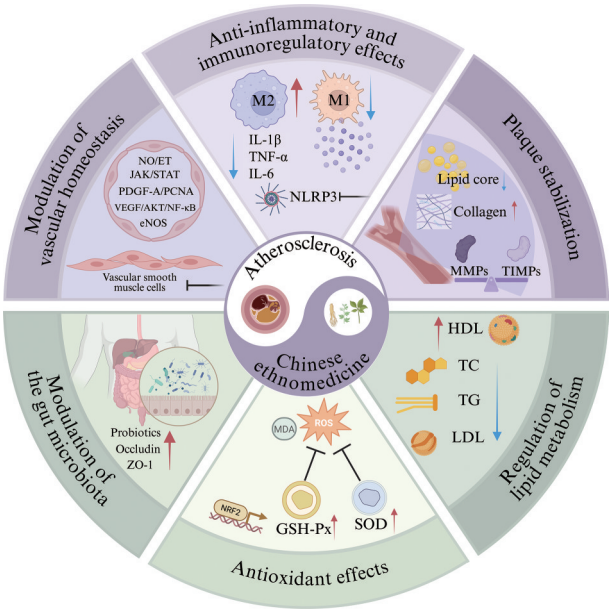


Figure 2 The mechanism of action of Chinese ethnomedicine in treating atherosclerosis

2.1 调节肠道菌群–代谢轴 “肠道–血管轴”在AS发病中的作用日益受到关注, 菌群紊乱及其代谢失调是其核心机制[46]。多项研究揭示了民族药通过靶向此轴发挥抗AS作用的潜力。在调控菌群结构方面, 苗药粗壮女贞水提物可增加放线菌门、乳酸杆菌和双歧杆菌丰度, 降低普雷沃氏菌属比例[47]; 蒙药通拉嘎-5能提高罗斯氏菌、优杆菌和乳酸杆菌等有益菌相对丰度[13]; 藏药滇结香则可富集乳酸杆菌、普拉梭菌和嗜黏蛋白阿克曼菌等有益菌[27]。此外, 蒙药沙棘黄酮可改善菌群alpha多样性, 调节厚壁菌门与拟杆菌门等关键菌门的

相对丰度[48]; 而蒙药冠心七味滴丸[49]、壮药广山楂叶总黄酮[50]与田蓟苷[51]则被证实可显著调节拟杆菌门与厚壁菌门的相对丰度。在代谢物调控方面, 苗药粗壮女贞水提物[47]直接减少盲肠内容物中三甲胺 (trimethylamine, TMA) 生成, 并降低血清三甲胺*N*-氧化物 (trimethylamine *N*-oxide, TMAO) 水平, 维吾尔药香青兰总黄酮亦可降低血清TMAO水平[19]; 蒙药通拉嘎-5通过菌群调控促进胆固醇代谢从而抑制AS的发生[13]。在肠道屏障功能维护上, 藏药滇结香[27]与松萝[52]可上调回肠组织紧密连接蛋白闭锁小带蛋白-1 (zonula occludens-1, ZO-1) 与occludin表达, 增强肠黏膜屏障完整性。值得注意的是, 滇结香的抗AS效应经粪菌移植实验证实由肠道菌群介导[27], 为阐释菌群在民族药治疗AS的作用机制中提供了有力证据。

2.2 调控脂质代谢与胆固醇逆向转运 脂质代谢紊乱作为AS形成的核心驱动因素, 直接导致脂质在血管壁沉积[53]; 因此, 改善脂质代谢失衡已成为多数民族药延缓AS进程的关键。研究表明, 多种民族药均能通过下调总胆固醇 (total cholesterol, TC)、甘油三酯 (triglyceride, TG)、低密度脂蛋白胆固醇 (low-density lipoprotein cholesterol, LDL-C) 水平, 上调高密度脂蛋白胆固醇 (high-density lipoprotein cholesterol, HDL-C) 水平, 或调控血脂相关蛋白表达, 改善脂质代谢紊乱以控制AS的脂质沉积进程[20,28,35,40,43,54-63]。在机制上, 蒙药保利尔胶囊通过激活肝脏X受体*α* (liver X receptor alpha, LXR*α*), 显著上调三磷酸腺苷结合盒转运体A1 (ATP-binding cassette transporter A1, ABCA1)、三磷酸腺苷结合盒转运体G5/G8 (ATP-binding cassette transporter G5/G8, ABCG5/ABCG8) 及胆固醇7*α*-羟化酶 (cytochrome P450 family 7 subfamily A member 1, CYP7A1) 的表达, 直接促进胆固醇向HDL的转运及其向胆汁与粪便中的排泄, 进而改善AS小鼠的病理进程[11]。值得注意的是, 其活性成分胡椒碱与阿托伐他汀或依折麦布 (两种广泛使用的降胆固醇和抗动脉粥样硬化药物) 联用展现出协同增效作用。蒙药通拉嘎-5[13]可减少小鼠血管内膜及主动脉根部脂质沉积面积, 减轻肝脏组织脂肪。其作用机制可能与上调肝脏中胆固醇代谢相关蛋白的表达有关, 包括过氧化物酶体增殖物激活受体*α* (peroxisome proliferator-activated receptor alpha, PPAR*α*)、清道夫受体B1 (scavenger receptor B1, SR-B1)、载脂蛋白A-I (apolipoprotein A-I, Apo-AI) 的表达; 其中, PPAR*α*可通过多途径来发挥抗AS作用[64]。此外, 蒙药冠心七味片[65]的含药血清可调控过氧化物酶体增殖物激活受体*γ* (peroxisome proliferator-activated receptor gamma, PPAR*γ*)/ABCA1/ABCG1脂代谢, 进而改善动脉粥样硬化; 而蒙药清血八味片[14]的作用机制则主要涉及甘油三酯、甘油磷脂及胆固醇酯代谢途径。藏药三味檀香散[24]可调节脂肪组织巨噬细胞的脂质代谢功能, 特别是增强溶酶体活性和胆固醇外流能力, 显著提升餐后HDL-C水平, 从而发挥抗动脉粥样硬化作用。其活性成分 (如七叶苷、桑色素) 被认为是潜在的溶酶体激活剂, 具有开发为新型调脂药物的潜力。与此同时, 藏药红景天中的红景天苷通过逆转AS小鼠中多种异常表达的脂质分子 (磷脂酰胆碱、磷脂酰丝氨酸、烷基二酰基甘油及烯基二酰基甘油), 使其恢复至正常水平[66]。在载脂蛋白E敲除 (apolipoprotein E knockout, *ApoE*$^{-/-}$) 小鼠模型中, 苗药粗壮女贞水提液通过上调肝脏SR-B1与回肠ABCG8的表达, 并下调回肠尼曼−匹克C1样1 (Niemann-Pick C1-like 1, NPC1L1) 的表达, 改善血脂代谢紊乱, 其作用机制可能涉及对胆固醇吸收与外排平衡的调控[47]。在维吾尔族民族药药对研究中, 榄桲−罗勒总黄酮联合用药显著减轻了棕榈酸 (palmitic acid, PA) 诱导人脐静脉内皮细胞 (human umbilical vein endothelial cells, HUVEC) 脂质损伤, 并降低了高胆固醇诱导的斑马鱼体身体质量指数 (body mass index, BMI), 减少了脂肪沉积[67]。除此之外, 源自维吾尔药香青兰的田蓟苷能够经由激活转录因子固醇调节元件结合蛋白2 (sterol regulatory element-binding protein 2, SREBP2), 有效上调肝细胞低密度脂蛋白受体 (low-density lipoprotein receptor, LDLR) 的表达, 进而增强其对LDL-C的摄取与清除能力, 展现出抗AS的潜力[51]。

2.3 拮抗氧化应激 机体氧化还原稳态的破坏是加速细胞衰老并推动动脉粥样硬化病理进程的关键驱动力[68,69]。多项研究证实[29,41,42,57,58,63,70,71], 民族药可显著提升超氧化物歧化酶 (superoxide dismutase, SOD) 和谷胱甘肽过氧化物酶 (glutathione peroxidase, GSH-Px) 等内源性抗氧化酶活性, 并降低丙二醛 (malondialdehyde, MDA) 水平, 从而提升机体的整体抗氧化能力, 发挥抗动脉粥样硬化作用。在信号通路调控层面, 三七皂苷作为壮药三七的核心活性成分, 可通过减少泛素特异性蛋白酶2 (ubiquitin specific peptidase 2, USP2) 介导的Kelch样ECH关联蛋白1 (Kelch-like ECH-associated protein 1, Keap1) 去泛素化促进Keap1降解, 进而激活核因子E2相关因子2 (nuclear factor erythroid 2-related factor 2, Nrf2) 信号通路, 改善铁代谢并减少脂质过氧化, 最终发挥抗动脉粥样硬化作用[72]。同样, 蒙药沙棘-5味散也被证实可激活Nrf2信号通路, 并显著上调其下游靶基因醌氧化还

原酶1 [NAD(P)H quinone dehydrogenase 1, *NQO1*] 的表达水平, 从而增强抗氧化功能[73]; 壮药积雪草活性成分积雪草酸则通过抑制活性氧 (reactive oxygen species, ROS) 生成及诱导型一氧化氮合酶 (inducible nitric oxide synthase, iNOS) 表达, 并经由PPARγ/核因子κB (nuclear factor kappa-B, NF-κB) 信号轴发挥协同抗氧化与抗炎效应[74]。此外, 维吾尔药新塔花总黄酮[20]通过下调血管内皮生长因子/蛋白激酶B/NF-κB (vascular endothelial growth factor/protein kinase B/NF-κB, VEGF/AKT/NF-κB) 信号通路减轻氧化应激与炎症反应; 而榅桲−罗勒总黄酮则通过抑制NADPH氧化酶4 (NADPH oxidase 4, NOX4) 的表达, 从源头上减少ROS的生成[67]。综上, 民族药通过多靶点、多通路协同调控氧化应激, 进而发挥其抗AS效应。

2.4 抑制炎症反应与免疫调节 炎症作为动脉粥样硬化的典型特征, 贯穿于疾病发生发展的全过程[75,76]。其中, 氧化低密度脂蛋白等致病因子持续刺激血管内皮细胞并激活免疫细胞, 促使炎症细胞因子大量释放, 同时驱动巨噬细胞向促炎M1表型极化, 共同加剧AS进展[77]。在免疫细胞调控方面, 民族药主要通过调节巨噬细胞极化状态发挥作用, 其蒙药沏其日甘-8通过调控干扰素调节因子1/C-X-C motif趋化因子配体10 (interferon regulatory factor 1/C-X-C motif chemokine ligand 10, *Irf-1*/*Cxcl10*) 基因表达, 促进巨噬细胞由促炎M1型向抗炎M2型转化, 表现为CD68、iNOS表达下调和CD206表达上调[78]; 蒙药五味沙棘散通过调节M1/M2极化平衡, 有效抑制炎症反应并减少主动脉斑块内泡沫细胞浸润[79]; 而藏药藏红花的活性成分藏红花素可通过促进M2型巨噬细胞极化并抑制NF-κB的p65核转位, 实现抗炎作用[80]。另有研究表明, 从壮药蓬莪术中提取的蓬莪术二醇可通过调控C-X-C motif趋化因子配体12/C-X-C motif趋化因子受体4 (C-X-C motif chemokine ligand 12/C-X-C motif chemokine receptor 4, CXCL12/CXCR4), 抑制单核细胞与内皮细胞的黏附, 从源头控制炎症细胞浸润[81]。在炎症信号通路调控层面, 靶向Janus激酶 (Janus kinase, JAK) 通路的治疗策略近年来展现出显著潜力[82]; 研究发现维吾尔药苦艾提取物可靶向Janus激酶2/信号转导子和转录激活子3 (Janus kinase 2/signal transducer and activator of transcription 3, JAK2/STAT3) 通路进而改善AS[60], 而香青兰总黄酮亦可调控JAK/STAT信号, 下调相关炎症因子表达[19]。壮药积雪草主要活性成分积雪草酸则通过激活PPARγ进而抑制NF-κB信号通路, 该机制可被PPARγ抑制剂GW9662特异性逆转[74]; 同样, 蒙药朱日亨滴丸也被证实是一种PPARγ激动剂, 可通过调节该靶点改善炎症[15]。此外, 苗药粗壮女贞水提物则通过c-Jun氨基末端激酶/激活蛋白-1 (c-Jun N-terminal kinase/activator protein-1, JNK/AP-1) 通路抑制内皮细胞炎症反应[47]; 朝医方太阴调胃汤通过调节促炎/抗炎平衡, 降低C-反应蛋白 (C-reactive protein, CRP) 和肿瘤坏死因子-α (tumor necrosis factor-alpha, TNF-α) 同时提高白细胞介素-10 (interleukin-10, IL-10) 表达[42]。藏药红景天苷抑制NOD样受体蛋白3 (NOD-like receptor family pyrin domain containing 3, NLRP3) 炎症小体活化及随后的半胱天冬酶-1 (Caspase-1) 依赖性细胞焦亡, 有效减少了白细胞介素-1β (interleukin-1 beta, IL-1β) 与白细胞介素-18 (interleukin-18, IL-18) 的成熟释放[83]。

2.5 调节血管稳态 血管稳态失衡是动脉粥样硬化的重要病理基础, 涉及内皮功能障碍、炎症反应、平滑肌细胞异常增殖与表型转化等多重机制[84-87], 而民族药在此环节展现出明确的干预作用。在调节血管功能方面, 蒙药达日布-8对AS兔模型的一氧化氮/内皮素 (nitric oxide/endothelin, NO/ET) 具有调控作用[88], 五味沙棘散则抑制VEGF过度表达及激活内皮型一氧化氮合酶/一氧化氮 (endothelial nitric oxide synthase/nitric oxide, eNOS/NO) 通路改善内皮功能[79]; 藏药额尔敦−乌日勒则通过降低ET等多种血管内皮因子、上调血清前列环素2 (PGI_2) 并抑制血管内皮凝血纤溶物质的表达, 综合改善动脉粥样硬化血管内皮功能[89]; 蒙药通拉嘎-5进一步通过PPARα-Apo-AI信号轴提升HDL水平, 并降低IL-6、IL-1β等促炎因子, 协同改善内皮代谢[13]。在抑制内皮炎症与细胞凋亡方面, 藏药红景天苷通过直接结合缺氧诱导因子-1α (hypoxia inducible factor-1alpha, HIF-1α), 抑制Caspase-1依赖性焦亡与脂质积累, 减轻内皮炎症损伤; 维吾尔药新塔花总黄酮[20]可调控VEGF/AKT/NF-κB通路, 同时抑制细胞间黏附分子-1 (intercellular cell adhesion molecule-1, ICAM-1) 和血管细胞黏附分子-1 (vascular cell adhesion molecule-1, VCAM-1) 的表达, 其表达水平的降低会抑制单核细胞的迁移和黏附, 拮抗动脉粥样硬化血管的增殖。维吾尔药香青兰总黄酮通过抑制JAK/STAT信号通路, 降低TMAO与促炎因子水平, 缓解AS内皮炎症状态[19]。在调控血管平滑肌细胞方面, 蒙药荜茇活性成分荜茇宁可抑制其增殖与迁移, 并通过上调收缩表型蛋白、下调合成表型蛋白, 促进其表型转化, 其机制与抑制Ras/Raf/MEK/ERK1/2通路有关[90]; 维吾尔药艾菲提蒙汤则通过抑制血小板衍生生长因子-A/增殖细胞核抗原 (platelet derived growth factor-A/proliferating cell nuclear antigen, PDGF-A/PCNA) 表达, 阻断平滑肌

异常增殖, 共同维持血管结构与功能稳态[58]; 壮药金边蚂蟥活性成分菲牛蛭素通过促进PCNA和抑制Caspase-3蛋白的表达来抑制血管平滑肌细胞的异常增殖、迁移和凋亡, 达到治疗动脉粥样硬化的效果[91]。

2.6 改善斑块稳定性 斑块的稳定性是决定动脉粥样硬化性疾病临床预后的关键因素[92]。研究表明, 多种民族药可通过干预斑块组成与结构, 增强其稳定性。基质金属蛋白酶是降解胶原、弹性蛋白的关键酶, 其活性过高或与TIMPs比值失衡, 会增加斑块的不稳定性[93,94]。然而, 藏药藏红花水提取物可降低MMP-2、MMP-3、MMP-9活性及MMP-2/TIMP-2比值, 并提升斑块内胶原与弹性蛋白含量[26]; 彝药塔若散可调节MMP-9/TIMP-1比值, 维持基质稳定[43]; 维吾尔药香青兰总黄酮通过抑制转化生长因子-β1/果蝇母源抗生存基因同源物 (transforming growth factor-beta 1/mothers against decapentaplegic homolog, TGF-β1/Smad) 信号通路下调MMP-2与MMP-9表达[95]; 与之相似, 蒙药当贡-3亦可有效下调MMP-2、MMP-9的表达, 进而抑制基质降解[54]。在调控细胞功能方面, 蒙药荜茇活性成分荜茇宁可上调血管平滑肌细胞中α-平滑肌肌动蛋白 (alpha-smooth muscle actin, α-SMA)、平滑肌22α (smooth muscle protein 22-alpha, SM22α)、平滑肌肌球蛋白重链11 (myosin heavy chain 11, MYH11) 等收缩型蛋白, 同时抑制骨桥蛋白 (osteopontin, OPN)、MMP-2、MMP-9等合成型蛋白表达, 从而诱导其向收缩表型转化, 延缓斑块进展[90]; 维吾尔石榴皮提取物介导的斑块坏死减少被证明与Mer酪氨酸激酶 (Mer tyrosine kinase, Mertk) 裂解减弱及病灶胞葬作用增强相关[96]; 壮药蓬莪术二醇则通过干预CXCL12/CXCR4信号轴抑制单核细胞与内皮细胞黏附, 减少炎症细胞在斑块内聚集[81]。藏药然那桑培丸改善AS小鼠动脉斑块内平滑肌、巨噬细胞、胶原、脂质含量[25]。此外, 在调节脂质代谢与自噬方面, 蒙药朱日亨滴丸可显著回调斑块中TAG、神经酰胺 (ceramide, Cer) 及葡萄糖神经酰胺 (glucosylceramide, GlcCer) 等脂质成分, 改善斑块易损性[97]; 壮药水蛭可促进斑块内自噬水平, 表现为自噬蛋白Beclin-1与微管相关蛋白1轻链3-II (microtubule-associated protein 1A/1B-light chain 3-II, LC3-II) 表达上调, 自噬体数量增多, 斑块面积相应减小[37]。藏药三味檀香散可通过提高溶酶体活性和巨噬细胞胆固醇外流来减弱动脉粥样硬化斑块的形成[24]; 红景天活性成分红景天苷则可直接结合HIF-1α抑制细胞焦亡通路, 从而减轻巨噬细胞及动脉粥样硬化斑块中的脂质蓄积[83]。基于上述讨论, 表1[11,13-15,19,20,24,26-29,34,35,37,39-44,47-52,54-63,65-67,70-74,78-81,83,88-91,96-99]对前述相关的民族药、对应的活性成分、植物来源及药用部位进行了系统梳理, 并总结了其抗动脉粥样硬化的主要作用机制。

Table 1 Mechanisms of Chinese ethnomedicine in treatment of AS

Ethnic medical system	Ethnomedicine/ component	Source material	Key mechanisms of action	Ref.
Mongolian medicine	Danggong-3	*Angelica sinensis*, *Caesalpinia sappan*, *Pterocarpus santalinus*	Improves lipid profile and enhances cholesterol clearance; promotes plaque stability	[54]
	Bright Salt-4 Decoction	Halite, *Terminalia chebula* Retz., *Piper longum* L., *Zingiber officinale* Rosc.	Regulates blood lipid levels and mitigates oxidative stress	[55]
	The New-No.2	*Ziziphus mauritiana* Lam., *Ferula* spp., *Aquilaria sinensis* (Lour.) Spreng., *Bos mutus*, *Myristica fragrans* Houtt., and 11 other ingredients	Alleviates dysregulated lipid metabolism and diminishes plaque formation	[56]
	Guanxin Shutong Capsule	*Choerospondias axillaris*, *Salvia miltiorrhiza* Bge., *Syzygium aromaticum* and 2 other ingredients	Modulates lipid metabolism and enhances antioxidant capacity	[57]
	Guanxin Qiwei Tablets	*Salvia miltiorrhiza* Bge., *Choerospondias axillaris*, and 5 other ingredients	Modulates lipid metabolism	[65]
	Baolier Capsule	*Salvia miltiorrhiza* Bge., *Choerospondias axillaris*, and 13 other ingredients	Modulates lipid metabolism and promotes cholesterol efflux	[11]
	Tonglaga-5	*Punica granatum* L., *Carthamus tinctorius* L. and 3 other ingredients	Improves lipid metabolism, regulates inflammation, and modulates gut microbiota	[13]
	Qingxue Bawei Tablet	Bovis Calculus Artifactus, Arnebiae Radix, *Gardenia jasminoides*, and other ingredients	Regulates inflammation and remodels lipid composition	[14]
	Qiqirigan-8	*Hippophae rhamnoides* L., *Caesalpinia sappan* L., *Piper longum* L., *Kaempferia galanga* L., and other ingredients	Suppresses inflammation and promotes reparative macrophage polarization	[78]
	Wuwei Shaji Powder	*Hippophae rhamnoides* L., *Aucklandia lappa* Decne, *Gardenia jasminoides* J. Ellis, and other ingredients	Regulates inflammation, enhances systemic antioxidant defense, restores macrophage homeostasis, and protects endothelial function	[73, 79]

Continued

Ethnic medical system	Ethnomedicine/ component	Source material	Key mechanisms of action	Ref.
	Daribu-8	*Hippophae rhamnoides* L., *Kaempferia galanga* L., *Piper longum* L. and other ingredients	Repairs endothelial damage	[88]
	Piperlonguminine	*Piper longum* L.	Inhibits abnormal vascular smooth muscle cells proliferation and maintains vascular structural stability	[90]
	Hippophae rhamnoides L.	*Hippophae rhamnoides* L.	Restructures the microbial community	[48]
	Zhuriheng Pills	*Choerospondias axillaris*, *Myristica fragrans* Houtt., and other ingredients	Suppresses inflammation, modulates lipid metabolism and stabilizes plaques	[15, 97]
	Guanxin Qiwei Dropping Pills	*Choerospondias axillaris*, *Hippophae rhamnoides* L., *Myristicae semen*, *Santalum album* L., *Dalbergia odorifera*, and other ingredients	Improves lipid metabolism, suppresses inflammation and oxidative stress, and modulates the gut microbiota	[49]
Tibetan medicine	*Sibiraea angustata* (Rehder) Hand.-Mazz.	*Sibiraea angustata* (Rehder) Hand.-Mazz.	Modulates lipid profiles, exerts antioxidant effects, and attenuates inflammatory cytokines	[61]
	Phyllanthus emblica L.	*Phyllanthus emblica* L.	Ameliorates hepatic cholesterol accumulation and inflammatory factors	[28]
	San-wei-tan-xiang Capsule	*Choerospondias axillaris*, *Santalum album* L., *Myristicae Semen*	Promotes cholesterol elimination and attenuates atherosclerotic plaque formation	[24]
	Salidroside	*Rhodiola crenulata* (Hook. f. & Thomson) H. Ohba	Exhibits anti-inflammatory, lipid-regulatory, cytoprotective, and plaque-stabilizing effects	[66, 83]
	Crocin	*Crocus sativus* L.	Suppresses inflammation and maintains immune homeostasis	[80]
	Crocus sativus L.	*Crocus sativus* L.	Increases atherosclerotic plaque stability	[26]
	Usnea diffracta Vain.	*Usnea diffracta* Vain.	Improves intestinal barrier; restructures the gut microbiota; promotes lipid metabolism; reduces plaque area; regulates inflammatory signaling pathways	[52, 98]
	Eerdun-Geriletu	Margarita, Bovis Calculus Artifactus, Moschus Artificialis, *Carthamus tinctorius* L. and other ingredients	Promotes endothelial homeostasis and vascular function	[89]
	Edgeworthia gardneri (Wall.) Meisn	*Edgeworthia gardneri* (Wall.) Meisn	Strengthens intestinal barrier integrity, modulates gut microbiota, exerts antioxidant effects	[27]
Uygur medicine	Aifeitimeng Tang	*Cuscuta chinensis* Lam., *Terminalia chebula* Retz., and other ingredients	Regulates blood lipids and enhances antioxidant enzyme activity	[58]
	Ziziphora clinopodioides Lam.	*Ziziphora clinopodioides* Lam.	Improves lipid metabolism, protects endothelium, and alleviates oxidative stress	[20]
	Yangxin Dawaimixike Honey Paste	Moschus, Margarita, *Crocus sativus* L., *Santalum album* L., *Pterocarpus santalinus* L. f., *Styrax benzoin* Dryand., *Cinnamomum cassia* (L.) J. Presl, *Aquilaria sinensis*, bamboo sugar, and other ingredients	Exerts lipid-lowering, antioxidant and anti-inflammatory effects	[59]
	Artemisia absinthium L.	*Artemisia absinthium L.*	Exerts anti-inflammatory effects and improves local immune status	[60]
	Quince-Basil	*Cydonia oblonga* Mill. and *Ocimum basilicum* L.	Reduces lipid deposition and oxidative damage	[67]
	Tilianin	*Dracocephalum moldavica* L.	Enhances cholesterol clearance and modulates intestinal microbiota	[51]
	Dracocephalum moldavica L.	*Dracocephalum moldavica* L.	Exerts anti-inflammatory, endothelial-protective, and modulates microbiota-metabolite axis	[19]
	Punica granatum L.	*Punica granatum* L.	Reduces necrotic core formation and stabilizes plaques	[96]
	Morus nigra L.	*Morus nigra* L.	Enhances antioxidative capacity	[71]
Zhuang medicine	Zhuangtong Yin	*Euonymus fortunei*, *Panax notoginseng* and *Campanumoea javanica*	Exerts anti-inflammatory effects	[34]
	Gynostemma pentaphyllum	*Gynostemma pentaphyllum*	Alleviates vascular lesions and improves lipid metabolism	[35]

Continued

Ethnic medical system	Ethnomedicine/ component	Source material	Key mechanisms of action	Ref.
	Hemsleya chinensis	*Hemsleya chinensis*	Regulates blood lipid levels, exerts anti-inflammatory effects, and protects vascular endothelial cell function	[62]
	Panax notoginseng saponins	*Panax notoginseng*	Exhibits antioxidant activity and reduces lipid peroxidation	[72]
	Asiatic acid	*Centella asiatica*	Exhibits anti-inflammatory and antioxidant effects	[74]
	Zedoarondiol	*Curcuma phaeocaulis*	Reduces inflammatory cell adhesion and promotes plaque stability	[81]
	Bufrudin	*Hirudinaria manillensis*	Modulates lipid metabolism and inhibits abnormal vascular smooth muscle cells proliferation	[91]
	Malus doumeri (Bois) A. Chev.	*Malus doumeri*	Modulates microbial composition and lipid metabolism	[50]
	Hirudo	*Hirudo nipponica* Whitman	Enhances autophagy and stabilizes plaques	[37]
Miao medicine	Yindan Xinnaotong Capsule	*Ginkgo biloba* L., leaf, *Salvia miltiorrhiza* Bge., *Erigeron breviscapus* (vant.) Hand.-Mazz., *Panax notoginseng*, and other ingredients	Lowers lipids and modulates vascular homeostasis	[40, 99]
	Rosa damascena	*Rosa damascena*	Enhances antioxidant system	[70]
	Dengzhan Shengmai Capsule	*Erigeron breviscapus*, *Panax ginseng*, and other ingredients	Reduces carotid intima-media thickness, enhances plaque stability, and lowers lipid levels	[39]
	Ligustrum robustum	*Ligustrum robustum*	Exerts anti-inflammatory and endothelial-protective effects, and improves microbiota-metabolism interactions	[47]
Traditional medicine of the Korean ethnic group in China	Mahuangdingtong Tang	*Ephedra sinica* and other ingredients	Enhances antioxidative capability	[41]
	Chaoyi Taeeumjowui Tang	*Coix lacryma-jobi* L. and other ingredients	Restores inflammatory balance and reduces oxidative injury	[42]
Dai medicine	*Toddalia asiatica* (L.) Lam.	*Toddalia asiatica* (L.) Lam.	Regulates lipid levels and exhibits antioxidant activity	[63]
	Yajie Yizai (Xin) Recipe	*Eupatorium heterophyllum*, *Cyathula officinalis* Kuan, and other ingredients	Mitigates oxidative damage	[29]
Yi medicine	Taruosan	*Taxillus chinensis* (DC.) Danser and *Zanthoxylum bungeanum*, and other ingredients	Balances extracellular matrix turnover and stabilizes plaques	[43]
Wa medicine	Nie Liang Gei	*Polygalajaponica* Houtt.	Regulates lipid metabolism	[44]

3 民族药治疗AS的临床研究进展

民族医药在治疗AS中展现出一定前景, 其临床应用价值正逐步通过一系列临床研究得到初步验证。一项为期12个月、纳入150例患者的随机对照非劣效性研究证实, 在改善颈动脉内膜中层厚度 (intima-media thickness, IMT) 方面, 苗药灯盏生脉胶囊疗效非劣于阿司匹林, 且胃肠道不良事件发生率更低, 安全性更优[39]。另一项纳入109例受试者的临床观察表明, 壮药壮通饮对血清炎症因子具有调节作用, 可显著降低CRP水平[34]; 蒙药清血八味散与氨氯地平的联合治疗方案 (70例受试者) 则在改善血栓前状态分子标志物[如纤维蛋白原 (fibrinogen, FIB)、*D*-二聚体] 及颈动脉粥样硬化影像学指标方面表现出协同增效作用[100]。此外, 针对蒙药沙棘-5味散 (95例)[101]、苗药银丹心脑通软胶囊联合普罗布考 (70例)[102]及佤药聂良给 (60例)[44]的临床研究, 也均报告了这些民族药在调节AS患者脂质代谢及稳定动脉斑块等方面的积极效应。综上, 现有临床证据初步证实了民族药治疗AS的应用潜力, 但未来仍需开展大规模、多中心、随机对照的高质量临床试验, 进一步验证其疗效与安全性, 为民族药在AS临床实践中的规范应用提供更充分的循证医学依据。

4 总结与展望

基于不同民族医学的理论体系, AS的核心病机在于内在生理失衡所致“病理产物”阻滞脉络。其治疗强调“调节机体平衡”与“清除病理产物”相结合的原则, 该理念在以自然元素和生命能量为理论基础的四大民族医学中高度一致。在机制上, 民族药通过改善肠道菌群、调节脂质代谢、拮抗氧化应激、抑制炎症反应、调节血管稳态及稳定斑块等六大相互关联的核心环节, 构成一个多靶点、多维度协同的药理作用网络, 体现出其整体干预的优势。同时, 多项临床研究观察到其在

改善颈动脉内膜中层厚度、调节血脂与炎症指标方面的积极作用, 为其临床应用提供了初步依据。

尽管研究取得一定进展, 但民族药在治疗AS的现代化进程及其获得国际医学界广泛认可方面仍面临诸多挑战。其一, 理论体系的现代化阐释不足, 民族医学理论 (如“三根”“三因”“四塔”等) 植根于特定的文化与哲学背景, 其概念与现代医学语言体系存在隔阂, 如何实现理论的有效对接与科学转化, 是当前研究的关键瓶颈。其二, 民族药复方成分复杂, 其“多靶点”作用机制研究仍具挑战, 难以明确不同机制在整体药效中的相对贡献及其是否构成民族药的特有作用模式, 且活性成分在体内的代谢过程、相互作用及药理作用尚未被完全阐明。其三, 现有临床研究多属单中心、小样本探索, 导致高级别循证医学证据尚显匮乏, 限制了其临床推广与应用; 同时, 长期用药的系统安全性评价亦有待加强。

面向未来, 可整合系统生物学、人工智能及多组学数据分析等前沿技术, 基于民族医药临床观测与基础实验数据, 构建“病–证–方–靶”多维关联网络, 探索民族医药传统理论与现代医学的内在关联, 进而表征其传统理论的科学内涵[103-105]。在机制解析层面, 可借鉴中医药领域的前沿研究策略, 通过超高效液相色谱–质谱联用、代谢组学、计算药学等技术系统解析复方中的活性成分群及其作用特征, 结合靶标验证实验与单细胞多组学技术阐明其协同作用机制, 可采取“药效评价–靶细胞确认–靶标识别–生物学功能确证”的研究模式[106-109]。此外, 亟需构建新型实验方法, 融合人工智能及计算模型, 以科学识别并量化各作用机制在整体药效中的贡献权重。在临床验证层面, 须重点推进大规模、多中心、前瞻性随机对照临床试验, 建立契合民族药整体作用特点的疗效评价标准, 完善药物安全性评价体系及药物相互作用研究技术规范。借助这种多层次、跨学科的协同创新, 民族医药有望实现从传统经验医学到现代证据医学体系的系统性转变。这不仅能为AS治疗体系提供独具民族特色的治疗方案, 还能为现代整合医学的创新发展增添独特价值。

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利益冲突: 所有作者均声明本文无相关利益冲突。

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