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微塑料对生物体生殖发育的毒性作用及其分子机制研究进展

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摘要: 微塑料在环境中持续降解,对全球生态及生物健康构成了严重威胁。除累积于各生态系统外,微塑料可通过多种途径进入生物体,对动物神经系统、呼吸系统及消化系统产生不良影响。已有研究结果表明,微塑料具有生殖发育毒性,能干扰配子发生、胚胎发育及子代健康。本文综述了微塑料在不同生物模型中的生殖发育毒性研究进展,重点剖析了氧化应激、炎症反应、内分泌干扰及表观遗传调控等潜在分子机制。此外,本文对基于抗氧化剂、中草药及益生菌等的毒性缓解策略的开发与应用进行了展望。本文旨在为微塑料诱发的生殖发育毒性的毒理研究及环境风险评估提供科学参考。

关键词: 微塑料;生殖发育毒性;毒理机制;解毒途径;经济动物
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Research progress on the toxic effects of microplastics on the reproductive development of organisms and their molecular mechanisms

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Abstract: Microplastics continuously degrade in the environment, posing a serious threat to global ecology and biological health. Besides accumulating in various ecosystems, microplastics can enter organisms through multiple pathways, adversely affecting the nervous, respiratory, and digestive systems of animals. Existing studies have confirmed that microplastics exhibit reproductive and developmental toxicity and can interfere with gametogenesis, embryonic development, and offspring health. We reviewed the research progress on the reproductive and developmental toxicity of microplastics in different biological models, with a focus on analyzing potential molecular mechanisms such as oxidative stress, inflammatory response, endocrine disruption, and epigenetic regulation. Furthermore, we discussed the prospects for the development and application of toxicity mitigation strategies, including antioxidants, Chinese herbal medicines, and probiotics. This paper aims to provide a scientific reference for the study of the reproductive and developmental toxicology of microplastics and environmental risk assessment.

Key words: microplastics; reproductive and developmental toxicity; toxicological mechanisms; detoxification pathways; economic animals

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1 微塑料污染对生物健康的影响

微塑料被定义为直径小于 5 mm 的塑料颗粒或碎片,肉眼难以分辨且在环境中广泛分布[1]。随着塑料产量的激增,微塑料广泛分布于土壤、大气及地表水源等多种环境介质中,无处不在。这类污染物不仅存在于海洋和河流中,也通过农业地膜残留、污水排放等方式进入陆地生态系统。微塑料具有持久性和难降解性,通过长期的释放与迁移,最终进入人体及动植物体内,导致生物体机能紊乱,甚至引发疾病与死亡,由此造成的微塑料污染已成为当前主要的环境问题之一[2-3]。

有研究结果表明,不同类型和规格的微塑料能够通过饮食摄入[4-5]、吸入[6-7]以及皮肤直接接触[8]等途径进入生物组织乃至细胞,并在生物体内长期沉积。毒理学研究结果表明,微塑料在生物体内能够通过破坏下丘脑-垂体轴进而显示出神经毒性、细胞毒性、生殖毒性和免疫毒性,最终引起多系统的损伤[9]。值得注意的是,在经济动物如猪、牛、羊、鲤鱼、牡蛎等体内也检测到了不同大小和浓度的微塑料颗粒,并显示出不同程度的炎症、生殖障碍和发育异常[10-13]。越来越多的研究结果表明,经济动物体内的微塑料能够通过食物链传递并最终沉积在动物产品(如肉制品和奶制品)中[9-10,14]。作为食物链顶端的人类,对肉类、蛋类及奶制品的需求日益增长,其健康不可避免地受到微塑料的威胁。因此,深入研究微塑料的生物毒性及其作用机制,并开发有效的毒性缓解药物,具有重要的科学意义和应用价值。

近年来,微塑料的生殖发育毒性逐渐受到学术界的高度关注。微塑料可通过氧化应激、内质网应激、炎症反应、细胞凋亡、DNA 损伤、自噬及内分泌干扰等多种机制诱发生殖发育毒性。在哺乳动物中,针对微塑料对雄性动物生殖毒性的研究较为深入,其损害主要表现为破坏血睾屏障、抑制精子发生、导致精子畸形与 DNA 损伤等。相比之下,针对微塑料对雌性动物生殖毒性的研究较少,其毒理作用主要集中于损害卵母细胞成熟与卵泡生长、促进颗粒细胞凋亡、削弱卵巢储备功能及扰乱母体内分泌代谢等。此外,有研究发现微塑料可跨越母胎屏障,进而影响子代的存活和发育[15-16]。

当前,关于微塑料诱发的生殖发育毒性的研究主要集中于低等生物及水生生物,且对毒理机制的探索仍不够深入[17]。因此,亟需开展更多基础研究与临床试验,以揭示微塑料对生物体生殖发育的毒性作用机制,并研发有效的保护性干预措施,从而缓解微塑料对各类海陆生物的生殖损害。

2 微塑料的分布、传播与常见毒性

2.1 微塑料的环境分布与生物体摄入途径

塑料污染是当前海洋与陆地生态系统面临的重要环境问题之一,塑料降解后可分解为大量微塑料,释放至水、土壤和大气等环境介质中,并在其中迁移与转化,最终进入生物体内[13,18]。消化道摄入是微塑料进入生物体内的主要途径。小颗粒微塑料通过受污染食物被生物体摄入后,可经食道进入胃,进而穿过肠道屏障进入淋巴系统[19]或穿透肠道上皮细胞进入血液循环系统[20],最终积聚在体内器官中产生毒性效应。

2.2 微塑料的多途径人体暴露与器官毒性

微塑料可通过多种途径进入人体,并导致多器官毒性效应。有研究结果表明,微塑料已经在人体的血栓、血液样本中被检测到,且与炎症性肠病的发病与加重密切相关[21]。微塑料能够通过血脑屏障和胎盘,对人体神经系统和母体-胎儿健康造成威胁[22]。大气中的微塑料可通过呼吸作用进入生物体内,沉积于肺泡区域并干扰肺泡-毛细血管气体交换过程[23]。此外,微塑料可通过扩散或细胞吞噬等途径进入呼吸道上皮细胞,进而诱发炎症,造成肺组织损伤和呼吸系统异常[24-26]。目前在人类肺组织和痰液中已经鉴定出多种纤维状微塑料和颗粒状微塑料[27-28]。无处不在的微塑料也能够穿透皮肤这层屏障[29]。有研究结果表明,粒径小于 100 nm 的纳米级塑料颗粒能够穿透上皮组织进入生物体内,而较大的微塑料颗粒(粒径大于 100 nm)通常难以穿过完整的角质层,更多是滞留在皮肤表面或毛囊口,当皮肤屏障完整时,微塑料的穿透是有限的,但如果皮肤存在炎症、损伤、过敏情况时,穿透量会显著增加[30-31]。医疗接触是人体微塑料暴露的另一重要途径。微塑料的主要来源包括注射器、口罩、输液管以及人体植入设备等医疗器械和设备。这些微塑料能够通过扩散作用转移到患者的肝脏、脾脏和肾脏组织中,最终影响器官功能[32]。研究证实,微塑料在人体内能够诱导炎症、氧化应激、细胞凋亡、内分泌紊乱和菌群失调等一系列毒性效应,并与多

种疾病或症状(炎症性肠病、心血管疾病、自身免疫疾病、糖尿病、神经退行性疾病和生殖发育障碍等)密切相关[25,33-34]。

2.3 农业生态系统中微塑料的污染及食物链传递

农业生产正受到塑料污染物的显著影响。在作物生产过程中,塑料地膜的广泛应用导致土壤中微塑料的大量残留与聚集,这些微塑料可扩散至生态系统中并被生物体摄入[35]。此外,微塑料可通过堆肥施肥的过程,随动物粪便一同进入土壤环境,进而威胁作物生长[36]。值得注意的是,微塑料能够积聚在各种经济动物体内,并经过食物链传递到人体内。此外,微塑料还能够吸收有害物质(如双酚、邻苯二甲酸盐、二□恶英、多环芳烃、多溴联苯醚、重金属和有机污染物),并充当传输介质。携带内分泌干扰物的微塑料能够严重紊乱机体内激素受体和内分泌腺的作用,从而破坏下丘脑-垂体轴,诱导氧化应激、神经毒性和生殖毒性等[9]。目前,已经在牛和羊的可食用部分(如肉、肝脏和牛肚)中以及加工肉制品、禽蛋和奶制品中均检测到了微塑料[10]。通过微光谱衰减全反射分析,研究人员还在牛肉汉堡中发现了 18 种不同类型的微塑料,其中聚碳酸酯、聚乙烯和聚丙烯最为丰富[14]。在反刍动物体内及其粪便中也检测到了微塑料,其原因可能在于农场内大量使用塑料制品,如饲料加工、包装及储存过程中所用的塑料器具。此外,研究人员从反刍动物和猪的饲料中检测到多种微塑料聚合物,且所有饲料样本的微塑料检出率均为 100%[15,37]。

作为农业生产的重要组成部分,畜牧养殖业扮演着日益重要的角色。微塑料对畜牧业尤其是牲畜健康的影响逐渐引起人们的注意。研究人员发现,微塑料聚苯乙烯能够严重影响猪的肉质和骨骼肌发育,能够显著降低仔猪的肉红度指数以及Ⅰ型肌纤维密度。代谢组学分析发现,微塑料导致仔猪肌肉中肉香化合物肌肽、β-丙氨酸、棕榈酸和烟酰胺的含量均降低,并通过抑制血栓反应蛋白 1(THBS1)的降解,抑制仔猪肌肉血管的生成[38]。此外,微塑料能够影响猪肠道中肠神经系统神经元的数量和结构,造成肠道绒毛顶端损伤、细胞碎片化、黏液积聚以及嗜酸性粒细胞的浸润和充血[39]。由于肠道消化、免疫和屏障功能不成熟,仔猪更容易感染病原体并发生腹泻。有研究人员发现,聚苯乙烯的暴露增加了断奶仔猪腹泻的发生率,并损害其肠道屏障功能,进而引发肠道氧化应激和炎症[40]。另有研究结果表明,微塑料对猪肠道健康的影响具有剂量依赖性[41]。研究人员也发现了微塑料对猪心血管系统的影响。具体而言,微塑料能够通过提高衰老相关蛋白质的表达水平以及细胞内的氧化应激水平,从而促进猪冠状动脉内皮细胞的过早衰老[42]。此外,微塑料颗粒也能够对猪肾细胞产生毒理学影响。微塑料能够以时间-剂量依赖性方式被猪肾细胞内化,并通过引发炎症反应和氧化应激诱导细胞衰老[43]。聚苯乙烯的暴露还可引起羔羊胃肠道的损伤和消化系统紊乱并诱发炎症,最终导致羔羊日增质量和肉质下降[44]。总之,微塑料对家畜生长发育及畜牧业生产的影响日益凸显,深入研究其毒理机制至关重要,同时需采取多方面措施以减轻微塑料对畜牧业生产的损害。

3 微塑料的生殖发育毒性

生殖系统是微塑料毒性作用的重要靶标。生殖发育毒性是指生物体在接触某种物质或混合物后,其性功能、生育能力及后代发育受到损害的一种毒性效应[45-46]。近年来,越来越多的证据表明,微塑料暴露与人类及动物生殖能力下降密切相关。

3.1 微塑料对雄性生殖系统的影响

针对微塑料对雄性生殖系统影响的研究较为深入。微塑料能够在睾丸中积累,通过破坏血睾屏障、抑制精子发生,导致精子数量和活力下降。在人类精液和尿液中已检测到多种微塑料,其与精子质量下降及男性生殖能力降低显著相关。例如,聚四氟乙烯暴露与精液质量下降显著相关。接触聚四氟乙烯后,男性精子总数减少,精子浓度下降,且性功能显著减退。这一发现揭示了微塑料潜在的生殖健康风险,并引起了人们对微塑料日常暴露途径的广泛关注[47-48]。动物试验结果表明,微塑料暴露可导致雄性动物体质量增加、生殖激素分泌紊乱、睾丸质量下降及结构异常,最终引发不育。微塑料对不育雄鼠的睾丸毒性甚至比可育雄鼠更为严重,微塑料通过提高特定蛋白质的泛素化水平来降低精子活力,抑制获能过程[49-52]。

3.2 微塑料对雌性生殖系统的影响

相较于雄性生殖系统,微塑料对雌性生殖系统影响的研究也逐渐增多。聚苯乙烯微塑料作为一种内分泌干扰物,可严重干扰生殖系统功能,诱发多囊

卵巢综合征(PCOS)等生殖内分泌疾病。有研究发现,通过饮用水摄入的聚苯乙烯可诱发多囊卵巢综合征和长期卵巢纤维化,病理特征显示为促卵泡激素和β-雌二醇水平降低以及黄体生成素和睾酮水平升高。卵巢组织学分析结果显示,微塑料暴露后的女性体内成熟卵母细胞数量减少,且伴有囊性病变:卵泡膜解体、透明带内陷、卵膜肥大、嗜碱性颗粒堆积以及卵母细胞增生。此外,多囊卵巢综合征相关标志物(如 *tox3*、*dennd1a*、*fem1a*)的表达显著受抑,进而诱导了氧化应激、炎症以及卵巢纤维化[53]。

3.3 微塑料对胚胎发育的影响

孕期和胚胎期是生殖发育极为敏感的阶段。微塑料能够跨越母胎屏障,进入子宫内膜引起炎症和细胞凋亡,导致胎盘代谢功能紊乱、易位及胎儿生长迟缓[54-56]。有研究发现,微塑料可沉积在胎盘、羊水和胎便中,与复发性流产、胎儿出生体质量降低及死胎等不良妊娠结局相关[57-62]。另有研究结果表明,哺乳期母体暴露于聚苯乙烯中,也会产生严重的跨代生殖发育毒性。以使用塑料瓶制备婴儿配方奶粉过程中检测到的微塑料浓度为参照,暴露于该浓度的雌性小鼠表现出青春期延迟、发情周期紊乱、卵泡发育异常、卵巢类固醇生成中断、睾酮水平升高、生育能力下降和卵巢炎症[63]。在模式生物中,微塑料暴露会导致斑马鱼胚胎孵化率降低、畸形率升高[64-65],同时影响斑马鱼子代的自发性尾卷曲、昼夜运动等行为模式,并诱发细胞器功能障碍、心包水肿、骨骼畸形、色素沉着异常、视觉结构的完整性受损等[66-71]。

3.4 微塑料对生物体生长发育和生殖功能的跨代影响

微塑料的毒性不仅局限于亲代,还具有显著的跨代效应。小鼠在妊娠期或哺乳期暴露于微塑料,其后代往往表现出出生体质量下降、生长发育迟缓以及生殖功能障碍,子一代雄性小鼠可能出现精子发生异常。转录组学分析结果表明,子一代雄性小鼠的生殖系统发育和表观遗传修饰相关机制受到微塑料的显著影响。这种跨代毒性提示微塑料对生物种群繁衍存在潜在长期威胁[63]。

4 微塑料诱导生殖发育毒性的作用机制

微塑料诱导生殖发育毒性的分子机制复杂且多样,目前的研究主要聚焦于氧化应激、炎症反应、内分泌干扰、表观遗传调控等核心通路。

4.1 氧化应激

氧化应激是微塑料发挥生殖毒性的核心机制之一。微塑料暴露会导致生殖细胞内活性氧(ROS)过量产生,进而攻击脂质、蛋白质和脱氧核糖核酸(DNA)。在雄性个体中,微塑料通过激活线粒体 PINK1/Parkin 自噬通路,提高细胞内氧化应激水平,降低腺苷三磷酸(ATP)含量和线粒体膜电位,破坏线粒体结构,最终导致精子功能受损[72-73]。在雌性个体中,微塑料显著增加卵母细胞中的 ROS 水平,诱发氧化应激和线粒体功能障碍,导致卵母细胞质量下降和 DNA 损伤。此外,氧化应激还会激活下游的凋亡信号通路,促进生殖细胞死亡[74]。

4.2 炎症反应

炎症反应在微塑料引起的生殖损伤中扮演关键角色。微塑料暴露会激活体内的炎症小体(如 NLRP3)和转录因子(如 NF-κB),导致促炎因子(如 TNF-α、IL-6、IL-1β)的高表达。在雄性个体中,这种炎症环境会破坏血睾屏障的完整性,损伤睾丸组织,抑制精子发生。在雌性个体中,微塑料诱导的卵巢炎症会加速卵泡闭锁和颗粒细胞凋亡。此外,微塑料还可能通过扰乱肠道菌群,引发全身性低度炎症,进而通过“肠-睾丸轴”或“肠-卵巢轴”间接影响生殖功能[75]。

4.3 内分泌干扰

微塑料本身及其吸附的污染物具有内分泌干扰效应。它们能够模拟或拮抗内源性激素,扰乱下丘脑-垂体-性腺(HPG)轴的正常功能。有研究结果表明,微塑料暴露会影响血清中睾酮、雌二醇、黄体生成素和卵泡刺激素等关键生殖激素的水平。例如,微塑料通过干预促黄体激素介导的 LHR/cAMP/PKA/StAR 通路,导致睾酮合成量减少。这种内分泌干扰作用不仅影响性腺的发育和功能,还会导致生殖周期紊乱和性成熟异常[76]。

4.4 表观遗传调控

表观遗传调控是微塑料跨代毒性的重要机制。研究发现,微塑料暴露会导致生殖细胞中 DNA 甲基化、组蛋白修饰等表观遗传标志物的改变。这些改变可能不涉及 DNA 序列的变异,但会影响基因的表达模式,并可能遗传给后代。例如,在线虫模型中,微塑料暴露会导致与生殖相关的基因表达受抑,并

且这种影响可持续多代[77]。在哺乳动物中,雄性子代的生殖障碍与表观遗传修饰机制的异常密切相关,揭示了微塑料影响种群繁衍的深层次分子基础。

5 微塑料诱导的生物体生殖发育毒性的缓解途径

面对微塑料带来的严峻挑战,寻找有效的毒性缓解策略已成为研究热点。目前的缓解途径主要集中在应用抗氧化剂以及利用天然产物和微生物进行干预等方面。

5.1 抗氧化剂的应用

面对微塑料带来的生殖发育危害和生物安全威胁,人类开展了众多应对性研究,并不断将研究成果转化为生产生活中用以缓解微塑料毒性的实际手段。鉴于氧化应激在微塑料毒性中的核心作用,补充抗氧化剂成为一种直接的干预手段。*N*-乙酰半胱氨酸(NAC)作为一种抗氧化剂,能有效预防微塑料引起的血管内皮细胞衰老,减轻胎儿脑氧化损伤[78]。此外,白藜芦醇[沉默信息调节因子 1(Sirt1)激活剂]等药物也能缓解微塑料诱导的生殖系统损伤,其机制可能是通过清除过量自由基,维持细胞的氧化还原稳态[42]。

5.2 中草药的干预

多种中草药成分及其提取物被发现对微塑料生殖毒性具有明显的缓解作用。鼠李糖苷类化合物是一种广泛存在于植物中的多糖、糖苷,它能够通过恢复雄性白化大鼠的激素水平、调节细胞凋亡和减轻炎症反应,缓解聚苯乙烯微塑料引起的睾丸组织学和功能损伤[79]。虫草素(Cordycepin)是第一个从真菌中分离出来的核苷类抗生素,可以与微塑料竞争性结合,减轻微塑料暴露下的精细胞氧化应激[80]。银杏素和山柰素等黄酮类化合物具有抗氧化特性,通过调节谷胱甘肽过氧化物酶(*GPx*)、过氧化氢酶(*CAT*)、超氧化物歧化酶(*SOD*)及谷胱甘肽还原酶(*GSR*)等抗氧化酶的活性,并降低脂质过氧化标志物丙二醛(MDA)的含量,从而抑制活性氧(ROS)的堆积。此外,银杏素和山柰素等黄酮类化合物还能发挥抗炎和抗凋亡作用,恢复微塑料暴露后的精子数量、活力及雄激素水平,降低精子畸形率,改善睾丸组织病理学损伤。山柰素的作用还与激活 Nrf-2 通路、恢复抗氧化酶表达密切相关。益肾通络方是一种中药方剂,能够通过 PI3K/Akt 通路保护精子 DNA 完整性[81-82]。此外,牛磺酸也被证实可有效逆转微塑料对卵母细胞质量的损害,能够有效恢复受损卵子的线粒体功能,降低活性氧水平,改善纺锤体异常、染色体异常、皮质颗粒分布紊乱等缺陷,进而保障卵子成熟和早期胚胎发育[83]。

5.3 益生菌的作用

肠道微生态与生殖健康密切相关。研究发现,微塑料暴露会导致肠道微生物结构紊乱,而补充益生菌(如乳杆菌、长双歧杆菌)可以通过调节“肠道-生殖轴”,改善睾丸的生精功能。益生菌能够减少炎症因子的释放,缓解氧化应激,从而改善精子质量[84]。这为通过调节肠道菌群来对抗微塑料生殖毒性提供了新的思路。

5.4 其他缓解微塑料生殖毒性的潜在保护剂

除了上述物质,外源硫化氢(H_2S)、褪黑素等也显示出对微塑料诱导的生殖系统损伤的潜在保护作用。褪黑素主要通过恢复生长激素/胰岛素样生长因子的表达,减少氧化应激和细胞凋亡,从而缓解微塑料导致的跨代毒性。外源硫化氢可以增强细胞对氧化应激的耐受力,能够在睾丸中通过调节 Nrf2/PGC-1α 信号来缓解线粒体凋亡和自噬,最终消除微塑料诱导的体外生殖发育毒性[85]。

6 总结与展望

微塑料的全球性污染对生物多样性和人类生殖健康构成严重威胁。生殖系统关乎人类生生不息的繁衍,因此针对微塑料生殖发育毒性的基础研究以及针对其毒性缓解策略的应用性研究至关重要。农业生产事关国家经济社会发展和人民生活,然而越来越多的研究发现微塑料能够进入土壤、农业用水以及饲料等生产资料中,严重危害经济动物的繁殖与生长。目前从肉制品等食物中已检测出微塑料,表明人类健康也因此受到了不可避免的威胁。本文综述了微塑料在雄性生殖系统、雌性生殖系统、胚胎发育及跨代遗传中的毒性表现,并深入剖析了氧化应激、炎症反应、内分泌干扰及表观遗传调控等分子机制。同时,总结了微塑料诱导的生物体生殖发育毒性的缓解途径。尽管已有研究取得了一定进展,但仍存在诸多不足。首先,目前的毒理学研究主要集中于斑马鱼、小鼠等模式生物及体外试验,针对人体及大型经济动物(如牛、羊)的体内研究相对匮乏。由于生物体代谢和环境暴露的复杂性,体外试

验结果难以完全反映真实情况,因此亟需开展更多哺乳动物体内的验证性研究。其次,实际环境中生物体往往暴露于多种微塑料及复合污染物中,单一聚苯乙烯微塑料的研究结果可能低估了其联合毒性,微塑料与其他污染物的协同作用机制尚待明确。此外,微塑料的跨代毒性研究尚处于起步阶段,表观遗传机制的细节及其长期影响仍需深入探索。

未来研究应重点关注以下几个方面:一是加强对经济动物和人体微塑料暴露的流行病学调查及毒理学机制研究;二是深入探讨微塑料与重金属、有机污染物等复合暴露的联合毒性效应;三是建立长期的跨代毒性模型,持续追踪后代的生殖表观遗传改变;四是开发高效、低毒的解毒药物或功能性饲料添加剂,并推动其在农业生产中的应用。同时,应加强对塑料产品的使用监管和回收处理,从源头上减少微塑料排放,以最大程度降低其对生态系统和人类健康的危害。

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