

# 臭氧和温度交互作用及其协同健康影响研究进展

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**摘要** 气候变化是21世纪人类所面临的最大环境挑战。人类活动是引起臭氧污染和全球变暖的最主要原因,二者同根同源并且能够彼此影响。未来气候变暖的“气候惩罚”效应会进一步促进人为排放较强区域臭氧浓度的升高,而臭氧可以通过其正向辐射强迫作用致使大气升温。臭氧污染和全球温度变化对于人体健康具有潜在的联合作用,流行病学研究主要通过分层分析和纳入交互作用项的方式探究二者协同健康影响。根据现有的研究证据,高温与臭氧复合暴露增加人群死亡风险的研究证据最为充分,低温和臭氧复合暴露的健康影响还存在较大的不确定性,温度和臭氧协同作用超额死亡风险定量评价从1.73%~12.31%不等。心肺疾病患者和老年人是二者复合暴露的潜在易感人群。然而,目前对于这种协同作用超额风险的定量研究证据比较有限,研究结果仍存在较大的不确定性。未来仍需要多中心、大样本量的流行病学研究量化这种协同作用的大小,从而为更加准确、全面评估疾病负担和制定公共卫生政策提供依据。

**关键词** 温度, 臭氧, 协同健康效应, 死亡风险, 气候变化

气候变化是人类现阶段面临的最严峻的环境挑战之一,对人体健康具有显著危害。据联合国政府间气候变化专门委员会第六次评估报告(The United Nations Intergovernmental Panel on Climate Change 6th Assessment Report, IPCC AR6)显示,全球地表温度相比于工业革命前已经上升1.1°C<sup>[1]</sup>。全球变暖呈现加速趋势,预计到2035年,全球平均地表温度将进一步上升0.3~0.7°C,同时,极热天气事件出现的频率将迅速增加<sup>[2]</sup>。一项纳入了五大洲750个城市,涵盖全球46.3%人口的研究显示,2000~2019年,不适温度与每年超过500万超额死亡有关,平均每10万居民中有74例气温相关死亡,占全球总死因的9.43%<sup>[3]</sup>。极端天气事件频发,通过直接或间接的方式对人群健康产生广泛而复杂的影响。

在全球气候变化的背景下,环境污染问题也日益

凸显。近地面臭氧(ozone, O<sub>3</sub>)是大气氧化性的主要来源,也是危害人体健康的关键污染物。越来越多的证据显示,臭氧暴露的健康危害独立于PM<sub>2.5</sub>的健康效应。伴随着臭氧浓度水平的增加,其潜在健康威胁也在逐年递增。根据全球疾病负担研究<sup>[4]</sup>,2019年,全球约有36.5万的过早死亡和621万的伤残调整寿命年可以归因于环境臭氧暴露。2021和2022年我国臭氧的长期和短期暴露导致的成人过早死亡人数分别为13万和8万<sup>[5]</sup>。Westervelt等人<sup>[6]</sup>估计,到2050年,气候变化将导致中国的臭氧污染进一步恶化11%,导致6万多人过早死亡。

全球温度变化和臭氧污染复合暴露是现阶段最常见的复合暴露事件之一。气象条件改变可以通过影响排放、大气化学和污染物运输来影响地表空气质量<sup>[7]</sup>,而臭氧作为一种光化学产物<sup>[8,9]</sup>,与温度变化具有很强

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的关联性。气候变化背景下，不适宜温度和臭氧污染复合暴露事件出现的频率将进一步增加，为了应对当前和未来全球温度变化和臭氧污染所带来的挑战，我们亟需相关研究明晰和评估二者复合暴露是否会导致额外的健康危害。因此，本文旨在梳理现阶段臭氧污染与全球温度变化之间相互影响以及二者协同作用于人体健康的科学证据，以健康为驱动力，为气候变化与臭氧污染协同治理提供科学依据。

## 1 臭氧与全球温度变化的交互作用

### 1.1 近地面臭氧对全球温度变化的影响

与其他重要的温室气体(如二氧化碳和甲烷)不同，近地面臭氧是一种由氮氧化物和挥发性有机物等前体物质在光照下反应生成的二次污染物。臭氧本身是一种大气污染物，同时也是一种典型的短寿命气候强迫因子，能够直接影响天气<sup>[10]</sup>。与温室气体相比，臭氧的寿命通常较短，在对流层中表现为随季节变化的非均匀分布。臭氧在红外区域的9.6 μm波段有一个显著的吸收峰，能有效吸收传入的太阳辐射和地球表面发出的长波辐射。在此波段，温室气体和水蒸气的吸收能力较弱，臭氧贡献了主要的增温作用。对流层臭氧浓度的变化通过扰乱大气的辐射平衡影响近地表温度、降水和大气环流模式等<sup>[11~14]</sup>，进而作用于人体和生态系统健康。

臭氧对于气候的影响主要来自于其正向辐射强迫(radiative forcing, RF)作用导致的温度上升。2021年IPCC AR6中臭氧有效辐射强迫最新估算值为0.47(0.24~0.71) W/m<sup>2</sup><sup>[15]</sup>。自前工业化时代以来，对流层臭氧浓度增加导致的正向RF约为二氧化碳总变暖效应的25%~40%和甲烷的75%。在对全球气候的影响上，臭氧也仅次于这两种主要温室气体<sup>[16]</sup>。

### 1.2 全球温度变化对于对流层臭氧浓度的影响

气候变化相关的温度变化可以通过影响气象物理条件、臭氧的化学生成和沉降以及臭氧前体物自然排放等途径影响臭氧浓度水平<sup>[17]</sup>。热浪和臭氧污染通常在温暖的季节同时发生，停滞的高压系统有利于热量和臭氧前体物的积累，目前已经成为全球范围内对健康威胁最大的复合事件之一<sup>[10]</sup>。同时，高温天气往往伴随着干燥的气候条件，这种条件通过加剧植被的干旱胁迫，限制了植物气孔吸收臭氧的能力，从而在热浪期

间显著促进了臭氧水平的升高<sup>[18,19]</sup>。随着野火等污染事件频率的增加，预计臭氧的背景浓度也将上升<sup>[10]</sup>。

IPCC AR6报告中提到，在参与CMIP6的地球系统模式中，4个模型集合的平均结果表明，随着全球变暖，水汽的增加将促进臭氧的化学去除，导致全球地表臭氧年均浓度在升温1.5~2.5°C时下降约1.2~2.3 ppb(1 ppb=10<sup>-9</sup>)。然而，这种浓度下降主要发生在海洋上空和较为清洁的陆地区域。地表温度每升高1°C，臭氧浓度可降低0.2~2 ppb。而在北美、欧洲和东亚等臭氧产生地区，模式研究预测未来气候变暖将导致夏季臭氧浓度普遍增加<sup>[20]</sup>。全球温升增加了污染区域臭氧前体物转化的效率<sup>[21]</sup>，使得在人为或自然源排放较高的区域，地表臭氧浓度随温度上升而增加。

总的来说，未来气候变化对近地面臭氧的影响呈现为“气候惩罚”效应。在未受污染地区，较高的水蒸气含量和温度会增强臭氧的化学分解，导致近地面臭氧背景水平降低<sup>[17]</sup>，而在污染地区，较高的地表温度将激发区域化学和局部排放的反馈作用，增加地表臭氧及其峰值强度。

## 2 全球温度变化与臭氧复合暴露对人群健康风险的协同作用

研究表明，在诸多经历过极端高温天气的城市中，与臭氧相关的健康问题增长显著<sup>[22~25]</sup>，常见的是臭氧污染与高温热浪同时发生，对人群健康构成重大威胁<sup>[26,27]</sup>。流行病学研究发现，当高臭氧暴露与高温同时出现时，其对人群健康的危害可能达到最大程度<sup>[27]</sup>。有研究通过在模型中加入或移除臭氧和温度变量，分析这些变量对健康效应强度和方向的影响。结果表明，在某些情况下，模型中加入臭氧变量并不改变温度与死亡率关联的估计值<sup>[28~30]</sup>；但也有研究发现，加入臭氧变量则略微降低了这一关联的估计值<sup>[31~33]</sup>。Basu等人<sup>[34]</sup>在加州两个县的研究中发现了臭氧的显著效应修饰作用，但在将研究范围扩大至9个县的综合分析中，则未发现臭氧混杂作用的证据。Zanobetti和Schwartz<sup>[35]</sup>在美国9个城市的研究中发现，臭氧的存在并未改变温度对健康的影响，但作为混杂因素考虑时，臭氧略微降低了高温对死亡率的效应估计。

近年来，越来越多的研究提出，臭氧等污染物在温度和死亡率之间的因果关系中可能扮演重要角色，因此仅将臭氧视为混杂因素可能并不妥当<sup>[36,37]</sup>。Reid等人<sup>[38]</sup>运用有向无环图深入分析了温度与臭氧-死亡关

系的因果结构,发现臭氧作为受温度影响的二次污染物,本身也会影响健康,因此不应简单地将其视为温度影响健康的混杂因素。在法国和韩国的最新时间序列研究中,臭氧被认为是温度影响人群死亡率的潜在中介机制<sup>[39,40]</sup>。还有研究应用贝叶斯核机器回归模型(Bayesian kernel machine regression model, BKMR)探究臭氧和温度对于江西省南昌市心脑血管疾病死亡风险评估,发现二者之间可能存在相互作用,尤其是对于脑血管死亡<sup>[41]</sup>。这些研究都强调了温度和臭氧复合暴露对死亡风险的协同作用在很大程度上被忽视了<sup>[42]</sup>,考虑温度修饰在精准量化臭氧的健康风险具有重要的意义<sup>[43]</sup>。

为了更全面理解臭氧和温度复合暴露对健康影响的研究现状,我们使用“臭氧”“温度”“气候变化”和“健康”等关键词,收集了自1990年以来发表在Web of Science和PubMed上共计2265篇文献。我们的纳入标准包括:考虑臭氧污染与温度复合暴露对人群死亡率的影响,用定性或定量方法表征臭氧与温度对人群死亡风险的协同作用,以及基于典型的流行病学研究方法。最终,我们筛选出39篇相关文献。根据这些文献的分析,我们将现有的研究分为两类:一类是比较不同温度或臭氧条件下健康效应的方向和强度,探索是否存在交互作用,共计纳入27项研究;另一类是通过引入温度和臭氧的交互作用项来量化这种协同效应,共计纳入12项研究。

## 2.1 不同温度/臭氧分层下健康风险存在差异

一项荟萃分析针对不同温度下臭氧与死亡风险的关系,发现在低温和高温环境下,臭氧均显著影响非意外死亡,臭氧暴露量每增加 $10 \mu\text{g}/\text{m}^3$ ,其合并效应分别为0.48%(95%置信区间:0.28%, 0.69%)和0.47%(0.32%, 0.63%)<sup>[44]</sup>。特别是在高温条件下,臭氧与心血管疾病死亡风险增加间的关联更为显著,合并结果为1.63%(1.14%, 2.13%),这一数值显著高于常温条件下0.27%(0.03%, 0.51%)增加<sup>[44]</sup>。在高温环境中,臭氧对呼吸系统疾病死亡的影响相对稳定,死亡风险的合并比值比(odds ratio, OR)为1.006(1.001, 1.012)。然而,在低温分层下,臭氧对呼吸系统健康的影响则表现出不一致性<sup>[45]</sup>。

表S1总结了现有的根据分层方法探讨温度与臭氧交互作用的流行病学研究。目前,相关研究已得到较为一致的结论,即相对于高温和臭氧的单独暴露,二者的

复合暴露会导致健康效应的显著增强。例如,Qian等人<sup>[46]</sup>在中国武汉开展的时间序列研究显示,臭氧与极端高温对非意外死亡率的交互作用具有统计学意义,该结果在Pascal等人<sup>[47]</sup>和Sartor等人<sup>[48,49]</sup>在欧洲开展的研究中得到了印证。同时,Zhang等人<sup>[50]</sup>通过一项包含我国13城市的研究发现,高温可以显著加强臭氧与心血管死亡的关联。Shi等人<sup>[51]</sup>在我国128个县以及Zhang等人<sup>[52,53]</sup>在成都开展的时间序列研究均发现,高温分层下,臭氧相关的非意外死亡、呼吸系统和心血管系统疾病死亡率进一步增加。

然而,Pattenden等人<sup>[54]</sup>对英国15城市不同温度分层下臭氧的全因死亡和死因别死亡的分析结果发现,臭氧和高温的协同效应仅在伦敦显著。一项对于美国60个社区的研究表明,尽管温度修饰了臭氧暴露与死亡率间的关联,但这种修饰作用在不同区域存在差异,在东北地区,臭氧与死亡的关联强度随温度升高而增强,但在东南地区该变化模式并不显著<sup>[55]</sup>。Jhun等人<sup>[56]</sup>纳入了美国97个社区的研究也未发现臭氧与温度对非意外死亡风险的协同作用。Lin和Liao<sup>[57]</sup>在中国台湾高雄开展的时间序列研究发现,暖日臭氧暴露与死亡风险呈现负相关。研究结果的差异可能归因于两个主要因素:首先,不同研究区域的污染物日均水平和气候条件存在差异;其次,许多现有研究采用的是人群水平的生态学研究,生态学谬误的存在可能增加了暴露评估的不确定性,从而导致暴露错分。

两项多中心研究发现,无论温度高于或低于舒适温度,臭氧暴露导致的死亡风险均会增加。Ren等人的研究纳入了美国95座城市,发现臭氧浓度每增加10 ppb,心血管疾病死亡率在不同温度水平下均呈现显著升高,低温下增加0.41%,中等温度下增加0.27%,高温下增加1.68%,提示当温度偏离舒适范围时,臭氧的危害增加。在对京津冀及周边地区39个县的研究中发现,无论是极端高温还是低温均会增强臭氧对于死亡的急性效应<sup>[59]</sup>。

某些研究特别指出,在较低温度的环境下,臭氧对健康的不良影响更显著。例如,一项在苏州的研究发现,在寒冷季节,臭氧对总死亡率和心血管死亡率的影响超过了温暖季节<sup>[60]</sup>。同样,Cheng和Kan<sup>[61]</sup>在上海的研究以及Liu等人<sup>[62]</sup>在广州的研究均发现,低温条件下臭氧暴露健康风险显著增加,而这种效应在高温环境下则不那么明显。

值得注意的是,臭氧与低温交互作用对健康的影

响主要在气候较为温暖的地区(例如中国的广州、上海和苏州)得到证实。这一趋势可能与不同区域人群对温度的适应性有关：习惯于温暖气候的人在遭遇低温时可能更敏感。另一方面可能是地域生活习惯的差异导致的暴露错分。由于臭氧是一种反应活性强的空气污染物，其室内浓度通常显著低于室外浓度，室内外比值介于0.1~0.4<sup>[63]</sup>。在气候温暖的城市，尽管夏季室外臭氧浓度可能更高，但居民可能由于室内温度调节而倾向关闭门窗，这样减少了臭氧暴露的机会。在寒冷季节，由于缺乏足够的取暖措施，这些地区的居民可能更容易受到低温和臭氧的共同影响。多中心研究观察到低温和臭氧的协同作用<sup>[58,59]</sup>，这表明该效应可能是真实存在的，而不仅仅是由于暴露偏倚导致。有研究显示，老年人对这种复合暴露更加易感<sup>[64]</sup>。因此，未来的研究需要深入探讨这一协同作用的真实性，以便更准确地进行因果推断，保护易感人群。

除了温度分层的考虑，部分研究还根据臭氧浓度的不同水平进行分层，探讨温度在不同臭氧浓度水平下对健康的影响。最新发布的一项荟萃分析显示，在高臭氧水平下，温度对全因和非意外死亡的超额风险分别为7.6%(6.3%，9.0%)和12.5%(4.7%，20.9%)，而在低臭氧水平下，则分别为5.1%(3.9%，6.3%)和3.6%(0.1%，7.2%)，表明高臭氧水平显著增强了温度相关死亡的风险<sup>[65]</sup>。然而，该研究并未观察到臭氧在极端天气事件，如热浪或寒潮中的健康效应。

全球范围的多中心研究揭示了臭氧水平上升显著改变了高温对心血管和呼吸系统死亡率的影响。在低、中、高三个不同的臭氧暴露水平下，心血管死亡率分别增加 1.6%(1.5%，1.6%)、5.1%(5.1%，5.2%)和 8.7%(8.7%，8.8%)<sup>[66]</sup>。Analitis等人<sup>[67]</sup>在欧洲九座城市开展的研究发现，考虑臭氧暴露后，热浪相关死亡风险降低了15%~30%，热浪期间高臭氧暴露比低臭氧暴露水平下每日死亡率增加54%，且这种交互作用在75至84岁人群中最为显著。在臭氧浓度较低的环境中，气温每升高1°C，日总死亡人数增加1.84%，而在高臭氧环境下，这一比例进一步增至2.20%<sup>[68]</sup>。De Sario<sup>[69]</sup>、Scortichini<sup>[70]</sup>以及Ren等人<sup>[71]</sup>在各自的多中心研究中均发现，高臭氧水平增强了温度与死亡率之间的相关性。

同时，有研究按照污染物和温度层次进行分析。Pascal等人<sup>[72]</sup>和Fu<sup>[73]</sup>的研究分别在法国17城市和中国石家庄发现，在臭氧浓度较高的日子里，高温与死亡风险的关联更强。在欧洲的另一项研究中也得到了类似

的结论，即在臭氧水平较高的情况下，高温显著加剧了臭氧对总自然死亡的影响<sup>[74]</sup>。

总体而言，现有的基于温度与臭氧分层的研究为温度和臭氧对健康协同作用提供了支持性证据，即在高温或高臭氧条件下，不良健康结局的发生频率更高。低温条件下臭氧对健康影响的研究证据相对有限，主要集中于较温暖地区，但这些发现的有效性可能受到暴露错分的影响，未来的研究需要进一步深入分析和论证这些发现背后的因果关系。

## 2.2 温度与臭氧协同健康影响的定性定量评价

在分层分析的研究中已经发现，温度与臭氧的复合暴露可能对健康具有协同作用，但缺乏相应的量化评价。通过对这一过程的定量分析，我们能够更深入理解两者复合暴露带来的健康风险进而明确其对公共健康的影响。这不仅有助于明确哪些群体最为易感，同时为建立预警系统提供科学依据。

表S2对当前通过建立温度和臭氧交互作用项方法探究其协同健康效应的流行病学研究进行了总结。在相关研究中，臭氧与高温复合暴露对健康的协同效应获得了广泛且一致的证据支持。例如，Dear等人<sup>[75]</sup>对2003年欧洲夏季的持续热浪进行的分析揭示了温度和臭氧之间的显著交互作用。在意大利热那亚，Parodi等人<sup>[76]</sup>也发现了类似的结论，在温暖的季节，臭氧暴露对次日死亡率和心血管系统死亡率的影响增强，特别是在老年群体中。Burkart等人<sup>[77]</sup>在德国柏林和葡萄牙本利斯的研究通过引入交互项的模型证实，高水平臭氧环境下，高温的影响显著加剧。东北亚16座城市的时间序列研究发现，臭氧与高温复合暴露对心肺疾病死亡的协同作用显著，其超额风险(relative excess risk due to interaction，RERI)为1.9%<sup>[78]</sup>。在上海浦东区，夏季臭氧浓度升高10 μg/m<sup>3</sup>，慢性阻塞性肺疾病(Chronic Obstructive Pulmonary Disease, COPD)死亡风险(Risk Ratio, RR)为1.0173；臭氧对COPD死亡的影响在热浪期间较非热浪时期显著增强<sup>[79]</sup>。Zong等人<sup>[80]</sup>通过将文献中获取的温度和臭氧对于死亡率作用的系数纳入模型，分离出热浪和臭氧对于死亡率的协同效应为12.31%。Filleul等人<sup>[81]</sup>发现臭氧与高温对死亡率表现出正向协同作用，但联合作用大小表现出地区差异，从勒阿弗尔的10.58%增加到巴黎的174.68%。这种地区差异在其他多中心研究中也有所体现，在美国，北方城市协同效应往往更加明显<sup>[82,83]</sup>。一项针对英国15城市的研究，在伦

敦和卡迪夫两个城市的单独效应中发现了显著的正向协同作用，但在其他13个城市中和合并15个城市后，这种效应并不显著<sup>[54]</sup>。

我国几项对于单一城市的研究也有类似发现。在苏州的一项响应面分析结果显示，臭氧和平均温度的张量积平滑项具有统计学意义( $P<0.050$ )，提示臭氧与总死亡率和心血管疾病死亡率之间可能存在交互作用。一项在广州越秀、荔湾两区针对非意外死亡开展的时间序列研究发现，在寒冷季节或者日均气温低于25分位时，死亡风险受到臭氧与日平均气温交互作用的影响<sup>[62]</sup>。Xie等人<sup>[84]</sup>在我国广州开展的时间序列研究结果显示，高温与高浓度臭氧对冠心病死亡风险存在协同放大作用，RERI为0.103。

综上所述，臭氧与温度的复合暴露对公共健康构成重要威胁<sup>[78~81]</sup>，尤其在气候变化的背景下。未来的研究需要着重于这种交互作用的深入分析和量化评估，以便更好的理解其对人群健康的影响，为制定有效的预防措施和政策提供科学依据。

### 3 温度和臭氧协同健康作用的潜在生物学机制

臭氧与温度可能通过多种方式互相作用，产生共同的健康影响。包括环境臭氧浓度和温度同时升高导致的臭氧暴露水平增加，以及二者相互关联的生物学机制。例如，夏季热浪可能导致空气停滞，使得臭氧等污染物在光化学反应的作用下积累增多<sup>[85]</sup>。在温暖的季节，人们可能会因为户外活动增加或者开窗通风，而更多地暴露于高温和高臭氧环境下，特别是对于易感人群<sup>[42]</sup>。另外，当环境温度偏离人体舒适区间时，人体会激活体温调节机制，如通过调整皮肤毛孔舒缩和调整换气频率来适应，这也可能改变污染物吸入量。此外，在热应激条件下，机体的体温调节反应会降低应对环境风险因素的适应能力，高活性的臭氧分子对呼吸道造成的氧化损伤可能降低机体对于呼吸道感染的抵抗力，进一步加剧高温的不良健康效应。臭氧和高温还可以通过激活相同的生物学路径，如炎症反应，共同造成不良健康影响<sup>[86~88]</sup>。

### 参考文献

目前，仅有两项流行病学研究专门探讨了臭氧和温度复合暴露对健康的潜在影响。Ren等人<sup>[89]</sup>开展的队列研究显示，在臭氧浓度较高的环境下，暖季温度与心率变异性之间的相关性显著增强，且不受PM<sub>2.5</sub>水平的影响，提示高温和臭氧复合暴露可能通过自主神经系统影响心血管健康。另一项纳入了16名年龄在21~36岁之间健康志愿者的随机交叉对照研究显示<sup>[90]</sup>，臭氧诱导的全身效应随环境温度的变化而有所不同，尽管对呼吸功能的直接影响不明显，但在中等温度下，臭氧可能激活纤溶途径，在高温下则可能干扰纤溶系统的正常功能。这些结果为理解温度和臭氧如何通过相互作用影响人群健康提供了生物学基础。

### 4 总结与展望

在全球变暖的背景下，环境臭氧浓度和温度间存在着重要交互作用，尤其在人为排放较为严重地区，这种交互作用将加剧暖季臭氧污染。未来人们经历不适宜温度和臭氧污染复合暴露的频率和强度会进一步增加。现有的研究通过分析不同臭氧和温度分层下的健康效应变化，以及创建交互效应模型，来探讨二者的协同作用。研究普遍表明，不适宜温度与臭氧复合暴露可能进一步增加人群死亡风险，高温环境下的研究证据最为充分，而低温与臭氧复合暴露的协同作用还存在较大的不确定性。心肺疾病患者和老年人是这种复合暴露的潜在易感人群。然而，目前对于这种协同作用超额风险的定量研究证据比较有限，且异质性较强，使得风险评估面临较高的不确定性。因此，仍需要进行更多高质量的流行病学研究来弥补现有知识的不足。考虑到空间和时间的巨大差异，精细化设计的多中心流行病学研究将是探索臭氧和温度对健康协同影响的有效手段。全球范围内不同的暴露条件为我们提供了独特的机会来详细研究温度和臭氧污染之间的相互作用。这些研究不仅可以推动减缓气候变化和改善城市空气质量的努力，而且对于理解气候变化如何影响空气污染、温度及其综合健康影响至关重要，对我们适应未来气候变化具有重要意义。

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## 补充材料

表S1 分层分析探讨温度和臭氧联合健康效应的流行病学研究总结

表S2 交互作用探讨温度和臭氧联合健康效应的文献总结

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Summary for “臭氧和温度交互作用及其协同健康影响研究进展”

# Advances in ozone and temperature interactions and their synergistic health effects

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Climate change is one of the most formidable health challenges of the 21st century. Human activities, particularly the combustion of fossil fuels, contribute significantly to both ozone pollution and global warming. These phenomena not only originate from the same sources but also interact dynamically. The “climate penalty” effect, which is associated with global warming, is expected to increase ozone levels in regions with substantial human-generated emissions. Moreover, ozone exacerbates atmospheric warming as a greenhouse gas due to its positive radiative forcing. The concurrent exposure to increased temperatures and heightened ozone pollution represents one of the most prevalent combined environmental threats currently. As climate change progresses, the frequency and severity of simultaneous exposure to elevated temperatures and increased ozone levels are expected to rise, posing significant health challenges both now and in the future. Therefore, there is an urgent need for research to determine whether this dual exposure contributes to additional health risks. To enhance our understanding of the health impacts of combined ozone and temperature exposures, we conducted a comprehensive review of epidemiological studies from SCI-indexed journals dating back to 1990, ultimately including 39 studies in our review. Recent epidemiologic research focusing on this combined effect has primarily used stratified analyses and interaction terms to explore their synergistic effects.

In studies stratified by temperature or ozone, the strongest evidence emerged from the simultaneous exposure to both ozone and elevated temperature, indicating an increased risk of mortality. However, the health consequences of combined exposure to lower temperatures and ozone remain less understood. Variability in exposure assessment and population adaptation to climate contribute significantly to the heterogeneity of research findings. Notably, two multicenter studies have observed an increased risk of ozone-related mortality when temperatures deviate from comfortable levels, no matter higher or lower. Particularly vulnerable groups include individuals with cardiovascular and respiratory conditions, and the elderly, who face heightened mortality risks from this dual exposure.

Efforts have been made to assess the synergistic health impacts of ozone and temperature both qualitatively and quantitatively by establishing interaction terms. The synergistic health effects of combined exposure to ozone and heat are supported by extensive and consistent evidence. Research indicates that the excess mortality risk due to co-exposure to ozone and temperature ranges from 1.73% to 12.31%. Nevertheless, the full extent of this risk has yet to be definitively established, highlighting the ongoing need for further investigation.

Ozone and temperature may interact in multiple ways to produce shared health effects. The mechanisms underpinning the synergistic health impacts include higher ozone exposure resulting from the simultaneous occurrence of elevated ambient ozone concentrations and temperatures, along with interconnected biological mechanisms. To date, only two studies have explored the biological mechanisms behind these synergistic effects. Combined exposure to high temperatures and ozone may impact cardiovascular health by disrupting the normal functioning of the autonomic nervous system and the fibrinolytic system.

Addressing the synergistic health effects of temperature and ozone is critical for accurately assessing health risks in future climate change scenarios. Well-designed multicenter epidemiological studies offer a powerful means to further explore the synergistic health effects of ozone and temperature. The varied exposure conditions around the world provide a unique opportunity to examine the interactions between temperature and ozone pollution in depth. This will play a crucial role in driving climate change mitigation and improving urban air quality, which are essential for adapting to future climate changes.

**temperature, climate change, ozone, synergistic health effects, mortality**

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