· 环境与职业卫生 ·

低剂量环境镉暴露与慢性肾脏病风险：流行病学证据的矛盾与整合

肖瑶，谢林伸
四川大学华西公共卫生学院/华西第四医院，四川 成都 610041

摘要：关于低剂量环境镉暴露与慢性肾脏病风险的人群研究，当前的流行病学证据存在一定的矛盾，这构成了当前风险评估的核心不确定性。本文旨在系统梳理并整合这些矛盾发现。通过评述近期主要研究，本文首先呈现了支持与否定该关联的关键证据。进而，文章重点剖析了导致证据异质性的多层次根源，包括暴露与结局测量的方法学差异、高血压等强混杂因素的干扰、多金属混合暴露的复杂性，以及人群年龄与遗传易感性的效应修饰作用。最后，基于此分析，本文提出未来研究需推动范式转变：即从单一暴露评估转向混合暴露模型，从追求全人群统一风险转向识别易感亚群，并以此推动建立更精准的公共卫生防控策略。本综述为厘清低剂量镉的肾脏健康风险、整合矛盾证据提供了关键的分析框架与方向指引。

关键词：镉暴露；慢性肾脏病；肾损伤；联合暴露；生物标志物
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Environmental cadmium exposure: mechanisms from low - dose accumulation to kidney damage and population risks

XIAO Yao, XIE Lin - shen
West China School of Public Health / West China Fourth Hospital, Sichuan University, Chengdu, Sichuan 610041, *China*

Abstract: Population studies on the association between low - dose environmental cadmium exposure and the risk of chronic kidney disease present conflicting epidemiological evidence, constituting a core uncertainty in current risk assessments. This paper aims to systematically review and synthesize these conflicting findings. By reviewing recent major studies, it first presents key evidence supporting and refuting this association. Subsequently, the article focuses on analyzing the multi - level sources of evidence heterogeneity, including methodological differences in exposure and outcome measurement, interference from strong confounders such as hypertension, the complexity of mixed exposure to multiple metals, and the effect - modifying roles of population age and genetic susceptibility. Finally, based on this analysis, the paper proposes that future research should drive a paradigm shift: moving from single - exposure assessments to mixed - exposure models, and from pursuing uniform risks across entire populations to identifying susceptible subgroups. This shift will facilitate the development of more precise public health prevention and control strategies. This review provides a critical analytical framework and directional guidance for clarifying the renal health risks of low - dose cadmium exposure and integrating conflicting evidence.

Keywords: Cadmium exposure; Chronic kidney disease; Kidney injury; Combined exposure; Biomarkers

镉(Cd)是一种广泛存在于环境(如受污染的稻米、烟草烟雾)中的有毒重金属，可通过食物链进入人体，并在体内长期蓄积[1-4]。作为职业性致癌物，有研究显示，2007 - 2017 年间 Cd 的暴露水平持续上升[5]。环境中包括 Cd 在内的重金属暴露，如今已成为全球工业化国家公共卫生领域面临的一大切实威胁[6]。其肾毒性源于独特的毒代动力学：Cd 在肝脏形成低分子量镉金属硫蛋白复合物后，经肾小球滤过并被近端小管细胞主动重吸收，导致游离 Cd 离子在肾皮质特异性蓄积，使肾脏成为慢性暴露的核心靶器官[7-13]。

蓄积的 Cd 离子可诱发氧化应激、炎症反应、细胞凋亡及代谢重编程等一系列分子事件，最终导致肾小管损伤、间质纤维化及肾功能下降，这构成了 Cd 致肾损害的经典病理生理框架[14-21]。然而，这一明确的毒理学机制在人群流行病学研究中，尤其是针对低剂量环境暴露时，并未转化为一致的风险证据。大量关于环境 Cd 暴露与慢性肾脏病(Chronic Kidney

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作者简介：肖瑶(2000—)，女，硕士在读，研究方向：职业中毒
通信作者：谢林伸，E - mail：linshenxie@163. com

Disease,CKD)关联的研究结论存在显著矛盾,这种异质性掩盖了真实的暴露 - 反应关系,也导致了公共卫生风险评估的不确定性。因此,本文旨在系统梳理这些矛盾的流行病学证据,并深入剖析其产生的多层次根源,以期为厘清低剂量 Cd 暴露的肾脏健康风险提供更清晰的学术视角。

1 低剂量镉暴露与慢性肾脏病风险:主要流行病学证据梳理

肾损伤是以肾小球滤过率(eGFR)下降为特点的临床综合征,可导致肾小管上皮细胞损伤、微血管内皮修复不良以及持续免疫炎性反应,从而增加 CKD 和终末期肾病的发生风险。CKD 是一种进行性且无法治愈的疾病,会随着时间的推移损害肾功能[22]。全球疾病负担(GBD)研究表明,CKD 是全球死亡的主要原因之一[23]。据显示每年新增晚期 CKD 人数 12 224 例,每年因 Cd 相关 CKD 死亡人数可达 2 064 人[24]。众多横断面及队列研究为环境 Cd 暴露与 CKD 风险的正向关联提供了证据。研究表明,血 Cd 或尿 Cd 水平升高与肾功能下降、蛋白尿风险增加及 CKD 患病率显著相关,且这种关联在低剂量暴露范围内依然存在,其剂量 - 反应关系可能呈非线性[25-30]。值得注意的是,部分研究指出,即使 Cd 暴露水平低于现行的安全参考值,仍可观察到肾小管损伤标志物升高或 eGFR 下降的风险增加[31-34]。这些发现提示,普通人群的长期低剂量 Cd 暴露可能构成不可忽视的健康威胁,并对现有安全阈值提出了审慎评估的要求。

然而,Cd 暴露与 CKD 的流行病学关联远非一致,相当数量的研究未能重复上述阳性结果。在瑞典的一项前瞻性队列研究中,研究者发现 Cd 与 CKD 的发生无显著关联[35]。在台湾的一项工业区周边乡镇的研究中发现,尿 Cd 水平与 CKD 的风险之间无统计学意义,尚未发现二者之间存在独立且显著的关联[36]。目前尚缺乏令人信服的证据支持环境 Cd 暴露会显著增加人群进展为 CKD 的风险[37-38]。这些不一致的发现构成了当前领域的关键争议。

2 研究异质性的来源与解读

2.1 *方法学挑战:暴露与结局的测量异质性* 肾脏是人体最重要的器官之一,起着至关重要的生理功能,兼具排泄、代谢与内分泌三重核心功能[13]。肾脏通过肾小球滤过和近端肾小管的重吸收来维持水、酸碱和电解质的平衡。随年龄增长,肾脏呈现“结构与功能双减退”的渐进性衰老轨迹。目前评价肾脏损伤常用的生化指标主要分为两类,一类是反映肾小球功能的指标,如估算 eGFR、尿白蛋白/尿肌酐比值(urinary albumin to creatinine ratio,ACR)等;另一类是反映肾小管功能的指标,如尿 N - 乙酰 - β - D - 氨基葡萄糖苷酶(尿 NAG 酶)、尿 β2 微球蛋白、尿 α1 微球蛋白、尿视黄醇结合球蛋白等[39]。Cd 暴露评估通常因血液和尿液的直接性和便捷性,故而二者是评估 Cd 暴露水平的首选指标。尿 Cd 更可能反映慢性暴露,血 Cd 则反映近期暴露[40]。不同的评估标准带来的研究结局可能不同,例如有研究表明血 Cd 是新发慢性肾脏疾病的独立危险因素,而尿 Cd 则与 CKD 无显著关联[41]。故选择贴切的评估标准极为重要。

2.2 *混杂控制:高血压、糖尿病等强效因素的干扰* 研究结论的矛盾,部分源于对关键混杂因素控制的充分性存疑。在普通人群中,高血压和糖尿病是 CKD 最主要且强势的驱动因素。若流行病学研究未能精确测量并充分校正这些因素,其巨大的负面效应极易掩盖或混淆 Cd 暴露本身可能存在的、相对微弱的独立风险。这一点在特定人群中表现得尤为明显。例如,一项针对原发性高血压患者的研究发现,在调整相关变量后,尿 Cd 水平与估算 eGFR 之间并无显著关联[42]。该结果表明,在已存在强肾损伤风险(如高血压)的人群中,环境水平 Cd 暴露的附加效应可能被主导疾病所淹没,从而在统计学上难以被察觉。这警示我们,研究人群的基础疾病谱是解释结果异质性的关键:在健康人群或早期研究中观察到的关联,可能在已患病人群或校正更严格的研究中消失。

2.3 *暴露场景:从单一镉到多金属混合暴露的复杂性* 传统风险评估常基于单一金属模型,但这与现实环境中共存多种重金属的暴露场景严重脱节。日益增多的证据表明,Cd 的肾毒性风险必须在混合暴露的复杂体系中重新审视。

首先,Cd 与其他金属(如铅、砷)的联合暴露普遍存在,且常表现出协同增强效应。例如,一项中国台湾的病例对照研究显示,Cd、铅、砷三者共暴露时,其导致 CKD 的风险远高于单一金属效应的简单相加[27]。这强烈提示,单独评估 Cd 可能会严重低估其在真实环境混合物中的实际健康威胁[43-44]。

更重要的是,这种联合效应使得在单一金属分析框架下,Cd 的独立贡献可能被掩盖或扭曲。一项前瞻性队列研究提供了关键洞见:尽管在该研究人群中,单独的血浆 Cd 水平与肾功能下降无显著关联,但当将其置于多金属混合物中评估时,Cd 却是驱动肾毒性的关键贡献因子之一[45]。这一“单独不显,混合显效”的现象,深刻揭示了传统分析模型的局限性。因此,要厘清低剂量 Cd 暴露的真实风险,必须超越单一暴露模型,转向采用如贝叶斯核机器回归(BKMR)

或加权分位数和(WQS)等专门设计用于解构混合暴露效应的统计模型。这些方法有助于识别关键毒性成分、揭示金属间的交互作用,并将流行病学证据推向更贴近真实世界复杂性的新阶段。然而,这些模型本身对共线性敏感、结果解释复杂,其应用也构成了新的方法学挑战。

2.4　人群易感性:年龄、遗传与基础疾病的效应修饰

研究人群的内在差异,是导致 Cd 肾毒性流行病学关联出现矛盾的关键解释因素之一。不同亚人群对 Cd 暴露的生物学反应可能存在显著不同,若忽视这种效应修饰作用,整体研究结论易产生偏倚或掩盖特定群体的真实风险。年龄是最显著的效应修饰因子。生命两端的群体,儿童与老年人对 Cd 暴露尤为敏感,但机理不同。儿童因肾脏发育未成熟、代谢系统不完善,对重金属的毒性更为敏感,研究证实其环境水平的血 Cd 即可与早期肾小管损伤标志物升高相关[46]。而老年人则主要面临 Cd 的终身累积负荷与自身肾功能生理性下降的双重压力,横断面研究显示其 Cd 暴露与肾功能损伤的关联强度更高[25]。因此,一项在中年健康雇员中未发现关联的研究,与一项在老年社区人群中报告阳性结果的研究,其结论可能并无根本冲突,而是反映了不同年龄结构人群的风险差异。更深层的差异来源于个体遗传背景。涉及金属解毒、代谢及氧化应激反应通路的相关基因多态性,可能决定了个体对 Cd 毒性的内在易感性。然而,这部分难以测量的遗传混杂因素,在大部分流行病学研究中均未被有效控制或分析,成为导致结果不一致的潜在但重要的"暗箱"。

因此,要调和现有研究的矛盾,未来工作必须正视并系统评估人群异质性。这需要在研究设计与分析中,主动进行人群分层或纳入基因与环境交互作用的考察,从而更精准地识别高风险亚群,而非寻求一个适用于全人群的单一风险估计值。

3　总　结

综上所述,关于低剂量环境 Cd 暴露与 CKD 风险的流行病学证据呈现出显著矛盾,这并非简单的"是"或"否"的问题,而是深刻反映了环境健康研究在迈向精准风险评估时所面临的复杂性与多维挑战。本文系统揭示,证据异质性源于方法学设计、强混杂因素的干扰、真实世界混合暴露的复杂性以及人群内在易感性差异等多个层次的交互作用。因此,整合现有矛盾证据的关键,不在于寻求更多重复性研究,而在于推动研究范式的根本转变,即从评估单一暴露转向解析混合效应,从寻求全人群统一风险转向识别易感亚群,从横断面关联分析转向强化因果推断。通过采纳更精细的测量工具、更先进的统计模型与更具分层意识的公共卫生策略,方能在不确定性中厘清风险,为制定科学、精准的 Cd 暴露防控政策奠定坚实的证据基础。Cd 致肾损害是一个涉及环境、生物、社会等多维度的复杂问题,未来需通过跨学科合作、多层次研究与国际协作,共同应对这一全球性公共卫生挑战。

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