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空气污染对过敏性鼻炎影响的研究进展

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摘要 空气污染与过敏性鼻炎的发病及症状加重具有关联性, 但相关致病污染物、影响机制、空气污染与过敏原协同作用等尚缺乏系统性的总结分析. 本文系统分析了空气污染对过敏性鼻炎的影响机制、不同类别空气污染物对过敏性鼻炎的影响, 以及空气污染与其他因素对过敏性鼻炎存在的协同作用. 结果显示: 空气污染物可通过过敏原作用或过敏原增强作用在致敏阶段产生影响, 还可通过氧化应激、炎症反应或免疫系统调节在致敏或效应阶段产生影响. 包括$PM_{2.5}$、PM_{10}、黑碳在内的颗粒物以及SO_2、NO_2、CO、O_3、挥发性有机物(VOCs)在内的室外气态污染物和室内挥发性有机物会通过不同机制诱发炎症反应, 影响过敏性鼻炎; 此外, 空气污染物可与过敏原、温度及湿度等气象因素和遗传因素协同作用影响过敏性鼻炎, 而佩戴口罩、使用空气净化器等干预措施可减轻空气污染对过敏性鼻炎的不利影响. 本文可为我国空气、气候及疾病管控相关政策的制定实施提供参考, 并为个人过敏性鼻炎的预防与患者的风险控制提供科学依据. 最后, 我们建议采取更加多样的方法研究更多不同种类空气污染物与过敏性鼻炎间的关联, 深入研究相关的影响机制, 并探索可行的干预措施.

关键词 空气污染, 过敏性鼻炎, 影响机制, 气象因素, 协同作用

过敏性鼻炎(allergic rhinitis)是由吸入过敏原引起、免疫球蛋白E(IgE)介导反应所致的一种鼻黏膜炎症性疾病, 症状包括打喷嚏、鼻塞、鼻痒和流鼻涕, 有时还会伴有眼痒、流泪等眼部症状[1]. 在我国有8%以上的成人患有过敏性鼻炎, 且在不同城市间存在显著差异[2,3]. 在全球范围内过敏性鼻炎各国发病率在5%~50%之间[4], 其中发达国家和城市地区更高, 且近年来呈现上升趋势[5,6]. 过敏性鼻炎常与过敏性结膜炎、过敏性哮喘等共病, 给个人生活和社会经济造成负担[7,8], 而且因其难以根治、多诱因、多症状的特性, 已成为一个全球公共健康问题. 在欧盟国家过敏性鼻炎对工作效率的影响估计每年会造成300~500亿欧元的损失[9,10], 在中国北京成人过敏性鼻炎患者每年的总社会成本约为4.4亿欧元[11]. 过敏性鼻炎与遗传因素、环境因素、生活方式等均存在关联, 越来越多的证据表明空气污染会增加过敏性鼻炎的发病风险、加重过敏性鼻炎症状[12~14]. 近年来作为一种重要的环境和外源性病因而备受关注, 其对过敏性鼻炎的影响及机制等问题也成为研究关注的焦点[15~18].

在此背景下, 更多的研究采用了队列研究等高证据等级的研究方法来探求空气污染物与过敏性鼻炎间的关联[19,20], 且通过动物实验探究污染物影响机制的

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研究也不断增多. 由于空气污染的复杂性, 当前研究的目标更多关注于混合空气污染物, 且个人生活环境的不同也使得其他环境因素成为研究的对象. 但目前研究的空气污染物种类仍较少, 很多污染物的影响机制尚不明晰, 且与其他因素的协同作用研究尚不完善, 系统性地总结现有研究结果并更深入地研究空气污染对过敏性鼻炎的影响对疾病的预防与控制十分必要.

相较于以往研究缺乏空气污染对过敏性鼻炎的影响机制及协同因素的系统总结, 本文围绕空气污染对过敏性鼻炎的影响、机制和相关协同因素进行分析, 使用“过敏性鼻炎”“变应性鼻炎”与“空气污染”“空气污染物”“颗粒物”“臭氧”“二氧化氮”“氮氧化物”“二氧化硫”“一氧化碳”“室内污染物”“挥发性有机物”“过敏原”“气象”“温度”“湿度”“风”“遗传”“干预”这些检索词的中英文组合在PubMed、Web of Science、Scopus和CNKI进行了文献检索, 根据文献篇名、关键词和摘要进行了筛选, 去除了无明确污染物类型或可能协同因素的文献, 着重归纳了国内外近5年的最新研究进展并进行了分析总结, 采取定性综述的方法分析了当前研究存在的不足, 基于结果提出了下一步可能的研究方向, 旨在为空气污染影响下的过敏性鼻炎的发病机制、影响因素、预防措施等方面的研究提供参考, 以更好地减轻过敏性鼻炎对患者和社会的疾病负担, 也为我国空气、气候及疾病管控相关政策的制定实施提供依据.

1 空气污染对过敏性鼻炎的影响机制

过敏性鼻炎可分为季节性和常年性2种[4,21]. 季节性过敏性鼻炎又被称为“花粉热”, 发作呈季节性, 主要由花粉、真菌等季节性过敏原所致; 常年性过敏性鼻炎发作呈常年性, 主要由尘螨、动物毛发皮屑、蟑螂等室内常年存在的过敏原所致. 空气污染对2种过敏性鼻炎均会产生影响, 这与它们相同的发病机制有关. 当人体首次暴露于过敏原时会导致过敏原特异性IgE的生成[22,23]. 它们会与效应细胞(如肥大细胞)表面的高亲和力IgE受体结合, 完成致敏过程[23]. 当致敏的机体再次暴露于相同过敏原时, 过敏原会与肥大细胞上的IgE结合, 从而激活肥大细胞并使其脱颗粒. 短时间内预存的和新合成的炎症介质被释放[24](图1), 这些介质与鼻黏膜、鼻黏膜感觉神经末梢、血管和腺体相互作用, 导致过敏性鼻炎急性症状的出现, 即效应阶段.

研究表明空气污染可能会在以上过程的不同阶段发挥作用从而对过敏性鼻炎造成影响, 其可能的影响机制主要有5种, 空气污染可发挥过敏原作用或过敏原

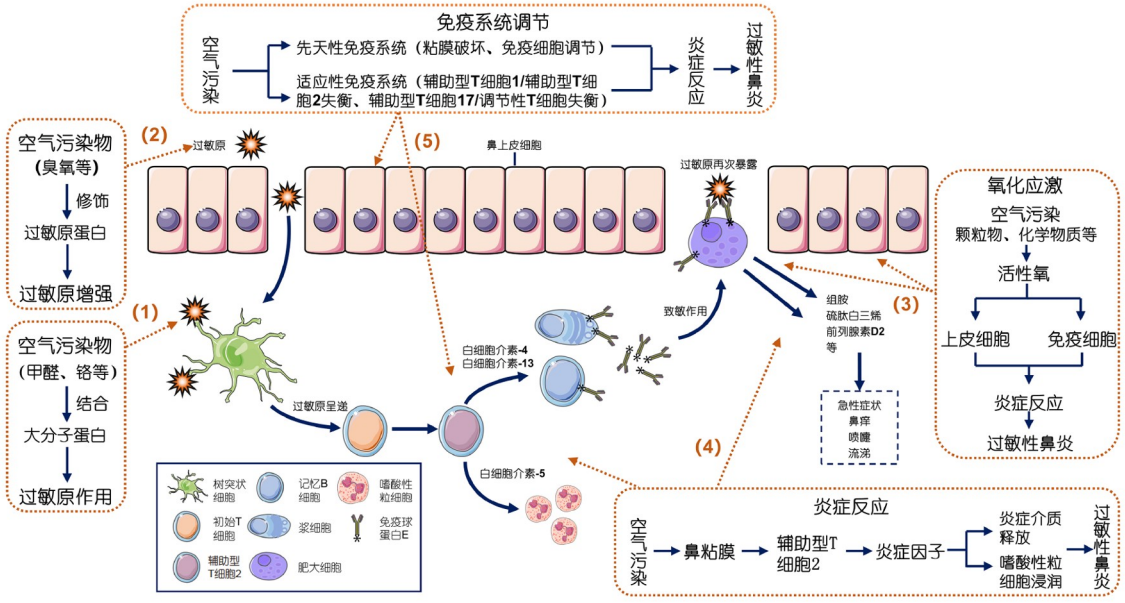

图 1 (网络版彩色)空气污染对过敏性鼻炎的影响机制. 序号对应正文不同机制: (1) 代表过敏原作用; (2) 代表过敏原增强作用; (3) 代表氧化应激; (4) 代表炎症反应; (5) 代表免疫系统调节

Figure 1 (Color online) The effect mechanism of air pollution on allergic rhinitis. Serial numbers correspond to different mechanisms in the main text: (1) represents acting like allergen; (2) represents allergen enhancement; (3) represents oxidative stress; (4) represents inflammatory response; (5) represents immune system regulation

增强作用从而在致敏阶段产生影响, 还可通过氧化应激、炎症反应或免疫系统调节对致敏或效应阶段产生影响, 具体机制如下(图1):

(1) 过敏原作用. 甲醛、三价铬等空气污染物作为一种小分子, 可以与较大的蛋白质分子结合, 形成新的抗原, 这些抗原可能会引发特异性IgE的形成, IgE与肥大细胞结合, 导致肥大细胞脱颗粒从而使过敏反应相关的炎症介质释放, 引起过敏性鼻炎症状[25,26]. 这在职业暴露中比较常见, 工作中接触到的飞灰、扬尘、废气等很可能存在这类物质, 它们与蛋白的缀合物会直接诱发过敏性鼻炎[27].

(2) 过敏原增强作用. 空气污染物可通过化学修饰改变过敏原蛋白质的结构, 从而增强过敏原的致敏作用. 研究表明过敏原蛋白质硝化可以增强其致敏性[17,28], 这在动物实验中得到了验证[29]. 活性氮物种也会激活某些过敏原蛋白的氧化防御机制, 从而增强过敏原活性[30].

(3) 氧化应激. 空气污染中的颗粒物和化学物质可以使细胞产生氧化应激作用, 即产生更多的自由基和氧化物质, 这些物质可以损伤鼻黏膜细胞, 促进炎症反应的发生, 进而导致过敏性鼻炎发病, 动物实验也证明了该机制[31].

(4) 炎症反应. 空气污染会刺激鼻黏膜和呼吸道, 从而引起多种细胞的炎症反应, 促进白细胞介素等炎性因子的生成与释放, 这会进一步促进肥大细胞脱颗粒及组胺等炎症介质的释放, 并导致嗜酸性粒细胞显著浸润, 从而加剧过敏性鼻炎, 这在动物和人群研究中均得到了证实[32~34].

(5) 免疫系统调节. 研究表明空气污染可能对免疫系统产生影响, 这包括先天性免疫系统和适应性免疫系统. 污染物会破坏鼻上皮细胞屏障的完整性, 从而对一系列免疫细胞产生影响, 进而导致炎症反应的发生[34,35]. 空气污染可通过影响辅助型T细胞2(Th2)相关细胞因子的产生来实现适应性免疫的调节, 它们会影响过敏反应相关的特异性IgE的生成, 也会影响辅助型T细胞1(Th1)/Th2细胞间的平衡, 从而影响炎症反应进而对过敏性鼻炎产生影响[36,37], 此外, 空气污染物也可能通过影响辅助型T细胞17(Th17)/调节性T细胞(Tregs)平衡调节过敏性鼻炎[38,39].

需要注意的是, 空气污染对过敏性鼻炎的影响是一个复杂的过程, 涉及多个机制的相互作用, 但多数影响最终都会诱发炎症反应, 因为炎症介质的释放直接影响着过敏性鼻炎发病. 此外, 不同的污染物和个体差异也可能导致不同的影响. 目前对于影响机制的研究多为单一空气污染物的动物实验, 机制通路研究有限, 混合空气污染物的影响机制可能会更为复杂, 此外空气污染是否会通过诸如神经调节等机制影响过敏性鼻炎尚不明晰, 因此, 仍然需要进行更深入的研究, 以全面了解空气污染与过敏性鼻炎之间的关系.

2 不同类别空气污染物对过敏性鼻炎的影响

2.1 不同粒径颗粒物对过敏性鼻炎的影响

不同粒径颗粒物与过敏性鼻炎间的相关性存在区别, 研究多集中于$PM_{2.5}$和PM_{10}, 它们已被证明对过敏性鼻炎存在不利影响. 共整理了28项颗粒物影响过敏性鼻炎的流行病学研究, 其中发现$PM_{2.5}$和PM_{10}与过敏性鼻炎存在关联的研究分别有22和15项, 这些研究包括横断面、队列、时间序列研究等多种, 研究对象包括许多国家的成人与儿童(表S1). 动物实验表明$PM_{2.5}$会通过影响氧化应激及炎症反应从而增强机体对过敏原的免疫反应[40,41]. $PM_{2.5}$及PM_{10}也会通过破坏鼻黏膜屏障影响免疫系统, 加重炎症反应及鼻部症状[34]. 目前$PM_{2.5}$及PM_{10}与过敏性鼻炎间存在关联在流行病学研究中得到了充分的证实, 如Luo等人[42]对英国地区379,488名非过敏性鼻炎参与者开展了一项前瞻性队列研究, 在12.5年的随访期间, $PM_{2.5}$浓度每升高5 μg m^{-3}, 过敏性鼻炎的风险比(HR)为1.51(95%CI: 1.27~1.79), PM_{10}浓度每升高10 μg m^{-3}, 过敏性鼻炎的HR为1.45 (95%CI: 1.20~1.74). 综合以往的荟萃分析表明, $PM_{2.5}$对过敏性鼻炎的影响比PM_{10}更加显著, 特别是在亚洲地区, 在儿童中的影响也更强[43~45]. 此外, 相较于$PM_{2.5}$和PM_{10}, PM_1及超细颗粒物($PM_{0.1}$)对过敏性鼻炎是否存在影响并不明确, 它们因为粒径小可进入肺部, 很少积聚在鼻腔或其他上呼吸道部位, 所以可能并不参与过敏性鼻炎相关的免疫过程.

2.2 颗粒物不同化学组分对过敏性鼻炎的影响

对于颗粒物中不同化学组分对过敏性鼻炎的影响研究仍相对较少, 共整理了4项颗粒物化学组分与过敏性鼻炎存在关联的研究. 黑碳(BC)作为颗粒物中的重要组分, 会对人体多系统造成影响[46], 其与过敏性鼻炎间的关联在近些年也得到了广泛关注. BC会造成免疫基因表观遗传变化从而加重炎症反应, 进而影响过敏

性鼻炎，这在人口腔黏膜细胞和鼻上皮细胞的分析研究中得到了证实[47,48]. 在流行病学研究中，BC被发现对成人或儿童过敏性鼻炎患者均存在影响，Savouré等人[49]在法国开展的一项成人队列研究发现，BC的年平均暴露量的增加与当前成人鼻炎患病率的增加有显著关联. Chen等人[50]在中国开展的一项多中心研究发现，母亲在怀孕期间接触$PM_{2.5}$和化学组分会对学龄前儿童过敏性鼻炎产生影响，其中BC的影响最大. 除此以外，PM中的一些盐类、金属元素及半挥发性有机物也会对过敏性鼻炎造成影响，Li等人[51]在中国太原对9名过敏性鼻炎成人患者开展的一项定组研究中发现，暴露于$PM_{2.5}$中的部分化学组分(苯并[a]芘、多环芳烃、钒、铬、铜、砷、硒、镉和铅)会增强氧化应激作用，从而加重过敏性鼻炎患者的炎症症状. 一项前瞻性队列研究也证明了$PM_{2.5}$中的铵盐和硝酸盐对过敏性鼻炎发病存在影响[52]. 但总体来看BC及其他化学组分对过敏性鼻炎的影响及机制研究仍存在一定空白，需要更多精细化的研究来说明.

2.3 室外气态污染物对过敏性鼻炎的影响

对于室外气态污染物与过敏性鼻炎的关联研究在很早便已开始，早期研究主要关注工业或交通相关的气态污染物，如SO_2、CO和NO_2，随着O_3污染的加剧，其对过敏性鼻炎影响的研究也不断增多. 近年来，关于室外气态污染物对过敏性鼻炎的影响研究也主要集中于以上4种污染物上，我们近期的一项meta分析研究也证明了这点[53]. 共整理了22项室外气态污染物影响过敏性鼻炎的流行病学研究，其中发现NO_2、SO_2、CO和O_3存在关联的研究分别有16、7、6和7项，这些研究包括横断面、队列、时间序列研究等多种，研究范围包括中国在内的许多国家，对象包括成人与儿童(表S1).

SO_2早期已被证实与过敏性鼻炎存在关联[54]. 这种影响与工业排放关联密切，Kim等人[55]在韩国工业特区蔚山市针对小学生开展的一项追踪调查中也发现，SO_2与过敏性鼻炎的高风险有关. SO_2对过敏性鼻炎的影响与炎症反应和Th1/Th2细胞平衡有关，动物实验表明短期接触SO_2会加重嗜酸性粒细胞浸润和Th1、Th2、Th17炎症，从而加重过敏性鼻炎症状，但长期接触SO_2可降低血清特异性IgE(sIgE)水平从而减轻过敏性鼻炎症状[56]. 此外，SO_2对呼吸道的刺激和过敏性鼻炎患病的影响还与温度存在关系，在温度更高的环境中SO_2的影响会更强[57]. 目前，对于SO_2影响过敏性鼻炎的机制研究仍需继续，对于暴露时间不同是否会产生不同影响仍需验证.

早期便有研究证明NO_2会对鼻黏膜中的免疫细胞造成影响从而影响过敏性鼻炎[58]. 随着交通等排放的NO_2不断增加，NO_2带来的影响更加明显. Wang等人[59]在中国新乡市开展的一项空气污染对过敏性鼻炎门诊量影响的时间序列研究发现，NO_2浓度每增加10 μg m^{-3}，当日过敏性鼻炎门诊患者数量增加4.54%，NO_2浓度与过敏性鼻炎门诊量存在较强的相关性. NO_2会通过影响炎症反应从而对过敏性鼻炎产生影响，这可以通过影响细胞因子的释放实现，NO_2暴露会增加上皮细胞中白细胞介素(IL)-6的释放，当与过敏原共同暴露时，更会显著增加IL-6和IL-8的释放[60]，这解释了NO_2增加过敏性鼻炎发病风险的部分机制，是否存在其他机制仍需进一步研究.

CO对人体的健康影响主要集中于心血管系统与下呼吸道，现有研究也发现了其对过敏性鼻炎存在影响. Kwon等人[61]在韩国首尔开展的一项横断面研究发现，过敏性鼻炎发病率仅与CO水平之间存在弱关联. 但目前CO对过敏性鼻炎的影响研究结果存在不一致，且缺乏机制方面的研究，关于机制方面的动物研究或临床试验仍相对匮乏，所以关于CO对过敏性鼻炎的影响机制仍需继续探索.

早期研究即表明O_3会对呼吸道产生刺激作用，与过敏性鼻炎存在关联[62]. 近期国内的许多流行病学研究证明了O_3对过敏性鼻炎存在不利影响，这在儿童中较为明显，Zhou等人[63]在中国东北7市儿童中开展的一项横断面研究发现，环境O_3的长期暴露与儿童过敏性鼻炎的高风险显著相关. 近年来许多研究开展了动物实验以探究O_3影响过敏性鼻炎的机制，目前的结果表明其主要通过氧化应激和炎症反应实现. Zhang等人[64]发现O_3会加剧炎性小体核苷酸结合寡聚化结构域样受体蛋白3(NLRP3)在鼻黏膜组织中的表达，加重Th2炎性因子的表达从而加重炎症反应. Sun等人[65,66]也通过动物实验先后发现了O_3对炎症反应和氧化应激的影响. 此外，O_3会对免疫系统造成影响，长期暴露于臭氧会对鼻上皮屏障造成不可逆的损伤，免疫系统的破坏也会引发氧化应激和炎症反应[67]. 相较于其他3种气态污染物，O_3与过敏性鼻炎的关联研究仍相对较少，但动物实验已证明了其对过敏性鼻炎的部分影响机制，在后续过敏性鼻炎的相关研究中，应更加重视O_3可能带来的

影响.

目前, 关于室外气态污染物与过敏性鼻炎的研究多为包含颗粒物在内的多种污染物混合暴露的情况, 且多数研究表明了其中2种以上污染物与过敏性鼻炎的风险增加相关[42,59,68]. 整体而言, 相较于气态污染物, 颗粒物对过敏性鼻炎的影响似乎更强, 且周期更长; 在气态污染物中, NO_2和SO_2与过敏性鼻炎的关联更显著, 且在发展中国家的影响比发达国家强, 一个重要原因是工业、交通的排放使得这些物质在发展中国家的浓度更高, O_3和CO对过敏性鼻炎的影响相对次要[32,45,53,69]. 但不同污染物之间是否存在相互影响尚不明确, 且共同暴露下的影响机制与单独暴露时是否存在变化也缺乏系统研究, 还需要后期继续探索.

2.4 室内气态污染物对过敏性鼻炎的影响

多数人特别是儿童在室内的停留时间远比室外的长, 所以室内污染物对于过敏性鼻炎的影响同样重要. 目前对于室内气态污染物的研究主要集中于挥发性有机物(volatile organic compounds, VOCs)上, 因为其室内含量一般超过室外. 目前, 已有研究发现了室内VOCs与过敏性鼻炎相关, 分别在法国和印度尼西亚开展的2项横断面研究发现了苯和甲醛与过敏性鼻炎存在关联[70,71]. 除了非生物来源之外, 生物挥发性有机化合物(microbial volatile organic compounds, MVOCs)也与过敏性鼻炎相关. 一项横断面研究发现, 在成人中, 脂肪族MVOCs(1-辛烯-3-醇)的暴露与过敏性鼻炎发病增加存在正相关[72]. 一些属于内分泌干扰物的VOCs也被发现与过敏性鼻炎相关, 它们除了对人体内分泌系统造成影响外, 还能通过调节细胞因子的合成、Th1/Th2平衡和IgE生成对过敏免疫反应产生潜在影响[73]. 这在动物实验中也得到了验证[74]. 但目前室内VOCs与过敏性鼻炎的关联研究仍相对较少, 且仍缺乏对于机制方面的探索[75,76], 多数研究采用横断面这类证据等级较低的研究方法, 有必要考虑进行更多诸如前瞻性队列等高质量的研究, 并明确相关的影响机制.

3 空气污染与其他因素对过敏性鼻炎的协同作用

3.1 过敏原

散布在环境中的过敏原是诱发过敏性鼻炎最直接的原因, 许多研究指出空气污染会与过敏原协同作用从而促进致敏(图2), 关于空气污染与过敏原协同作用的动物或人群研究已有许多. 花粉是诱发过敏性鼻炎的重要因素, 因其对花粉过敏患者的影响十分显著, 且粒径较大, 容易与空气中的颗粒物结合或受到气态污染物影响, 多数研究以花粉为目标来探讨与空气污染的协同作用. 空气污染可增加花粉过敏原的释放, 并通过过敏原的修饰增强其致敏潜能, Motta等人[77]研究了空气污染对草花粉中过敏原释放的影响, 发现交通排放的NO_2和O_3会增加草花粉过敏原的释放, 并增加空气中花粉过敏原的生物利用率, 从而增加过敏性鼻炎的发病风险. 空气污染还充当着免疫佐剂的角色, 通过影响免疫系统加剧过敏反应, 主要包括破坏上皮屏障促进过敏原与免疫细胞的接触、诱导氧化应激和免疫细胞的活化、与过敏原共暴露从而加强Th2细胞的应答[35,78,79]. Czarnobilska等人[80]研究了30名过敏性鼻炎患者不同暴露场景的过敏反应情况, 发现桦树花粉和$PM_{2.5}$对过敏性鼻炎患者有协同作用, $PM_{2.5}$会激活外周血中的嗜碱性粒细胞, 加强过敏原对嗜碱性粒细胞的刺激, 从而加剧过敏反应, 接触污染物越多, 患者嗜碱性粒细胞对过敏原和PM的协同反应就越高. 整体来看, 空气污染与过敏原已被证明存在协同作用并对过敏性鼻炎产生影响, 但还存在不一致的研究结果[81], 且对于机制的探索仍有待深入.

3.2 气象因素

研究表明气象因素对过敏性鼻炎的发病存在影响, 空气污染可能与气象因素协同作用从而影响过敏性鼻炎(图2), 它们之间的协同作用可能体现在2个方面: 一是气象因素通过直接影响空气污染物的种类、浓度、分布间接影响过敏性鼻炎发病与严重程度; 二是气象因素可以对过敏原或人体产生影响, 改变过敏原浓度或调节人体免疫功能及过敏易感性, 从而协同空气污染对过敏性鼻炎产生影响.

气象因素对空气污染的影响包括气温、气湿、风力、降水和日照等多种因素, 它们会通过影响大气流动性、影响物质的合成与分解以及湿沉降作用等对PM、O_3、NO_2和SO_2等空气污染物产生影响[82~87].

气象因素还会对人体免疫产生影响. 气温能影响机体的免疫调节功能, 造成机体免疫功能的紊乱, 并改变对过敏原的应激性, 从而诱发或加重过敏性鼻炎. Cheng等人[88]开展的一项关于寒流对儿科过敏性鼻炎门诊影响的时间序列研究发现, 不同滞后天数的寒流

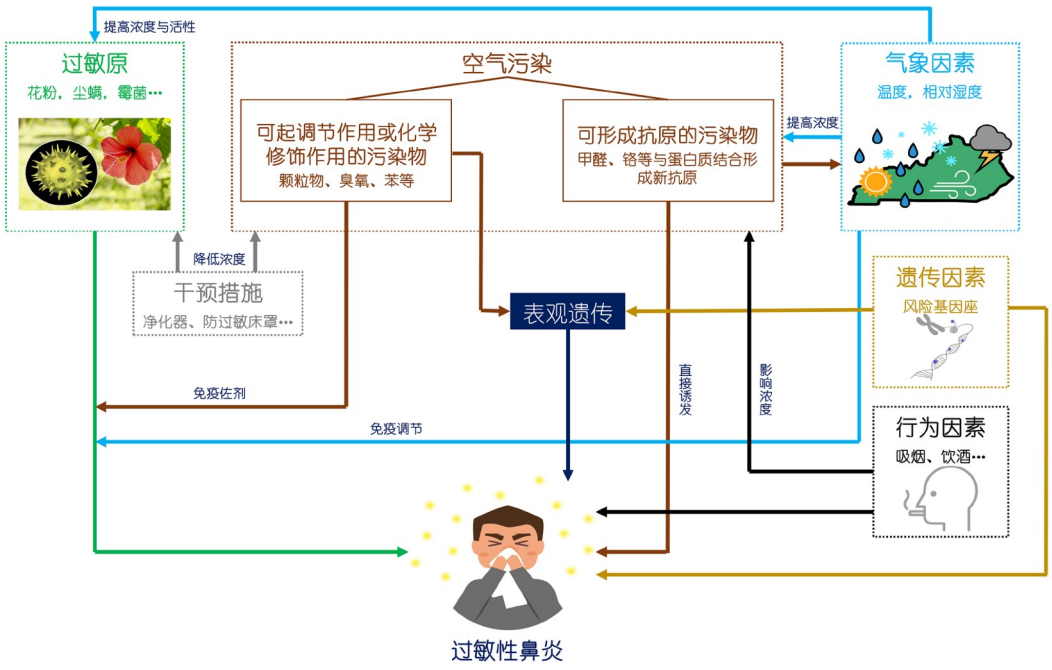

图 2 (网络彩色版)空气污染与其他因素对过敏性鼻炎的协同作用
Figure 2 (Color online) The synergistic effect of air pollution and other factors on allergic rhinitis

对儿科过敏性鼻炎日门诊量均有影响，寒流可能会大大增加儿科过敏性鼻炎的门诊量. 这种气温对人体机能的影响会加剧空气污染的影响. 研究表明, 在低温条件下, PM_{10}、NO_2和O_3浓度的增加对过敏性鼻炎门诊量增加的影响会变大[89]. 此外, 气温也会影响过敏原蛋白的活性以及过敏原的浓度, 特别是花粉. 气温的变化会使植物应激产生大量花粉，因气温升高导致花粉季提前到来会延长花粉季的持续时间, 且树木花粉的过敏性明显加强[90], 这会加剧空气污染与过敏原的协同作用.

空气的相对湿度也会对过敏性鼻炎造成影响，相对湿度较低时, 干燥的空气会使鼻黏膜变得脆弱, 更易于损伤; 相对湿度较高时, 屋尘螨、霉菌过敏原更易生长, 更易诱发过敏性鼻炎. Duan等人[91]开展的一项关于相对湿度对过敏性鼻炎的影响的时间序列研究发现, 高相对湿度和低相对湿度都造成医院过敏性鼻炎门诊量的增加, 增加的比例分别为4.07%和5.22%. 相对湿度对人体和过敏原的影响间接加剧了空气污染对过敏性鼻炎的影响. 一项新的时间序列研究表明在低温和高相对湿度下，空气污染对过敏性鼻炎的影响可能会增强[92]. 出生前, 特别是在子宫内暴露于潮湿环境中对过敏性鼻炎的发展起着关键作用[93]. 在室内环境中, 潮湿对过敏性鼻炎的影响比其他环境因素更加突出，其与室内污染物及过敏原的协同作用会加剧过敏性鼻炎发病[94].

总体而言，空气污染与气象因素的协同作用是多方面的，但空气污染与气象因素对过敏性鼻炎的协同作用的直接证据仍较少，且已有研究仅部分解释了气象因素与空气污染协同作用的机制，还需要进一步研究来阐明其内在联系.

3.3 遗传因素

研究表明许多基因与多种免疫相关疾病(包括过敏性和自身免疫性疾病)有关[95]. 一项大型全基因组关联研究(genome-wide association studies, GWAS)和人类白细胞抗原(HLA)精细定位研究发现了41个过敏性鼻炎的显著风险基因座, 包括20个新基因座, 这些新基因座大多在先天和适应性免疫过程中具有功能[96]. 一项新的前瞻性队列研究表明高遗传风险人群长期暴露于空气污染物的过敏性鼻炎发病风险更高[42,95]，这表明空气污染与遗传因素可能存在协同作用. 近年来的研究主要发现了空气污染会影响表观遗传进而影响过敏性鼻炎. 在中国上海开展的一项针对儿童的病例对照研究发现$PM_{2.5}$暴露水平与γ干扰素(IFN-γ)启动子区的甲基化水平呈正相关，$PM_{2.5}$对儿童过敏性鼻炎的影响可

能是通过IFN-γ启动子区的表观遗传学修饰介导的[97]. 一项动物实验也表明$PM_{2.5}$暴露可通过增加$CD4^+$T细胞中IFN-γ启动子的DNA甲基化, 从而加剧过敏性鼻炎[98]. 对于空气污染与遗传因素特别是表观遗传协同影响过敏性鼻炎仍是值得研究的方向, 特别是机制方面.

3.4 干预措施

鉴于当前空气污染对过敏性鼻炎患病的不利影响, 除了药物干预、免疫疗法等, 个人可以采取适当的措施进行自我保护以降低空气污染带来的影响(图2). 一方面可以从接触途径上减少与空气污染或过敏原的接触, 如佩戴口罩; 另一方面可以从改善环境着手, 减少环境中空气污染物或过敏原的浓度, 调节室内温度、湿度或保持卫生清洁等. 很多研究表明空气净化器可以有效去除室内空气污染物和过敏原, 对过敏性鼻炎患者的症状存在改善效果. Reisman等人[99]开展的随机对照试验表明高效微粒空气净化器(HEPA)对室内0.3 μm以上的PM去除效果可达70%, 并可减少常年性过敏性鼻炎患者的呼吸道症状. Park等人[100]在美国加利福尼亚州过敏性鼻炎儿童患者间开展的随机对照试验表明HEPA干预降低了室内$PM_{2.5}$水平, 并显著改善了过敏性鼻炎儿童的鼻部症状. 除此以外, 也有研究表明使用防过敏原床罩、枕头等床上用品也可以减少个人对室内过敏原或污染物的暴露量, 从而减缓过敏性鼻炎症状[101,102]. 室外环境于个人而言是不可控的, 但可以减少在花粉季或空气污染严重时期的外出. 此外, 国家也可以出台相关政策对区域空气环境进行改善, 以降低空气污染带来的危害.

4 总结与展望

4.1 总结

本文总结了不同空气污染物对过敏性鼻炎的影响及其机制, 以及空气污染与其他因素对过敏性鼻炎的协同作用的研究结果. 已有的研究结果表明: 空气污染对过敏性鼻炎的影响机制包括过敏原作用、过敏原增强作用、炎症反应、氧化应激和免疫系统调节; PM及其化学组分、室外气态污染物、室内VOCs均对过敏性鼻炎存在影响, 且影响机制存在不同; 空气污染会与过敏原、气象因素、遗传因素协同作用于过敏性鼻炎, 而一些干预措施可以减轻空气污染对过敏性鼻炎的影响.

4.2 研究前沿建议

虽然目前已有研究证明了空气污染与过敏性鼻炎存在关联, 但研究的污染物种类仍较少, 且研究方法存在较大差异, 高质量研究相对缺乏, 空气污染对过敏性鼻炎的影响仍存在不确定性. 此外, 对于影响机制的研究仍不充分, 特别是混合污染物的影响, 与其他因素的协同作用同样缺乏高质量的研究与机制方面的探索, 基于以上问题, 我们提出以下几点未来的研究建议: (1) 研究超细颗粒物等其他粒径的PM以及不同化学组分、室内VOCs、空气中的新污染物对过敏性鼻炎的影响, 发觉更多可能存在影响的污染物, 并通过研究设计或统计分析, 探究单一或混合污染物的不同影响; (2) 应用暴露组学、微生物组、行为组等多种组学, 并结合机器学习等手段, 探寻人体内空气污染物对过敏性鼻炎的影响机制及特征生物标志物; (3) 将空气污染影响过敏性鼻炎机制的研究拓展到神经调节等可能机制方面, 并进一步开展空气污染对过敏性鼻炎可能存在的结膜炎、哮喘等共病关联的研究; (4) 过敏性鼻炎难以根治, 除了药物治疗、免疫疗法等, 针对个人的有效干预措施仍较缺乏. 未来应开展空气净化设备、过敏防护装备等干预措施去除室内空气污染物、过敏原的效率研究, 并探索其在中国空气环境与室内条件下用于过敏性鼻炎预防或干预的可能性.

参考文献

1 Wise S K, Damask C, Greenhawt M, et al. A synopsis of guidance for allergic rhinitis diagnosis and management from ICAR 2023. J Allergy Clin Immunol-Pract, 2023, 11: 773–796

2 Zhang X, Zhang M, Sui H, et al. Prevalence and risk factors of allergic rhinitis among Chinese adults: A nationwide representative cross-sectional study. World Allergy Organization J, 2023, 16: 100744

3 Chen Y, Zhu J, Lyu J, et al. Association of maternal prepregnancy weight and gestational weight gain with children's allergic diseases. JAMA Netw Open, 2020, 3: e2015643

4 Wise S K, Damask C, Roland L T, et al. International consensus statement on allergy and rhinology: Allergic rhinitis – 2023. Int Forum Allergy

Rhinol, 2023, 13: 293–859
5 Zhang Y, Zhang L. Increasing prevalence of allergic rhinitis in China. Allergy Asthma Immunol Res, 2019, 11: 156–169
6 Li Q, Zhang X, Feng Q, et al. Common allergens and immune responses associated with allergic rhinitis in China. J Asthma Allergy, 2023, Volume 16: 851–861
7 Bousquet J, van Cauwenberge P, Khaltaev N. Allergic rhinitis and its impact on asthma. J Allergy Clin Immunol, 2001, 108: S147–S334
8 Greiner A N, Hellings P W, Rotiroti G, et al. Allergic rhinitis. Lancet, 2011, 378: 2112–2122
9 Zuberbier T, Lötvall J, Simoens S, et al. Economic burden of inadequate management of allergic diseases in the European Union: A GA^2 LEN review. Allergy, 2014, 69: 1275–1279
10 Vandenplas O, Vinnikov D, Blanc P D, et al. Impact of rhinitis on work productivity: A systematic review. J Allergy Clin Immunol-Pract, 2018, 6: 1274–1286.e9
11 Li X, Xu X, Li J, et al. Direct and indirect costs of allergic and non-allergic rhinitis to adults in Beijing, China. Clin Transl All, 2022, 12: e12148
12 Norbäck D, Lu C, Wang J, et al. Asthma and rhinitis among Chinese children — Indoor and outdoor air pollution and indicators of socioeconomic status (SES). Environ Int, 2018, 115: 1–8
13 Lu C, Wang F, Liu Z, et al. Intrauterine and early postnatal exposure to air pollution associated with childhood allergic rhinitis. Chemosphere, 2023, 336: 139296
14 Hu X, Jiang W, Deng Q, et al. Early life exposure to environmental pollution increases childhood asthma, allergy and infection (in Chinese). Chin Sci Bull, 2018, 63: 954–967 [路婵, 胡旭, 缪玉峰, 等. 生命早期环境污染暴露增加儿童过敏与感染风险. 科学通报, 2018, 63: 954–967]
15 Zhang Y, Lan F, Zhang L. Advances and highlights in allergic rhinitis. Allergy, 2021, 76: 3383–3389
16 Li C H, Sayeau K, Ellis A K. Air pollution and allergic rhinitis: Role in symptom exacerbation and strategies for management. J Asthma Allergy, 2020, Volume 13: 285–292
17 Eguiluz-Gracia I, Mathioudakis A G, Bartel S, et al. The need for clean air: The way air pollution and climate change affect allergic rhinitis and asthma. Allergy, 2020, 75: 2170–2184
18 Bousquet J, Grattan C E, Akdis C A, et al. Highlights and recent developments in allergic diseases in EAACI journals (2019). Clin Transl Allergy, 2020, 10: 56
19 Feng C, Shao Y, Ye T, et al. Associations between long-term exposure to $PM_{2.5}$ chemical constituents and allergic diseases: Evidence from a large cohort study in China. Sci Total Environ, 2023, 904: 166755
20 Tingskov Pedersen C E, Eliasen A U, Ketzel M, et al. Prenatal exposure to ambient air pollution is associated with early life immune perturbations. J Allergy Clin Immunol, 2023, 151: 212–221
21 Brożek J L, Bousquet J, Agache I, et al. Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines—2016 revision. J Allergy Clin Immunol, 2017, 140: 950–958
22 Humbert M, Bousquet J, Bachert C, et al. IgE-mediated multimorbidities in allergic asthma and the potential for omalizumab therapy. J Allergy Clin Immunol-Pract, 2019, 7: 1418–1429
23 Bousquet J, Anto J M, Bachert C, et al. Allergic rhinitis. Nat Rev Dis Primers, 2020, 6: 95
24 Palomares Ó, Sánchez-Ramón S, Dávila I, et al. dIvergEnt: How IgE axis contributes to the continuum of allergic asthma and Anti-IgE therapies. Int J Mol Sci, 2017, 18: 1328
25 McGwin Jr. G, Lienert J, Kennedy Jr. J I. Formaldehyde exposure and asthma in children: A systematic review. Environ Health Perspect, 2010, 118: 313–317
26 Yon D K, Hwang S, Lee S W, et al. Indoor exposure and sensitization to formaldehyde among inner-city children with increased risk for asthma and rhinitis. Am J Respir Crit Care Med, 2019, 200: 388–393
27 Kurt O K, Basaran N. Occupational exposure to metals and solvents: Allergy and airway diseases. Curr Allergy Asthma Rep, 2020, 20: 38
28 Li A, Qiu X, Jiang X, et al. Alteration of the health effects of bioaerosols by chemical modification in the atmosphere: A review. Fundamental Res, 2024, 4: 463–470
29 Samadi N, Klems M, Heiden D, et al. Nitrated food proteins induce a regulatory immune response associated with allergy prevention after oral exposure in a Balb/c mouse food allergy model. Allergy, 2020, 75: 412–422
30 Verscheure P, Honnay O, Speybroeck N, et al. Impact of environmental nitrogen pollution on pollen allergy: A scoping review. Sci Total Environ, 2023, 893: 164801
31 Wang Y, Tang N, Mao M, et al. Fine particulate matter ($PM_{2.5}$) promotes IgE-mediated mast cell activation through ROS/Gadd45b/JNK axis. J Dermatol Sci, 2021, 102: 47–57
32 Rosario Filho N A, Satoris R A, Scala W R. Allergic rhinitis aggravated by air pollutants in Latin America: A systematic review. World Allergy Organization J, 2021, 14: 100574
33 Mohd Isa K N, Hashim Z, Jalaludin J, et al. The effects of indoor pollutants exposure on allergy and lung inflammation: An activation state of

neutrophils and eosinophils in sputum. Int J Environ Res Public Health, 2020, 17: 5413
34 Xian M, Ma S, Wang K, et al. Particulate matter 2.5 causes deficiency in barrier integrity in human nasal epithelial cells. Allergy Asthma Immunol Res, 2020, 12: 56–71
35 Reinmuth-Selzle K, Kampf C J, Lucas K, et al. Air pollution and climate change effects on allergies in the anthropocene: Abundance, interaction, and modification of allergens and adjuvants. Environ Sci Technol, 2017, 51: 4119–4141
36 Nagappan A, Park S B, Lee S J, et al. Mechanistic implications of biomass-derived particulate matter for immunity and immune disorders. Toxics, 2021, 9: 18
37 Sun N, Han Z, Wang H, et al. Effects of ursolic acid on the expression of Th1–Th2-related cytokines in a rat model of allergic rhinitis after $PM_{2.5}$ exposure. Am J RhinolAllergy, 2020, 34: 587–596
38 Van Nguyen T, Piao C H, Fan Y J, et al. Anti-allergic rhinitis activity of α-lipoic acid via balancing Th17/Treg expression and enhancing Nrf2/HO-1 pathway signaling. Sci Rep, 2020, 10: 12528
39 Wang C, Wang D, Zhao H, et al. Traffic-related $PM_{2.5}$ and diverse constituents disturb the balance of Th17/Treg cells by STAT3/RORγt-STAT5/Foxp3 signaling pathway in a rat model of asthma. Int Immunopharmacol, 2021, 96: 107788
40 Li J, Zhang Y, Zhang L, et al. Fine particulate matter exposure exacerbated nasal mucosal damage in allergic rhinitis mice via NLRP3 mediated pyroptosis. Ecotoxicol Environ Saf, 2021, 228: 112998
41 Piao C H, Fan Y, Nguyen T V, et al. $PM_{2.5}$ exacerbates oxidative stress and inflammatory response through the Nrf2/NF-κB signaling pathway in OVA-induced allergic rhinitis mouse model. Int J Mol Sci, 2021, 22: 8173
42 Luo P, Ying J, Li J, et al. Air pollution and allergic rhinitis: Findings from a prospective cohort study. Environ Sci Technol, 2023, 57: 15835–15845
43 Lin L, Li T, Sun M, et al. Effect of particulate matter exposure on the prevalence of allergic rhinitis in children: A systematic review and meta-analysis. Chemosphere, 2021, 268: 128841
44 Jia X, Shen Z, Liu R, et al. Association of fine particulate matter to allergic rhinitis: A systematic review and meta-analysis. Eur J Inflamm, 2022, 20: 1721727X2210898
45 Wang H, Li X B, Chu X J, et al. Ambient air pollutants increase the risk of immunoglobulin E–mediated allergic diseases: A systematic review and meta-analysis. Environ Sci Pollut Res, 2022, 29: 49534–49552
46 Janssen N A H, Gerlofs-Nijland M E, Lanki T, et al. Health effects of black carbon. In: Copenhagen: World Health Organization. Regional Office for Europe, 2012
47 Jung K H, Lovinsky-Desir S, Yan B, et al. Effect of personal exposure to black carbon on changes in allergic asthma gene methylation measured 5 days later in urban children: Importance of allergic sensitization. Clin Epigenet, 2017, 9: 61
48 Li Y, Ouyang Y, Jiao J, et al. Exposure to environmental black carbon exacerbates nasal epithelial inflammation via the reactive oxygen species (ROS)–nucleotide-binding, oligomerization domain-like receptor family, pyrin domain containing 3 (NLRP3)-caspase-1-interleukin 1β (IL-1β) pathway. Int Forum Allergy Rhinol, 2021, 11: 773–783
49 Savouré M, Lequy É, Bousquet J, et al. Long-term exposures to $PM_{2.5}$, black carbon and NO_2 and prevalence of current rhinitis in French adults: The Constances Cohort. Environ Int, 2021, 157: 106839
50 Chen T, Norback D, Deng Q, et al. Maternal exposure to $PM_{2.5}$/BC during pregnancy predisposes children to allergic rhinitis which varies by regions and exclusive breastfeeding. Environ Int, 2022, 165: 107315
51 Li X, Wu H, Xing W, et al. Short-term association of fine particulate matter and its constituents with oxidative stress, symptoms and quality of life in patients with allergic rhinitis: A panel study. Environ Int, 2023, 182: 108319
52 Jia P, Feng C, Ye T, et al. $PM_{2.5}$ chemical constituents and allergic rhinitis: Findings from a prospective cohort study in China. Allergy, 2023, 78: 1703–1706
53 Li S, Wu W, Wang G, et al. Association between exposure to air pollution and risk of allergic rhinitis: A systematic review and meta-analysis. Environ Res, 2022, 205: 112472
54 Iwasawa S, Kikuchi Y, Nishiwaki Y, et al. Effects of SO_2 on respiratory system of adult Miyakejima resident 2 years after returning to the island. J Occupational Health, 2009, 51: 38–47
55 Kim S H, Lee J, Oh I, et al. Allergic rhinitis is associated with atmospheric SO_2: Follow-up study of children from elementary schools in Ulsan, Korea. PLoS ONE, 2021, 16: e0248624
56 Ye M, Liu H, Li H, et al. Long-term exposure to sulfur dioxide before sensitization decreased the production of specific IgE in HDM-sensitized allergic rhinitis mice. J Inflamm Res, 2022, 15: 2477–2490
57 Chen C C, Chiu H F, Yang C Y. Air pollution exposure and daily clinical visits for allergic rhinitis in a subtropical city: Taipei, Taiwan. J Toxicol Environ Health A, 2016, 79: 494–501
58 Wang J H, Devalia J L, Duddle J M, et al. Effect of six-hour exposure to nitrogen dioxide on early-phase nasal response to allergen challenge in

patients with a history of seasonal allergic rhinitis. J Allergy Clin Immunol, 1995, 96: 669–676
59 Wang J, Lu M, An Z, et al. Associations between air pollution and outpatient visits for allergic rhinitis in Xinxiang, China. Environ Sci Pollut Res, 2020, 27: 23565–23574
60 Koehler C, Paulus M, Ginzkey C, et al. The proinflammatory potential of nitrogen dioxide and its influence on the house dust mite allergen der p 1. Int Arch Allergy Immunol, 2016, 171: 27–35
61 Kwon M Y, Lee J S, Park S. The effect of outdoor air pollutants and greenness on allergic rhinitis incidence rates: A cross-sectional study in Seoul, Korea. Int J Sustain Dev World Ecol, 2019, 26: 258–267
62 Jörres R, Nowak D, Magnussen H. The effect of ozone exposure on allergen responsiveness in subjects with asthma or rhinitis.. Am J Respir Crit Care Med, 1996, 153: 56–64
63 Zhou P E, Qian Z, McMillin S, et al. Relationships between long-term ozone exposure and allergic rhinitis and bronchitic symptoms in Chinese children. Toxics, 2021, 9: 221
64 Zhang X, Sun N, Huang Y, et al. Role of NLRP3 in the exacerbation of ozone-induced allergic rhinitis. Ecotoxicol Environ Saf, 2023, 265: 115506
65 Sun N, Niu Y, Zhang R, et al. Ozone inhalation induces exacerbation of eosinophilic airway inflammation and Th2-skew immune response in a rat model of AR. Biomed Pharmacother, 2021, 137: 111261
66 Sun N, Huang Y, Zhang X, et al. Involvements of Nrf2 and oxidative stress in the ozone-elicited exacerbation in an allergic rhinitis model. Ecotoxicol Environ Saf, 2023, 255: 114822
67 Sokolowska M, Quesniaux V F J, Akdis C A, et al. Acute respiratory barrier disruption by ozone exposure in mice. Front Immunol, 2019, 10: 2169
68 Meng Y, Wang C, Zhang L. Recent developments and highlights in allergic rhinitis. Allergy, 2019, 74: 2320–2328
69 Zou Q Y, Shen Y, Ke X, et al. Exposure to air pollution and risk of prevalence of childhood allergic rhinitis: A meta-analysis. Int J Pediatr Otorhinolaryngol, 2018, 112: 82–90
70 Penard-Morand C, Raherison C, Charpin D, et al. Long-term exposure to close-proximity air pollution and asthma and allergies in urban children. Eur Respir J, 2010, 36: 33–40
71 Prasasti C I, Haryanto B, Latif M T. Association of VOCs, $PM_{2.5}$ and household environmental exposure with children's respiratory allergies. Air Qual Atmos Health, 2021, 14: 1279–1287
72 Araki A, Kanazawa A, Kawai T, et al. The relationship between exposure to microbial volatile organic compound and allergy prevalence in single-family homes. Sci Total Environ, 2012, 423: 18–26
73 Chalubinski M, Kowalski M L. Endocrine disrupters – potential modulators of the immune system and allergic response. Allergy, 2006, 61: 1326–1335
74 Wang Y, Gu Z, Cao Z, et al. Nonylphenol can aggravate allergic rhinitis in a murine model by regulating important Th cell subtypes and their associated cytokines. Int Immunopharmacol, 2019, 70: 260–267
75 Tagiyeva N, Sheikh A. Domestic exposure to volatile organic compounds in relation to asthma and allergy in children and adults. Expert Rev Clin Immunol, 2014, 10: 1611–1639
76 Nurmatov U B, Tagiyeva N, Semple S, et al. Volatile organic compounds and risk of asthma and allergy: A systematic review. Eur Respir Rev, 2015, 24: 92–101
77 Motta A C, Marliere M, Peltre G, et al. Traffic-related air pollutants induce the release of allergen-containing cytoplasmic granules from grass pollen. Int Arch Allergy Immunol, 2006, 139: 294–298
78 Baldacci S, Maio S, Cerrai S, et al. Allergy and asthma: Effects of the exposure to particulate matter and biological allergens. Respir Med, 2015, 109: 1089–1104
79 Deng S Z, Jalaludin B B, Antó J M, et al. Climate change, air pollution, and allergic respiratory diseases: A call to action for health professionals. Chin Med J, 2020, 133: 1552–1560
80 Czarnobilska E, Bulanda M, Bulanda D, et al. The influence of air pollution on the development of allergic inflammation in the airways in Krakow's atopic and non-atopic residents. J Clin Med, 2021, 10: 2383
81 Annesi-Maesano I, Rouve S, Desqueyroux H, et al. Grass pollen counts, air pollution levels and allergic rhinitis severity. Int Arch Allergy Immunol, 2012, 158: 397–404
82 Wang J, Wang Y, Liu H, et al. Diagnostic identification of the impact of meteorological conditions on $PM_{2.5}$ concentrations in Beijing. Atmos Environ, 2013, 81: 158–165
83 He J, Gong S, Yu Y, et al. Air pollution characteristics and their relation to meteorological conditions during 2014–2015 in major Chinese cities. Environ Pollution, 2017, 223: 484–496
84 Chen Z, Chen D, Zhao C, et al. Influence of meteorological conditions on $PM_{2.5}$ concentrations across China: A review of methodology and

mechanism. Environ Int, 2020, 139: 105558
85 Chen Z, Li R, Chen D, et al. Understanding the causal influence of major meteorological factors on ground ozone concentrations across China. J Cleaner Production, 2020, 242: 118498
86 Xue R, Wang S, Li D, et al. Spatio-temporal variations in NO_2 and SO_2 over Shanghai and Chongming Eco-Island measured by Ozone Monitoring Instrument (OMI) during 2008–2017. J Cleaner Production, 2020, 258: 120563
87 Hu M, Wang Y, Wang S, et al. Spatial-temporal heterogeneity of air pollution and its relationship with meteorological factors in the Pearl River Delta, China. Atmos Environ, 2021, 254: 118415
88 Cheng Q, Wang X, Wei Q, et al. The short-term effects of cold spells on pediatric outpatient admission for allergic rhinitis in Hefei, China. Sci Total Environ, 2019, 664: 374–380
89 Wang X, Gao C, Xia Y, et al. Effect of air pollutants and meteorological factors on daily outpatient visits of allergic rhinitis in Hohhot, China. J Asthma Allergy, 2023, Volume 16: 1217–1228
90 D'Amato G, Holgate S T, Pawankar R, et al. Meteorological conditions, climate change, new emerging factors, and asthma and related allergic disorders. A statement of the World Allergy Organization. World Allergy Organization J, 2015, 8: 25
91 Duan J, Wang X, Zhao D, et al. Risk effects of high and low relative humidity on allergic rhinitis: Time series study. Environ Res, 2019, 173: 373–378
92 Wu R, Guo Q, Fan J, et al. Association between air pollution and outpatient visits for allergic rhinitis: Effect modification by ambient temperature and relative humidity. Sci Total Environ, 2022, 821: 152960
93 Lu C, Liu Z, Liao H, et al. Effects of early life exposure to home environmental factors on childhood allergic rhinitis: Modifications by outdoor air pollution and temperature. Ecotoxicol Environ Saf, 2022, 244: 114076
94 Xu X, Liu X, Li J, et al. Environmental risk factors, protective factors, and biomarkers for allergic rhinitis: A systematic umbrella review of the evidence. Clinic Rev Allerg Immunol, 2023, 65: 188–205
95 Choi B Y, Han M, Kwak J W, et al. Genetics and epigenetics in allergic rhinitis. Genes, 2021, 12: 2004
96 Waage J, Standl M, Curtin J A, et al. Genome-wide association and HLA fine-mapping studies identify risk loci and genetic pathways underlying allergic rhinitis. Nat Genet, 2018, 50: 1072–1080
97 Li Y, Mu Z, Wang H, et al. The role of particulate matters on methylation of IFN-γ and IL-4 promoter genes in pediatric allergic rhinitis. Oncotarget, 2018, 9: 17406–17419
98 Li Y, Zhou J, Rui X, et al. $PM_{2.5}$ exposure exacerbates allergic rhinitis in mice by increasing DNA methylation in the IFN-γ gene promoter in $CD4^+$T cells via the ERK-DNMT pathway. Toxicol Lett, 2019, 301: 98–107
99 Reisman R E, Mauriello P M, Davis G B, et al. A double-blind study of the effectiveness of a high-efficiency particulate air (HEPA) filter in the treatment of patients with perennial allergic rhinitis and asthma. J Allergy Clin Immunol, 1990, 85: 1050–1057
100 Park H K, Cheng K C, Tetteh A O, et al. Effectiveness of air purifier on health outcomes and indoor particles in homes of children with allergic diseases in Fresno, California: A pilot study. J Asthma, 2017, 54: 341–346
101 Terreehorst I, Hak E, Oosting A J, et al. Evaluation of impermeable covers for bedding in patients with allergic rhinitis. N Engl J Med, 2003, 349: 237–246
102 Stillerman A, Nachtsheim C, Li W, et al. Efficacy of a novel air filtration pillow for avoidance of perennial allergens in symptomatic adults. Ann Allergy Asthma Immunol, 2010, 104: 440–449

Summary for “空气污染对过敏性鼻炎影响的研究进展”

Research progress on the impact of air pollution on allergic rhinitis

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Allergic rhinitis is an inflammatory disease of the nasal mucosa caused by an immunoglobulin E (IgE) mediated response to inhaled allergens. The global prevalence of allergic rhinitis ranges from 5% to 50%, and it has been on the rise in recent years. Allergic rhinitis is often comorbid with allergic conjunctivitis and asthma, causing a burden on personal life and socio-economic well-being. Except for genetics, environment, and lifestyle, increasing evidence showed that air pollution is an important factor that increases the risk of allergic rhinitis and exacerbates its symptoms. Due to the complexity of air pollution, current research focuses more on mixed air pollutants, and other environmental factors have also become research objects. However, the types of air pollutants studied are still limited, and the mechanisms of many pollutants' effects are not yet clear. Moreover, research on the synergistic effects with other factors is not complete. Therefore, summaries of existing research results and deeper studies on the impact of air pollution on allergic rhinitis are crucial for the prevention and control of the disease.

This study analyzes the effects, mechanisms, and related synergistic factors of air pollution on allergic rhinitis. Multiple keyword combinations such as “allergic rhinitis” and “air pollution”, “allergens”, “meteorology”, “genetics” or “intervention” were used to screen relevant literature in four domestic and foreign databases. We excluded studies lacking explicit pollutant types or potential synergistic factors, focusing on summarizing and analyzing the latest research developments over the past five years. A qualitative review method was used to identify current research deficiencies and propose potential future research directions based on the findings.

Our conclusions are as follows: air pollution may affect different stages of the disease process, impacting allergic rhinitis by acting as allergens or allergen enhancers during sensitization, and influencing the sensitization or effector stages through oxidative stress, inflammatory responses, or immune system regulation. These effects may involve the interaction of multiple mechanisms, but most of them ultimately lead to inflammatory response. Epidemiological studies have found associations between particulate matter ($PM_{2.5}$, PM_{10}, and their chemical components) and outdoor gaseous pollutants (SO_2, NO_2, O_3, CO) with allergic rhinitis across different regions and populations. And some volatile organic compounds are associated with allergic rhinitis in indoor environments. These pollutants can affect allergic rhinitis through different mechanisms mentioned above, which have been corroborated by animal studies. Moreover, air pollution also interacts synergistically with other environmental factors. Environmental allergens are the direct cause of allergic rhinitis, and air pollution can synergize with allergens through multiple pathways to promote sensitization. Air pollution can also interact with meteorological factors such as temperature and humidity and epigenetics factors to influence allergic rhinitis, while interventions such as wearing masks and using air purifiers can reduce the adverse effects of air pollution on allergic rhinitis.

Finally, although there have been studies that have shown a correlation between air pollution and allergic rhinitis, the types of pollutants studied remains limited. Furthermore, there are considerable differences in research methodologies, and high-quality research is relatively lacking. The impact of air pollution on allergic rhinitis still remains uncertain. Therefore, we suggest that future research should identify more potential pollutants that may impact allergic rhinitis and investigate the distinct effects of individual and mixed pollutants. Methods such as omics or machine learning should be used to explore the effect mechanism and biomarkers of air pollution on allergic rhinitis in the human body. Given the incurable of allergic rhinitis, it is imperative to develop more effective personalized intervention measures.

air pollution, allergic rhinitis, effect mechanism, meteorological factor, synergistic reaction

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