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空气污染与鼻炎–哮喘共病：流行病学及机制研究进展

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摘　要

鼻炎与哮喘常表现为共病状态。空气污染作为重要的环境风险因素，可显著增加鼻炎、哮喘及二者共病的发病风险，且污染物与"鼻炎单独"或"鼻炎–哮喘共病"的关联强度高于"哮喘单独"。因此，本文从耳鼻喉科视角出发，整合流行病学证据，初步探讨空气污染对鼻炎–哮喘共病的作用机制，为临床防控提供科学依据。

关键词

空气污染，鼻炎–哮喘共病，流行病学，机制

Air Pollution and Rhinitis-Asthma Comorbidity: Research Advances in Epidemiology and Mechanisms

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Abstract

Rhinitis and asthma often co-occur. As a major environmental risk factor, air pollution can significantly increase the risk of developing rhinitis, asthma, and their co-occurrence; moreover, the association between pollutants and "rhinitis alone" or "rhinitis-asthma comorbidity" is stronger than that with "asthma alone". Therefore, this paper adopts an otolaryngological perspective to synthesize

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epidemiological evidence and preliminarily explore the mechanisms underlying the role of air pollution in rhinitis-asthma comorbidity, thereby providing a scientific basis for clinical prevention and control.

Keywords

Air Pollution, Rhinitis-Asthma Comorbidity, Epidemiology, Mechanisms

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1. 引言

空气污染作为全球性环境的健康威胁，与上呼吸道炎症性疾病的关联已得到证实。系统综述与荟萃分析证实，NO_2、$PM_{2.5}$、PM_{10}等污染物显著增加过敏性鼻炎、哮喘及慢性鼻窦炎的患病风险[1]。空气污染促进过敏致敏与症状恶化[2]，且其健康效应在弱势群体中呈现不均衡分布。

近年来，鼻炎与哮喘均为全球高发的慢性呼吸道疾病，疾病负担在持续上升。大量的流行病学证据表明二者常共存，且共病状态与鼻炎持续性关联有密切关系[3]。许多研究将鼻炎与哮喘孤立分析[4]，揭示鼻炎单独与鼻炎–哮喘共病存在显著的临床与免疫学差异[5]，对空气污染与鼻炎–哮喘共病的关联及潜在机制缺乏系统探讨[6]。

2. 空气污染与鼻炎–哮喘共病的流行病学特征

2.1. 共病的流行病学现状

“鼻炎–哮喘共病”指鼻炎与哮喘同时存在。全球数据显示，哮喘与鼻炎共病患病率差异显著，过敏性鼻炎与哮喘共病的比值比达 2.42 [7]。鼻炎单独、哮喘单独及二者共病的临床表型在各国均存在明确区分[5]，并且空气污染对过敏性鼻炎的影响在发展中国家强于发达国家，欧洲地区效应强于亚洲[8]。然而，共病存在明显识别不足，哮喘患者中鼻炎常被低估，部分自报“仅哮喘”者实际存在鼻部症状与用药[9]。

2.2. 短期与长期暴露

研究显示，长期暴露于NO_2、$PM_{2.5}$等污染物与“鼻炎单独”或“鼻炎–哮喘共病”的关联强度高于“哮喘单独”，其中，“鼻炎单独组”的效应值最高[10]。荟萃分析显示，空气污染使过敏性鼻炎、哮喘、慢性鼻窦炎患病率分别达 16%、11%、12% [1]。污染物成分也具有差异化作用，硝酸盐组分对鼻炎影响突出，而有机物组分与过敏性哮喘及结膜炎关联更强[9]。

短期污染暴露可触发呼吸道症状急性加重，加剧过敏性鼻炎患者的氧化应激与炎症标志物水平[11]。澳大利亚儿童急诊数据显示，空气污染暴露后数小时内哮喘急性发作风险即升高，学龄前儿童效应更显著[12]。此外，慢性鼻窦炎合并哮喘患者对$PM_{2.5}$、O_3的敏感性更高，污染物每增加一个 IQR，鼻窦症状评分显著上升，而无哮喘者无此效应，共病状态可能放大污染的急性危害。

3. 上呼吸道的始动作用

3.1. 屏障破坏

鼻腔作为气道第一道防线，其黏膜上皮直接暴露于高浓度环境刺激物。柴油颗粒物(DEP)、$PM_{2.5}$等

可下调 ZO-1 等关键连接蛋白表达，破坏呼吸道上皮屏障完整性[13]，接触固有层免疫细胞，诱发异常免疫应答。使得原本低剂量的过敏原更易穿透黏膜，为污染高峰后过敏症状常迅速加重的临床现象提供了证据[14]。

3.2. 警报素的释放

受损鼻上皮细胞迅速释放 IL-25、IL-33 等上皮源性细胞因子[15]。这些因子激活树突状细胞及 2 型固有淋巴细胞等，驱动 Th2 型免疫应答，并通过血液循环或气道内播散将炎症信号传递至下呼吸道[15]。此类警报素在过敏性鼻炎合并哮喘患者的上下气道中显著高表达[16]。

3.3. 鼻–支气管交互通路

后鼻滴漏通路：含组胺、白三烯、IL-5 等炎症介质的鼻分泌物经后鼻孔流入下气道，持续刺激支气管黏膜，加重气道炎症[17]。

神经反射通路：污染物或炎症刺激激活三叉神经–迷走神经反射弧，诱发支气管平滑肌收缩与腺体分泌增加，参与哮喘急性发作[18]。

系统性炎症播散通路：鼻黏膜释放的炎症因子入血后可影响骨髓造血，促进嗜酸性粒细胞生成[19]。

4. 污染物的直接致病作用

4.1. 氧化应激

短期暴露于 $PM_{2.5}$ 细颗粒物可诱导鼻黏膜和气道氧化应激，促进 IL-6、TNF-α 等促炎细胞因子的释放，加重过敏性鼻炎和哮喘的炎症状态[11]，同时促进气道高反应性和重塑[20]。

4.2. 屏障损伤

PM、臭氧、NO_2 可直接损伤鼻黏膜和支气管上皮的物理屏障，导致上皮细胞紧密连接蛋白表达下调、纤毛功能受损，增加过敏原和微生物的穿透机会，促使原本无害剂量的过敏原诱发过敏反应[21]，进而加重鼻炎与哮喘的共病状态[22] [23]。

4.3. 免疫应答

污染暴露可刺激气道上皮细胞释放 IL-25、IL-33 等警报素，激活 2 型固有淋巴细胞(ILC2)和 Th2 细胞，促进 IL-4、IL-5、IL-13 等细胞因子分泌，该类细胞因子是鼻炎和哮喘共病的关键免疫介质[20] [24]。

4.4. 表观遗传

孕期暴露于 NO_2、PM 与子代体内免疫扰动相关，影响脐带血中 T 细胞分化、DNA 甲基化和基因表达，进而增加儿童期发生鼻炎和哮喘共病的风险[6]。

4.5. 菌群失调

空气污染物可导致气道菌群失调，破坏局部的免疫调控网络，进而促进 Th2 炎症和鼻–气道超敏反应[23]。

5. 污染物与其他因素的协同作用

5.1. 过敏原

化学修饰作用：O_3 与 NO_2 可在环境浓度下诱导花粉及尘螨过敏原蛋白发生硝化修饰，导致蛋白构象

改变，显著提升其与 IgE 的结合能力，增强过敏原致敏性。硝化修饰可促进过敏原寡聚化，并影响树突状细胞功能，推动 Th2 型免疫应答偏移，抑制 Th1 反应，从而强化致敏过程[25]。

物理载体作用：细颗粒物及柴油机尾气颗粒(DEP)可吸附花粉碎片或过敏原，形成“过敏原－颗粒复合体”，增强其在呼吸道的沉积效率与滞留时间，并促进深层肺组织穿透[25]。DEP 本身可刺激 IgE 合成与 Th2 细胞因子释放，协同放大过敏炎症反应[26]。流行病学研究证实，花粉与空气污染物存在显著交互作用，共同加剧过敏性哮喘患者的鼻部、眼部及支气管症状，并增加急救药物使用频率[27] [28]。

5.2. 气象因素

低温可导致鼻腔血管收缩、黏液纤毛清除功能下降，延长污染物与过敏原在黏膜表面的滞留时间，加重上皮损伤；低温联合低湿或高湿环境可进一步放大空气污染对过敏性鼻炎门诊量的影响[29]。升温可能增强空气污染物对过敏原的化学修饰作用[30]；同时，大气 CO_2 浓度升高促进豚草等致敏植物花粉产量增加，延长花粉季[31]。气候变暖亦通过改变花粉季节长度、分布区域及真菌孢子释放模式，间接加剧过敏负荷[32] [33]。

5.3. 呼吸道病毒

O_3 可抑制分泌型白细胞蛋白酶抑制剂(SLPI)，激活病毒血凝素切割所需蛋白酶，增强流感病毒等呼吸道病毒的入侵能力[34]。流行病学证据表明，在寒冷季节，多种空气污染物显著放大病毒暴露对儿童哮喘相关住院的影响；温暖季节中，$PM_{2.5}$ 与 O_3 亦显示协同效应，且 5 岁以下儿童尤为敏感[32]。

6. 总结与展望

哮喘的共病状态体现了“同一气道，同一疾病”的病理生理连续性[35]。鼻腔黏膜作为吸入性污染物与过敏原的首道防线，在空气污染驱动的上下气道炎症进展中发挥始动作用。机制层面，空气污染物可诱发氧化应激与鼻黏膜上皮屏障功能受损，促进上皮源性促炎细胞因子的释放，加剧嗜酸性粒细胞浸润与 IgE 介导的炎症反应[20] [23] [36]。此外，污染物诱导的上气道炎症可通过后鼻滴漏、鼻－支气管神经反射以及系统性炎症介质的播散等途径向下气道传递，构成上下气道炎症进展的通路[37]。同时，空气污染物与过敏原、气象因素及呼吸道病毒之间存在协同效应，进一步放大过敏性疾病的风险与临床严重程度[28] [29]。

未来，不同共病表型的差异化分子机制与免疫特征有待进一步探究[3] [5]，将鼻炎的早期识别与干预纳入哮喘的预防与管理，减轻空气污染背景下呼吸过敏性疾病的全球负担。

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