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引发心血管疾病的新风险——微纳米塑料*

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摘要 近年来，随着塑料制品的大规模使用，塑料污染程度加剧，逐渐成为一个日益严重的全球性问题。塑料制品释放的微纳米塑料（microplastics and nanoplastics，MNPs）作为新兴的环境污染物广泛存在于生物体和环境中，这些塑料颗粒通过3种暴露途径进入人体：呼吸摄入、食物链的生物积累和转移以及皮肤接触，继而产生毒性效应。MNPs的物理特性（形状、大小、表面特性）会随环境变化而动态转化；MNPs充当化学物质载体可分为两种机制：一是从外界吸附而来的污染物，二是在商业生产过程中人工添加的化学剂（阻燃剂、色素等）。研究表明，MNPs对人体健康产生不利影响，现在已经在脑、肠道、肝脏、血液等组织器官中发现了MNPs。最新临床研究表明，MNPs是引发心血管疾病（cardiovascular diseases，CVDs）的一个新风险，其参与心肌纤维化等发生发展。CVDs是心脏、动脉、静脉、毛细血管疾病的统称，是导致人类致残和死亡的主要疾病之一。CVDs发病率和复发率较高，并发症较多，降低了患者生活品质和幸福指数，并且呈现年轻化趋势，因此，早期预防极为重要。本文综述了MNPs的性质及其对心血管系统的潜在威胁，旨在探讨MNPs通过何种生理效应、毒性机制及相关通路引发CVDs，重点讨论了其增强氧化应激，促进促炎症因子表达，形成慢性炎症微环境，吸附毒金属和有机物等有毒物质联合相互作用的毒性过程。其中，污水灌溉、大气沉降等过程是重金属与MNPs共污染农业土壤的主要因素，重金属与MNPs相互作用会抑制农作物生长，并促进重金属在植物中的吸收，再通过食物链进入人体，甚至诱发急性冠脉综合征等CVDs。此外，本文例举了MNPs对心血管功能的长期影响，探讨了当前MNPs影响心血管系统方面研究的局限性以及未来的研究方向。

关键词 塑料颗粒，微塑料，纳米塑料，心血管疾病
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20世纪以来，塑料制品因其较好的耐用性、轻便性和可塑性被广泛应用于日常家居业、包装业、医疗业等领域。尽管塑料有许多优点，但其破坏生态环境、危害人体健康等情况也不容忽视，其中塑料废品的回收和处理问题尤为突出。大量塑料废弃物的老化与分解，会释放微塑料（定义为长度小于5 mm的微小塑料颗粒，直径1 μm~5 mm）和纳米塑料（定义为长度小于1 000 nm的塑料颗粒），两者统称为微纳米塑料（microplastics and nanoplastics，MNPs）[1]。微塑料又可分为初级微塑料和次级微塑料，初级微塑料是为了满足某些经济需求（如用于商业产品的包装）由人工生产出来，次级微塑料则是大型塑料制品经紫外线照射、机械磨损和生物降解等因素致其碎裂而产生[2]。

MNPs在环境中普遍存在，各种生物都可能摄入这些塑料微粒，进而对其健康和生态系统的动态平衡产生不良影响[3]。MNPs同样会对人类健康构成威胁。最近的研究表明，这些微粒会进入食物链，最后在人体多种组织器官、体液中积累，可能引起广泛的毒理学影响，目前已经在肠道[4]、肺[5]、肝脏、肾脏、大脑等组织器官中发现微塑料[6]，这意味着MNPs可能侵入人体消化系统，改变肠道菌群并诱导肝毒性[7]，并相应地对呼吸系统[8]、神经系统及免疫系统[6]健康造成一定程度

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的威胁，甚至在胎盘[9]、母乳[10]血液[11]中也发现了MNPs，表明MNPs可能通过生殖系统及血液循环传递至下一代，并更易诱发心血管疾病（cardiovascular diseases，CVDs）。CVDs包括心脏及血管结构功能异常和（微）血管部位的相关疾病，是全球死亡率最高的疾病，号称人体健康的头号杀手。某些危险因素会诱发CVDs，包括心肌纤维损伤、细胞焦亡、细胞炎症和线粒体及能量代谢异常，MNPs与这些引发CVDs的危险因素存在关联[12]。此外，MNPs中可能含有的塑料添加剂（如双酚、邻苯二甲酸盐和全氟烷基和多氟烷基化合物（per- and polyfluoroalkyl substances，PFAS））是内分泌干扰化学物质，通过暴露途径在体内积累，可能引起甲状腺功能、生殖系统、内分泌和代谢失调，增加失眠、肥胖、糖尿病、癌症等风险[13-14]。本文旨在综述MNPs对心血管系统造成的风险，并讨论其毒性机制，以期为后续深入研究提供更明确的方向和思路。

1　MNPs的人体暴露途径

暴露途径指有害物质或危险因素通过某些途径接触并进入人体，引起人体健康风险的过程。MNPs通过空气吸入、食物链、皮肤接触等途径进入人体[15]，再由血液循环经过全身各个器官，在体内累积，最终诱发一系列威胁人类健康的问题（图1），其中吸入或摄入途径是人体暴露于MNPs的主要途径，例如，汽车尾气会导致吸入暴露，它们释放的MNPs弥漫在大气中，易于被人体吸入。皮肤接触为次要暴露途径，研究发现，颗粒物很少能够穿透皮肤，并且毛囊也有紧密的屏障阻止颗粒物侵入细胞[16]。

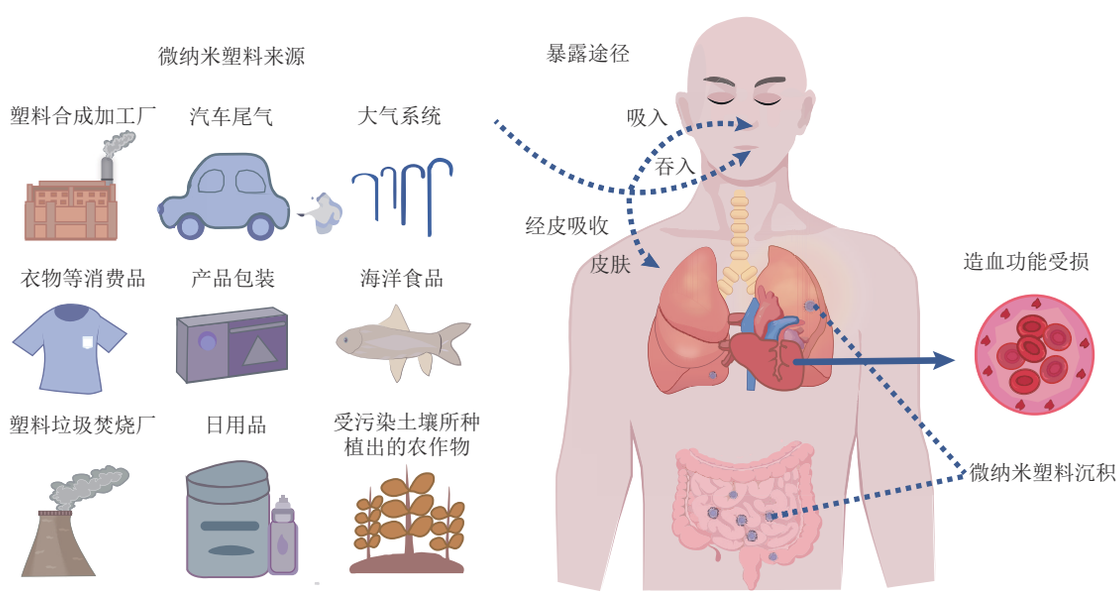

Fig. 1　Sources and exposure pathways of microplastics and nanoplastics

图1　微纳米塑料来源与暴露途径

1.1　呼吸途径

研究表明，MNPs颗粒具有较强的生物屏障穿透能力，可经呼吸道进入血流[15]。人体通过鼻腔-气道-肺途径，吸入MNPs，后者最终在肺部累积。空气中的MNPs将改变血清代谢物、鼻腔和肺部微生物群的组成，导致网格结构及功能通路发生明显变化[7]。人类日常生活中充斥着各式各样的塑料制品，人们无法避免接触到它们。微塑料主要以纤维的方式存在于灰尘中，其中含有与室内环境中的纺织品相似的聚合物，室内空气中的微塑料纤维含量是室外环境的两倍，主要聚合物为聚丙烯，空调引起的气流湍流会导致室内空气中的微塑料粉尘再悬浮与迁移，婴儿通过吸入灰尘而摄入的微塑料较高于成人与青少年，这可能与婴儿时常手口接触有

关[17-19]。有研究发现，基于北京3个城市功能区的粉尘摄入和皮肤接触情况，估算人类在环境中微塑料的年接触量，若按在室外活动2 h/d计算，儿童的年接触量为7.37×10^{4}个，成人的年接触量为1.06×10^{5}个，此数据与人类从食物和水中摄入的微塑料量相当，通过呼吸途径进入人体的大气微塑料可能会对人体健康产生影响，值得关注[20]。

1.2 饮食摄入途径

饮食摄入途径是MNPs进入人体内最普遍途径，食物中的MNPs会通过口腔-食道-胃肠道途径进入人体，显著改变胃肠道微生物菌群的结构和功能，对人体造成健康风险。小于10 μm的MNPs能够穿过胃肠道（gastrointestinal，GI）系统的上皮屏障，进入血液和淋巴系统[21]。而无法穿过真皮屏障的较大微米塑料以两种途径排出体外：通过打喷嚏、咳嗽、鼻涕、痰液从呼吸道排出或通过粪便从胃肠道排出[22-23]。目前，在常见的由聚合物（聚丙烯、聚乙烯、聚苯乙烯等）制成的外卖容器中发现了微塑料，据估计，食用外卖食品4~7次/周的人可能摄入约12~203片微塑料[24]。最近的一项基于长期微塑料摄入的小鼠模型研究证实，微塑料会对造血系统造成严重损害，破坏造血干细胞（hematopoietic stem cells，HSCs）的自我更新和重建能力，微塑料主要沉积于小鼠胃肠道中，破坏小肠绒毛结构，减少肠道中理研菌科（Rikenellaceae）和次黄嘌呤的含量，长期摄入微塑料会显著抑制HSCs中的HPRT-Wnt信号通路，而Rikenellaceae菌对造血系统可能有保护作用，这些菌的减少导致HSCs重建能力下降与功能受损[25]，损害造血系统正常功能。

1.3 皮肤暴露

塑料作为常用的包装材料，在日常生活中随处可见，日化用品如牙膏、洗面奶、化妆品、护肤品等均含有MNPs，长期使用这些护理产品使得MNPs经皮肤吸收[26-27]，或皮肤有伤口及破损时，一些MNPs可能通过皮肤暴露途径进入人体。皮肤是人体最大的器官，一些塑料制品中含有的阻燃剂或塑化剂（如多溴二苯醚和六溴环十二烷）可能会随微塑料与皮肤的接触进入人体，微塑料中多溴二苯醚的经皮吸收率会因MNPs的大小与添加剂（止汗剂、保湿剂等）的存在、皮肤湿度的高低产生不同程度改变，造成MNPs中的化学添加剂进入人体，可能引发疾病等不良后果[28]。

2 MNPs毒性机制研究及其对心血管系统的影响

MNPs既可因其本身性质直接产生毒性，也会与其他有害物质相互作用间接诱导毒性，MNPs经多种物理、化学、生物因素处理后会发生老化，老化后的塑料颗粒具有一定毒性[29]。以光老化的MNPs为例，其吸附环境中的重金属、有机污染物等有害物质能力增强，对生物产生联合毒性效应[30]，光老化的MNPs可能会被引入表面功能团，理化性质发生改变，进而与生物体内蛋白质、脂类等分子发生相互作用，这种作用会加剧氧化反应并破坏细胞膜结构，从而使生物体细胞发生损伤[31]，引发炎症，致使生物体抵抗力下降[32-33]。

同时，MNPs与CVDs之间存在潜在联系。MNPs的心血管毒性是由其性质（类型、大小和结构）、暴露剂量和持续时间、生物体的生命阶段、性别和种类以及与其他环境污染的相互作用所决定[34]。MNPs能够直接穿透循环系统或通过间接机制影响心血管健康，例如慢性暴露于MNPs后的小鼠心肌组织受损，并且心肌组织中纤维化和脂质积累程度增加，在转录组水平，基因的m6A修饰介导参与MNPs诱导心脏毒性过程[35]。不同粒径大小的MNPs对人体产生不同程度的毒性影响：0.5 μm的微塑料在器官中诱导炎症与机械损伤，5 μm的微塑料导致与神经炎症相关的肠道菌群失调和代谢紊乱[36]，纳米塑料与微塑料均具有组织间迁移能力，80 nm的纳米塑料比1 μm微塑料具有更强的跨组织转运能力，但1 μm微塑料会引发更严重的肺毒性[8]。血栓形成是CVDs的另一个重要发病机制，MNPs能够促使血小板聚集，加快血栓形成。MNPs刺激血小板聚集的能力取决于多种因素，包括其大小、表面电荷/修饰及其浓度[37-38]。研究发现，在颈动脉斑块中检测到MNPs的患者，在34个月的随访中，因心肌梗死、中风等复合原因而死亡的风险更高，且斑块中MNPs含量越高，炎症标志物水平越高[11]，这表明MNPs对人体心血管系统有负面影响。另外，从细胞和分子层面来看，MNPs可通过引起内皮功能障碍[39-44]、诱发氧化应激与慢性炎症[45-51]、扰乱脂质代谢[14, 52]、形成蛋白质冠（protein corona，PC）[53-54]等多重机制干扰心血管稳态与功能，加速其病理过程（图2）。

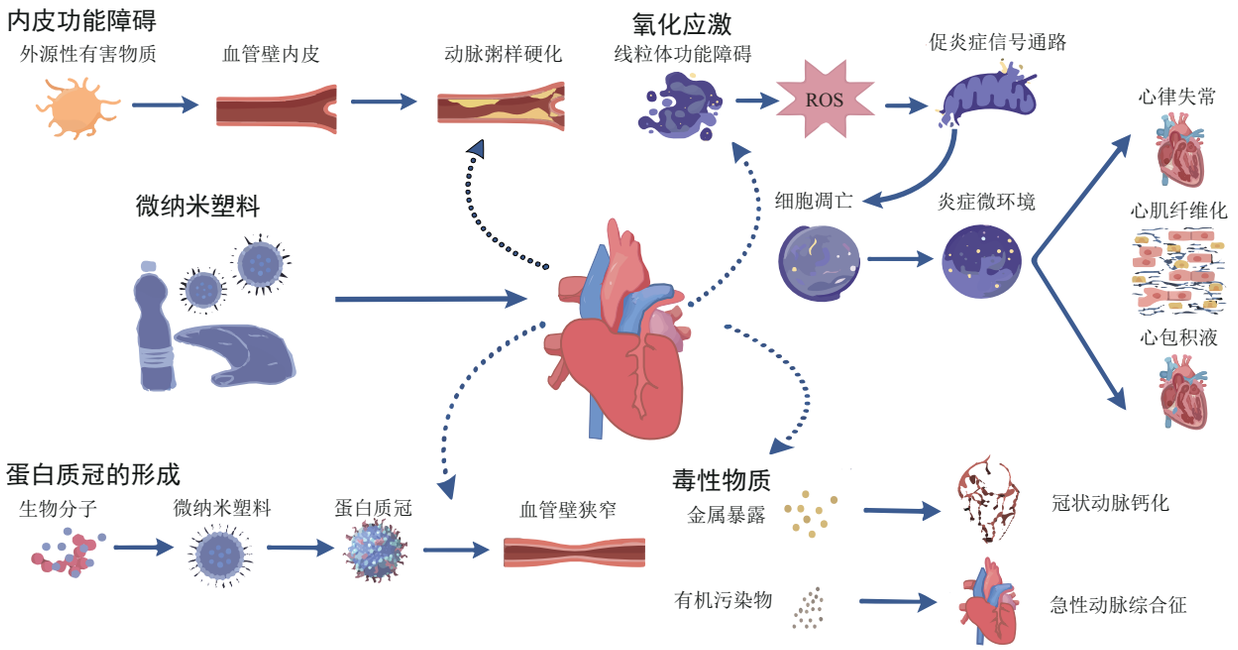

Fig. 2　The toxic mechanism of microplastics and nanoplastics and its effects on the cardiovascular system

图2　微纳米塑料毒性机制及其对心血管系统的影响

ROS：活性氧类（reactive oxygen species）。

2.1　内皮功能障碍

血管内皮是维持正常血管弹性、调节血管表面张力和防止凝血异常的重要生理屏障[39]。MNPs进入血液后，可直接与内皮细胞相互作用，破坏其细胞骨架和紧密连接蛋白（如ZO-1、Occludin）的完整性，导致细胞间隙增大和生理屏障功能减弱，聚苯乙烯微塑料（polystyrene microplastics，PS-MPs）能够破坏血脑屏障的通透性，使谷胱甘肽代谢紊乱，进而促进脑微血管细胞铁死亡和氧化应激，降低ZO-1蛋白表达和分布[40]。这种内皮功能障碍使外源性有害物质（如低密度脂蛋白胆固醇、炎症细胞等）更容易穿过内皮层沉积于血管壁，从而加速动脉粥样硬化进程[41]。同时，内皮细胞受损会减少一氧化氮合酶的合成与释放，降低血管舒张功能，诱发血管痉挛和高血压等[42]。

动脉粥样硬化的核心环节之一在于脂质在动脉内膜下沉积[43]。MNPs可能通过干扰脂质代谢相关基因和信号通路，诱导血管壁中脂质代谢紊乱及过度沉积致使内皮屏障受损，例如，MNPs中可能含有的双酚会引起血脂代谢异常，增加脂质积累与CVDs的发病[52]。内皮屏障受损后低密度脂蛋白胆固醇更易进入内膜并被氧化，随后诱导巨噬细胞摄取大量低密度脂蛋白胆固醇，形成泡沫细胞，最终导致斑块体积和不稳定性增加，斑块不稳定性增加意味着更高的CVDs风险（如心肌梗死或脑卒中）[44]。人脐静脉内皮细胞在1 μm粒径，100 mg/L较高浓度的PS-MPs实验环境中暴露48 h，人脐静脉内皮细胞活性显著降低[55]。这些研究结果表明，高剂量的MNPs可引起细胞活性降低和血管内皮损伤，增加发生CVDs的风险。

2.2　氧化应激和炎症反应

在多种因素作用下，MNPs的表面结构会发生变化，产生自由基并对人体产生一定影响，活性氧类（reactive oxygen species，ROS）的生成与人体通过生物抗氧化防御系统抵消ROS的能力之间的平衡改变会导致氧化应激，这种失衡是各种环境应激源（如MNPs等）诱发细胞损伤的核心机制[56]。塑料颗粒可通过引发线粒体功能障碍、激活还原型辅酶II氧化酶和中断氧化还原敏感信号级联放大等多种途径生成过量ROS[57]。ROS水平升高会对关键的细胞大分子（包括脂质、蛋白质和脱氧核糖核酸）造成氧化损伤，破坏细胞结构和功能，促使细胞凋亡，进而导致局部氧化应激水平上升[58]。MNPs氧化应激后可激活核因子κB（nuclear factor κB，NF-κB）、核苷酸结合结构域富含亮氨酸重复序列和含热蛋白结构域受体3（nucleotide-binding

domain leucine-rich repeat and pyrin domain-containing receptor 3，NLRP3）炎性小体等促炎症信号通路途径，诱导分泌多种促炎症因子（如白介素-6、肿瘤坏死因子α），形成慢性炎症微环境[45-48]。该微环境能够促进多种炎症反应的产生，使动脉粥样硬化斑块中泡沫细胞与炎性细胞聚集，进一步增加血管损伤和CVDs风险，例如，心脏肌节内MNPs可以诱发氧化应激和随后的细胞凋亡，可能导致心律失常[49-51]、心脏功能受损、心包积液和心肌纤维化，加速细胞衰老，引发炎症，抑制一系列心血管代谢功能，最终导致某些临床疾病。

2.3 蛋白质冠（PC）形成与免疫识别改变

纳米塑料进入血液循环后，由于其强结合力，血浆蛋白等生物分子将迅速附着于其表面，从而形成PC，这将改变纳米塑料颗粒物理化学性质，影响其毒性，诱导蛋白质结构与功能变化，促发炎症、增强氧化应激及促癌[53-54]。PC的形成也会改变纳米塑料与免疫细胞、内皮细胞及平滑肌细胞的相互作用，还会改变纳米塑料表面性质及免疫识别模式，刺激或减轻免疫效应[59]，可能影响其在血管内的分布；纳米塑料可能会聚集在特定血管位点，有一定风险干扰正常免疫调控和细胞间通讯、细胞摄取和清除效率，加速其蓄积于血管壁的过程，造成管腔逐渐变窄，血管组织损伤加剧，最终诱发多种CVDs。

2.4 MNPs吸附有毒物质后的相互作用毒性

MNPs吸附有毒物质（金属、有机物等）产生相互作用毒性，进入人体后发挥其毒性效应，损害人体健康，金属接触是一个新发现的引发CVDs的危险因素，这可能与其加速动脉粥样硬化进展有关[60]。有研究显示，塑料添加剂溴化阻燃剂六溴环十二烷促进重金属在微塑料上的富集及其在陆地海洋间的迁移，添加剂的使用可能改变微塑料原本的吸附性能[61]，从而显著地提升微塑料对环境及人体健康的影响。一定浓度的含铬老化PS-MPs颗粒的理化性质会发生显著改变，诱导小鼠氧化应激，驱动自噬反应和炎症-焦亡反应[62]。土壤中镉与微塑料相互作用，提高了其进入植物的效率，增强了植物的氧化应激，并可能通过食物链进入人体并对人类健康构成潜在风险：当微塑料与镉结合时，会显著增强镉的生物可及性和被吸收效率，使其积聚于器官内[63]，增加CVDs风险。最新研究发现，金属暴露通常与冠状动脉钙化的程度呈正相关，是引起CVDs的风险因素[64]。这些研究结果表明，MNPs吸附金属元素产生毒性效应，促进CVDs发生发展。

此外，MNPs对有机物具有较高的亲和力，能吸附和积累环境中的有机污染物，因此，MNPs在环境中的迁移常常伴随有机污染物的富集、转移与释放[65]。研究发现，细颗粒物（PM2.5）的5种主要组分（有机物、黑碳、硝酸盐、硫酸盐和铵盐）均与急性冠脉综合征发作显著相关。其中，有机物与黑碳可能发挥关键作用[66]，而微塑料中富集着大量有机物，如邻苯二甲酸酯[67]、多溴二苯醚[28]、六溴环十二烷[28]等，可能诱发急性动脉综合征，并增加心脏病、中风的风险。以上研究结果为微塑料吸附有机物危害人类心血管健康提供了强有力的流行病学证据。

2.5 MNPs诱导表观遗传调控紊乱

一项研究通过甲基化敏感扩增多态性（methylation-sensitive amplification polymorphism，MSAP）技术，揭示了MNPs会诱导聚对苯二甲酸乙二醇酯（polyethylene terephthalate，PET）MNPs污染水域的紫萍（*Spirodela polyrhiza*（L.）Schleid）DNA高甲基化，表明该淡水物种正保护其基因组[68]。MNPs可以在生殖组织中蓄积，包括睾丸、卵巢滤泡液，通过表观遗传修饰等分子机制破坏内分泌功能，引起发育毒性，影响胚胎发育，产生跨代影响效应[69]。实验室研究发现，MNPs能穿过胎盘屏障沉积在后代组织中，在怀孕期间暴露于雾化的聚十二内酰胺（PA-12）MNPs环境中的大鼠分娩2周以后，在其代表性雌性及雄性幼崽的全身组织中观察到PA-12 MNPs，表明了早期生命阶段暴露于MNPs可能通过表观遗传修饰增强后代发生心血管代谢性疾病的风险[70]。

3 讨论与展望

MNPs尺寸微小，降解时间长，且无处不在，不仅会对生态环境产生一定影响，也会通过食物链等多种暴露途径在人体器官中蓄积。MNPs累积达到一定剂量可能在人体中引发一系列生物学反应，对生命健康产生不利影响，特别是在CVDs、糖尿病和癌症等领域。开展更多的研究来揭示MNPs与CVDs间错综复杂的关联以及建立检测和分析这些微粒的标准化方法至关重要。

目前，MNPs对心血管系统毒性机制的研究尚处于探索阶段，存在一定局限性：临床证据与样本数据较少。动物模型已揭示部分MNPs的毒性机

制，例如免疫细胞吞噬循环系统中的微塑料形成血栓，导致小鼠血流减少与神经功能异常[71]，以及MNPs在心血管系统中通过诱发氧化应激等多种机制协同作用，最终形成不利于血管健康的病理环境。临床实验多局限于特定患者，如意大利团队选择257名接受过颈动脉内膜剥脱术的患者作为研究分析对象，在他们的颈动脉斑块中发现了MNPs[11]，但因研究样本量较小，未控制饮食等因素，且该实验为观察性研究，不能确切证明MNPs与CVDs之间的因果关系，需要更多跨学科实验团队协作，验证MNPs剂量与效应的关系，并明确其毒性机制。

大量MNPs毒性研究是在实验室中进行的，而实验室中设置的MNPs浓度通常较高，暴露时间较短，颗粒与尺寸较为单一，使用动物模型模拟人体在MNPs中暴露情况与真实情况可能存在差异。应当增强实验环境的真实性，设定更贴近真实情况的暴露条件，采用长时间低剂量的类器官模型或疾病背景动物模型进行验证。同时，还需考虑实验的可重复性和标准化以及数据可靠性，如使用同一批次的MNPs，设置空白组与对照组等。

由于目前缺乏统一的MNPs检测标准体系，并且不同检测方法的灵敏性、精确性差异较大，数据单位可能也各不相同，导致不同研究的数据难以直接比较。MNPs的实际环境检测方法、采样方法与追踪定量技术研究尚处于初期阶段，需要进一步结合实际，使用高稳定性、高可靠性和高灵敏性的方法来对环境和生物体中的MNPs进行定性和定量，并深入探究其对生物体的潜在健康风险。现有研究探讨了干预MNPs摄入的方法，例如把自来水煮沸过滤后再饮用，MNPs可以与碳酸钙凝结在一起共沉淀至烧水壶底部，沸腾的硬水可去除至少80%的MNPs[72]。尽量少吃或不吃外卖，外卖一般用一次性塑料盒包装，塑料制品热接触后的浸出液对心血管系统存在潜在影响[73]。此外，需进一步思考如何利用酶工程及合成生物学技术来设计智能、高效的生物降解MNPs绿色途径，研发相应产品替代塑料以减少其广泛使用。最后，MNPs对心血管健康的影响可能与个体年龄、饮食习惯、基因差异等因素有关，对相关研究结果的分析解读需要全面且慎重。

总之，未来的研究应重点聚焦于MNPs的生物累积效应、食物链传递以及MNPs长期暴露对生态环境与人体健康的影响。实验过程中需设置长时间低剂量MNPs暴露条件，采用与人类亲缘相近的动物建模，设计并使用MNPs实时监测仪观察微纳米塑料在其体内的代谢情况，开展临床研究，明确引发相关疾病的毒性机制，推广行之有效的干预MNPs进入人体的良策，或为将来探寻新MNPs靶点药物奠定基础。

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A New Risk of Cardiovascular Disease —— Micro-nanoplastics*

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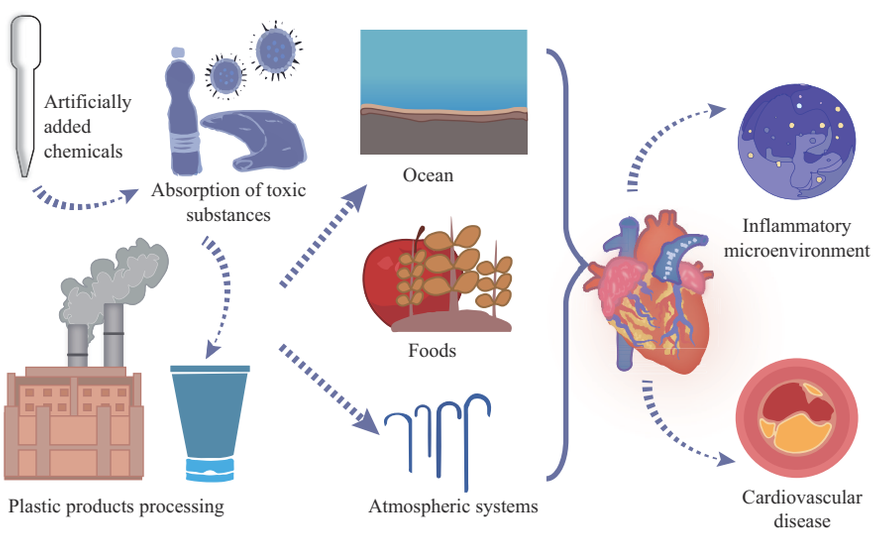
Graphical abstract



Abstract In recent years, with the large-scale use of plastic products, the degree of plastic pollution has increased, becoming a serious global problem. Microplastics and nanoplastics (MNPs), as emerging environmental pollutants, are widely found in organisms and the environment. These plastic particles enter the human body through 3 exposure pathways: breathing, the food chain's bioaccumulation and transfer, and skin contact, thereby exerting toxic effects. The physical attributes of MNPs, including their shape, size, and surface characteristics, are not static but rather undergo dynamic transformations in response to changing environmental conditions. These changes can significantly influence their behavior and interactions within different ecosystems. When considering MNPs as carriers of chemicals, two primary mechanisms can be distinguished. (1) MNPs have the capacity to adsorb pollutants from their surrounding environment. These pollutants may encompass a wide range of substances, such as heavy metals, organic compounds, and other contaminants that are commonly found

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in water, soil, or air. (2) MNPs may also carry chemical agents that are artificially introduced during their commercial production process. For example, flame retardants and pigments are often added to plastics to enhance their performance or appearance. These artificially added chemicals can remain associated with MNPs throughout their lifecycle and may contribute to their overall toxicological impact. Cardiovascular diseases (CVDs) are a general term for diseases of the heart, arteries, veins, and capillaries, and are one of the main causes of disability and death. CVDs have higher incidence, mortality, and recurrence rates, and more complications, which reduce the quality of life and happiness of patients, the phenomenon is gradually showing a trend of early onset, therefore early-stage prevention for CVDs is of critical importance. This article reviews the properties of MNPs and their potential threats to the cardiovascular system, aiming to explore how MNPs cause CVDs through certain physiological effects, toxicity mechanisms, and related pathways. Our review primarily focus on elucidating several critical mechanisms through which MNPs exert their adverse effects. Specifically, the review examines how the enhancement of oxidative stress can trigger the expression of pro-inflammatory factors, which in turn leads to the formation of a chronic inflammatory microenvironment within biological systems. Additionally, MNPs possess the capacity to adsorb toxic metals and organic substances from their surroundings. Furthermore, the review summarizes that sewage irrigation and atmospheric deposition are significant factors contributing to the co-pollution of heavy metals with MNPs in environmental settings. The interaction between heavy metals and MNPs has been shown to have detrimental effects on agricultural productivity, as it can inhibit crop growth and simultaneously increase the absorption rate of heavy metals in plants. When these contaminated plants enter the food chain, the accumulated heavy metals can ultimately be ingested by humans. This process poses a potential risk for inducing acute coronary syndrome and other CVDs, thereby underscoring the importance of understanding and mitigating the impact of MNPs on human health. In addition, our review also gives examples of the long-term effects of MNPs on cardiovascular function and the adverse consequences such as arrhythmia and atherosclerosis, the limitations of the current studies of MNPs affecting cardiovascular system health and future directions are also explored.

Key words　plastic particles, microplastics, nanoplastics, cardiovascular disease
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