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人体微塑料污染特征及健康风险研究进展

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摘要: 微塑料污染不仅是一个环境问题, 更是一个社会问题。目前针对环境微塑料来源、丰度和分布, 已开展了大量研究, 但对人体微塑料暴露水平和潜在健康风险的认识仍非常有限。基于文献计量学方法, 系统地梳理总结了当前人体微塑料的暴露途径, 揭示了人体微塑料污染的赋存特征及潜在健康危害。结果表明, 人体内微塑料主要以粒径小于 50 μm 的聚乙烯(PE)、聚丙烯(PP)、和聚对苯二甲酸乙二醇酯(PET)等人工合成聚合物为主。环境微塑料主要通过食物和呼吸途径进入人体, 并在肺部和肠胃组织累积, 其中小粒径微塑料可通过循环系统分布于各组织器官。基于实验的毒理学实验结果表明, 微塑料不仅影响细胞膜完整性、免疫应激、肠道菌群和能量代谢, 对生殖系统也具有潜在危害。为科学评估微塑料污染的健康风险, 还需进一步加强微塑料毒理效应及其发生机制研究, 构建微塑料健康风险评估框架, 以科学依据支撑微塑料污染防治。

关键词: 微塑料; 暴露途径; 毒理效应; 分子机制; 健康风险

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Research Progress on Characteristics of Human Microplastic Pollution and Health Risks

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Abstract: Microplastic pollution is not only an environmental problem but also a social problem. Many studies have been conducted on the sources, abundance, and distribution of microplastics in the environment, but an understanding of human exposure levels and potential health risks remains very limited. Based on the bibliometric methods, the present review systematically summarized the exposure pathways of microplastics in humans, and then the characteristics and potential adverse impacts on human health were expounded upon. Available literature showed that microplastics in human bodies were mainly concentrated on sizes smaller than 50 μm , and polyethylene (PE), polypropylene (PP), and polyethylene terephthalate (PET) were the main polymers. Microplastics in environments entered human bodies mainly through food and respiratory pathways, then accumulated in lung and gastrointestinal tissues. Most importantly, small-sized microplastics could distribute in tissues and organs via the circulatory system. The results from lab-based toxicological experiments showed that microplastics not only posed threats to cell membrane integrity, immune stress, gut microbiota, and energy metabolism but also had potentially adverse impacts on the reproductive system. To further understand the health risks of microplastic pollution, it is necessary to promote research on the toxicological effects of microplastics as well as the inner mechanisms and also to establish risk assessment frameworks for evaluating microplastic pollution. These works are crucial to preventing the risks of microplastic pollution with scientific evidence.

Key words: microplastics; exposure routes; toxicological effects; molecular mechanism; health risk

自 1972 年 Carpenter 等^[1]在大西洋马尾藻海域首次发现塑料碎片到 2004 年英国学者 Thompson 等^[2]首次提出“微塑料”概念以来,微塑料在河流、河口、近岸海域甚至极地大洋以及多种陆生和水生生物体相继检出,微塑料污染也逐渐受到全社会的广泛关注,并被联合国环境大会列入全球亟待解决的第二大环境问题。微塑料是指粒径小于 5 mm 的塑料微粒,按照形状可分为球形、颗粒、碎片、薄膜、纤维以及不规则状等,材质则包括聚乙烯(polyethylene, PE)、聚丙烯(polypropylene, PP)和聚苯乙烯(polystyrene, PS)等各种人工合成高分子聚合物。环境微塑料的来源可分为原生微塑料和次生微塑料,其中原生微塑料是指因需要生产使用的微塑料,如作为原材料的树脂颗粒、向化妆品中添加的塑料微珠等;次生微塑料是指非故意产生的微塑料,如大块塑料老化降解产生的碎片、织物洗涤脱落的纤维和轮胎磨损产生的

颗粒等。微塑料因具环境持久性、生物蓄积性和危害隐蔽性等特征,被我国列入《新污染物治理行动方案》(国办发[2022]15号)以加强污染防治。

微塑料体积小,但比表面积相对较大,并随着表面老化吸附污染物的能力增大,是环境污染物和病原微生物进入生物体的重要载体。实验室研究结果表明,微塑料会对生物体产生多方面的不利影响,如诱发免疫反应^[3]、氧化应激和神经毒性^[4]、发育毒性^[5]和胚胎毒性^[6]等。同时,由于微塑料自身的物理和化学属性及其吸附污染物具有的“特洛伊木马”效应^[6],微塑料与环境污染物形成的复合污染会潜在危害生物健康。有研究发现,微塑料与邻苯二甲酸酯联

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合暴露不仅导致小鼠产生严重的肠道炎症和代谢紊乱^[7],还会产生明显的生殖毒性^[8];另外,微塑料与四溴双酚 A 共同暴露还会干扰人体肠道微生物群的代谢途径^[9]. 近年在人体排泄物、肺部、胎盘以及血液^[10]中相继检出不同材质微塑料,暗示微塑料可能随着血液进入人体各组织器官,更是引起了全社会对于环境微塑料健康危害的广泛担忧.

为加深对环境微塑料污染健康危害的理解,本文对近年环境微塑料污染与人体健康的相关研究进行了系统梳理,识别微塑料危害人体健康的研究方向,以期提高对环境微塑料污染风险的认知,并为科学评估环境微塑料污染的潜在健康风险提供有益参考.

1 人体摄入微塑料的途径

环境微塑料可通过多种途径进入人体,其中经口摄入和呼吸吸入是人体的主要暴露途径,而皮肤接触是人体的一个潜在暴露途径.

1.1 食物摄入

从目前研究结果来看,人体非主动摄入微塑料是不可避免的. Cox 等^[11]测算发现,一个成年男性每天经口摄入 142 个微塑料,每年的摄入量为 $(51\,814 \pm 8\,172)$ 个微塑料,其中日常饮用水和食物是人体摄入微塑料的主要来源. Mohamed 等^[12]研究发现利用人体终生暴露模型预测儿童和成人每天通过食物和饮用水摄入微塑料($1 \sim 5\,000\ \mu\text{m}$)的中位数分别为 553 个 $\cdot(\text{人}\cdot\text{d})^{-1}$ 和 883 个 $\cdot(\text{人}\cdot\text{d})^{-1}$,当他们分别长到 18 岁和 70 岁时,微塑料不可逆积累量可高达 8.32×10^3 个和 5.01×10^4 个,这与自来水和食物中广泛检出微塑料密不可分. 另有研究结果表明,自来水中微塑料丰度为 5.45 个 $\cdot\text{L}^{-1}$ ^[13],而塑料瓶装水中的微塑料丰度则可达到 118 ~ 325 个 $\cdot\text{L}^{-1}$ ^[14, 15],是自来水的 20 倍以上. 因此有研究估算通过饮用水(尤其瓶装水)摄入的微塑料贡献了人体摄入微塑料总量的 88% 以上^[16]. 同样,牛奶中也检出了微塑料 $[(7 \pm 2.83)\text{个}\cdot\text{L}^{-1}]$ ^[17],但明显低于蜂蜜 $[(166 \pm 147)\text{个}\cdot\text{kg}^{-1}]$ 和糖 $[(217 \pm 123)\text{个}\cdot\text{kg}^{-1}]$ 中微塑料的赋存水平^[18]. 值得注意的是,作为日常消费食物中不可或缺的食材,食盐中也广泛检出了微塑料,并且海盐中微塑料丰度(550 ~ 681 个 $\cdot\text{kg}^{-1}$)明显高于湖盐(43 ~ 363 个 $\cdot\text{kg}^{-1}$)和岩盐(7 ~ 204 个 $\cdot\text{kg}^{-1}$)^[19]. 有研究者推测,若每人每天食用 5 g 盐,则仅通过海盐摄入的微塑料每人每年就可累积达到 216 个^[20]. 此外,食品中的微塑料含量也与食品的生产、加工、包装到消费整个过程有关,如通过清洗方式可将生米中微塑料 $[3.7\text{ mg}\cdot(100\text{ g})^{-1}]$ 减少 25% $[2.8\text{ mg}\cdot(100\text{ g})^{-1}]$;但若将生米直接加工成速熟

米,则速熟米中微塑料含量 $[13.3\text{ mg}\cdot(100\text{ g})^{-1}]$ 几乎是生米的 4 倍^[21]. 在当前有关饮品和食物中微塑料的研究显示,一次性茶包可向茶水中释放约 116 亿个纳米塑料微粒,是已知人体摄入微塑料丰度最高的饮料^[22].

1.2 呼吸摄入

根据调查的空气中微塑料丰度,粗略估算一个成年人每天通过呼吸能够摄入 170 个微塑料,每年摄入量大约为 $(61\,928 \pm 68\,865)$ 个^[11],可以看出通过呼吸摄入的微塑料明显高于通过食物摄入,说明呼吸摄入和经口摄入都是微塑料进入人体的主要途径. 尽管目前有关大气中微塑料污染水平的研究相对较少,但有限研究仍显示空气中微塑料污染发生的普遍性并具有明显的区域差异性. 调查发现,我国北方地区空气中微塑料丰度 $[(393 \pm 145)\text{个}\cdot\text{m}^{-3}]$ ^[23]明显高于南方地区空气中微塑料的丰度 $[(267 \pm 117)\text{个}\cdot\text{m}^{-3}]$ ^[24],特别是广东东莞地区微塑料丰度 $[(36 \pm 7)\text{个}\cdot\text{m}^{-3}]$ 约是北方地区的 1/10^[25]. 与我国相比,其他国家空气微塑料赋存丰度则显示出了较低水平,如伊朗布什尔港空气中微塑料丰度仅为 5.2 个 $\cdot\text{m}^{-3}$ ^[26],而西班牙马德里地区空气中微塑料丰度也仅达 13.9 个 $\cdot\text{m}^{-3}$ ^[27]. 此外,有研究发现同一区域室内外空气中微塑料丰度有着明显的差别,如中国台湾室内空气中微塑料丰度 $[(46 \pm 55)\text{个}\cdot\text{m}^{-3}]$ 略高于室外 $[(28 \pm 24)\text{个}\cdot\text{m}^{-3}]$ ^[28],而法国巴黎室内空气中以纤维为主的微塑料丰度(1 ~ 60 个 $\cdot\text{m}^{-3}$)则明显高于室外(0.3 ~ 1.5 个 $\cdot\text{m}^{-3}$)^[29],这与中国温州室内空气中微塑料丰度 $[(1\,583 \pm 1\,180)\text{个}\cdot\text{m}^{-3}]$ 明显高于室外 $[(189 \pm 85)\text{个}\cdot\text{m}^{-3}]$ ^[11]的研究结论相一致,这可能是与室内空间较小并且空气流通相对较差有关. 尽管有研究认为呼吸摄入微颗粒是人体微塑料的主要来源,但是目前仍没有直接证据表明呼吸摄入的微塑料与人体健康之间的潜在关系.

1.3 皮肤接触

虽然经口摄入和呼吸吸入是微塑料进入人体的主要途径,但面部清洗剂、沐浴露^[30]、化妆品以及其他个人护理品中^[31]广泛检测出的纳米塑料微粒,经皮肤的潜在暴露途径也逐渐引起了研究者的关注. 尽管微/纳塑料经皮肤进入人体发生概率极小,但它们仍有可能通过伤口、汗腺或毛囊进入皮肤^[32]. Alvarez-Román 等^[33]探究纳塑料如何通过皮肤接触进入组织时,发现粒径 20 nm 的 PS 塑料颗粒主要累积在皮肤的毛囊中,但很难渗透到角质层;同样, Campbell 等^[34]也发现 20 ~ 200 nm 的 PS 颗粒只能渗透进入皮肤顶层 2 ~ 3 μm ,而不能进入皮下组织. 然而,暴露在紫外条件下会使皮肤损伤,皮肤抵抗能力

变弱,这将增加纳塑料颗粒通过皮肤被人体吸收的可能^[35]。为了预防穿透皮肤吸收的暴露风险,塑料微粒经皮肤进入人体途径仍需关注并开展潜在健康影响研究。

1.4 特定人群的暴露途径

与成人相比,儿童尤其婴儿可能处于更高的环境微塑料暴露水平,如长时间在室内活动以及特殊的饮食方式。有研究发现,婴儿使用的奶瓶释放的微塑料可高达 $1\,620\text{万个}\cdot\text{L}^{-1}$,并且灭菌和高温消毒处理还会进一步促进微塑料的释放^[36]。另外,有研究发现婴儿日常使用的塑料奶瓶、水瓶经过100个开关循环,会从塑料奶瓶瓶盖和瓶颈之间释放 $(53\pm 9.4)\sim(393\pm 57.5)\text{个}\cdot\text{mL}^{-1}$ 的微塑料,与塑料水瓶释放量 $[(100\pm 23.3)\sim(209\pm 38.4)\text{个}\cdot\text{mL}^{-1}]$ ^[37]相近,进而也导致婴儿和儿童比成人处于更多来源的微塑料暴露。此外,随着有研究陆续在孕妇胎盘中检测出微塑料,微塑料对孕妇及新生儿健康影响也受到了格外关注。深入调查孕妇、新生儿和儿童微塑料的特定暴露途径和摄入水平,探索可能发生的潜在健康危害及其作用机制,从根本上改善微塑料污染水平,是亟待解决的科学难题和社会问题。

2 人体微塑料的赋存特征

环境及食物和饮用水中广泛检出微塑料,也就意味着人体也处于微塑料的暴露环境之中。如表1所示,近年相继在人体不同组织器官、体液和排泄物中检出各种材质、形状和丰度的微塑料也证实了这个结论。

2.1 消化系统中微塑料赋存特征

粪便中检出微塑料是人体处于微塑料暴露的直接证据。Zhang等^[38]调查了18~25岁男性粪便样本,发现其中95.8%样品含有以PP为主要材质、粒径在20~800 μm 的微塑料 $(1\sim 36\text{个}\cdot\text{g}^{-1})$,并进一步通过相关性检验分析发现,参与者体内微塑料丰度与他们日常消费带包装的水和饮料中微塑料丰度具有较强的相关性($P=0.029$),但由于实验中没有考虑更多性别和地区对实验结果的影响,该研究结果仍存在一定局限性。Schwabl等^[39]调查33~65岁健康人群粪便时,同样发现以PP和聚对苯二甲酸乙二醇酯(polyethylene terephthalate, PET)为主要材质、粒径在50~500 μm 的9种塑料微粒 $(10\sim 20\text{个}\cdot\text{g}^{-1})$,其研究结果暗示人体微塑料来源的多样性,即除了食品和水,食品加工、包装或制备,空气沉降物也是人体微塑料可能来源。Luqman等^[40]研究发现20~50岁健康人群每克粪便中约有3.33~13.99 μg 微塑料,材质主要是高密度聚乙烯(high-density polyethylene,

HDPE),同时检出的PP、PET等材质可能与参与者食用了不同食品和饮用水等产品有关。相似地,Wibowo等^[41]调查印度尼西亚某地区20~50岁年龄段健康人群粪便的微塑料,丰度与Luqman等^[40]的结果相近 $(6.49\sim 16.55\text{ }\mu\text{g}\cdot\text{g}^{-1})$,但主要材质除HDPE以外还存在较高丰度的PP,认为这不仅与参与调查者牙膏中丰度较高的HDPE颗粒有关外,还与当地人群广泛食用的豆豉有关。Zhang等^[42]研究发现婴儿粪便中PET微塑料含量 $(5.7\sim 8.2\text{ ng}\cdot\text{g}^{-1})$ 明显高于成人 $(1.6\text{ ng}\cdot\text{g}^{-1})$,这可能与婴儿接触了更多塑料包装饮食和玩具有关,暗示婴儿比成人处于更高的微塑料暴露环境。Ibrahim等^[43]在11个结肠切除术标本中发现了不同颜色的微塑料 $(12.7\sim 43.5\text{个}\cdot\text{g}^{-1})$,进一步证实了微塑料可以积累在消化道中。另外,Horvatits等^[44]在肝硬化患者的肝脏 $(3.2\text{个}\cdot\text{g}^{-1})$ 和脾脏 $(0.9\text{个}\cdot\text{g}^{-1})$ 中检出了微塑料,肾脏中并未有微塑料的存在,然而在无基础肝脏疾病患者的肝脏、肾脏和脾脏均未发现微塑料,但微塑料积累与肝脏疾病之间的潜在关系还需进一步研究,以便揭示微塑料污染对人体健康的潜在危害。

2.2 呼吸系统中微塑料赋存特征

同样,各种微塑料在人体呼吸系统中也被相继检出。Abbasi等^[45]以漱口的方式采集了2000个唾液样本,检测发现微塑料的平均丰度高达 $0.33\text{个}\cdot\text{人}^{-1}$,主要是粒径小于100 μm 的纤维,材质以PP和PE为主;Huang等^[46]在22名肺部疾病患者的痰液中也发现了微塑料,丰度为 $18.75\sim 91.75\text{个}\cdot(10\text{ mL})^{-1}$,材质主要是聚氨酯(polyurethane, PU)和聚酯(polyester, PES)等,并且检出到的微塑料粒径均小于500 μm ,这可能与检测方式有关。两项研究结果均再次证实微塑料可以通过呼吸进入呼吸道,也可以随着唾液和痰液排出,但最后仍有部分微塑料会进入肺组织,由此对肺功能产生的影响是一个全社会都非常关注的健康问题。Pauly等^[47]早在1998年通过显微镜和偏振光探究114例肿瘤患者的肺组织样本,87%的样本中存在微纤维;而文献[48]更是在2021年利用拉曼(Raman)光谱技术首次在人体肺组织样本中检出并确定了微塑料,即在13个尸检的肺组织样本中检出了33个粒径小于5.5 μm 的微塑料颗粒和4个8.12~16.8 μm 的微纤维,并且发现PP和PE是以上微塑料的主要材质。相似地,Jenner等^[49]在11例肺组织样本共检出了39个微塑料 $[(0.69\pm 0.84)\text{个}\cdot\text{g}^{-1}]$,并且微塑料广泛分布在整个肺组织,包括上区域、中区域和下区域,材质也以PP、PET和PE为主,这与文献[48]的结论相似,而49%的微塑料形状呈纤维状,这与Pauly等^[47]调查的结果一致。以上研

表 1 人体内微塑料存在特征¹⁾
Table 1 Characteristics of microplastics in human bodies

组织/器官/体液/排泄物	地点	年龄(岁)	人数	性别(男:女)	丰度	材质	粒径/ μm	形状	检测方法	文献
粪便	印度尼西亚泗水沿海地区	20~50	11	9:2	$3.33 \sim 13.99 \mu\text{g} \cdot \text{g}^{-1}$	LDPE、LLDPE、PP、PS 和 PET	—	—	Raman	[40]
	欧洲、亚洲	33~65	8	3:5	$10 \sim 20 \text{ 个} \cdot \text{g}^{-1}$	PP、PET	50~500	碎片和薄膜	FTIR	[39]
	印度尼西亚农业社区	20~50	11	5:6	$6.49 \sim 16.55 \mu\text{g} \cdot \text{g}^{-1}$	PP、PET、PS、PE、HDPE 和 LDPE	—	—	Raman	[41]
	中国北京	18~25	26	26:0	$1 \sim 36 \text{ 个} \cdot \text{g}^{-1}$	PP、PET、PS、PE、PVC、PC、PA 和 PU	20~800	—	FTIR	[38]
	美国(纽约)	1	6	—	PET: $5.7 \sim 8.2 \mu\text{g} \cdot \text{g}^{-1}$ PC: $0.049 \sim 2.1 \mu\text{g} \cdot \text{g}^{-1}$	PET、PC	—	—	LC-MS/MS	[42]
结肠	马来西亚半岛	34~88	11	6:5	$12.7 \sim 43.5 \text{ 个} \cdot \text{g}^{-1}$	PC、PA 和 PP	—	—	FTIR	[44]
痰	中国(广东)	17~93	22	17:5	$18.75 \sim 91.75 \text{ 个} \cdot 10 \text{ mL}^{-1}$	PU、PES、alkyd varnish、Chlorinated polyethylene	20~500	—	LDIR、FTIR	[46]
	中国(沈阳)	20~45	16	16:0	—	PVC 和 PA	—	纤维	LDIR	[55]
肺	英国(城堡山医院)	32~77	11	6:5	$(0.69 \pm 0.84) \text{ 个} \cdot \text{g}^{-1}$	PP、PET	平均长度: 223.10 ± 436.16 平均宽度: 22.21 ± 20.32	纤维、碎片和薄膜	FTIR	[49]
	巴西(圣保罗)	48~94	20	7:13	—	PP、PE、棉花	颗粒: $1.90 \sim 5.56$ 纤维: $8.13 \sim 16.8$	颗粒、纤维	Raman	[48]
肝脏	德国	56	6	4:2	$3.2 \text{ 个} \cdot \text{g}^{-1}$	PS、PVC 和 PET	3.0~29.5	—	Raman	[43]
	意大利(罗马)	—	6	0:6	—	PP	5~10	球状、不规则	Raman	[48]
	德国(柏林)	—	2	0:2	—	PE、PP 和 PU	>50	—	FTIR	[47]
胎盘	意大利(罗马)	25~42	10	0:10	—	—	2.1~18.5	—	Raman	[50]
	伊朗(德黑兰)	25~29	43	0:43	$2 \sim 38 \text{ 个} \cdot \text{个}^{-1}$	PE、PS	7.3~27.6	纤维、碎片、薄膜和颗粒	Raman	[56]
	中国(无锡)	23~36	17	0:17	$0.28 \sim 9.55 \text{ 个} \cdot \text{g}^{-1}$	PVC、PP 和 PBS	20.3~307.9	碎片和纤维	LDIR	[57]
	中国(上海)	30~35	18	0:18	$18 \text{ 个} \cdot \text{g}^{-1}$ (平均)	PA、PU	20~50	—	LDIR	[58]
唾液	伊朗	—	2 000	—	$0.33 \text{ 个} \cdot \text{人}^{-1}$	PE、PET	<100	纤维	Raman	[45]
血液	荷兰	—	22	—	$1.6 \mu\text{g} \cdot \text{mL}^{-1}$	PE、PET 和 PS	≥ 700	—	Py-GC/MS	[10]

1) LDIR:激光红外成像系统(laser direct Infrared), FTIR: 傅里叶变换红外光谱(fourier-transform infrared spectroscopy), LC-MS/MS:液相色谱串联质谱, Py-GC/MS:热裂解-气相色谱/质谱, LDPE:低密度聚乙烯(low-density polyethylene), LLDPE:线性低密度聚乙烯(linear-low-density polyethylene), PC:聚碳酸酯(polycarbonate), PA:尼龙(polyamide), PES:聚酯(polyester), PVC:聚氯乙烯(polyvinylchloride), PBS:聚丁二酸丁二醇酯(polybutylene succinate); “—”表示数据未报告

究结果证实了呼吸系统是人体摄入微塑料的途径,而肺部 是微塑料在人体内积累的主要器官之一.

2.3 生殖系统中微塑料赋存特征

2020 年, Ragusa 等^[50]用拉曼光谱首次 在人体胎盘中检出了微塑料,即在胎儿侧 检出 4 个微塑料,母体侧检出 5 个微塑料,绒毛侧检出 3 个微塑料,材质

均以 PP 为主,并且证实了人体胎盘中与微塑料兼容的片段在细胞室中的存在和定位,推测它们与胞浆内细胞器(线粒体和内质网)的重要超微结构的改变之间可能存在一定的相关性^[51]. 相似的, Braun 等^[52]用傅里叶变换红外光谱(FT-IR)同时在胎盘组织和胎粪中检出了以 PP 和 PE 为主要材质和粒径大于 50

μm 的微塑料。需要指出的是,母体和胎儿之间存在胎盘屏障,尽管胎盘中检出的微塑料说明微塑料已经穿透了胎盘屏障,但由此对胎儿发育和健康的影响还需更多的证据,以科学数据回应社会关切。

2.4 血液系统中微塑料赋存特征

最近,Leslie 等^[10]在采集的 22 名身体健康志愿者全血中检出了以 PET 和 PE 为主要材质、并且粒径大于 700 nm 的塑料微粒($1.6\text{ }\mu\text{g}\cdot\text{mL}^{-1}$);与之相比,Wu 等^[53]更是在 26 个血栓样品中的 16 个样品检出了 87 个 2.1~26 μm 的 PE 微塑料及其他聚合物材质的微粒,暗示微塑料已经进入血液并可能会通过血液循环系统进入全身各组织器官,最终在肝脏、脾脏和其

他器官中积累^[54],这势必引起社会对环境微塑料污染影响人体健康的担忧。不可否认,随着高灵敏检测技术发展和社会关注度持续升高,微塑料将在人体的更多组织器官中被检出,并且微塑料的检出丰度和材质类型也会呈增加趋势,这对科学评估环境微塑料污染的健康风险提出了迫切需求。

3 微塑料污染的潜在健康危害

目前尽管还缺少微塑料污染危害人体健康的直接的科学证据,但现有的体内实验结果(表 2)和体外实验结果仍在一定程度上证实了微塑料污染对人体健康的潜在不利影响(图 1)。

表 2 微塑料对小鼠健康的影响¹⁾
Table 2 Impacts of microplastics on mice

材质	暴露剂量	暴露时间/d	反应	文献
PS	$20\text{ }\mu\text{g}\cdot(\text{g}\cdot\text{d})^{-1}$	7	肠道菌群多样性显著降低	[72]
	$0.5、5\text{ 和 }50\text{ mg}\cdot(\text{kg}\cdot\text{d})^{-1}$	90	结肠炎症	[73]
	$1\text{ }000\text{ }\mu\text{g}\cdot\text{L}^{-1}$	35	改变小鼠肠道菌群组成,诱导肝脏脂质紊乱	[57]
	$20\text{ mL}\cdot\text{kg}^{-1}$	28	肠道屏障功能障碍	[55]
	$1\text{ }000\text{ }\mu\text{g}\cdot\text{L}^{-1}$	42	肠道菌群失调、屏障功能障碍、代谢紊乱	[56]
	$20\text{ }\mu\text{g}\cdot\text{L}^{-1}$	28	加剧了糖尿病小鼠肾脏代谢紊乱	[74]
	$0.5、5\text{ 和 }50\text{ mg}\cdot(\text{kg}\cdot\text{d})^{-1}$	90	肝脏损伤	[75]
	$15、50\text{ 和 }300\text{ mg}\cdot\text{kg}^{-1}$	14	肝功和脂代谢异常	[76]
	$0.1\text{ mg}\cdot\text{d}^{-1}$	28	肝脏氧化应激、能量代谢和脂代谢紊乱	[77]
	$0.1、1\text{ 和 }10\text{ mg}\cdot\text{L}^{-1}$	21 和 56	生殖毒性、发育毒性	[78]
	$0.01、0.1\text{ 和 }1\text{ mg}\cdot\text{d}^{-1}$	42	睾丸组织损伤、精子质量和数量下降	[79]
	300 μg	24	影响哮喘小鼠呼吸系统	[64]
	250 μg	GD5.5、7.5 和 11.5	对妊娠造成不良影响	[59]
	$100\text{ mg}\cdot\text{L}^{-1}\text{ 和 }1\text{ }000\text{ mg}\cdot\text{L}^{-1}$	PND0	后代代谢紊乱	[80]
	$0.015、0.15\text{ 和 }1.5\text{ mg}\cdot\text{d}^{-1}$	90	精子活力和浓度降低,精子异常升高,引起雄性生殖毒性	[81]
	$5\text{ mg}\cdot(\text{kg}\cdot\text{bw})^{-1}$	28	雄性生殖功能造成潜在毒性	[82]
PVC、PS 和 PC	2%	61	雄性生殖毒性	[83]
PU 和 PS	20 μL	14	对暴发性肝炎小鼠肝脏损伤具有促进作用	[84]
PE	6、60 和 $600\text{ mg}\cdot\text{d}^{-1}$	35	诱发肠道菌群失调和炎症	[58]
PE 和 PS	0.3 % 和 3 %	27	小肠显微结构破坏,生长减慢	[85]
PE 和 PP	1%	60	畸形率增加	[86]

1) GD 5.5:怀孕第 5.5 d;PND0:生产出子代的第 0 d;bw:体重(body weight);a%:暴露剂量的百分数,即每 100g 饲料中添加目标微塑料的质量

3.1 微塑料对组织和器官的影响

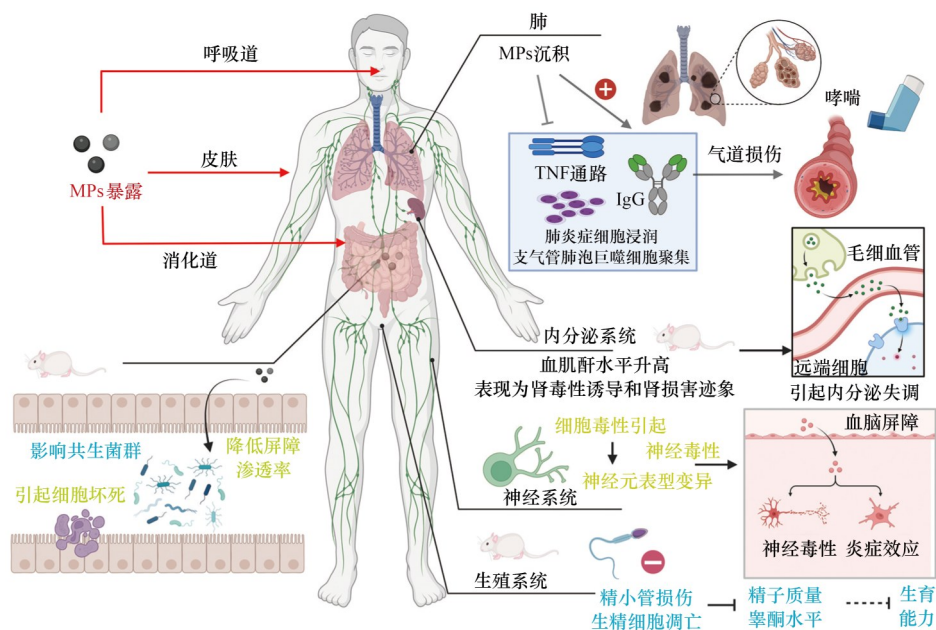
3.1.1 肠道

有研究发现微塑料不仅能够影响小鼠的肠道屏障功能^[59]、减少肠道粘液分泌^[60],还能改变肠道微生物群落构成^[61]和导致肠道菌群失调^[62]等。但 Stock 等^[63]在组织学水平上并没有检出 PS 微塑料对小鼠肠道造成损伤,Stock 等^[64]还利用人体细胞系 Caco-2、HepG2 和 HepaRG 研究 4 种塑料微粒(PE、PP、PET 和 PVC,粒径范围为 1~4 μm 和 10~20 μm)对肠道和肝脏的细胞影响,结果发现低浓度并不会诱发细胞的

急性毒性,而只有高浓度才会引起细胞发生明显毒性效应,并且产生的毒理效应与微塑料的形状和材质无关。以上的研究结论导致微塑料对小鼠健康影响的不确定性增加,因此还需要开展更深入系统的研究以阐明微塑料对生物包括人体健康影响。

3.1.2 肺

PS 微塑料通过诱导人的肺上皮 BEAS-2B 细胞活性氧的生成,对细胞产生毒性和炎症作用,对呼吸系统造成潜在不利影响^[65]。Xu 等^[66]评估两种不同尺寸(25 nm 和 70 nm)纳米 PS 微粒(PS-NPs)对人肺泡上皮



图中所涉及微塑料对组织和系统的影响均为以小鼠为实验动物研究的结果,正号和负号分别表示上调和下调

图1 微塑料对人体健康潜在影响

Fig. 1 Potential impacts of microplastics on human health

A549细胞系的影响,发现PS显著影响细胞活力,引起细胞周期S停滞,激活炎症基因转录,改变与细胞周期和促凋亡相关的蛋白质表达,触发了TNF- α 相关的细胞凋亡途径. Goodman等^[67]研究发现粒径1 μm 和10 μm 的PS能够导致人肺泡A-549细胞的增殖延缓、代谢活性下降,同时细胞形态也发生了明显变化. Lu等^[68]研究发现微塑料还导致正常小鼠肺炎细胞浸润、支气管肺泡巨噬细胞聚集和支气管肺泡灌洗液(BALF)中TNF- α 和血浆IgG1水平升高,也会导致哮喘小鼠加剧症状. Yang等^[69]使用两种类型的人肺上皮细胞(Ad12-SV40 2B, BEAS-2B)和人肺泡上皮细胞(HPAEPiC)来研究肺损伤与PS-NPs之间的关联,发现长时间暴露PS-NPs后会产生组织损伤和肺部疾病,PS的暴露可能会降低肺的修复能力并导致组织损伤,并且氧化还原失衡是PS诱导的肺损伤的主要因素. Zhang等^[70]也同样指出了氧化应激在纳米塑料诱导肺损伤机制中的潜在作用. Li等^[71]研究发现吸入PS微塑料通过激活小鼠的氧化应激和Wnt/ β -连环蛋白信号通路诱导肺纤维化,数据显示抗氧化剂褪黑激素可以缓解PS诱导的肺纤维化.

3.2 微塑料对机体系统的影响

3.2.1 对神经系统的影响

Ban等^[87]将PS-NPs暴露于培养的神经细胞-人神经母细胞瘤细胞SH-SY5Y来研究细胞毒性,结果表明PS暴露诱导细胞毒性的产生,促进了细胞分化为神经元表型,细胞表现出神经突触生长收缩,细胞核形态改变和肿胀以及细胞内成分溢出. Shan等^[88]研

究发现PS-NPs存在于小胶质细胞中,并诱导小鼠大脑中的小胶质细胞活化和神经元损伤而产生神经毒性. Chu等^[89]通过研究25 nm的PS是否会导致学习功能障碍发现,在小鼠暴露于PS后,氧化应激水平和DNA损伤增加的同时,突触功能也严重损伤;而Wang等^[90]研究也发现PS微塑料暴露可以通过诱导氧化应激和降低乙酰胆碱水平来影响学习和记忆功能. 更严重的是, Liu等^[91]研究发现铁和微塑料的共同暴露可能会通过扰乱脑组织中铁稳态和诱导认知相关大脑区域的铁中毒加重认知障碍.

3.2.2 对内分泌系统的影响

Fan等^[92]通过研究PS-NPs在包括肝、肾、脾和胰腺在内的各个器官中的积累,发现口服PS-NPs诱导内脏器官损伤,主要毒性为肝功能损害和脂质代谢异常,显著增加了氧化应激,扰乱了PI3K/Akt途径,导致胰岛素抵抗和小鼠肝脏中的血浆葡萄糖增加. Amereh等^[80]探究长期暴露于PS-NPs对大鼠甲状腺内分泌状态和生化应激的潜在影响,结果表明大鼠暴露于PS-NPs后,血清肌酐水平升高,出现肾毒性诱导和肾损伤的迹象,证明暴露PS-NPs可引起甲状腺内分泌紊乱以及代谢缺陷.

3.2.3 对免疫系统的影响

Wang等^[93]研究发现饮用水中的微塑料通过降低脾脏重量、CD⁸⁺T细胞数量和提高CD⁴⁺/CD⁸⁺T细胞比率显著损害小鼠免疫功能. Choi等^[94]研究发现PS微塑料通过释放化学试剂使免疫细胞的急性炎症增加20倍. Li等^[95]研究发现纳米塑料渗透到脾淋巴细胞

中,降低了细胞活力,诱导细胞凋亡,上调细胞凋亡相关蛋白表达,引发活性氧的产生,改变线粒体膜电位,损害线粒体功能,抑制了T细胞表面活化和T细胞标志物的表达,同时抑制了CD8⁺T细胞的分化和辅助性T细胞因子的表达。

3.2.4 对生殖系统的影响

微塑料通过免疫系统导致小鼠妊娠紊乱^[81],诱发小鼠代谢异常^[96],还能够诱导雌性小鼠卵巢颗粒细胞凋亡,甚至降低小鼠受孕成功率,与对照小鼠相比,投喂微塑料的雌小鼠产幼崽的数量、幼崽的性别比和幼崽的体质量均发生了显著变化^[4,97]。同样,微塑料还能在雄性小鼠睾丸累积,进而导致精小管损伤、生精细胞凋亡^[98],最终降低小鼠精子质量和睾酮水平^[99]进而对繁殖能力产生潜在影响。Nie等^[100]的研究发现60 nm或900 nm的PS可以穿过小鼠胎盘并影响发育中的小鼠胎儿,而Graefmueller等^[101]更是利用胎盘灌注模型发现50~300 nm的PS主要聚集在胎盘组织的合体滋养层中,因此推测合体是影响纳米塑料在胎盘组织中迁移和累积的主要因素,与此同时,NPs的表面电荷或修饰以及人体蛋白形成的蛋白冠^[102]也会影响NPs的迁移和累积。Wick等^[103]研究发现尽管240 nm的PS能够被胎盘吸收,但不会影响胎盘外植体的存活能力。Wei等^[104]为了揭示微塑料对生殖系统的影响,利用PS微塑料暴露雄性和雌性小鼠的结果表明,PS暴露后雌鼠卵巢产生的氧化应激和活性氧水平显著高于睾丸,并且雌鼠卵巢大小和卵泡数量均显著减少,而雄鼠睾丸中活附睾精子和生精细胞的数量显著减少,精子畸形率增加。同样,Xie等^[105]研究发现PS通过氧化应激和p38 MAPK信号通路激活诱导小鼠生殖毒性,而精子代谢相关酶琥珀酸脱氢酶(SDH)和乳酸脱氢酶(LDH)的活性降低,血清睾酮含量也同时显著降低。

3.3 微塑料对人体健康的潜在影响

微塑料体积小并且难降解,一旦进入人体可能会通过长时间的累积对人体健康产生不利影响。Barboza等^[106]研究发现尺寸小于130 μm的微塑料能够转移并进入淋巴和循环系统,而欧洲食品安全局(EFSA)^[107]研究表明小于10 μm的微塑料甚至可以穿透器官,小于1.5 μm的微塑料则能够进入所有器官,并通过循环系统进入各组织器官。最近,Chen等^[108]研究发现肺组织中存在多种微纤维,并且微纤维丰度随着年龄增长而累积。该研究还发现微纳米塑料在肺组织中长期摩擦甚至会导致呼吸道发生磨玻璃结节(GGNs)疾病。此外,人体肿瘤中存在的微塑料多于正常组织,暗示长期暴露于微塑料可能会诱导组织发生病变。Zarus等^[109]研究发现棉屑、纺织和

PVC制造这3个行业的工人患有肺炎、肺癌、直肠癌和肝癌等疾病与职业吸入的微塑料粉尘有关;Boag等^[110]研究也发现空气中的纤维可导致呼吸困难和肺活量降低等一系列呼吸疾病。此外,Yan等^[111]分析发现肠炎患者粪便中以聚对苯二甲酸乙二醇酯、乙烯和聚酰胺为主的微塑料纤维和碎片 $[41.8 \text{ 个} \cdot (\text{g} \cdot \text{dm})^{-1}]$ 明显高于健康人群 $[28.0 \text{ 个} \cdot (\text{g} \cdot \text{dm})^{-1}]$,并且粪便中微塑料含量与肠炎的严重程度呈正相关,据此推测环境微塑料污染可能会导致肠炎等疾病的发生。同样,Wu等^[53]研究发现血栓微塑料数量与血小板数量呈明显的正相关,即微塑料可能是通过血小板促进血栓形成而带来健康风险,但是具体发生机制还待进一步研究。虽然一些动物体内实验和细胞模型表明微塑料可诱导氧化应激、引起细胞炎症和生长发育毒性等,但目前还缺乏微塑料与人体健康是否相关的研究,因此还没有直接证据表明微塑料可对人体产生健康危害,这一领域仍存在很大的知识空白。

4 展望

(1)揭示环境微塑料污染水平对人体健康的影响。目前虽有大量的体内和体外研究表明微塑料对人体细胞和小鼠健康产生各种不利影响,但这些研究结果均是基于控制条件下的高浓度微塑料短时间暴露条件下取得的,并且微塑料的材质类型和形态特征与环境微塑料存在较大差距,尚无法证实环境水平微塑料对人体健康的影响。因此,急需基于现有研究手段构建环境微塑料暴露人群队列,探索环境微塑料在人体内的潜在累积行为,揭示环境水平长期暴露下微塑料对人体健康的潜在影响,进而为环境微塑料污染防治提供直接证据,这也是当前亟待解决的科学问题和社会问题。

(2)探索微塑料影响人体健康的发生机制。微塑料是集物理和化学于一体的复合污染物,兼具不溶的颗粒属性和亲脂的化学属性(自身携带的塑料添加剂及其表面吸附的环境污染物),一旦进入人体就可能同时从物理、化学甚至吸附的微生物等多个途径危害人体健康,并且还会受到微塑料的粒径、聚合物材质、形态特征和老化等多个因素的影响。因此,基于现有体外研究结果进一步阐明微塑料影响人体健康的效应终点并筛选敏感评价指标,建立环境微塑料暴露的剂量-效应关系,揭示微塑料危害人体健康的毒理发生机制和作用途径,建立环境微塑料污染的环境质量标准,为预防环境微塑料污染危害提供科学依据。

(3)发展并建立环境微塑料污染的健康风险评

估框架. 与可溶的持久性有毒有害污染物不同, 微塑料因其兼具颗粒不溶性和携带可溶性污染物的双重属性, 可通过多个途径影响和危害人体健康, 并可同时产生多个危害终点, 即适用于可溶性化学污染物的健康风险评估框架并不完全适用于评估微塑料污染的健康风险. 因此, 急需在微塑料生物毒性的危害识别、暴露水平和风险表征基础上, 建立科学评估微塑料健康风险的框架, 并结合现有知识和研究结果评估环境微塑料污染的潜在人体健康风险, 利用科学数据回应社会关切并指导微塑料污染防治.

5 结论

(1) 微塑料通过食物包括饮用水、呼吸以及皮肤接触等途径进入人体, 并通过循环系统进入各组织器官甚至突破胎盘屏障, 进而引起公众对微塑料污染影响人体健康的日益担忧.

(2) 通过体外细胞实验和动物活体实验, 在细胞、组织、器官和系统水平上证实微塑料污染对人体健康的潜在影响, 但环境水平微塑料对人体健康的影响仍缺少直接的科学证据.

(3) 环境微塑料污染对人体的暴露途径、健康危害、发生机制及其潜在健康风险是当前亟待解决的关键科学问题, 也是提升微塑料污染精准防治的科学需求.

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