UNITED STATES OF AMERICA
BEFORE THE
CONSUMER PRODUCT SAFETY COMMISSION

Consumer Product Safety Commission: Request for Information on Chronic Hazards Associated with Gas Ranges and Proposed Solutions	**)**	**CPSC-2023-0009**

RESPONSE OF THE AMERICAN GAS ASSOCIATION TO REQUEST FOR INFORMATION

Dated: May 8, 2023

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The American Gas Association submits these comments on the U.S. Consumer Product Safety Commission's ("CPSC" or "Commission") notice of its *Request for Information on Chronic Hazards Associated with Gas Ranges and Proposed Solutions* (the "CPSC RFI" or "RFI").[1] These comments are responsive to the questions the CPSC posed in the RFI and provide the Commission with objective, data driven analysis and studies to assist it in its investigation of whether there are any chronic hazards associated with gas stoves.

In addition to this filing with an exhibit provided electronically via regulation.gov, the American Gas Association is submitting the documents listed in the appendix by uploading them to regulations.gov in separate batches to meet the file size requirements.

I. THE INTEREST OF THE AMERICAN GAS ASSOCIATION

The American Gas Association ("AGA"), founded in 1918, represents more than 200 local energy companies that help deliver clean natural gas throughout the United States. There are more than 78 million residential, commercial, and industrial natural gas consumers in the U.S., of which 95 percent — more than 73 million consumers — receive their gas through AGA members. AGA is an advocate for natural gas utility companies and their customers and provides a broad range of programs and services for its member natural gas pipelines, marketers, gatherers, international natural gas companies, and industry associates. Today, natural gas meets more than one-third of the United States' energy needs.[2] AGA's members serve commercial consumers and residential consumers, the majority of which use natural gas cooking appliances, and the AGA therefore has a direct and vital interest in this proceeding.

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[1] CPSC-2023-0009, *Request for Information on Chronic Hazards Associated with Gas Ranges and Proposed Solutions*, 88 Fed. Reg. 14150 (March 7, 2023).

[2] For more information, please visit www.aga.org.

A. AGA and its Members Have a Long History of Investing In and Prioritizing Safety

Nearly 187 million Americans and 5.5 million businesses use natural gas because it is affordable, reliable, safe and essential to protecting the environment. America's natural gas utilities are innovative and committed to the safety of natural gas consumers and the public through new and modernized infrastructure and advanced technologies that maintain reliable, resilient, affordable, and safe delivery of natural gas.

The natural gas distribution system is both safe and efficient. According to the Department of Transportation's Pipeline and Hazardous Materials Safety Administration, the federal regulator responsible for developing and enforcing pipeline safety regulations, pipelines are the safest, most environmentally-friendly, and most efficient and reliable mode of transportation for gas.[3] Not only are gas pipelines a safe way to deliver energy, they are also an extremely efficient method of delivering energy to customers, with 92 percent efficiency. As little as .1 percent of the natural gas delivered nationwide is lost to fugitive emissions from local distribution systems. These safety and environmental metrics will only improve as natural gas utilities continue to spend billions of dollars each year on enhancing the safety and efficiency of natural gas distribution and transmission systems.

As for natural gas appliances, AGA and its members have been at the forefront of the development of safety standards and installation codes for more than half a century – since before the creation of the Consumer Product Safety Commission. Today, they are instrumental in supporting the continuing development of appliance safety standards and installation codes by national consensus standard making bodies. These voluntary standards are widely adopted

[3] *See*, https://primis.phmsa.dot.gov/comm/SafetyStandards.htm.

throughout North America and help ensure that the gas appliances and equipment available to and installed for consumers are safe, reliable, and efficient.

In the 1970s, AGA and its members supported voluntary consensus standards for appliance safety accredited by the American National Standards Institute ("ANSI"), including the design performance standard for Household Cooking Gas Appliances, ANSI Z21.1-2018/CSA 1.1-2018. The ANSI design standards are rigorous, and in the case of natural gas cooking appliances, products cannot be certified until they are evaluated by an independent third-party testing agency, and even then, products continue to be tested annually on a random basis to ensure continued compliance. Many state and local authorities require ANSI-certified gas appliances.

The ANSI standards committees that develop and maintain these voluntary standards for gas appliances comprise a broad cross-section of representatives from private and public entities, including consumers, manufacturers, utilities, installers, government agencies, testing laboratories, etc. AGA in fact served as an early administrator of the ANSI standards development process, which includes provisions that require an open and transparent development process.

AGA is also the cosponsor of the two main natural gas appliance installation codes adopted in the United States, the International Fuel Gas Code (IFGC) and the National Fuel Gas Code (NFGC) also known as ANSI 223.1, NFPA 54. Both installation codes were developed using a consensus-based approach in an open and transparent process. These installation codes help ensure natural gas appliances are installed safely and properly.

In addition, AGA has an extensive history of participating with the CPSC on consumer education programs and voluntary standards development on related products. AGA and its members have worked closely with the CPSC staff in developing consumer safety messages to

minimize potential problems with the operation of both natural gas and electric appliances. The AGA also has participated in standards development for products such as carbon monoxide alarms to ensure consistent performance and compliance with safety standards.

This history demonstrates that AGA and the natural gas industry support appliance safety standards and installation codes based on objective scientific and technical information and an open and transparent process – and that voluntary consensus standards such as those described above are both possible and effective. Indeed, CPSC staff have actively participated in the development of these voluntary standards.

B. Current Voluntary Standards Development Efforts

Currently, AGA staff is participating in the CPSC-led effort that started in 2021 and the CPSC titled the "Indoor Air Quality associated with Gas Ranges Working Group". It was created to examine the relationship between gas ranges and indoor air quality (IAQ). The CPSC established three Task Groups under the Working Group that have each been assigned discrete tasks related to existing industry codes and standards for gas ranges, air concentration guidelines for certain identified emissions from cooking activities, and test procedures for evaluating emissions from gas ranges in light of potential air concentrations. These three task groups, which have a range of participants with different backgrounds and interests, have been meeting regularly over the past two years and have made substantial progress in collecting and analyzing information.

Task Group #1 was assigned the task of identifying industry codes and standards that could be affected by new IAQ testing requirements for ranges. The task group reviewed both national and international standards for gas ranges and their components. The task group determined that

the critical standard that could be affected by IAQ testing requirements is CSA/ANSI Z21.1 • CSA 1.1, *Household Gas Cooking Appliances*.

Task Group #2 was tasked with determining air concentrations guidelines for the three identified emissions: CO, NO_2 and $PM_{2.5}$. Air concentration guidelines from the United States Environmental Protection Agency ("USEPA"), the World Health Organization, Health Canada, and other sources have been reviewed to help establish appropriate indoor air concentrations guidelines.

Task Group #3 was tasked with developing laboratory test procedures/methods to determine how a gas cooking range will be tested to determine if the emissions typically produced by the gas appliance itself (as opposed to cooking activities) result in air concentrations that remain below the guideline levels identified by Task Group #2. AGA staff chairs this working group.

AGA supports this voluntary effort to obtain and analyze data and information that can be useful in future standards development proceedings. AGA appreciates the efforts of the CPSC to evaluate any potential consumer safety or health risks related to gas range use in the current Request for Information, but suggests that it may be more appropriate at this stage to let the CPSC's "Indoor Air Quality associated with Gas Ranges Working Group" and the voluntary standard setting process run its course. Regardless, the AGA believes that an objective scientific evaluation and an open process are critical components of any evaluation and standard development.

II. COMMENTS

The CPSC has called for information relating to "chronic hazards associated with gas ranges and proposed solutions" but fails to identify any chronic hazards let alone support for such claim. The CPSC's characterization presumes that there are "chronic hazards" that need to be

addressed. In reality, the core underlying question of whether or not "chronic hazards" are associated with gas ranges has not been answered.

There have been decades of research examining the association of gas ranges with health outcomes, with the main focus on respiratory illnesses. Similarly, there is extensive scientific literature examining specific health outcomes (*e.g.*, asthma) with the goal of identifying triggers or root causes. Moreover, government agencies and independent panels of experts have also reviewed the information on gas range use and health effects over the years. These groups have not reached any conclusions that gas range use causes any adverse health effects.

The CPSC should evaluate recent claims that gas range use is associated with chronic health hazards with the current studies' relevance or limitations in mind. Recent studies purporting to raise concerns about gas ranges and health outcomes cannot be evaluated in isolation, but must be systematically and rigorously evaluated in the context of the overall body of scientific evidence, including consensus public health literature. For the CPSC to determine that there are "chronic health hazards associated with gas ranges," the CPSC should identify where earlier examinations of these issues, including by the CPSC itself, were insufficient or flawed. In short, any consideration of new evidence must be conducted through a process of weighing and evaluating scientific evidence through systematic review from experts across multiple disciplines within a transparent and iterative process.

The AGA respectfully submits that the available body of scientific research does not provide sufficient or consistent evidence demonstrating that there are chronic hazards from gas ranges, *i.e.*, that pollutant emissions associated with gas ranges adversely affect human health, and that potential hazards from gas ranges are meaningfully different from those associated with

appliance alternatives (*e.g.*, electric ranges). To demonstrate hazards from gas ranges, there must be consistent, validated evidence on the emissions from gas ranges (differentiated from the process of cooking), how those emissions would typically impact indoor air quality, and whether there are any adverse health effects from indoor air exposures from gas ranges (differentiated from other indoor sources). There is a paucity of data on some of these issues. On others, available studies are highly heterogeneous and inconsistent. In other cases, the data that has been collected and validated is not sufficient to demonstrate hazards from gas ranges. In other words, despite decades of examination of these topics, there is still insufficient evidence to draw the conclusion that there are hazards from gas range use. Notably, no studies or reports have demonstrated a causal association between gas cooking and childhood asthma or wheeze, which are the most studied adverse health effects – nor have reports provided adequate evidence for any other adverse health effect.

For emission rates of some potential pollutants, for example, there are only limited data, especially for currently available ranges and cooktops. To be sure, gas ranges produce combustion emissions of recognized pollutants, some of which are not emitted by electric ranges. Overall, the data on typical emissions of these pollutants are limited, and sampling methods and equipment used vary in reliability and accuracy. Potential chronic exposures, however, are heavily mitigated by the relatively short duration of cooking typically performed in residences. The typical range or cooktop is off and not in use more than 95 percent of the time in typical residences. Furthermore, the impact of any emissions on indoor air quality – both in the kitchens where cooking takes place as well as elsewhere in the residences – appears to be highly dependent on whether exhaust hoods and vent fans are employed and other factors such as size of the indoor space, open versus closed construction designs, and building ventilation.

Likewise, there are only limited data on measured indoor air concentrations associated with the use of natural gas cooking appliances. These studies, however, show the benefits of ventilation and that using ventilation maintains levels of pollutants below health-based guidelines. Pollutant air concentrations from gas range emissions have also been modeled, but the typically conservative assumptions built into such modeling make it unlikely that those results are representative of real-world indoor air concentrations for most settings. Indoor air quality is highly individual and variable. It depends on factors such as outdoor air, building type and size, layout, natural and mechanical ventilation, indoor air pollution sources such as cleaning products, pet dander, dust, furniture and flooring, and individual behaviors (such as smoking). In modeling indoor air concentrations, moreover, the assumptions used in the model – such as contributions from outdoor air, the time spent cooking, air exchange rate, room size, and decay rate of various substances – can significantly affect the results.

Also, it must be demonstrated that indoor air concentrations are likely to lead to exposures that would result in adverse health effects. There are substances in the environment around us all the time, but whether they are a hazard to human health depends on the dose (the level, duration, and frequency of exposure) and toxicity (at what dose could adverse health effects occur) of the substance. Indeed, a recent comprehensive examination of the existing literature of epidemiology studies on the relationship between gas cooking and potential adverse health effects (*Li et al.*, 2023, attached as an Exhibit to these comments) concludes that the evidence does not demonstrate any strong, consistent association between gas cooking and childhood asthma or wheeze, the most studied respiratory effects to date. Available studies have shown inconsistent results, and most have null findings, *i.e.*, they do not find statistically significant associations between gas cooking and childhood asthma or wheeze. Given the high heterogeneity of studies, their generally low

quality (*e.g.*, failure to account for key confounding factors such as environmental tobacco smoke; reliance on self-reporting for exposures or outcomes), and the fact that most studies are not designed to examine effects over time (temporality), there is no scientific basis to infer causation.

Claims that children in homes with gas stoves have an increased risk of asthma symptoms frequently reference a 2013 "meta-analysis" of literature by *Lin et al.*, which analyzed studies that for the most part used self-reported information on gas appliance use as a proxy for exposure and not objective measurements of usage or exposure. Reliance on that analysis to demonstrate chronic hazards is misplaced – that report only found an association, and the underlying studies on which it relied as well as studies since that time do not support a causal conclusion, as explained more below. In short, there is no demonstrated health effect caused by the presence of natural gas cooking appliances in homes.

In sum, available data do not establish a "chronic hazard" associated with gas ranges, and thus there is no need for a "solution." As discussed more below (see responses to Questions 1(b) and 1(e)), studies have consistently shown the benefits of ventilation for reducing emissions associated with *cooking activities* and for the overall improvement of indoor air quality, and education relating to ventilation *for all stove types* (gas and electric) could be beneficial.

Below, the AGA addresses specific questions posed by the CPSC in the RFI, recognizing that many of these responses are informed by the overarching issues discussed above.

Question 1: Please provide information related to the scope and scale of potential chronic chemical hazards, exposures, and risks associated with gas range use.

(a) Please provide information related to habits and practices surrounding consumers' use of gas ranges in residential settings. How many U.S. homes have gas ranges? What is the duration of gas range usage per cooking event, per day (*i.e.*, minutes to hours)? How many days per week/year is a gas range used? Provide information on infrequent, high-end usage events such as using the oven-cleaning cycle or preparing for large holiday gatherings.

Response to Question 1(a):

According to the 2020 Residential Energy Consumption Survey,[4] about one-third of the residences in the U.S. have natural gas-fired appliances in their kitchen. There are a reported 34.67 million ranges (units with both an oven and a cooktop), 7.61 million separate cooktops, and 2.84 million separate wall ovens that use natural gas fuel. Most of these appliances are located in single-family detached buildings (63% of the ranges, 89% of the cooktops, and 70% of the ovens).[5]

As for daily or weekly durations of cooking with gas ranges, relevant information is available from a couple of sources. Data on participation in general cooking-related activities (time spent preparing and cleaning up food, cooking food, cleaning dishes) and time spent in the kitchen generally – but not specifically in relation to gas range use – have been compiled and analyzed by the U.S. Environmental Protection Agency (USEPA) and published in their Exposure Factors Handbook.[6] The cooking-related data in the Exposure Factors Handbook were obtained by the USEPA from the National Human Activity Pattern Survey, which provides detailed information about typical activities, often with a breakdown by age, race, day of the week, etc. Key applicable

[4] Residential Energy Consumption Survey (RECS), 2020 RECS Survey Data. U.S. Energy Information Administration. https://www.eia.gov/consumption/residential/data/2020/.

[5] Residential Energy Consumption Survey (RECS), 2020 RECS Survey Data. U.S. Energy Information Administration. https://www.eia.gov/consumption/residential/data/2020/.

[6] U.S. Environmental Protection Agency (USEPA), 2011. Exposure Factors Handbook, Chapter 16 – Activity Factors. EPA/600/R-09/052F. USEPA, ORD, Washington, DC. October.

information on time spent each day on various cooking-related activities, by age group, is summarized in the Table below for the 50th and 90th percentiles. Examining these compiled statistics indicates that time spent cooking with gas per day is, on average, approximately 15 to 20 minutes. For example, the Table below shows adults (aged 18 to 64) spend 1.0 (50th percentile) to 3.3 hours-per-day (90th percentile) in the kitchen, but more than 85 percent of that time appears to be spent in food preparation and post-cooking clean-up activities. This means that well below an hour each day is spent on actual cooking. Confirming this point, additional statistics indicate time spent cooking by adults (aged 18 to 64) ranges from 18- to 43-minutes-per-day on average, but no more than 20-minutes-per-day are spent near an open flame (*e.g.*, a natural gas-fired cooktop) or near food while fried, grilled, or barbequed. Based on this data, **15 to 20 minutes per day** is a reasonable estimate of the average time spent cooking with gas daily.

The reasonableness of this estimate is confirmed by a web-based cooking survey coordinated by Lawrence Berkeley National Laboratory (LBNL) and completed in 2010/2011 by predominantly California residents on the duration of cooking-related activities, including the use of gas ranges.[7] Given the low participation rate of the survey (372 respondents), there is some uncertainty as to the representativeness of the results for larger populations compared to the data from the National Human Activity Pattern Survey, which represents nearly 10,000 respondents.[8] Nonetheless, the LBNL survey results for cooking durations per day, based on responses to the duration of cooktop and oven use for breakfast, lunch, dinner, and other cooking "within the past 24 hours," are generally consistent with the data discussed above from the Exposure Factors

[7] Klug, V.L., Lobscheid, A.B. and Singer, B.C., 2011. Cooking appliance use in California homes – data collected from a web-based survey. Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-5028E. August.
[8] USEPA, 2011. Exposure Factors Handbook, Chapter 16 – Activity Factors. EPA/600/R-09/052F. USEPA, ORD, Washington, DC. October.

Handbook.[9] Notably, most respondents indicated use of cooktops of 16 to 30 minutes for the cooking of dinners, with cooktops predominantly not used for the cooking of breakfasts, lunches, and "other cooking." Similarly, over 90 percent of respondents indicated ovens were not used for the cooking of breakfasts, lunches, or other cooking within the past 24 hours, and "zero minutes" was the most common response to the duration of oven use for dinners (45 percent of respondents); the most common non-zero response for oven use for cooking of dinners was 16 to 30 minutes.

The LBNL survey also compiled data on the number of days per week cooktops were used, which indicated most respondents use their cooktops 5 to 6 days per week for the cooking of dinners, but only 1 to 2 days per week for the cooking of breakfasts, lunches, or "other cooking." Compiled data on the number of days per week ovens were used indicated most respondents use their ovens for cooking dinners 1 to 2 days per week, but "never" was the most common response for cooking breakfasts, lunches, or "other cooking." Based on the LBNL survey, **5 to 6 days per week** is a reasonable estimate of the number of days per week spent cooking with gas.

No sources were identified that provided compiled data on the frequency or duration of high-end usage events such as the oven-cleaning cycle or preparing for large holiday gatherings.

[9] The latest complete edition of the Exposure Factors Handbook was released in 2011. Since October 2017, the USEPA has released updates to chapters individually as new data becomes available, but Chapter 16 on "Activity Factors," from which the information presented in this response was obtained, has not been updated since October 2011.

Time Spent in Various Activities (adapted from USEPA, 2011)

Activity	Age Range	Time Spent (minutes-per-day)	
		50th Percentile	90th Percentile
In kitchen	3 to <6	30	105
	6 to <11	30	105
	11 to <16	24	90
	16 to <21	15	90
	18 to 64	60	200
Food Preparation	18 to 64	35	110
Food Clean-up	18 to 64	30	60
Dishes/Laundry	18 to 64	30	150
Cooking	12 to 17	11	NR
	18 to 24	18	NR
	24 to 44	38	NR
	45 to 64	43	NR
	65 and over	50	NR
Near Food while Fried, Grilled or Barbequed	1 to 4	20	45
	5 to 11	15	60
	12 to 17	20	60
	18 to 64	20	120
	>64	20	60
Near Open Flames	1 to 4	10	121
	5 to 11	15	90
	12 to 17	23	60
	18 to 64	20	121
	>64	17	120

NR = not reported; > = greater than; < = less than

(b) Please provide information on related (non-cookware) products used in conjunction with gas ranges and their frequency of use. Examples of related products of interest are range hoods (in general), CO alarms, smoke alarms, chemical sensors other than CO, portable air filters, etc.

Response to Question 1(b):

It is well-known that ventilation plays an important role in mitigating indoor air emissions associated with cooking activities, including both gas combustion-related emissions and cooking-related emissions.

Measurement-based studies, conducted in the laboratory[10,11,12] as well as residential settings[13,14] by researchers at Lawrence Berkeley National Laboratory, Health Canada, and National Research Council Canada, demonstrate the effectiveness of vented kitchen range exhaust for reducing both kitchen and whole-house air concentrations of pollutants. Modeling-based exposure simulations conducted by the Lawrence Berkeley National Laboratory (LBNL, 2020), which were funded by the California Energy Commission, also demonstrate that effective kitchen exhaust ventilation, and specifically outdoor-vented kitchen range hoods, can significantly reduce indoor air concentrations of NO_2 and PM associated with gas-fired kitchen appliances and cooking

[10] Singer BC; Sherman AD; Hotchi T; Sullivan DP. 2011. "Pollutant Removal Efficiency of Residential Cooking Exhaust Hoods." LBNL-4902E. Lawrence Berkeley National Laboratory, Berkeley, CA.

[11] Delp WW; Singer BC. 2012. "Performance Assessment of U.S. Residential Cooking Exhaust Hoods." LBNL-5545E. Lawrence Berkeley National Laboratory, Berkeley, CA.

[12] Dobbin, NA; Sun, L; Wallace, L; Kulka, R; You, H; Shin, T; Aubin, D; St-Jean, M; Singer, B.C. 2018. "The benefit of kitchen exhaust fan use after cooking – An experimental assessment." Building and Environment. Vol. 135, pp286-296.

[13] Singer BC; Delp WW; Price PN; Apte MG. 2012. "Performance of Installed Cooking Exhaust Devices." LBNL-5265E-r1(3). Lawrence Berkeley National Laboratory, Berkeley, CA.

[14] Singer, BC; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2016. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-1006385. October. *subsequently published as* Singer, BC; Pass, RZ; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2017. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Building and Environment. 122:215-229. doi: 10.1016/j.buildenv.2017.06.021.

activities.[15] The performance of range hoods can vary depending on device design, exhaust air flow rate, and placement over burners. Some key points from these studies of kitchen range hoods include:

- Both measurement-based studies and modeling simulations demonstrate the effectiveness of vented kitchen range exhaust for mitigating NO_2 and PM emissions from cooking activities on kitchen ranges. For example, the *Dobbin et al.* (2018) study demonstrated the effectiveness of three different kitchen range hoods operated under both low and high flow rates for maintaining short-term NO_2 kitchen air concentrations during cooking activities below reference ambient air health-based guidelines.

- Capture efficiencies (CE) for many of the range hoods evaluated were >75% when operated on medium or higher fan settings, indicating the potential for effective removal of pollutants generated from cooking activities.

- In general, above-the-cooktop devices with a flat bottom and no capture hood, including fan/microwave combination devices, had lower CEs compared to devices with capture hoods at the same flow rates.

- Burner coverage, defined as the degree to which the range hood covers the burner being used, is an important predictor of CE. Accordingly, CE is generally higher for cooking on back burners compared to front burners. Coverage of front burners is often poor for under-the-cabinet range hoods (including range/microwave combination devices), which do not

[15] Lawrence Berkeley National Laboratory (LBNL). 2020. "Simulations of Short-Term Exposure to NO_2 and $PM_{2.5}$ to Inform Capture Efficiency Standards (Final)." Report to California Energy Commission; US Dept. of Energy (US DOE) Sustainable Energy and Environmental Systems Dept., 27p., March 30.

extend over the front burners so as not to interfere with the stove operator or impede access to the microwave.

- Exhaust hood airflow is an important predictor of CE, but due to device design and installation considerations (including but not limited to those described above), meeting minimum standards for airflow is insufficient to guarantee high CE.

- Noise is an important variable that impacts usage of range hoods, although there does not appear to be quantitative data on how noise affects consumer usage. For the high CE range hoods tested in these studies, most generated noise at levels that would interfere with normal conversation, which could limit their use by consumers. There were some devices that were relatively quiet that still performed well with CE >70%.

- In addition, other studies[16,17] demonstrate that options other than vented range hoods can help reduce pollutant concentrations from cooking activities, including gas-combustion related emissions. For example, the *Paulin et al.* (2014)[18] home intervention study, which investigated the efficacy of interventions for reducing indoor NO_2 concentrations in homes with unvented gas stoves, demonstrated the effectiveness of air filtration devices for reducing indoor levels of both NO_2 and PM. HEPA air filtration devices are well-

[16] Chen, C.-F.; Hsu, C.-H.; Chang, Y.-J.; Lee, C.-H.; Lee, D.L. 2022. "Efficacy of HEPA Air Cleaner on Improving Indoor Particulate Matter 2.5 Concentration." Int. J. Environ. Res. Public Health 19, 11517. https://doi.org/10.3390/ijerph191811517.

[17] Matthaios, VN; Rooney, D; Harrison, RM; Koutrakis, P; Bloss, WJ. 2023. "NO_2 levels inside vehicle cabins with pollen and activated carbon filters: A real world targeted intervention to estimate NO_2 exposure reduction potential." Sci Total Environ 860:160395. doi: 10.1016/j.scitotenv.2022.160395.

[18] Paulin, LM; Diette, GB; Scott, M; McCormack, MC; Matsui, EC; Curtin-Brosnan, J; Williams, DL; Kidd-Taylor, A; Shea, M; Breysse, PN; Hansel, NN. 2014. "Home interventions are effective at decreasing indoor nitrogen dioxide concentrations." Indoor Air 24(4):416-424. doi: 10.1111/ina.12085.

established for $PM_{2.5}$ removal, while the addition of an activated carbon filter can result in effective filtration of NO_2.

(c) Please provide information on the naming/definition, requirements for, frequency of use, and performance of different kinds of range hoods (*i.e.*, external venting, recirculating, wall-mounted canopy, under-cabinet, microwave/combo, island/ceiling, or retractable downdraft, etc.).

Response to Question 1(c):

The AGA has no response to this question at this time.

(d) Please provide information on the frequency of occurrence of reported gas leaks associated with gas range defects or related equipment or installation defects.

Response to Question 1(d):

The AGA has no response to this question at this time.

(e) Please provide details on chronic chemical hazards associated with gas range use. Specify the nature of the chronic chemical hazard, the brand of gas range (if available), and additional context for settings and conditions related to exposures if available.

Response to Question 1(e):

The AGA is not aware of evidence of chronic chemical hazards associated with gas range use. As discussed more below (see also Responses to Questions 2(d) and 2(e)), both cooking activities themselves and the use of gas ranges have certain related emissions that affect indoor air quality, but the available evidence does not show that the use of gas ranges causes adverse health effects – and it thus is misleading to suggest that there is a "hazard." As noted above, ranges typically are off 95% of the time or more, and the chronic, long-term impacts on indoor air quality are not substantial from a health-based perspective. With this in mind, the AGA addresses below

information that is available on certain pollutant emissions that have been associated with gas range use as well as cooking activities more generally.

Gas range use can result to some degree in emissions of NO_2, $PM_{2.5,}$ CO, and formaldehyde. These pollutants are everyday constituents of indoor and ambient air due to a multitude of common sources, including both natural and anthropogenic sources. Any combustion source, whether indoors (*e.g.*, woodstove or fireplace, furnace, candles, cigarette smoking) or outdoors (*e.g.*, motor vehicle, fire pit, construction equipment, forest fire), can be a source of these pollutants. Many other common activities also contribute to these pollutant concentrations, including cleaning and cleaning products, mold, pet dander, dust, pollen, and human movement. And, as explained below, some of these pollutants are emitted primarily from the cooking process itself, regardless of the fuel source. Thus, the critical question is not whether these common air constituents are present, but at what concentrations they are present as a result of gas range use.

It is well accepted that food preparation methods such as frying, grilling, sautéing, and broiling are sources of $PM_{2.5}$ emissions, regardless of whether a gas or electric cooking appliance is used. Cooking-related $PM_{2.5}$ emissions are generated by the heating of cooking oils, burning of food, and mechanical cooking processes like frying and sautéing. Study findings overall indicate that factors besides the appliance energy source, including food preparation method, types of cooking oils used, and types of food cooked, are generally the key drivers of cooking-related $PM_{2.5}$ emissions.[19] Consistent with this, in their analysis of 541 cooking events from 13 studies, researchers from the Lawrence Berkeley National Laboratory (LBNL) reported that stove fuel type

[19] Lawrence Berkeley National Laboratory (LBNL). 2012. "Compilation of Published $PM_{2.5}$ Emission Rates for Cooking, Candles and Incense for Use in Modeling of Exposures in Residences." Report to US Dept. of Energy (US DOE); US Dept. of Housing and Urban Development (HUD); US EPA; California Energy Commission. Environmental Energy Technologies Division, 29p., August.

"did not show significant differences in emission rates [of $PM_{2.5}$] when data from multiple studies were compared."[20] In addition, in boiling and simmering experiments conducted on gas cooktops and in gas ovens in nine California homes where pots of water were used as heat sinks to eliminate emissions associated with food preparation (*i.e.*, no use of food or oils), *Singer et al.*[21] reported "negligible increases" in indoor $PM_{2.5}$ concentrations for the majority of experiments in their study, further supporting the key role of cooking oils and food cooking as $PM_{2.5}$ sources rather than fuel type. In fact, LBNL investigators, recognizing the body of evidence demonstrating that oven/range fuel type is not a driver of cooking-related $PM_{2.5}$ emissions, assumed that the same amount of PM would be emitted during cooking activities with gas, propane, or electric burners for their modeling simulations of range hood capture efficiency standards.[22] GTI Energy also examined the relative contributions of $PM_{2.5}$ from gas and electric ranges using a controlled cooktop test. Results show that $PM_{2.5}$ emissions are more a function of the cooking vessel and food product than the fuel type used, and that electric ranges could have higher $PM_{2.5}$ emissions than gas ranges for some circumstances.[23] Thus, available evidence indicates that gas range use is an insignificant contributor to indoor $PM_{2.5}$, regardless of the type of range used or the settings and conditions.

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[20] *Id.*

[21] Singer, BC; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2016. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-1006385. October. *subsequently published as* Singer, BC; Pass, RZ; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2017. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Building and Environment. 122:215-229. doi: 10.1016/j.buildenv.2017.06.021.

[22] Lawrence Berkeley National Laboratory (LBNL). 2020. "Simulations of Short-Term Exposure to NO2 and PM2.5 to Inform Capture Efficiency Standards (Final)." Report to California Energy Commission; US Dept. of Energy (US DOE) Sustainable Energy and Environmental Systems Dept., 27p., March 30.

[23] Johnson, F. 2022. "Residential Cooking IAQ Special Report: Cooking Emissions for Natural Gas, Propane, and Electric Range Tops." https://www.gti.energy/wp-content/uploads/2022/09/Residential-Cooking-Indoor-Air-Quality-Cooking-Emissions-for-NaturalGas-Propane-Electric-Range-Tops-whitepaper.pdf (gti.energy) (Last visited, May 8, 2023).

In addition, studies have demonstrated air emissions of other pollutants with *both* gas and electric cooking appliances. The *Fortmann et al.* study (sometimes referred to as the 2001 California Air Resources Board cooking and indoor air quality study) reported findings from 32 tests performed to measure indoor air quality impacts during typical cooking activities with gas and electric ranges.[24] This study demonstrated that air pollutants including $PM_{2.5}$, NO_2, CO, and formaldehyde are emitted during cooking with both gas and electric appliances. It showed that PM is produced during electric as well as gas cooking events (as discussed above), with some of the highest measured $PM_{2.5}$ concentrations for frying of tortillas in oil on an electric range. Findings also indicated NO_2 and CO emissions for both gas-range and some electric-range cooking activities, such as broiling. Findings for cooking experiments where fish was broiled using a gas range or an electric range, and for oven cleaning tests of gas and electric ranges, indicated similar generation of formaldehyde emissions with gas and electric ranges. In addition, *Mullen et al.*, reported air measurements in 352 California homes and found no evidence of increased indoor formaldehyde concentrations in homes with gas appliances.[25] These studies suggest that both gas and electric ranges can contribute to certain indoor pollutant concentrations depending on the food cooked and method of cooking.

As NO_2 is a byproduct of combustion, gas range use can expectedly contribute to greater NO_2 emissions than electric range use. However, studies of long-term NO_2 concentrations (*i.e.*, average concentrations of one week or more) in homes with gas ranges as compared to homes with electric ranges have demonstrated no meaningful difference in NO_2 concentrations from a health-

[24] Fortmann, R., Kariher, P. and Clayton, R., 2001. Indoor air quality: Residential cooking exposures.

[25] Mullen, NA; Li, J; Russell, ML; Spears, M; Less, BD; Singer, BC. 2016. "Results of the California Healthy Homes Indoor Air Quality Study of 2011-2013: Impact of natural gas appliances on air pollutant concentrations." Indoor Air 26(2):231-245. doi: 10.1111/ina.12190.

based perspective. Measured concentrations from gas range use are below the long-term (annual) National Ambient Air Quality Standard (NAAQS) set by the U.S. Environmental Protection Agency, and furthermore, such concentrations appear to have decreased over time (see the response to Question 2(d) for more details). In limited measurement studies of short-term (1-hour) NO_2 concentrations emitted from gas ranges, results have shown concentrations could exceed the short-term (1-hour) NAAQS under certain conditions (higher-than-average cook times, in small, poorly ventilated kitchens) but can be controlled below reference ambient air guideline levels with exhaust hood ventilation (see the response to Question 2(d) for more details). Such mitigation is advisable for indoor cooking no matter what fuel type is employed (*e.g.*, gas or electric).

Two recent research publications measured levels of benzene and other volatile organic compounds (VOCs) in end-use, unburned natural gas samples collected from 69 residential locations in the Greater Boston (Massachusetts) metropolitan area (Michanowicz *et al*., 2022[26]) and from 159 residential stoves in seven California regions (*Lebel et al*., 2022[27]). *Michanowicz et al*. (2022) reported an arithmetic mean benzene concentration of 165 parts per billion by volume (ppbv), or 0.0000165%, for their analyses of Boston area natural gas samples, while *Lebel et al*. (2022) measured higher (typically low part per million [ppm]) levels for the California gas samples. Although these studies reported the presence of detectable levels of benzene in end-use natural gas samples in these two areas, they did not demonstrate that indoor air concentrations of benzene are typically elevated in homes with gas cooking appliances. Specifically, *Michanowicz*

[26] Michanowicz, DR; Dayalu, A; Nordgaard, CL; Buonocore, JJ; Fairchild, MW; Ackley, R; Schiff, JE; Liu, A; Phillips, NG; Schulman, A; Magavi, Z; Spengler, JD. 2022. "Home is where the pipeline ends: Characterization of volatile organic compounds present in natural gas at the point of the residential end user. " *Environ. Sci. Technol.* 56(14):10258-10268. Doi: 10.1021/acs.est.1c08298.

[27] Lebel, ED; Michanowicz, DR; Bilsback, KR; Hill, LAL; Goldman, JSW; Domen, JK; Jaeger, JM; Ruiz, A; Shonkoff, SBC. 2022. "Composition, emissions, and air quality impacts of hazardous air pollutants in unburned natural gas from residential stoves in California. " *Environ. Sci. Technol.* 56(22):15828-15838. doi: 10.1021/acs.est.2c02581.

et al. did not make any indoor air measurements, nor did they conduct an indoor air modeling analysis to determine the potential indoor air quality impacts of the measured levels of benzene (or other VOCs) in the natural gas samples. However, based on the measured levels of benzene and natural gas odorants in the natural gas samples, *Michanowicz et al*. estimated that indoor air benzene concentrations would be very small (mean of 0.004 ppbv) for levels of natural gas where gas odorants would be at their odor thresholds (and able to be smelled). This finding is supportive of a lack of meaningful indoor air quality impacts for the measured benzene concentrations in Boston-area natural gas samples under typical real-world settings.

Lebel et al. (2022) conducted an indoor air modeling analysis to estimate kitchen indoor air benzene concentrations from the measured benzene levels in the California natural gas samples. He did not, however, actually measure indoor air concentration levels (which he could have done). Instead, he conducted an indoor air modeling analysis based on multiple highly conservative and non-representative assumptions (*e.g.*, very low air change rates), producing hypothetical "worst case" predictions rather than representative real-world exposure estimates.

Even with this conservative approach, most modeling simulations yielded kitchen indoor air benzene concentrations below the California Office of Environmental Health Hazard Assessment (OEHHA) 8-hour reference exposure level (REL) of 0.94 parts per billion by volume (ppbv), which *Lebel et al*. used as a health-based exposure guideline for benzene. The *Lebel et al*. (2022) study findings do not support conclusions regarding the typical magnitude or frequency of benzene indoor air exposure levels. In short, there is no confirmed evidence (*i.e.*, indoor air measurements) of meaningful exposures to indoor air benzene concentration levels in association with gas range use in real-world settings.

(f) Please provide any additional information not requested above related to the scale and scope of potential chronic chemical hazards associated with gas range use.

Response to Question 1(f):

The AGA has no response to this question at this time.

Question 2: Please provide information related to data sources and approaches CPSC should consider when completing an evaluation of chronic chemical hazards, exposures, and risks related to gas range use.

(a) Please provide product testing information or studies that describe the emissions of chemical substances from gas ranges over time, including average and peak levels where available. Provide supporting information on sampling and analytical methods. Examples of such information include environmental conditions, and other material test information for laboratory-based environmental chamber testing and field-based testing in a representative home environment, such as temperature, humidity, chamber ventilation, heating, ventilation, air conditioning (HVAC), leakage in and out of the chamber or home, mixing characteristics, chamber and/or representative home dimensions and characteristics, emissions measurements, measurement methods, computer modeling, post processing methods, product use variations, sample locations, fuel pressure, and fuel composition.

Response to Question 2(a):

The AGA has no response to this question at this time as regards product testing information. For emissions and fuel composition related information, refer to the responses to Questions 1(a) and 1(e) above.

(b) Please provide product testing information or studies characterizing differences in product performance, design, or compliance over time and related impacts on emissions of chemical substances from the gas ranges. If available, provide supporting information on sampling and analytical methods. If available, identify the relevant product safety standard(s) the product met, with revision/edition (*e.g.*, ANSI Z21.1-2000 or other international standards).

Response to Question 2(b):

The AGA has no response to this question at this time.

(c) Please provide product testing information that explores the impact that using different gas fuel sources has on emitted chemical substances. If available, provide supporting information on sampling and analytical methods.

Response to Question 2(c):

The AGA has no response to this question at this time.

(d) Please provide research that explores the relationship between emissions from gas ranges and indoor air quality. This includes experimental chamber or observational field studies that reflect environmental air monitoring of chemical substances during and after gas range use and/or modeled estimates of indoor air concentrations based on chamber emission data. If available, provide average and peak levels and supporting information on methods.

Response to Question 2(d):

Studies of the effects of cooking with natural gas ranges on indoor air quality typically include short-term measurements, collected during/after the use of the ranges, of oxides of nitrogen (NO_X) and nitric oxide (NO), which are byproducts of combustion. Nitrogen dioxide (NO_2) is not typically measured directly but is calculated from the NO_X and NO measurement values. Studies have also examined fine particulate matter ($PM_{2.5}$) and even smaller, ultra-fine particles (UFP), as well as carbon monoxide (CO) and formaldehyde, which can be produced from incomplete natural gas combustion. The above pollutants are typically present in outdoor air and may arise from various indoor sources other than cooking. When measuring these various substances in indoor air from cooking with natural gas, any number of variables may affect the measurement results, including cooking duration (as longer cooking times lead to greater cooking-related emissions), room size and configuration, whether any food is cooked and if so, the type of food and method of cooking, and the influence of ventilation (both natural air change rates and mechanical ventilation such as exhaust fans).

How the measurements are performed has a significant effect on the usefulness and reliability of the data. For measurements of outdoor air quality, the U.S. Environmental Protection Agency (USEPA) has approved lists of methods and equipment shown to generate acceptable data (these are termed reference methods and equivalent methods). These methods have been used in some indoor air quality studies, as well. But because the necessary equipment is expensive and complicated, some researchers have opted to use simpler, less expensive equipment options, *e.g.*, so-called "low-cost sensors." The low-cost sensors are less sensitive than the equipment used in USEPA reference and equivalent methods, and the data generated by the low-cost sensors are less accurate and less precise. Thus, measurements performed using non-USEPA approved methods, while perhaps useful as a reference point, should not be considered with the same certainty or – in the case of low-cost sensors - be relied upon for policy decisions.

For longer-term indoor air quality studies (looking at weekly or monthly concentrations), the measurement results in such studies are not tied directly to gas ranges because the results can be greatly influenced by other indoor or outdoor sources. Relative contributions of gas ranges to indoor air concentrations of various pollutants therefore have been evaluated in these studies by comparing measured concentrations in the homes with gas ranges to measured concentrations in homes with electric ranges.

Research that explores the relationship between gas ranges and indoor air quality is discussed below, split into discussions on short-term studies (1-hour measurements during/immediately after cooking), and long-term studies (*i.e.*, weekly or monthly measurements).

Short-term Studies

Relatively few studies have been conducted within the U.S. or Canada that have measured short-term concentrations of NO_x/NO_2 and other pollutants of interest (*e.g.*, CO) emitted from gas ranges during or after cooking. *Fortmann et al.* conducted what is believed to be the first cooking emissions study in a controlled, residential setting in February 2000.[28] Prescribed cooking tests were performed with both gas and electric ranges in a small, single-story test house in northern California using cooking durations of over an hour. Studies by *Singer et al.*,[29] which were performed in nine northern California homes, and *Dobbin et al.*[30] which were performed in two Canadian research homes, are the main sources of more current data. A larger study funded by the State of California is expected to publish additional data in the coming years, using low-cost sensors to measure indoor concentrations of NO_x/NO_2.

Singer et al., 2016

Singer et al.[31] measured NO, NO_x, CO, carbon dioxide (CO_2), and $PM_{2.5}$ in the kitchen and a bedroom of nine homes (seven detached houses, one flat of a two-flat duplex, and a small apartment) in northern California. The instrumentation used during testing included USEPA-

[28] Fortmann, R., Kariher, P. and Clayton, R., 2001. Indoor air quality: Residential cooking exposures.

[29] Singer, BC; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2016. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-1006385. October. *subsequently published as* Singer, BC; Pass, RZ; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2017. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Building and Environment. 122:215-229. doi: 10.1016/j.buildenv.2017.06.021.

[30] Dobbin, NA; Sun, L; Wallace, L; Kulka, R; You, H; Shin, T; Aubin, D; St-Jean, M; Singer, B.C. 2018. "The benefit of kitchen exhaust fan use after cooking – An experimental assessment." Building and Environment. Vol. 135, pp286-296.

[31] Singer, BC; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2016. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-1006385. October. *subsequently published as* Singer, BC; Pass, RZ; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2017. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Building and Environment. 122:215-229. doi: 10.1016/j.buildenv.2017.06.021.

approved reference methods (*e.g.*, gas phase chemiluminescence analyzers) for measuring NO_x and NO. Gas range tops were operated for 30 minutes and gas ovens were operated for 50 minutes in this study. Based on the information provided in the response to Question 1(a), these operation times are higher than average household cook times; a reasonable estimate for the average daily time spent cooking with gas, whether that is from cooktops or ovens, is **15 – 20 minutes per day**. The test conditions thus employed emissions almost twice the average daily emissions for cooking with gas (assuming emissions are linear to time using gas). Measurements were made with exhaust hoods off and with exhaust hoods on while cook tops, ovens, and broilers were operated, without actual food being cooked, to obtain 1-hour and 4-hour time-integrated concentrations for comparison to outdoor air health-based guidelines. Specifically, the short-term NAAQS set by the USEPA for NO_2, CO and $PM_{2.5}$ were used as reference health-based guidelines.

Pollutant concentrations varied widely across and within homes, with negligible concentrations reported for CO (below the 1-hour NAAQS of 35 parts per million [ppm]) and $PM_{2.5}$ (below the 24-hour NAAQS of 35 micrograms per cubic meter [$\mu g/m^3$]), even with exhaust hoods off. Results for CO were consistent with those reported by *Fortmann et al.* where a gas range burner was operated for over an hour.[32] Results for $PM_{2.5}$ were consistent with expectations, given that no actual cooking of food took place during the study; previous cooking studies specifically investigating particulate emissions have shown that cooking activities themselves are the main source of $PM_{2.5}$ emissions, regardless of whether a gas or electric cooking appliance is

[32] Fortmann, R., Kariher, P. and Clayton, R., 2001. Indoor air quality: Residential cooking exposures.

used[33,34] (see response to Question 1(e) for additional information). A recent GTI Energy Study comparing particulate emissions from the pan frying of hamburgers on gas and electric ranges[35] found that electric ranges consistently contributed to higher particulate matter emissions compared with gas ranges, with the differences due to poorer control of temperature for the electric ranges. This contrasts with earlier reported studies, which found cooking with gas ranges emitted more particles than electric.[36,37]

Singer et al.[38] determined that 1-hour NO_2 concentrations, estimated with exhaust hoods off, exceeded the 1-hour NAAQS of 100 parts per billion (ppb) in the kitchens of four of the nine homes, specifically the smaller homes with enclosed kitchens, with the highest concentrations being associated with oven use rather than cooktop use. Reported values elsewhere in the residences were significantly lower. As noted above, NO_2 is difficult to measure directly, and therefore *Singer et al.* measured NO_x and NO and then estimated NO_2 concentrations from those values. As *Singer et al.* acknowledged, the reported NO_2 values were biased high to an unknown degree by other pollutants (*e.g.*, nitrous acid [HONO]) that were present. Studies measuring

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[33] Lawrence Berkeley National Laboratory (LBNL), 2012. "Compilation of Published $PM_{2.5}$ Emission Rates for Cooking, Candles and Incense for Use in Modeling of Exposures in Residences." Report to US Dept. of Energy (US DOE); US Dept. of Housing and Urban Development (HUD); US EPA; California Energy Commission. Environmental Energy Technologies Division, 29p., August.

[34] Abdullahi, K.L., Delgado-Saborit, J.M. and Harrison, R.M., 2013. Emissions and indoor concentrations of particulate matter and its specific chemical components from cooking: A review. Atmos Environ. Vol. 71:260-294.

[35] Johnson, F. 2022. "Residential Cooking IAQ Special Report: Cooking Emissions for Natural Gas, Propane, and Electric Range Tops." https://www.gti.energy/wp-content/uploads/2022/09/Residential-Cooking-Indoor-Air-Quality-Cooking-Emissions-for-NaturalGas-Propane-Electric-Range-Tops-whitepaper.pdf (gti.energy) (Last visited, May 8, 2023).

[36] Buonanno, G., Morawska, L. and Stabile, L., 2009. Particle emission factors during cooking activities. Atmos Environ. 43(20):3235-3242.

[37] Dennekamp, M., S. Howarth, C.A. Dick, J.W. Cherrie, K. Donaldson, and A. Seaton, 2001. Ultrafine particles and nitrogen oxides generated by gas and electric cooking. Occup Environ Med. 58(8):511-516.

[38] Singer, BC; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2016. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-1006385. October. *subsequently published as* Singer, BC; Pass, RZ; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2017. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Building and Environment. 122:215-229. doi: 10.1016/j.buildenv.2017.06.021.

HONO have found it can be over 80% of the reported NO_2 value.[39] The extent of the bias is not known with certainty but from other studies can be conservatively estimated to be roughly 33% on average.[40,41,42,43]

Peak NO_2 concentrations in most of the kitchens exceeded 200 ppb with higher peak concentrations measured in the kitchen of the small (280 square foot) apartment. However, comparisons of peak NO_2 concentrations, which are instantaneous and transient, to the 1-hour time-average NAAQS are inappropriate and can lead to misleading conclusions.

The use of exhaust hoods by *Singer et al.*[44] was found to yield substantial reductions in estimated NO_2 concentrations both in the kitchens and in the bedrooms. For exhaust hoods with airflows above 95 liters per second, reductions of up to 80 to 95 percent were found. *Singer et al.* concluded, "since cooking with electric burners also produces pollutants, kitchen exhaust ventilation should be available in all homes, and operated as a precaution whenever cooking occurs."

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[39] USEPA, 2016a. Integrated Science Assessment for Oxides of Nitrogen – Health Criteria. EPA/600/R-15/068. January. https://www.epa.gov/isa/integrated-science-assessment-isa-oxides-nitrogen-health-criteria

[40] Bottorff, B., Wang, C., Reidy, E., Rosales, C., Farmer, D.K., Vance, M.E., Abbatt, J.P.D. and Stevens, P.S., 2022. Comparison of Simultaneous Measurements of Indoor Nitrous Acid: Implications for the Spatial Distribution of Indoor HONO Emissions. Environ. Sci. Technol. Vol. 56, pp13573-13583.

[41] Collins, D.B., R.F. Hems, S. Zhou, C. Wang, E. Gringnon, M. Alavy, J.A. Siegel, and J.P.D. Abbatt, 2018. Evidence for gas-surface equilibrium control of indoor nitrous acid. Environ. Sci. Technol. Vol. 52, pp12419-12427.

[42] Liu, J., S. Li, J. Zeng, M. Mekic, Z. Yu, W. Zhou, G. Loisel, A. Gandolfo, W. Song, X. Wang, Z. Zhou, H. Haerrmann, X. Li, and S. Gligorovski, 2019. Assessing indoor gas phase oxidation capacity through real-time measurements of HONO and NO_X in Guanzhou, China. Environmental Science: Processes and Impacts. 21 (5), July.

[43] Wang, C., B. Bottorff, E. Reidy, C. M.R. Rosales, D.B. Collins, A. Novoselac, D.K. Farmer, M.E. Vance, P.S. Stevens, and J.P.D. Abbatt, 2020. Cooking, Bleach Cleaning, and Air Conditioning Strongly Impact Levels of HONO in a House. Environ. Sci. Technnol. Vol. 54, No. 21, pp13488-13497. November 3.

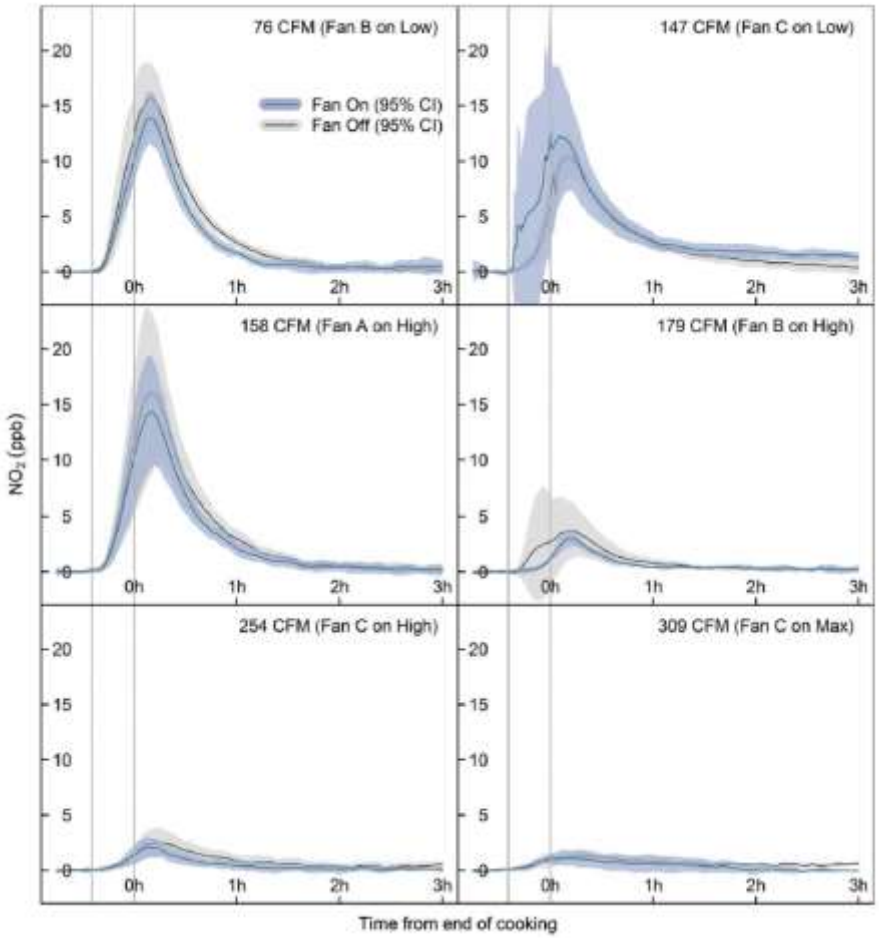
[44] Singer, BC; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2016. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-1006385. October. *subsequently published as* Singer, BC; Pass, RZ; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2017. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Building and Environment. 122:215-229. doi: 10.1016/j.buildenv.2017.06.021.

Dobbin et al., 2018

Dobbin et al.[45] reported indoor air NO, NO_2, $PM_{2.5}$, and ultrafine particulates (UFP) during 60 cooking tests in two identical Canadian research houses with very low ventilation rates (~0.08 air changes per hour [ACH]). The study emphasized the role of exhaust fans and ventilation. Various exhaust fans were tested, and air change rates in the kitchens associated with fan use were estimated to range from 0.72 to 2.30 ACH. The cooking test involved cooking of actual food (boiling of frozen broccoli and frying of hamburgers as a simulation of a "typical family meal") with the exhaust fans turned on during cooking. The duration of gas stove use was approximately 25 minutes. After the stove was turned off at the end of cooking, the tested exhaust fans were either turned off or left on for an additional three hours. Pollutant concentrations were monitored continuously during the entire 3-hour and 25-minute periods.

NO_2 air concentration values monitored during and after cooking, for all exhaust fans tested, were far below the reference 1-hour NAAQS of 100 ppb. The *peak* NO_2 value measured with the least effective fan used (generating an air exchange rate of 0.72 ACH) was roughly 15 ppb, while the peak values measured with the more effective fans and fan settings were much smaller, at about 1 to 4 ppb. The study found that NO_2 air concentration levels were reduced when: 1) fans were run at higher settings, 2) fans continued to operate after cooking had ended, and 3) when cooking was performed on back burners rather than front burners. The figure below is reproduced from the *Dobbin et al.* paper and shows that as air flows go up (from top to bottom), the maximum and average NO_2 concentrations go down.

[45] Dobbin, NA; Sun, L; Wallace, L; Kulka, R; You, H; Shin, T; Aubin, D; St-Jean, M; Singer, B.C. 2018. "The benefit of kitchen exhaust fan use after cooking – An experimental assessment." Building and Environment. Vol. 135, pp 286-296.



Data reported by *Dobbin et al.*[46] indicated $PM_{2.5}$ levels after cooking were less than 26 µg/m^3 and most values were less than 10 µg/m^3, which are all below the short-term, 24-hour NAAQS of 35 µg/m^3.

Modeled Estimates

Modeling is less helpful or relevant when actual measurements can be taken or have been taken. As a general matter, modeling of air quality is intended to be a conservative first step in

[46] Dobbin, NA; Sun, L; Wallace, L; Kulka, R; You, H; Shin, T; Aubin, D; St-Jean, M; Singer, B.C. 2018. "The benefit of kitchen exhaust fan use after cooking – An experimental assessment." Building and Enviroment. Vol. 135, pp 286-296.

evaluations, meaning that estimated indoor air concentration levels are likely overstated. And, as with any modeling exercise, the outputs are limited by the quality of the input information. It is thus advisable to follow up on modeled estimates with direct measurements when possible, especially if the modeling suggests potential air quality concerns.

Prior to the measurement work performed by *Singer et al.*[47] and *Dobbin et al.*,[48] some researchers used modeling to evaluate potential impacts of gas ranges on indoor air quality, such as the simulation-based assessment performed by *Logue et al*.[49] *Logue et al.* used a mass balance model to estimate indoor air concentrations of NO_2, CO, and formaldehyde from natural gas stoves in Southern California residences under both summertime and wintertime conditions.[50] The modeling also evaluated the potential benefits of exhaust hood use. The outputs in this modeling were affected by the input assumptions chosen, including the relatively high NO_2 concentrations present in outdoor air in Los Angeles,[51] which were incorporated into the simulation-based assessment. Building ventilation was randomly selected from data distributions for winter and summer seasons for different ages of homes (with higher values for the summer, likely due to more

[47] Singer, BC; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2016. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-1006385. October. *subsequently published as* Singer, BC; Pass, RZ; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2017. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Building and Environment. 122:215-229. doi: 10.1016/j.buildenv.2017.06.021.

[48] Dobbin, NA; Sun, L; Wallace, L; Kulka, R; You, H; Shin, T; Aubin, D; St-Jean, M; Singer, B.C. 2018. "The benefit of kitchen exhaust fan use after cooking – An experimental assessment." Building and Environment. Vol. 135, pp286-296.

[49] Logue, J.M., N.E. Klepeis, A.B. Lobscheid, and B.C. Singer, 2014. Pollutant Exposures from Natural Gas Cooking Burners: A Simulation-Based Assessment for Southern California. Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-6712E.

[50] Logue, J.M., N.E. Klepeis, A.B. Lobscheid, and B.C. Singer, 2014. Pollutant Exposures from Natural Gas Cooking Burners: A Simulation-Based Assessment for Southern California. Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-6712E.

[51] Zusman, M., A.J. Gassett, K. Kirwa, R.G. Barr, C.B. Cooper, M.K. Han, R.E. Kanner, K. Koehler, V.E. Ortega, R. Paine III, L. Paulin, C. Pirozzi, A. Rule, N.N. Hansel, and J.D. Kaufman, 2021. Modeling residential indoor concentrations of $PM_{2.5}$, NO_2, NO_X, and secondhand smoke in the Subpopulations and Intermediate Outcome Measures in COPD (SPIROMICS) Air study. Indoor Air. 31: 702-716.

window opening). The modeling results indicated that gas burner use could lead to elevated short-term (1-hour) NO_2 concentrations in simulated homes in which gas burners were used without kitchen exhaust ventilation. The subsequent studies by *Singer et al.*[52] and *Dobbin et al.*[53] were conducted to get real-world measurement data. Their work indicates that NO_2 concentrations could be elevated under certain conditions (higher than average cook times, in small, poorly ventilated kitchens, without the use of exhaust hoods), although the results may have also been biased high and this represents a very limited sample size. Importantly, however, their work also confirms that emissions were kept below reference health-based guidelines if exhaust fans were utilized, and in fact far below those guidelines in the *Dobbin et al*. study.

Summary of Short-Term Studies

In summary, *Singer et al.*[54] found that short-term (1-hour) NO_2 concentrations from gas range use could exceed 1-hour ambient air health-based guidelines (*e.g.*, the 1-hour NAAQS) under certain conditions (*i.e.*, higher than average cook times, in small, poorly ventilated kitchens, without the use of exhaust hoods), although the results from the study may have also been biased high (due to HONO measurement interference). *Singer et al.* reported negligible concentrations

[52] Singer, BC; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2016. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-1006385. October. *subsequently published as* Singer, BC; Pass, RZ; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2017. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Building and Environment. 122:215-229. doi: 10.1016/j.buildenv.2017.06.021.

[53] Dobbin, NA; Sun, L; Wallace, L; Kulka, R; You, H; Shin, T; Aubin, D; St-Jean, M; Singer, B.C. 2018. "The benefit of kitchen exhaust fan use after cooking – An experimental assessment." Building and Environment. Vol. 135, pp286-296.

[54] Singer, BC; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2016. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-1006385. October. *subsequently published as* Singer, BC; Pass, RZ; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2017. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Building and Environment. 122:215-229. doi: 10.1016/j.buildenv.2017.06.021.

for other pollutants including CO and $PM_{2.5}$, and substantial reductions in NO_2 concentrations were observed when exhaust hoods were used. *Dobbin et al.*[55] subsequently established that NO_2 levels can be kept well below the 1-hour NAAQS with the use of exhaust fans during cooking with gas.

Long-term Studies

More research has been conducted on long-term (*i.e.*, weekly or monthly average) concentrations of cooking-related pollutants in indoor air. In such studies, reported CO concentrations were insignificant (generally below 1 ppm in houses in the U.S. and England)[56], and there was no evidence of significantly increased indoor formaldehyde concentrations in homes with gas appliances (in studies conducted in California and Quebec City, Canada)[57,58,59]. Formaldehyde is a common indoor pollutant with many other sources (such as building materials, carpeting and flooring, paint, furniture, personal care products),[60] with levels notably dropping in California houses after 2008 (when regulations to limit formaldehyde emissions from composite wood products became operational).[61]

[55] Dobbin, NA; Sun, L; Wallace, L; Kulka, R; You, H; Shin, T; Aubin, D; St-Jean, M; Singer, B.C. 2018. "The benefit of kitchen exhaust fan use after cooking – An experimental assessment." Building and Environment. Vol. 135, pp286-296.

[56] Vardoulakis, S., E. Giagloglou, S. Steinle, A. Davis, A. Sleeuwenhoek, K.S. Galea, K. Dixon, and J.O. Crawford, 2020. Indoor Exposure to Selected Air Pollutants in the Home Environment: A Systematic Review. Int'l Journal of Env. Research and Public Health. Vol. 17, p8972.

[57] Mullen, N.A., J. Li, and B.C. Singer, 2012. Impact of Natural Gas Appliances on Pollutant Levels in California Homes. Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-5970E. December.

[58] Mullen, N.A., J. Li, M.L. Russel, M. Spears, B.D. Less, and B.C. Singer, 2016. Results of the California Healthy Homes Indoor Air Quality Study of 2011-2013: Impact of Natural Gas Appliances on Air Pollutant Concentrations. Indoor Air. Vol. 26, pp231-245.

[59] Gilbert, N.L. et al., 2006. Housing characteristics and indoor concentrations of nitrogen dioxide and formaldehyde in Quebec City, Canada. Environ. Res. 102, 1-8.

[60] Collins, D.B., R.F. Hems, S. Zhou, C. Wang, E. Gringnon, M. Alavy, J.A. Siegel, and J.P.D. Abbatt, 2018. Evidence for gas-surface equilibrium control of indoor nitrous acid. Environ. Sci. Technol. Vol. 52, pp12419-12427.

[61] Chan, W.R., Y-S Kim, B.D. Less, B.C Singer, and I.S. Walker, 2020. Ventilation and Air Quality in New California Homes with Gas Appliances and Mechanical Ventilation. California Energy Commission, Final Project Report. April.

For NO_x/NO_2, available studies indicate long-term (*e.g.*, weekly average) NO_2 concentrations can be higher in homes with gas stoves than homes with electric stoves but remain below the long-term annual outdoor NAAQS of 53 ppm, indicating the concentrations are not meaningfully increased and are not expected to pose increased health risk. Compiled results of reported average NO_2 concentrations in homes with gas stoves versus homes with electric stoves from a variety of studies with long-term measurement durations (approximately one to four weeks)[62,63,64,65,66,67] are summarized in the figure below.

[62] Belanger, K., Gent, J.F., Triche, E.W., Bracken, M.B. and Leaderer, B.P., 2006. Association of Indoor Nitrogen Dioxide Exposure with Respiratory Symptoms in Children with Asthma. Am. J. Respi. Crit. Care Med. 173: 297-303.

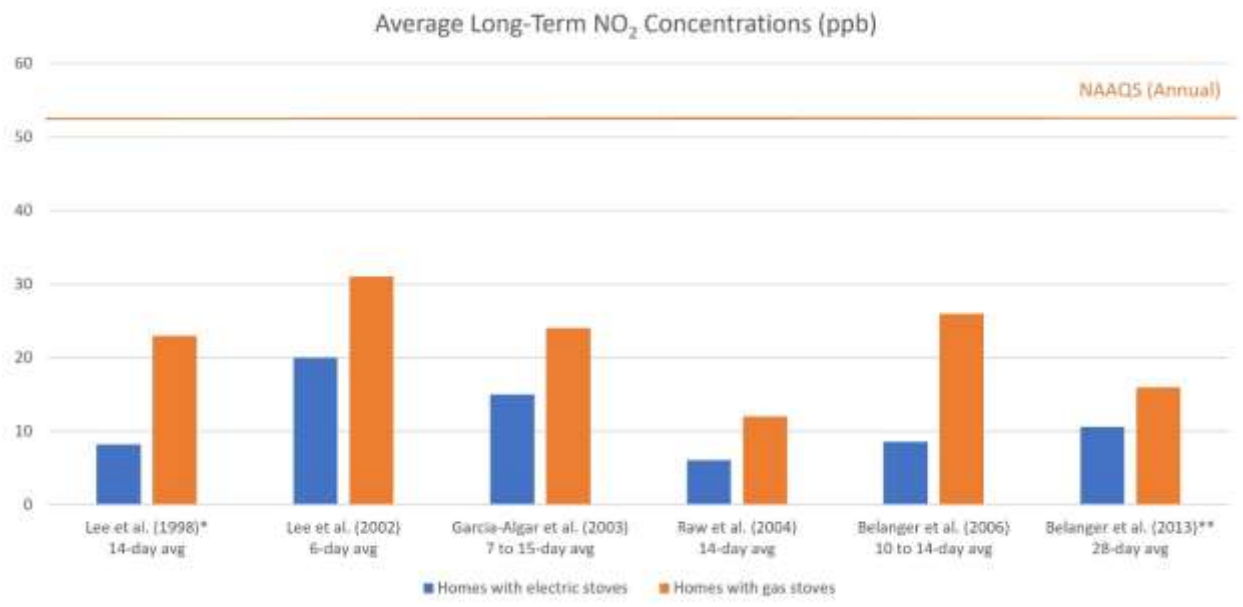
[63] Belanger, K., T.R. Holford, J.F. Gent, M.E. Hill, J.M. Kezik, and B.P. Leaderer, 2013. Household Levels of Nitrogen Dioxide and Pediatric Asthma Severity. Epidemiology. 24(2): 320-330.

[64] Garcia-Algar, O., M. Zapater, M., Figueroa, C., Vall, O., Basagana, X., Sunyer, J., Freixa, A., Guardino, X. and Pichini, S., 2003. Sources and Concentrations of Indoor Nitrogen Dioxide in Barcelona, Spain. J. Air & Waste Manag. Assoc. 53(11): 1312-1317.

[65] Lee, K., J.I. Levy, Y. Yanagisawa, and J.D. Spengler, 1998. The Boston Residential Nitrogen Dioxide Characterization Study: Classification and Prediction of Indoor NO2 Exposure. J. Air & Waste Manag. Assoc. 48: 736-742

[66] Lee, K., J. Xue, A.S. Geyh, H. Ozkaynak, B.P. Leaderer, C.J. Weschler, and J.D. Spengler, 2002. Nitrous Acid, Nitrogen Dioxide, and Ozone Concentrations in Residential Environments. Environ. Health. Perspect. 110(2): 145-149.

[67] Raw, G.J., S.K.D. Coward, V.M. Brown, and D.R. Crump, 2004. Exposure to Air Pollutants in English Homes. J. Expo. Anal. Environ. Epidemiol. 14 (Suppl. S1): S85–S94.



* Average concentration for homes with gas stoves from Lee *et al.* (1998), 23 ppb, is for homes with gas stoves without pilot lights. Average concentration for homes with gas stoves with gas-fed pilot lights from Lee *et al.* (1998) = 32 ppb.
** Average concentrations in Belanger *et al.* (2013) were only provided for homes with gas stoves and for all homes combined. Average concentration shown for homes with electric stoves is for all homes combined, 10.6 ppb.

An additional trend observable from the figure above is that of decreasing NO_2 concentrations over time. Such decreasing concentrations are likely attributable to several factors, including the phase-out of stoves with gas-fed pilot lights (gas appliances equipped with gas-fed pilot lights have not been manufactured since 2012, as mandated by the U.S. Department of Energy[68]), improvements in gas stove safety and efficiency, and reductions of outdoor air emissions levels (from electric utilities, other industrial fuel combustion sources, and on-road mobile sources[69]). For example, the research team of *Belanger et al.*[70] attributed the decrease in

[68] U.S. Department of Energy, 2009. 10 CFR Part 430. Energy Conservation Program: Energy Conservation Standards for Certain Consumer Products (Dishwashers, Dehumidifiers, Microwave Ovens, and Electric and Gas Ranges and Ovens) and for Certain Commercial and Industrial Equipment (Commercial Clothes Washers). Federal Register April 8, 2009 Vol 74(6): 16040-16096.
[69] USEPA, 2014. 2014 National Emissions Inventory (NEI) Data. https://www.epa.gov/air-emissions-inventories/2014-national-emissions-inventory-nei-data.
[70] Belanger, K., T.R. Holford, J.F. Gent, M.E. Hill, J.M. Kezik, and B.P. Leaderer, 2013. Household Levels of Nitrogen Dioxide and Pediatric Asthma Severity. Epidemiology. 24(2): 320-330.

NO_2 concentrations observed between their 2006 and 2013 studies to "the expanded use of high-efficiency gas appliances;" they measured a mean indoor NO_2 level of 25.8 ppb from single- and multi-family homes in Connecticut and Massachusetts from 1997 to 1999, compared to a mean level of 15.6 ppb from similar homes from 2006 to 2009.

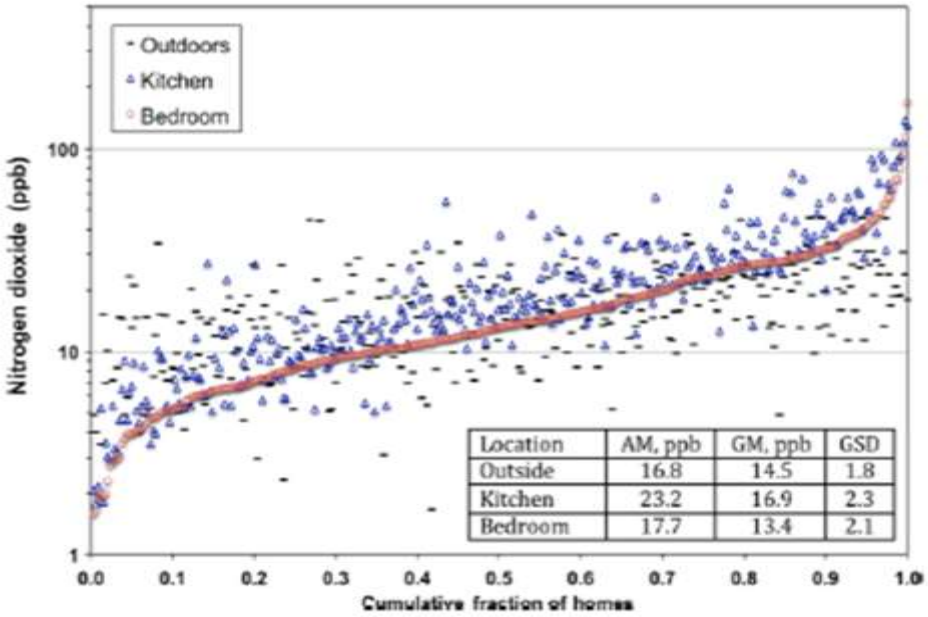
Moreover, in a recent study of 70 California homes, all with natural gas cooktops built between 2011 and 2017 after the 2008 California Title 24 Building Standards required dwelling unit mechanical ventilation, the 7-day median NO_2 concentration in these homes was 4.5 ppb.[71] This is comparable to the median 24-hour value of 3.2 ppb reported for 108 California homes, 98 percent of which had electric ranges, investigated as part of the 2009 California New Home Study (built between 2002 and 2004 without whole-dwelling mechanical ventilation).[72]

Additional research conducted by *Mullen et al.*[73] for 352 homes in California which employed passive samplers over a six-day sampling period to measure NO_x/NO_2 in kitchens, bedrooms, and outdoors identified outdoor air as a significant source of the NO_2 measured indoors as shown below in the figure reproduced from the *Mullen et al.* paper.

[71] Walker, I., B. Singer, and C. Rengie, 2019. Ventilation and Measured IAQ in new US homes. Lawrence Berkeley National Laboratory.

[72] Walker, I., B. Singer, and C. Rengie, 2019. Ventilation and Measured IAQ in new US homes. Lawrence Berkeley National Laboratory.

[73] Mullen, N.A., J. Li, M.L. Russel, M. Spears, B.D. Less, and B.C. Singer, 2016. Results of the California Healthy Homes Indoor Air Quality Study of 2011-2013: Impact of Natural Gas Appliances on Air Pollutant Concentrations. Indoor Air. Vol. 26, pp231-245.



Taken together, these studies of long-term NO_x and NO_2 concentrations demonstrate that homes with gas ranges do not have meaningfully elevated NO_2 concentrations as compared to homes with electric ranges. Both homes with gas ranges and electric ranges have long-term NO_2 indoor air concentrations that are below the U.S. outdoor air health-based guideline (NAAQS).

Summary

To sum up, the measurement evidence on short-term pollutant concentrations from gas range use is limited, particularly as to what is typical in most homes across the United States. A study of nine northern California homes (*Singer et al.*[74]) estimated short-term (1-hour) NO_2 concentrations from gas range use and found that, *without* the use of exhaust hoods, such concentrations could exceed 1-hour health-based guidelines (*e.g.*, the 1-hour NAAQS) under

[74] Singer, BC; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2016. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-1006385. October. *subsequently published as* Singer, BC; Pass, RZ; Delp, WW; Lorenzetti, DM; Maddalena, RL. 2017. "Pollutant concentrations and emission rates from natural gas cooking burners without and with range hood exhaust in nine California homes." Building and Environment. 122:215-229. doi: 10.1016/j.buildenv.2017.06.021.

certain specific conditions (*i.e.*, higher than average cook times, in small, poorly ventilated kitchens). NO_2 concentrations were not directly measured but rather inferred (as the difference between NO_x and NO measurements) and thus were likely biased high due to HONO measurement interference. And substantial reductions in NO_2 concentrations were observed when exhaust hoods were used. Following this work, another study evaluated short-term (1-hour) NO_2 concentrations from gas range use (*Dobbin et al.*[75]) involving multiple tests in two test homes, all with the use of exhaust fans during cooking, and consistently found that NO_2 air concentrations remained well below the 1-hour NAAQS guideline. Thus, the short-term studies of NO_2 are limited and may not have used typical cooking times or accounted for bias in measurements, but nevertheless confirm that ventilation mitigates emissions. As for other pollutants, *Singer et al.* reported negligible concentrations for CO and $PM_{2.5}$ levels, and *Dobbin et al.* reported $PM_{2.5}$ levels below the 24-hour NAAQS guideline. Moreover, previous cooking studies have demonstrated the difference in $PM_{2.5}$ emissions between gas and electric ranges alone is negligible, with particulate emissions driven much more heavily by the cooking itself (type of food, method of cooking, oil used) (see response to Question 1[e]). Moreover, modeling of short-term pollutant concentrations, conducted by *Logue et al.*,[76] shows the limits of air quality modeling, including its dependence on available data and assumptions, and demonstrates the need for more measurement studies.

Regarding long-term indoor pollutant concentrations, studies consistently show that NO_2 air concentrations in homes with gas stoves are below the long-term NAAQS guideline and that

[75] Dobbin, NA; Sun, L; Wallace, L; Kulka, R; You, H; Shin, T; Aubin, D; St-Jean, M; Singer, B.C. 2018. "The benefit of kitchen exhaust fan use after cooking – An experimental assessment." Building and Environment. Vol. 135, pp286-296.

[76] Logue, J.M., N.E. Klepeis, A.B. Lobscheid, and B.C. Singer, 2014. Pollutant Exposures from Natural Gas Cooking Burners: A Simulation-Based Assessment for Southern California. Ernest Orlando Lawrence Berkeley National Laboratory. LBNL-6712E.

long-term NO_2 concentrations appear to have decreased over time; that CO concentrations are insignificant; and that formaldehyde concentrations are not significantly increased l.

Adequate ventilation while cooking is beneficial regardless of stove type (gas or electric) to help reduce NO_2 and particulate emissions (and any other indoor pollutant concentrations), with ventilation to mitigate $PM_{2.5}$ necessitated more significantly by cooking methods, ingredients (oils), and food types than by the type of fuel used.

(e) Please provide studies that explore the relationship between the use, presence, or emissions from gas ranges and associated human health effects. This includes epidemiological studies that summarize exposures to gas ranges and observed health effects. If available, please provide test methods and raw data as well as the context and examples of the studies.

Response to Question 2(e):

Respiratory health effects have received the most attention with respect to gas cooking because of the potential for exposure to NO_2. Most of the epidemiology literature is focused on asthma and wheeze in children. *Lin et al.* (2013a) ten years ago conducted the most recent meta-analysis of gas cooking or indoor NO_2 and asthma or wheeze in children. A meta-analysis is a statistical analysis that combines the results of multiple scientific studies. While *Lin et al.* (2013a) found a weak statistically significant positive association between the presence of a gas stove and childhood asthma when they statistically combined results across studies, the authors found no statistically significant association between indoor NO_2 concentration levels and childhood asthma. As explained more below, the studies that were included in the *Lin et al.* meta-analysis were highly heterogeneous and of generally poor study quality (*e.g.*, failed to account for key confounding factors), making such a quantitative synthesis of data across these studies unreliable and inappropriate. Also, since the *Lin et al.* (2013a) paper was published, a large global analysis

of over 500,000 children from 47 countries (*Wong et al.*, 2013) found no association between gas stoves and asthma. The study was part of the International Study of Asthma and Allergies in Childhood (ISAAC). This study was cross-sectional, and of similar quality as the studies included in the *Lin et al.* (2013a) analysis, the majority of which were also cross sectional.

Li et al. (2023) (attached as an exhibit hereto) recently conducted a systematic review of 66 epidemiology studies on this topic, including all 41 studies in the *Lin et al.* (2013) meta-analysis. Those 66 studies, as well as the other studies referenced in this section, can be found in the appendix. Of the 66 studies, only a small proportion reported a statistically significant positive association; this was true of the available studies for all four relationships (gas stove – asthma; gas stove – wheeze; indoor NO_2 – asthma; indoor NO_2 – wheeze). Most studies, including the largest studies (*e.g.*, *Wong et al.* (2013); *Norback et al.* (2019); *Zacharasiewicz et al.* (1999)), reported null findings, meaning that there was no statistically significant result. Most of the gas cooking studies (*i.e.*, that evaluated the presence or use of a gas stove) were cross-sectional by design, meaning that they look at the presence of gas cooking and the outcome (asthma) at one moment in time, precluding causal inference. While a larger proportion of the studies that looked at indoor NO_2 concentration levels were cohort by design, meaning they looked at exposure and outcome over a time period, only a few cohort studies established temporality (by measuring exposure *prior* to the outcome), and those largely reported null findings. Overall, *Li et al.* conclude that the 66 studies do not provide consistent evidence of associations, nor do they support a causal conclusion between gas stove use/presence or indoor NO_2 and childhood asthma or wheeze.

With respect to the studies reviewed by *Li et al.* (2023), there is large variability across studies in terms of study region, age of children, gas cooking exposure definition, and asthma or wheeze outcome definition, making meta-analysis risk estimates that synthesize data across these

studies, such as those reported in *Lin et al.* (2013a), uncertain, indicating that such results should be interpreted with caution. Also, a large proportion of the studies to date are subject to multiple sources of bias and inaccuracy, primarily due to self-reported gas cooking exposure or respiratory outcomes, insufficient adjustment for key confounders (*e.g.*, environmental tobacco smoke, family history of asthma or allergies, socioeconomic status, or home environment), and unestablished temporality. For these reasons, one should not rely on the results from meta-analyses of the current literature on gas cooking or indoor NO_2 and respiratory outcomes.

More specifically, of the 29 studies that examined the association between gas stoves and childhood asthma, the measurements of gas cooking exposure relied on the objective observation of the presence of a gas cooking stove in only two studies; all other studies relied on self-reported information that was typically collected at one point in time. The definition of gas cooking exposure varied from "ever use gas for cooking" to "use gas generally/primarily for cooking" to "presence of gas cooking stove." There is rarely information on the frequency or duration of use, or if children were home or in proximity to the stoves during use.

With respect to the 20 studies that examined the association between indoor NO_2 concentration levels and asthma, all studies that measured indoor NO_2 did so using passive samplers, with six studies using the tube type samplers and seven studies using badge type samplers; one study did not specify the sampler type. Only some studies measured indoor NO_2 at multiple locations within each household. The averaging time for NO_2 measurements ranged from 1 day to 1 year. There are also other possible sources of NO_2 in homes in these studies such as outdoor air, resident smoking, and other non-cooking combustion sources.

For the studies examining asthma, the measurement of asthma outcome relied on contemporary physician diagnosis (during the study) in only two studies; all other studies that evaluated this relied on self-reported information, among which most were self-reported physician diagnosis. The definition of asthma varied widely among studies. As to the studies examining wheeze, most studies examined any wheeze as the only outcome type, six studies examined persistent wheeze only, and five studies examined both any wheeze and persistent wheeze. Only a few studies examined very specific wheeze outcomes, such as duration of wheezing, speech-limiting wheeze, sleep disturbance due to wheeze, exercise-induced wheeze, or wheeze with/without colds.

Even setting study limitations aside, taken together, the studies show no strong, statistically significant associations for any of the four associations examined (gas cooking – asthma; gas-cooking – wheeze; NO_2 – asthma; NO_2 – wheeze), weakening the likelihood of an underlying causal relationship. Most studies were cross-sectional by design and therefore could not establish temporality or a causal association. Only the indoor NO_2 studies addressed dose-response, in that they evaluated changes in risk associated with increases in NO_2 concentration. Only a few of these studies, however, reported statistically significant changes, and few examined the shape of the relationship (*e.g.*, linear *vs*. non-linear) of indoor NO_2 concentrations with asthma or wheeze. As a result, there is a lack of a well-characterized dose-response relationship between indoor NO_2 and asthma or wheeze in children, weakening the likelihood of an underlying causal relationship. And while NO_2 exposure above certain levels is associated in general with respiratory outcomes, it was not associated with asthma in studies evaluating indoor NO_2. Overall, these studies don't support the hypothesis that indoor NO_2 concentration levels associated with gas stove use are sufficient to increase asthma risk or wheeze in children. In sum, as *Li et al.* conclude by applying recognized

criteria for establishing causation, the epidemiology literature does not provide sufficient evidence to support causal relationships between gas cooking or indoor NO_2 and asthma or wheeze in children.[77]

In this regard, the recent *Gruenwald et al.* (2023) study that calculated a Population Attributable Fraction ("PAF") and estimated that "12.7% . . . of current childhood asthma in the US is attributable to gas stove use" is not valid. A PAF calculation is based on an assumption of a causal relationship and then extrapolates that risk to the population (*Rockhill et al.*, 1998). *Gruenwald's et al.* (2023) PAF calculation relied on the *Lin et al.* (2013a) risk estimates, which, as explained above, do not support causation; a PAF calculation is therefore unfounded and inappropriate.

(f) Please provide summaries of completed or ongoing research that produces information that can be used to evaluate potential associations between elevated exposures or disparate health outcomes and usage of gas ranges over time for vulnerable populations.

Response to Question 2(f):

The Cooking Electrification and Ventilation Improvements for Children's Asthma (CEVICA) study is currently being conducted in California (https://www.energizeinnovation.fund/projects/cooking-electrification-and-ventilation-improvements-childrens-asthma-cevica). This study is a randomized controlled trial that involves replacing gas cooking ranges with electric ranges with induction cooktops or providing countertop electric appliances in homes where gas ranges can't be replaced. It also evaluates the potential

[77] The Li *et al.* conclusions are consistent with the earlier findings of the National Academy of the Sciences report "Clearing the Air," which discusses the multi-factorial nature of asthma. *See* National Academies of Sciences, Engineering, and Medicine (NASEM). 2000. Clearing the Air: Asthma and Indoor Air Exposures; National Academies Press: Washington, DC; doi: 10.17226/9610).

benefit of ensuring adequate kitchen ventilation and education to improve indoor air quality and respiratory outcomes. This study is being conducted in the homes of children with asthma in three disadvantaged communities in California's Central Valley. This study could provide some information regarding the effects of cooking electrification and ventilation on indoor air quality and respiratory outcomes in vulnerable populations. However, because this study doesn't compare replacing older gas ranges with newer gas ranges, it will not provide evidence regarding health effects associated with modern gas stoves used with adequate ventilation. Since all participants have existing asthma, the study also cannot examine the relationship between gas stove use or ventilation and asthma onset.[78]

(g) Please provide any additional information, not requested above, related to the data sources and approaches CPSC should consider when completing an evaluation of chronic chemical hazards related to gas range use.

Response to Question 2(g):

As AGA noted above, it is not enough to identify pollutant emissions associated with gas combustion and with the use of gas cooking appliances. Cooking activities themselves, on both gas and electric stoves, generate certain emissions, and those emissions as well as overall indoor air concentration levels of various pollutants can be mitigated using ventilation. Moreover, the epidemiology literature evaluating gas stove use and NO2 levels and childhood asthma and wheeze does not demonstrate any causal association. Any approach to evaluating purported chronic

[78] The AGA is not aware of research showing disparate health outcomes caused by gas range use. While a couple studies report varying exposure levels among different population groups, most of the variances appear minor. Liu *et al.* (2021) published a systematic evaluation of outdoor air pollution exposure in the United States by race/ethnicity and income. They report that the exposures to $PM_{2.5}$ exhibit relatively little variability across groups. In 2010, the $PM_{2.5}$ exposures for the 50^{th} percentiles ranged from 9.3 ug/m3 for non-Hispanic whites to 10.0 ug/m3 for non-Hispanic blacks, versus a value of 9.5 ug/m3 for the entire population. For subgroups with greater exposures (75^{th} and 90^{th} percentiles), the exposures for each subgroup were essentially identical to one another. Liu *et al.* also report that the differences in exposures appear to be declining.

chemical hazards for gas range use must keep the overall body of scientific information in mind, recognizing its limitations, and consider whether any proposed solution should apply to electric ranges as well.

Question 3: Please provide information on proposed solutions related to any chronic chemical hazards, exposures, and risks associated with gas range use.

(a) Please provide information related to potential tradeoffs between different hazards (*i.e.*, chemical, fire, electrical, mechanical, or other) associated with the use of gas ranges, electric ranges (including older and newer models), and other large cooking appliances.

Response to Question 3(a):

Weighing the potential risks in the question above, *i.e.*, the "potential tradeoffs between different hazards. . . associated with the use of gas ranges [and] electric ranges," is by itself a complex task. Potential risks must be identified based on objective data and then weighed as to likelihood and severity of occurrence. There are some risks that occur more with electric ranges than with gas ranges, and it is not clear that replacing one type of range with another will lead to reduced risks, injuries, or harm to health – especially for risks driven more by the act of cooking than by the type of fuel source used.

Stepping back, however, the Commission should consider and evaluate whether restrictions on natural gas stoves or other natural gas appliances may create, incrementally, the risk of excessive energy dependence on one form of energy. Imbalanced dependence on an energy source or permitted use endangers energy security and therefore the essential life-sustaining public good it provides. Recent world events have highlighted the critical importance of energy security and reliability, which is an issue with national significance. Energy security includes safe, reliable, and affordable service and sufficient supplies to meet demand.

Our energy system must also be resilient to high impact events, *e.g.*, extreme weather events, geopolitical instability, or cyber attacks, that are occurring with increasing frequency. Such events create risks for each energy source, as well as for the system as a whole. Diversity of energy sources and energy use helps balance the energy system and ensure overall reliability during these types of events. Thus, elimination of natural gas as an energy source (by blocking infrastructure development or by prohibiting end use) would eliminate a method of mitigating the impacts of such events – in effect creating a new risk to individual consumers and to the system as a whole – with no corresponding benefit. And, in tight markets where supplies are scarce and prices high and volatile, those most likely to suffer hardship are those with the least economic resources, who are often historically disadvantaged groups within society.

Diversity of supply, generation, transmission, distribution and end use thus must be weighed as a positive factor providing tangible economic, life-sustaining, and security benefits. Indeed, efforts to eliminate fossil fuels, including any restrictions on the use of natural gas ranges, that are not based on firm, objective and scientific evidence impose a cost on society and consumers.

(b) Please provide information on existing solutions to reduce chronic chemical hazards associated with gas ranges and other large cooking appliances that have been developed but are not widely used throughout the market. Examples may include safety knobs, burner design, automatic or semi-automatic shut-off valves, or use of efficient externally vented range hoods. Where possible, please indicate cost estimates and why such solutions were not broadly adopted.

Response to Question 3(b):

The AGA has no response to this question at this time.

(c) Please provide information on technological advances that have been developed, or are underway, or could be developed in the future related to reducing chronic chemical hazards associated with gas ranges and other large cooking appliances (or related equipment) that are helpful for CPSC to consider for improving consumer safety and health. Please be specific and provide cost estimates where possible.

Response to Question 3(c):

The AGA has no response to this question at this time.

(d) Please provide information related to revision of voluntary standards for gas ranges. This includes data that would inform health-based emission limits for gas ranges. Such information would, for example, link robust and repeatable emissions testing data with estimated indoor air concentrations and toxicity reference values for CO, nitrogen dioxide, and fine particulate matter.

Response to Question 3(d):

As relayed in detail in section I, A and B, above, The CPSC's Indoor Air Quality associated with Gas Ranges Working Group ("Working Group") is in the midst of addressing the issues addressed in this query.

The Working Group was created to analyze indoor air quality associated with gas ranges and cooktops, and make coordinated recommendations based on data-driven, science-based, and public health-based analysis to determine if any proposals should be submitted to the appropriate voluntary standards or codes bodies.

The North American safety standards for residential gas cooking appliances is CSA/ANSI Z21.1-2018 • *CSA 1.1-2018, Household Gas Cooking Appliances* ("Z21.1 Standard") and has been identified by the Working Group as the likely appropriate voluntary standards or codes body.

The Z21.1 Standard applies to newly produced household gas cooking appliances, and applies to appliances using natural gas, manufactured gas, mixed gas, propane gas, LP gas-air

mixtures, and for use with either natural, manufactured, or mixed gas and convertible for use with propane gas.

The standard committees that develop the Z21.1 Standard follow the tenets of the ANSI Essential Requirements (https://www.ansi.org/american-national-standards/ans-introduction/essential-requirements) and Standards Council of Canada, which include balance of interest categories, openness to all interested and affected parties, and due process.

The ANSI Essential Requirements include several fundamental items, including:

- Lack of Dominance. This is defined as no more than one-third of the committee can represent any single interest category. At a minimum, the interest categories must include User, Producer, and General Interest. The standards developing organization ("SDO") may include additional, more specific, interest categories, for example utilities, testing laboratories, or engineers.

- Consensus. A majority is generallty the base threshold for establishing consensus.

- Due Process. All interested parties have the right to be heard and considered and there is an established transparent process for reviewing and responding to those comments.

- Openness. The process is open to all interested parties, meaning any individual have the right to submit comments, attend open meetings,

The ANSI process requires a public review period which generally ranges from 30 days to 60 days. Any comment submitted during the public review period must be addressed and the

proponent provided a technical response for all comments that are rejected. The SDO's ballot process requires that all comments be considered by the consensus committee.

The ANSI process also includes a final verification process to ensure that all requirements of both the SDO and ANSI were complied with during the revision process.

Since initially published in the 1920's, the CSA/ANSI Z21.1-2018 standard has been revised multiple times (as an ANSI-accredited standard it must be either revised or reaffirmed every five year) to address new technologies or safety issues.

The standard includes nearly 30 safety tests covering such the functionality of components such as ignition systems, burner and pilot light operation, burn potential (walls, ceiling, floor, cabinets, operator clothing, and exterior surfaces) along with knob, handles and glass temperatures.

An early commitment of the Working Group was to follow the spirit of the ANSI Essential Requirements (https://www.ansi.org/american-national-standards/ans-introduction/essential-requirements) by being open and balanced, facilitating lack of dominance, and providing due process. Thus far, the Working Group and Task Groups have made progress following the model provided by the ANSI Essential Requirements.

The efforts of the Working Group, consistent with the CPSC's statutory obligations, are working toward identifying whether and what recommendations the CPSC may make to amend the CSA/ANSI Z21.1-2018 • *CSA 1.1-2018, Household Gas Cooking Appliances.* The Working Group is followed a reasoned and balanced approach to identify the appropriate standards making body to which it may make recommendations and is now examining how testing may be

accomplished, and maximum emission exposure levels, AGA recommends that the CPSC should continue to pursue its efforts through the Working Group.

(e) Please provide information relating to the subject of mandatory standards for the sale of range hoods alongside gas ranges. Please provide information on what any such performance standards should be (*e.g.*, efficiency of removal of emitted substances), and the costs and benefits of such requirements.

Response to Question 3(e):

The AGA has no response to this question at this time.

(f) Please provide information on the effectiveness of different range hoods for reducing emissions and associated indoor air concentrations in air surrounding gas ranges (*i.e.*, personal breathing zone, room of use, etc.).

Response to Question 3(f):

The AGA has no response to this question at this time.

(g) Please provide information related to potential labeling for gas ranges that could provide information to educate consumers about potential hazards.

Response to Question 3(g):

The AGA does not believe that labeling for gas ranges should be considered at this time, when no "chronic chemical hazard" has been established. The AGA would note, however, that to the extent the Commission is concerned with cooking activities generally, any recommendations, *e.g.* on ventilation, should be for both gas and electric ranges.

(h) Please provide information on indoor air quality (IAQ) in home environments, both related to and separate from gas ranges.

Response to Question 3(h):

Recent reviews of indoor air quality (IAQ) highlight the range of sources of indoor air pollutants in residential buildings, and the complex set of factors and indoor processes that impact indoor air quality.[79,80,81,82,83] (Please see the response to Question 2(d) for information on indoor air quality related to gas ranges.) As discussed in these reviews, indoor air pollutants can be of indoor or outdoor origin; can be directly emitted by primary sources or can be secondarily formed, such as by indoor chemistry; and can be emitted continuously or episodically. Indoor air pollutants have many more sources than just cooking, cleaning, smoking, and outdoor air; indoor pollutant emission sources include consumer products like candles and air fresheners, building materials (wood, PVC pipes), home furnishings (vinyl flooring, carpets), home renovations including painting, hobbies (*e.g.*, woodworking, furniture refinishing, arts and crafts), pesticides, electronic equipment like printers, pets, and even humans themselves (both from behaviors, physical movement, as well as bodily emissions). Indoor exposure concentrations are modified by both building-related factors and by human activities. Building-related factors include, for example, ventilation conditions and building materials and furnishings, which can serve as both sources and sinks of indoor chemicals and also provide surfaces for indoor chemistry. Human activities include the use of personal care products, cleaning, cooking, and other individual behaviors such as smoking. Each of the reviews cited above highlights significant remaining data gaps regarding

[79] Baeza_Romero, MT; Dudzinska, MR; Amouei Torkmahalleh, M, *et al.* 2022. "A review of critical residential buildings parameters and activities when investigating indoor air quality and pollutants." *Indoor Air*. 32:e13144. doi: 10.1111/ina.13144.

[80] Vardoulakis, S; Giagloglou, E; Steinle, S, *et al.* 2020. "Indoor exposure to selected air pollutants in the home environment: a systematic review." *Int J Environ Res Public Health*. 17(23):8972.

[81] Mannan, M; Al-Ghamdi, SG. 2021. "Indoor Air Quality in Buildings: A Comprehensive Review on the Factors Influencing Air Pollution in Residential and Commercial Structure." *Int. J. Environ. Res. Public Health* 18, 3276. https://doi.org/10.3390/ijerph18063276.

[82] Weschler, CJ; Carslaw, N. 2018. "Indoor Chemistry." *Environ Sci Technol* 52(5):2419-2428. doi: 10.1021/acs.est.7b06387.

[83] National Academies of Sciences, Engineering, and Medicine (NASEM). 2022. Why Indoor Chemistry Matters; The National Academies Press: Washington, DC; doi: 10.17226/26228.

indoor air quality, which include indoor chemistry and its impacts on human exposure and health, how cooking-related emissions vary for different cooking styles, the role of building materials and furnishings as sinks of indoor pollutants, and how the human body interacts with the indoor environment. Due to its complexity and multi-factorial nature, indoor air quality is highly variable and individual. Below we discuss various indoor air quality issues separate from gas ranges in more detail.

Radon – Radon is a naturally-occurring element found in soil that can migrate as a gas and enter indoor spaces. Exposure to radon is responsible for about 21,000 lung cancer deaths each year in the US, making radon the 2nd leading cause of lung cancer (after tobacco smoking).[84] The U.S. Environmental Protection Agency (USEPA) recommends mitigating indoor air in residential spaces that is 4 picoCuries per liter (pCi/L) or higher.[85] Indoor air that is below 2 pCi/L is considered to be safe, but that level still poses a cancer risk of about 32 in 1,000 (3E-02) for smokers and a 4 in 1,000 risk for non-smokers (4E-03).[86]

Mold – Molds include all species of microscopic fungi that grow in the form of multicellular filaments. Molds can thrive on any organic matter with moisture management problems. They are part of the natural environment and can be found everywhere, both in indoor spaces and outdoors. Molds generally are not an issue unless mold growth is occurring. The best way to control mold growth is to control moisture levels. Reducing indoor humidity to levels below 60% will help to decrease mold growth indoors.[87] There are no USEPA or other federal limits set

[84] USEPA, 2023. Exposure to Radon Causes Lung Cancer in Non-smokers and Smokers Alike. https://www.epa.gov/radon/health-risk-radon. Last updated January 5, 2023.
[85] USEPA, 2016. A Citizen's Guide to Radon, The Guide to Protecting Yourself and Your Family from Radon. EPA 402/K-12/002.
[86] USEPA, 2023. Exposure to Radon Causes Lung Cancer in Non-smokers and Smokers Alike. https://www.epa.gov/radon/health-risk-radon. Last updated January 5, 2023.
[87] USEPA, 2012. A Brief Guide to Mold, Moisture, and Your Home. EPA 402-K-02-003. Reprinted 09/2012.

for mold or mold spores, so indoor air testing cannot be used to check a building's compliance with federal mold standards.

Pets and Other Biological Pollutant Sources – About two-thirds of U.S. households include a pet.[88] The presence of pets in indoor spaces can affect indoor air quality due to dirt and other items tracked in from outdoors by the pet and its shedding of hair and skin (dander). Proteins in pet emissions can be an allergy and/or asthma trigger. Other biological pollutants commonly encountered within residential spaces include house dust (and associated mites), plant pollens, and droppings & body parts from cockroaches, rodents, and other pests or insects. These items can be potent allergens.[89]

VOCs – VOCs are commonly found in indoor air due to a variety of sources, including infiltration of outdoor air and emissions from indoor sources such as cleaning supplies, furniture and building materials, food items such as bananas, etc. Past work by the USEPA in the 1980's found that about a dozen common organic pollutants were present in indoor air at 2- to 5-times their levels in outdoor air.[90] More recent data are summarized in the attached Table for selected VOCs frequently detected in outdoor air[91] and indoor air at schools and offices.[92] Indoor air data

[88] Forbes Advisor, 2023. Pet Ownership Statistics 2023. https://www.forbes.com/advisor/pet-insurance/pet-ownership-statistics. Accessed on April 20, 2023.
[89] USEPA, 2023. Biological Pollutants' Impact on Indoor Air Quality. https://www.epa.gov/indoor-air-quality-iaq/biological-pollutants-impact-indoor-air-quality. Accessed on April 20, 2023.
[90] USEPA, 2022. Volatile Organic Compounds' Impact on Indoor Air Quality. https://www.epa.gov/indoor-air-quality-iaq/volatile-organic-compounds-impact-indoor-air-quality. Last updated August 26, 2022.
[91] USEPA, 2018. 2015-2016 National Monitoring Programs Annual Report (UATMP, NATTS, and CSATAM). Final Report. Eastern Research Group, RTP, NC. July 2018.
[92] Rago, R., A. Rezendes, J. Peters, K. Chatterton, and A. Kammari, 2021. Indoor Air Background Levels of Volatile Organic Compounds and Air-Phase Petroleum Hydrocarbons in Office Buildings and Schools. Groundwater Monitoring & Remediation 41. no.2, pp27-47.

for existing residences is also shown,[93] but note that these data are not as current and are less complete (*e.g.*, data are reported for fewer VOCs, no data on frequency of detection is given).

Secondhand Smoke – Secondhand smoke is the smoke from a cigarette, cigar, or pipe, that is exhaled by a smoker. The testing of secondhand smoke has identified more than 4,000 substances contained in the smoke, including various compounds that can cause cancer.[94] The dangers of secondhand smoke are sufficient that people are generally advised not to smoke indoors or in vehicles to avoid exposing others.

Wood Smoke – Smoke from wood-burning stoves and fireplaces contains a mixture of potentially harmful gases and small particles. The small particles represent the greatest health risk associated with wood smoke exposure.[95] There has been increasing activity in recent years to ban or otherwise control wood-burning indoors at either the State level (*e.g.*, Washington, Oregon)[96,97] or for individual cities or metro areas (*e.g.*, Denver, Salt Lake City).[98,99]

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93 Hodgson, A.T. and Levin, H., 2003. Volatile Organic Compounds in Indoor Air: A Review of Concentrations Measured in North America Since 1990. Lawrence Berkeley National Laboratory, Berkeley, CA. LBNL-51715. April 21.

94 American Lung Association, U.S. Environmental Protections Agency (USEPA), Consumer Product Safety Commission, and American Medical Association, Undated. Indoor Air Pollution, An Introduction for Health Professionals.

95 USEPA, 2023. Smoke from Residential Wood Burning. https://www.epa.gov/indoor-air-quality-iaq/smoke-residential-wood-burning. Last updated February 16, 2023.

96 State of Washington, Department of Ecology, 2023. Wood stoves & other home heating. https://ecology.wa.gov/Air-Climate/Air-quality/Smoke-fire/wood-stove-info. Accessed on March 27, 2023.

97 State of Oregon, Department of Environmental Quality, 2023. Buying or Selling Wood Stoves. https://www.oregon.gov/deq/Residential/Pages/heatsmart.aspx. Accessed on March 27, 2023.

98 Colorado Department of Public Health & Environment, 2023. Indoor burning restrictions. https://cdphe.colorado.gov/indoor-burning-restrictions. https://cdphe.colorado.gov/indoor-burning-restrictions Accessed on March 27, 2023.

99 Utah Department of Environmental Quality, 2023. Is Pollution from Wood Smoke Going Down? All Your Burning Questions Answered. https://deq.utah.gov/air-quality/is-pollution-from-wood-smoke-going-downall-your-burning-questions-answered. Accessed on March 27, 2023.

Typical Measured Air Concentration Results (µg/m^3)

		Indoor Air - Existing Residences[1]		Indoor Air - Schools & Offices[2]			Outdoor Air[3]		
Chemical	Synonym	Median	Maximum	% Detection	Median	95th Percentile	% Detection	Median	Maximum
PETROLEUM HYRDROCARBONS									
Benzene		2.8	131	94	0.62	1.40	100	0.58	7.32
Toluene		12	177	100	2.24	14.9	100	1.06	130
Ethylbenzene		2.3	48	100	0.30	1.69	99.8	0.17	3.07
m-/p-Xylene		6.1	291	100	0.81	5.30	99.9	0.43	10.4
o-Xylene		2.3	61	100	0.32	1.94	99.4	0.19	3.18
Styrene		1.1	23	87	0.18	0.54	74.3	0.06	94.6
1,2,4-Trimethylbenzene		3.9		97	0.31	7.2	97.7	0.18	3.94
Hexane		1.8		41	0.70	2.97	99.9	1.52	27
Heptane		1.1		41	0.82	3.07	100	0.74	11.0
Naphthalene		0.47		23	0.26	0.84	100	0.049	0.403
CHLORINATED VOCs									
Tetrachloroethylene	PCE	1.02	47.5	64	0.173	1.74	78.3	0.081	13.6
Trichloroethylene	TCE	0.43	10.2	17	0.107	0.521	21.4	ND	5.80
cis-1,2-Dichloroethylene	cis-1,2-DCE			2	0.079	0.0888	0.07	ND	0.28
trans-1,2-Dichloroethylene	trans-1,2-DCE			1	0.079	0.079	11.3	ND	26.1
1,1-Dichloroethylene	1,1-DCE			1	0.079	0.079	15.7	ND	0.55
1,1,1-Trichloroethane		1.96	153	9	0.109	0.272	70.4	0.03	0.53
1,1-Dichloroethane	1,1-DCA			5	0.081	0.132	31.2	ND	20.7
Vinyl Chloride	VC	<0.03	0.41						
Chloroform		0.93	13	92	0.15	0.92	98.2	0.13	56.6
1,2-Dichloroethane		0.04	1.1	79	0.11	0.34	93.4	0.08	45.7
Carbon Tetrachloride		0.57	1.8	100	0.43	0.58	100	0.64	3.69
Methylene Chloride	Dichloromethane	7.3	257	30	4.86	16.7	100	0.41	1,490
CHLOROFLUOROCARBONS									
Trichlorofluoromethane	Freon 11			100	1.44	6.46	100	1.38	8.65
Dichlorodifluoromethane	Freon 12			97	1.89	3.28	100	2.56	6.73
Chloromethane	R-40			98	1.31	1.60	100	1.24	8.16
Chlorodifluoromethane	Freon 22			99	2.16	80.8			
1,1,2-Trichloro-1,2,2-trifluoroethane	Freon 113			99	0.575	0.74			
COMMON SOLVENTS									
Acetone				100	19.7	45.2	100	2.24	45.1
Ethanol				100	110	466			
Isopropyl Alcohol	2-Propanol			100	12.3	50.8			
Methanol				100	34.4	75.9			
2-Butanone	MEK			70	1.48	4.88	99.8	0.50	15.2

[1] Hodgson, A.T. and H. Levin, 2003. Volatile Organic Compounds in Indoor Air: A Review of Concentrations Measured in North America Since 1990. Lawrence Berkeley National Laboratory, Berkeley, CA. LBNL-51715. April 21.

[2] Rago, R., A. Rezendes, J. Peters, K. Chatterton, and A. Kammari, 2021. Indoor Air Background Levels of Volatile Organic Compounds and Air-Phase Petroleum Hydrocarbons in Office Buildings and Schools. Groundwater Monitoring & Remediation 41, no.2, pp27-47.

[3] U.S. Environmental Protection Agency, 2018. 2015-2016 National Monitoring Programs Annual Report (UATMP, NATTS, and CSATAM). Final Report. Eastern Research Group, RTP, NC. July.

(i) Please provide information regarding inclusion, enforcement, and compliance with any requirements in building codes or other local or state laws or regulations for exhaust hoods (internally and externally vented) above or near gas ranges.

Response to Question 3(i):

The AGA has no response to this question at this time.

(j) Please provide information on the costs and effectiveness of any other relevant voluntary or mandatory standards (such as for gas ranges or exhaust hoods found in model building codes).

Response to Question 3(j):

The AGA has no response to this question at this time.

(k) Please provide any additional information related to chronic hazards associated with gas ranges and proposed solutions to those hazards that CPSC should consider, not requested above.

Response to Question 3(k):

The AGA has no response to this question at this time, other than to note again that no "chronic hazards associated with gas ranges" have been shown, making any solution unnecessary.

III. CONCLUSION

The American Gas Association respectfully requests that the Consumer Product Safety Commission consider these comments and the information and literature provided. Viewed objectively as a whole, the literature does not demonstrate any chronic chemical hazards associated with gas range use, making any further action by the Commission in this regard unnecessary and inappropriate. Moreover, as the Commission is aware, the work of the Commission's Indoor Air Quality associated with Gas Ranges Working Group and its three Task Groups is ongoing, and is

constituted of a range of private and industry participants discussing voluntary standards that could address some of these issues. AGA respectfully suggests that the Commission should allow that process to play out before taking any action. If you have any questions regarding the AGA's submission, please do not hesitate to contact the undersigned.

Respectfully submitted,

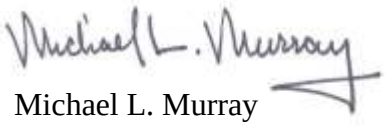

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EXHIBIT

Global Epidemiology

Gas cooking and respiratory outcomes in children: A systematic review

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Gas cooking and respiratory outcomes in children: A systematic review

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ABSTRACT

The most recent meta-analysis of gas cooking and respiratory outcomes in children was conducted by Lin et al. [93] in 2013. Since then, a number of epidemiology studies have been published on this topic. We conducted the first systematic review of this epidemiology literature that includes an in-depth evaluation of study heterogeneity and study quality, neither of which was systematically evaluated in earlier reviews. We reviewed a total of 66 relevant studies, including those in the Lin et al. [93] meta-analysis. Most of the studies are cross-sectional by design, precluding causal inference. Only a few are cohort studies that could establish temporality and they have largely reported null results. There is large variability across studies in terms of study region, age of children, gas cooking exposure definition, and asthma or wheeze outcome definition, precluding clear interpretations of meta-analysis estimates such as those reported in Lin et al. [93]. Further, our systematic study quality evaluation reveals that a large proportion of the studies to date are subject to multiple sources of bias and inaccuracy, primarily due to self-reported gas cooking exposure or respiratory outcomes, insufficient adjustment for key confounders (e.g., environmental tobacco smoke, family history of asthma or allergies, socioeconomic status or home environment), and unestablished temporality. We conclude that the epidemiology literature is limited by high heterogeneity and low study quality and, therefore, it does not provide sufficient evidence regarding causal relationships between gas cooking or indoor NO_2 and asthma or wheeze. We caution against over-interpreting the quantitative evidence synthesis estimates from meta-analyses of these studies.

Introduction

Nitrogen dioxide (NO_2) is commonly present in indoor air due to the presence of outdoor sources (e.g., mobile vehicles, industrial combustion) and indoor sources (e.g., tobacco use, fuel burning stoves or heating systems) [97]. In 2010, the World Health Organization (WHO) Guidelines for Indoor Air Quality [97] recommended a 1-h indoor NO_2 guideline of 200 $\mu g/m^3$ and an annual average indoor NO_2 guideline of 40 $\mu g/m^3$, which remained in the 2021 update [96].

The annual average guideline of 40 $\mu g/m^3$ was derived from the effect estimate from a meta-analysis by Hasselblad et al. [92]. Specifically, this meta-analysis included 11 epidemiology studies published in the 1970s and 1980s that examined associations between gas (vs. electric) stove use or indoor NO_2 concentrations and lower respiratory illness (e.g., wheeze, cough, bronchitis, phlegm) in children ≤12 years old. By assuming that the health outcomes examined across studies were similar enough, that all exposure contrasts could be converted to a 30 $\mu g/m^3$ increase in NO_2 concentration, and that key confounders were properly adjusted for in all studies, the authors estimated that exposure to a long-term increase of 30 $\mu g/m^3$ NO_2 was associated with a 1.2-times higher odds of having lower respiratory illness in children (odds ratio [OR] = 1.2, 95% confidence interval [CI]: 1.1–1.3) [92].

Twenty years later, Lin et al. [93] conducted another meta-analysis to quantitatively synthesize the evidence available through 2013, with a particular focus on asthma and wheeze as health outcomes. Lin et al. [93] included a total of 41 epidemiology studies that examined the associations between indoor NO_2 or gas cooking and asthma or wheeze in children (≤18 years), including those reviewed by Hasselblad et al. [92]. The authors reported statistically significantly positive associations between gas cooking and asthma (OR = 1.32, 95% CI. 1.18–1.48) and between indoor NO_2 and wheeze (OR = 1.12, 95% CI: 1.04–1.21 for a 15-parts-per-billion [ppb] increase in NO_2) and no statistically significant associations between indoor NO_2 and asthma (OR = 1.09, 95% CI: 0.91–1.31 for a 15-ppb increase in NO_2) or between gas cooking and

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Table 1
PECOS elements and corresponding inclusion and exclusion criteria.

PECOS element	Inclusion criteria	Exclusion criteria
Population	▪ Children (≤18 years of age) from any country or region	▪ Other age groups (e.g., adults) ▪ Children are not analyzed separately (e.g., 5 years of age and older)
Exposure	▪ Long-term (months to years) or short-term (hours to days) indoor exposure to NO_2 (in a concentration unit, e.g., ppb, $\mu g/m^3$) or gas cooking in family home	▪ Outdoor NO_2 exposure only ▪ Indoor exposure to other pollutants only ▪ Indoor NO_2 exposure in schools/classrooms only ▪ Includes other combustion sources (e.g., coal, wood, kerosene) ▪ Indoor gas cooking not the main source of NO_2 ▪ Prenatal exposure only ▪ Personal NO_2 exposures that include both indoor and outdoor NO_2
Comparator	▪ Exposure to less NO_2 ▪ Use electric stove, do not have a gas stove at home, do not use gas stoves usually	▪ Exposure contrast not given ▪ Comparators were non-electric combustion sources (e.g., coal, wood, kerosene, biomass)
Outcomes	▪ Asthma (newly diagnosed, ever-diagnosed, exacerbation) ▪ Wheeze (persistent, episodes within certain time windows)	▪ Other outcomes only
Study	▪ Primary epidemiology studies with data at the individual level (e.g., cohort, case-control, cross-sectional studies) ▪ Published in English	▪ Reviews, meta-analyses, commentaries, book chapters, or conference abstracts ▪ Ecological studies ▪ Non-human studies ▪ Methodological studies ▪ Studies with secondary analyses only (e.g., based on risk estimates from existing studies) ▪ Not published in English ▪ If no results presented

Notes
NO_2 = Nitrogen Dioxide; PECOS = Population, Exposure, Comparator, Outcomes, and Study Design; ppb = Parts per Billion.
Source: Morgan et al. [83].

wheeze (OR = 1.06, 95% CI: 0.99–1.13).

Quantitative evidence synthesis through a meta-analysis can help increase the statistical power to detect underlying associations, reconcile conflicting study results due to random variation, and generate summary effect estimates that are readily usable for policy-making. However, it is no substitute for a thorough understanding of what each individual study in the literature examined, how each study addressed its own research question, and to what extent each study is equipped to contribute to the knowledge base with respect to a specific research question. It is also not necessarily informative regarding causation. Hasselblad et al. [92] narratively described the design and finding of each of the 11 reviewed studies and tabulated the main study characteristics and results; Lin et al. [93] also tabulated the main study characteristics and results. Yet, there was no systematic study quality evaluation in either meta-analysis to determine the impact of individual studies' methodological limitations on the interpretation of their respective results or the quantitative evidence synthesis results for the literature as a whole. A study quality evaluation is now recognized as an essential component of systematic reviews and meta-analyses. As neither meta-analysis assessed study quality, they could not fully address whether any statistically significant associations were likely causal.

Furthermore, since the publication of the Lin et al. [93] meta-analysis, a number of new epidemiology studies that evaluated the associations between indoor NO_2 or gas cooking and asthma or wheeze in children have been published, including a large global analysis of phase three of the International Study of Asthma and Allergies in Childhood (ISAAC) for over 500,000 children from 47 countries [34]. To synthesize the evidence to date regarding the associations between indoor NO_2 or gas cooking and asthma or wheeze in children on the basis of an in-depth and systematic examination of study characteristics, results, and methodological strengths and limitations, we conducted a systematic review of relevant epidemiology studies published through June 1, 2022, including all 41 studies that contributed to the meta-analysis by Lin et al. [93].

Methods

The protocol for this systematic review was registered with the Open Science Framework (OSF) [18] on May 4, 2022.

Eligibility criteria and literature search

Study eligibility for this systematic review was determined based on inclusion and exclusion criteria structured by population, exposure, comparator, outcomes, and study design (PECOS) elements, as shown in Table 1.

To identify eligible studies, we systematically searched the PubMed, Scopus, and Lens.org databases for publications through June 1, 2022. In order to ensure that all studies captured in the Lin et al. [93] meta-analysis were captured in the present review, we performed, in each database, a main search using terms specifically for gas cooking or indoor NO_2 in relation to asthma or wheeze (detailed search strategies in Supplemental Table 1), as well as a supplementary search using terms for indoor risk factors in relation to asthma or wheeze (detailed search strategies in Supplemental Table 2).

Study selection and data collection

Titles, abstracts, and full article texts, as appropriate, of the relevant studies identified from the systematic literature search were independently screened by one reviewer (WL) and checked for accuracy by a second reviewer (TF or EA). Eligible primary studies identified directly through screening of the literature search results, as well as any additional primary studies identified from the reference lists of relevant reviews, were included. Non-eligible studies were excluded and the reasons were documented. Any disagreement between the two reviewers was noted and resolved through discussion.

For each included study, one reviewer (e.g., TF or EA) independently extracted data (e.g., study characteristics, study results); this was checked for accuracy by the other reviewer (e.g., EA or TF). Any

disagreement between the two reviewers was noted and resolved through discussion. If an included study only reported crude comparisons of exposure or outcome distributions or only generated crude effect estimates without adjusting for potential confounders, we briefly summarized the study for completeness, but did not tabulate the study information because we determined its results were unreliable for causal inference.

If a study reported multiple exposure-outcome pairs (i.e., NO_2-asthma, NO_2-wheeze, gas-asthma, gas-wheeze) of interest, each exposure-outcome pair was recorded as a separate record. If multiple effect estimates were reported for a single exposure-outcome pair, only the most fully adjusted one was extracted, unless the purpose of the most adjusted model was to evaluate potential mediation, effect modification, or sensitivity of the main study result, in which case a less adjusted one was extracted instead. In addition, if subgroup effect estimates were available and they differed meaningfully (e.g., the association was statistically significant in one subgroup but not the other), we extracted those data, as well.

Study quality evaluation

An evaluation of study quality was conducted to determine how reliable the results of each study are for addressing the corresponding research question. For each individual study included in the review and for each exposure-outcome pair, specific aspects of study quality were ranked as "high" or "low" according to a set of pre-determined criteria, as shown in Table 2. The ranking was independently performed by one reviewer (e.g., TF or EA) and then checked for accuracy by the other reviewer (e.g., EA or TF). Any disagreement between the two reviewers was noted and resolved through discussion. If an included study did not report adjusted effect estimates, its study quality was not tabulated.

Evidence synthesis

Evidence for each exposure-outcome pair was synthesized separately, taking into consideration study quality and heterogeneity across studies. Owing to the differences in the specific definitions of asthma and wheeze outcomes across studies, we classified asthma outcomes into three general categories (i.e., newly diagnosed asthma, ever-diagnosed asthma, and asthma exacerbation) and wheeze outcomes into two general categories (i.e., persistent wheeze and any wheeze). Within each exposure-outcome pair, studies that fell into the same health outcome category were considered more homogeneous (i.e., more likely to be examining the same underlying exposure-outcome relationship) than those that fell into different health outcome categories.

Evidence synthesis was performed within each health outcome category, as well as across all categories for comparison purposes. If several included studies were conducted in the same population, we primarily relied on the most recent study or the study reporting the most informative data for that population (e.g., greater population coverage, improved exposure estimates, and/or improved statistical analysis) for evidence synthesis. In addition, we explored potential heterogeneity of results by factors such as age group (e.g., ≤6 vs. >6–10 vs. >10 years), sex (male vs. female), study region (e.g., Europe vs. North America vs. Asia-Pacific), publication year (e.g., before 2013 vs. 2013 or later), study design (e.g., cohort vs. case-control vs. cross-sectional), and exposure contrast for gas cooking (e.g., gas vs. electric cooking, gas cooking/stove vs. not).

Guided by the Bradford Hill [4] considerations, we determined the overall plausibility of causality of the association between gas cooking or indoor NO_2 and asthma or wheeze through evaluations of strength of association, consistency, specificity, temporality, dose-response, biological plausibility, coherence, experiment, and analogy, taking into account study quality and associated possible non-causal explanations (i.e., information bias, confounding, selection bias, and reverse causation). The reporting of findings in this review follows the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist [71].

Results and discussion

Study selection

From the literature searches, we identified 1655 records from PubMed, 369 from Scopus, and 153 from Lens.org. Before title/abstract screening, 333 records were excluded because they were either duplicate records (n = 303) or records not in English (n = 30). Among the remaining 1844 records that were screened by title/abstract, 195 were kept and further screened by full-text, and 66 were eventually included in this review. Reasons for exclusion at each step are detailed in Fig. 1. The 66 studies included in this review contain all 41 studies that were previously included in the Lin et al. [93] meta-analysis, 5 studies that were published before 2013 but not captured in Lin et al. [93], and 20 new studies that were published since Lin et al. [93].

Table 2
Criteria for study quality evaluation.

Aspect		Criteria for high quality	Criteria for low quality
Exposure Assessment	NO_2	Objective, direct measure	Estimated or indirect measure
	Gas cooking	Objective measure (e.g., observed presence of gas stove)	Self-reported
Outcome Assessment	Asthma	Diagnosed, self-reported physician diagnosed, or self-reported and validated clinically	Self-reported symptoms
	Wheeze	Objective measure or short recall period (i.e., within 1 month) if self-reported	Self-reported with long (i.e., >1 month) recall period
Adjustment for Confounders		Adjusted for key confounders, including environmental tobacco smoke, family history of asthma/allergies/atopy, SES/home environment (e.g., dust mite, cockroach, pets, mold, wood stove, dampness, heating fuels, crowdedness, pillow/quilt/mattress, form of cooling), and outdoor NO_2 (e.g., season, region, traffic) (for NO_2 studies only) ([46,69])	Failed to adjust for key confounders
Sample Selection		No obvious sources of selection bias	Had obvious sources of selection bias: cohort – lost to follow-up (>25%); case-control – control selection; cross-sectional – inclusion/exclusion criteria, missing data (>25%)
Temporality		Exposure was measured before health outcome	Exposure was measured after or at the same time as the health outcome

Notes
NO_2 = Nitrogen Dioxide; SES = Socioeconomic Status.

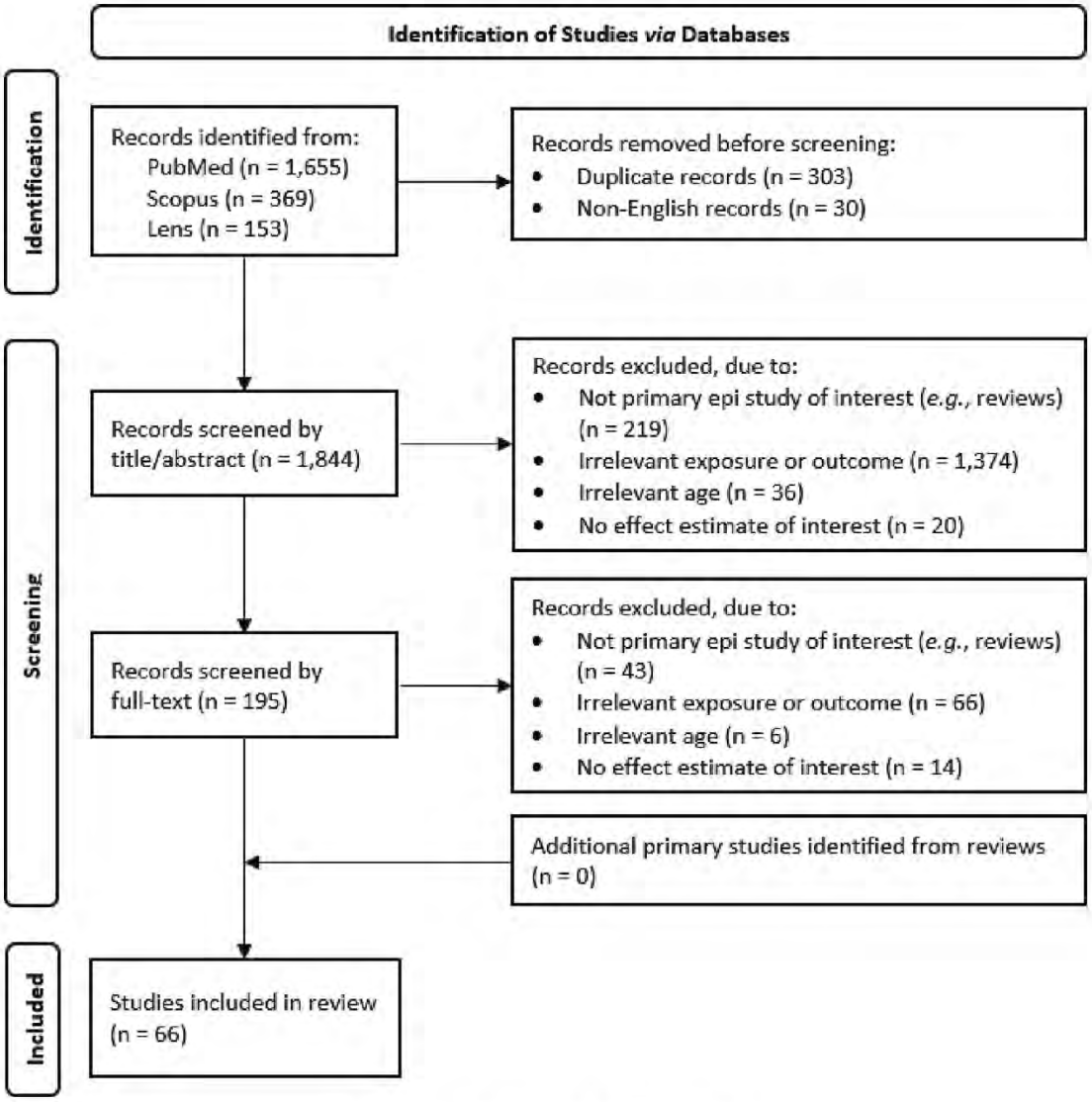

Fig 1. Flow chart of study selection process.

Gas cooking and asthma

We identified 29 studies that evaluated the association between gas cooking and asthma. In 6 of these studies [27,31,77,82,95,98], the authors only performed crude comparisons of exposure or outcome distributions or only generated crude effect estimates without adjusting for potential confounders. Of the 6 studies, 3 [27,31,95] compared the prevalence of gas cooking exposure between children with vs. without asthma, and the authors did not find statistically significant differences. The other 3 studies [77,82,98] compared the prevalence of asthma between children in homes using gas vs. electricity for cooking, and the authors did not find statistically significant differences. These crude comparison results are not reliable for causal inference. In addition, 1 study [68] performed adjusted statistical analysis but only reported that the gas cooking-asthma association was not statistically significant (i.e., $P \geq 0.05$) without specifying either the point estimate or associated 95% CI. The following discussions focus on the remaining 22 studies that generated specific confounder-adjusted effect estimates. Of the 22 studies, 15 were included in Lin et al. [93] and 7 were published since Lin et al. [93] (Table 3, Supplemental Tables 3–4).

As shown in Table 3 and Supplemental Table 3, the 22 studies are highly heterogeneous. Most of the studies used a cross-sectional design ($n = 16$) and few used a cohort design ($n = 4$) or a case-control design ($n = 2$). Notably, all 7 studies published since Lin et al. [93] used a cross-sectional study design. Of the 22 studies, 4 were conducted in Australia, 4 in China, 3 in Canada, 3 in Germany, 2 in Netherlands, 1 in Nigeria, 1 in Russia, 1 in Uganda, 1 in the United Kingdom [UK], 1 in the United States of America [USA], and the remaining study by Wong et al. [34] was conducted globally across 47 countries. Notably, while 10 of the 15 studies captured in Lin et al. [93] were conducted in North America or Europe, the 7 studies published since Lin et al. [93] consist of 4 studies in Asia (China), 2 studies in Africa (Nigeria and Uganda), and the global study by Wong et al. [34] that covers Africa, Asia-Pacific, Eastern Mediterranean, Indian subcontinent, Latin America, North America, Northern and Eastern Europe, Oceania, and Western Europe regions. Other than the global study that included over 250,000 children, the largest study is Norbäck et al. [19], which included over 39,000 children the sample sizes of the other studies were between 100 and 10,000. The study population overlapped between Lin et al. [94] and Willers et al. [87]. The study periods are between 1988 and 2018. The ages of children in these studies vary considerably between 0 and 19 years. While some studies (e.g., Eghomwanre et al. [5], Tavernier et al. [32], Hessel et al. [75]) examined wide ranges of ages (>10 years), other studies (e.g., Ponsonby et al. [7], Volkmer et al. [81], Willers et al. [87], Behrens et al. [88]) focused on very specific ages (within 2 years). All studies included both boys and girls. The majority of the studies were

Table 3
Epidemiology studies of gas cooking (exposed vs. unexposed) and asthma.

Citation	Study design	Country	Age (Years)	Sample size	% Exposed	Measure of association	Effect estimate	95% CI	Quality				
									E	O	C	S[3]	T
Ever diagnosed asthma													
Lin et al. [43]	Cross-sectional	China	5-13	2306	94	OR	3.18	0.42–24.23	L	H	H	H	L
Norback et al. [19]	Cross-sectional	China	3-6	39,782	75	OR	0.93	0.80–1.08	L	H	H	H	L
Casas et al. [57]	Cross-sectional	Germany	0–10	3222	12	OR	1.33	0.88–2.00	L	H	H	L[4]	L
Garrett et al. [70]	Cross-sectional	Australia	7-14	148	NR	OR	**2.23**	**.06–4.72**	H	H	L	L[3]	L
Holscher et al. [10]	Cross-sectional	Germany	5-14	2162	49	OR	0.59	0.26–1.33	L	H	L	H	L
McConnell et al. [79]	Cohort	USA	9–16	3535	77	HR (Ever wheeze)	1.20	0.70–2.00	L	H	L	L[1]	H
						HR (Never wheeze)	1.30	0.80–2.00					
Ponsonby et al. [7]	Cohort	Australia	7	851	1	RR (Adj. for family history)	1.44	0.85–2.45	L	L	L	H	H
						RR (Adj. for ETS)	**1.8**	**1.06–3.17**					
Zhang et al. [67]	Cross-sectional	China	1-8	2193	58[c]	OR	1.44	0.97–2.14	L	H	L	H	L
Hwang et al. [86]	Cross-sectional	China	3-6	2214	88	OR	**2.34**	**1.04–5.21**	L	H	L	L[4]	L
Volkmer et al. [81]	Cross-sectional	Australia	4-5	8154	41	OR (Adelaide)	**1.24**	**1.07–1.42**	L	L	L	H	L
Ponsonby et al. [8]	Cross-sectional	Australia	9-10	344	32	RR	1.20	0.91–1.58	L	L	L	L[4]	L
Wong et al. [34]	Cross-sectional	Global (47 countries)	6–7	97,726	74	OR	0.94	0.88–1.02	L	L	L	L[4]	L
			13–14	154,287	66		0.99	0.93–1.05					
Newly diagnosed asthma													
Lin et al. [94]	Cohort	Netherlands	0–8	3590	87	OR	1.10	0.85–1.43	L	H	H	H	H
Carlsten et al. [13] [d]	Cohort	Canada	0–7	380	10	OR	1.40	0.60–3.60	H	H	L	L[1]	H
Nantanda et al. [80] [e]	Cross-sectional	Uganda	0-5	614	2	OR	**3.80**	**1.20–13.30**	L	H	H	L[4]	L
Tavernier et al. [32]	Case-control	UK	4-17	200	NR	OR	0.69	0.24–1.95	L	H	L	L[2]	L
Asthma exacerbation													
Lin et al. [94]	Cohort	Netherlands	0–8	3590	87	OR	1.19	0.86–1.65	L	H	H	H	H
Behrens et al. [88]	Cross-sectional	Germany	6-7	2989	11	PR (Boys)	NE	–	L	H	H	H	L
						PR (Girls)	0.77	0.17–3.46					
Eghomwanre et al. [5]	Cross-sectional	Nigeria	≤17	304	NR	OR	2.00	0.25–15.64	L	H	H	H	L
						OR (Asthma)	**2.28**	**1.04–5.01**					
Spengler et al. [47]	Cross-sectional	Russia	8-12	5951	80	OR (Asthma-like symptoms)	1.19	0.94–1.52	L	H	H	H	L
Dekker et al. [14]	Cross-sectional	Canada	5–8	9841	5	OR	**1.95**	**1.41–2.68**	L	H	L	H	L
Garrett et al. [70]	Cross-sectional	Australia	7-14	148	NR	OR	1.73	0.77–3.90	H	H	L	L[3]	L
Hessel et al. [75]	Case-control	Canada	5-19	1035	5-7	OR	1.70	1.00–3.10	L	H	L	L[2]	L
Willers et al. [87]	Cross-sectional	Netherlands	4-5	2611	78	OR	1.50	0.90–2.49	L	L	H	L[4]	L
Wong et al. [34]	Cross-sectional	Global (47 countries)	6–7	97,726	74	OR	0.97	0.87–1.09	L	L	L	L[4]	L
			13–14	154,287	66		0.97	0.89–1.07					

Notes
– = Not Applicable; C = Adjustment of Confounders; CI = Confidence Interval; E = Exposure Assessment; ETS = Environmental Tobacco Smoke; H = High; HR = Hazard Ratio; L = Low; NE = Not Estimable; NR = Not Reported; O = Outcome Assessment; OR = Odds Ratio; PR = Prevalence Ratio; RR = Relative Risk; S = Sample Selection; SIDS = Sudden Infant Death Syndrome; T = Temporality; UK = United Kingdom; USA = United States of America.
Statistically significant results are bolded.
[a] L^1 = cohort study, lost to follow-up (>25%); L^2 = case-control study, control selection; L^3 = cross-sectional study, inclusion/exclusion criteria; L^4 = cross-sectional study, missing data (>25%).
[b] Study conducted among children at high-risk for SIDS.
[c] Calculated based on information provided in the study.
[d] Study conducted among children at high-risk for asthma (having family history of asthma or allergies).
[e] Study conducted among children with cough and/or difficulty in breathing plus fast breathing.

conducted in the general population, except three that were conducted among children with preexisting conditions (i.e., high risk for asthma in Carlsten et al. [13], high risk for sudden infant death syndrome [SIDS] in Ponsonby et al. [7], and cough and/or difficulty in breathing in Nantanda et al. [80]).

The measurement of gas cooking exposure relied on objective observation of the presence of gas cooking stove in only 2 studies [13,70]; all 20 other studies relied on self-reported information that was typically collected at one point in time. The definition of gas cooking exposure varied from ever use gas for cooking to generally/primarily use gas for cooking to presence of gas cooking stove. Few studies further specified whether "gas" was referring to natural gas (methane) or liquefied petroleum gas (LPG) (propane/butane). The prevalence of gas cooking exposure varied substantially across studies, ranging from 1.3% in Ponsonby et al. [7] to 94.09% in Lin et al. [43]. Among studies conducted in the same country, the prevalence of gas cooking exposure also varied – from 1.30% to 40.50% in Australia, from 58% to 94.09% in China, from 3.9% to 10% in Canada, from 10.90% to 48.70% in Germany, and from 77.90% to 86.50% in Netherlands. The measurement of asthma outcome relied on physician diagnosis during the study in only 2 studies [13,80]; all of the 20 other studies relied on self-reported information, among which most were self-reported physician diagnosis.

The definition of asthma outcome varied substantially across studies, with >10 different definitions used.

As shown in Table 3 and Supplemental Table 4, all studies examined gas cooking exposure as a binary variable, but the exposure contrast varied (e.g., gas vs. no gas, gas vs. electricity). There were 12 studies that examined ever-diagnosed asthma as the outcome type, 4 studies that examined newly diagnosed asthma, and 9 studies that examined asthma exacerbation. Overall, 15 of the 22 studies reported null results (i.e., results that are not statistically significant, regardless of the point estimate) and the other 7 studies [7,14,47,70,80,81,86] reported statistically significantly positive associations between gas cooking and asthma, with point estimates ranging from 1.24 to 3.80. Of the 7 studies that reported statistically significant findings, 3 [70,81,86] are cross-sectional studies of ever-diagnosed asthma, 1 [7] is a cohort study of ever-diagnosed asthma, 1 [80] is a cross-sectional study of newly diagnosed asthma (at time of study), and 2 [14,47] are cross-sectional studies of asthma exacerbation. Of these 7 studies, 3 [7,70,81] were conducted in Australia, whereas the other 4 studies were conducted in Russia [47], Canada [14], China [86], and Uganda [80], respectively. Of these 7 studies, 5 were captured in Lin et al. [93] and 2 were published since Lin et al. [93]. The newer studies generally adjusted for larger numbers of potential confounders than the studies captured in Lin et al. [93]. Given the high heterogeneity across the studies in this literature, we do not consider a meta-analysis to be appropriate for evidence synthesis.

The quality of studies in this literature is generally low, as a large proportion of the studies are subject to multiple sources of biases. As shown in Table 3, 20 of the 22 studies have low quality with respect to gas cooking exposure assessment, 18 studies cannot establish the temporal link between gas cooking and asthma, 13 studies have low quality with respect to confounding adjustment, 11 studies are prone to selection bias, and 5 studies have low quality with respect to asthma outcome assessment (studies that examined multiple outcome types were each only counted once). The 7 newer studies (all cross-sectional by design) are of similar quality as compared to the cross-sectional studies that were included in Lin et al. [93]. The distribution of study quality is also similar across asthma outcome types.

Temporality is a key aspect in both study quality evaluation and causal inference guided by the Bradford Hill [4] considerations (discussed below). Among the three different study designs (i.e., cohort, case-control, and cross-sectional) that have been used in this literature, the cohort study design is the only study design that could establish temporality between measured exposure vs. outcome and therefore it is the most reliable for making causal inference [51,84]. However, as noted above, only 4 of the 22 studies on which this review focused used a cohort study design that could establish temporality. These cohort studies mostly reported null results. Specifically, Carlsten et al. [13] examined the association between the presence of a gas cooking stove (yes vs. no) and newly diagnosed asthma in Canadian children aged 0–7 years and reported null result (OR = 1.40; 95% CI: 0.60–3.60) after adjusting for the key confounders (family history and socioeconomic status [SES]/home environment) as well as several other potential confounders. Lin et al. [94] examined the association between ever using gas for cooking (vs. never) and newly diagnosed asthma and asthma exacerbation, respectively, in Dutch children aged 0–8 years and reported null results (OR = 1.10, 95% CI: 0.85–1.43 and OR = 1.19; 95% CI: 0.86–1.65, respectively) after adjusting for all key confounders (environmental tobacco smoke [ETS], family history, and SES/home environment) as well as several other potential confounders. McConnell et al. [79] examined the association between the presence of a gas cooking stove (yes vs. no) and ever-diagnosed asthma in American children aged 9–16 years and reported null results (hazard ratio [HR] = 1.20; 95% CI: 0.70–2.00 among the subgroup with ever wheeze at baseline and HR = 1.30; 95% CI: 0.80–2.00 among the subgroup with never wheeze at baseline), after adjusting for the key confounder (SES) as well as several other potential confounders. Ponsonby et al. [7] examined the association between the presence of a gas cooker (yes vs. no) and ever-diagnosed asthma in Australian children aged 7 years and reported null result (relative risk [RR] = 1.44, 95% CI: 0.85–2.45) after adjusting for key confounder family history alone, but a statistically significantly positive association (RR = 1.84; 95% CI: 1.06–3.17) after adjusting for key confounder ETS exposure alone. While a positive finding (RR = 1.84; 95% CI: 1.06–3.17) was reported by Ponsonby et al. [7], it should not be overinterpreted given the fact that this positive finding was sensitive to confounder adjustment choices made within the study. It is also worth noting that the study by Ponsonby et al. [7] was conducted in a very specific subgroup of the population (children at high risk for SIDS) who were rarely exposed to gas cooking (prevalence = 1.30%), so the study results have limited generalizability to the general population. There is also large variability across the 4 cohort studies in terms of study region (four different countries), age of children (four different age ranges), gas cooking exposure definition (three measured presence of gas cooking stove vs. one measured ever use gas for cooking), and asthma outcome indicator (two effect estimates on newly diagnosed asthma, two on ever-diagnosed asthma, and one on asthma exacerbation), indicating that the only few cohort studies available to date are not necessarily examining the same underlying gas cooking-asthma relationship.

Guided by the Bradford Hill [4] considerations, we further discuss causal inference for the association between gas cooking and asthma in detail in Table 7. Overall, the epidemiology studies, including those with positive findings, largely cannot establish temporality. There are no large and precise effect estimates, and the observed associations lack consistency and specificity. An exposure-response relationship was not observed in the one study that evaluated it. No study has examined whether or how asthma risk or severity would change after removing or reducing gas cooking exposure. Experimental evidence is limited and does not sufficiently support any of the observed associations in epidemiology studies. We did not find a suitable analogy to address causality in this case. Taken together, we conclude that the evidence does not support causality.

In the meta-analysis by Lin et al. [93], all identified studies at the time were included, regardless of whether the studies reported adjusted effect estimates (vs. just performed crude comparisons), the study design, or study quality. Summarizing across these studies, Lin et al. [93] reported a statistically significantly positive association between gas cooking and asthma (OR = 1.32, 95% CI: 1.18–1.48). However, our systematic review shows that the literature to date, including the literature meta-analyzed by Lin et al. [93], is limited by the lack of reliable study designs (e.g., cohort), high heterogeneity across studies, and low study quality (primarily with respect to exposure assessment, temporality, confounding adjustment, and sample selection). Our detailed causal inference guided by the Bradford Hill [4] considerations also shows that the evidence does not support causality. As a result, the effect estimates from Lin et al. [93] should be interpreted with caution.

Gas cooking and wheeze

We identified 37 studies that evaluated the association between gas cooking and wheeze. In 6 of these studies [5,37,38,77,82,95], no adjusted effect estimates were reported. Of the 6 studies, 1 [95] compared the prevalence of gas cooking exposure between children with vs. without wheeze and did not find a statistically significant difference. The other 5 studies compared the prevalence of wheeze between gas cooking exposure groups, 4 of which [5,37,38,77] did not find statistically significant differences and 1 of which [82] observed a statistically significantly higher prevalence of wheeze among girls exposed to gas cooking (vs. electricity cooking, $P < 0.005$), but not among boys. These crude comparison results are not reliable for causal inference. In addition, 1 study [68] performed adjusted statistical analysis but only reported that the gas cooking-wheeze association was not statistically significant (i.e., $P \geq 0.05$) without specifying either the point estimate or

associated 95% CI. The following discussions focused on the remaining 30 studies that generated confounder-adjusted effect estimates. Of the 30 studies, 23 were included in Lin et al. [93] and 7 were published since Lin et al. [93] (Table 4, Supplemental Tables 5–6).

As shown in Table 4 and Supplemental Table 5, the 30 studies are highly heterogeneous. Most of the studies used a cross-sectional design ($n = 25$) and a few used a cohort design ($n = 3$) or a case-control design (n = 2). Similar to the studies captured in Lin et al. [93], the newer studies were predominantly cross-sectional by design (all but 1 cohort study). Of the 30 studies, 7 were conducted in China, 5 in USA, 4 in UK, 3 in Australia, 3 in Germany, 2 in Netherlands, 1 in Austria, 1 in Canada, 1 in New Zealand, 1 in Russia, 1 in South Africa, and the remaining study by Wong et al. [34] was conducted globally across 47 countries. Notably, while 16 of the 23 studies captured in Lin et al. [93] were conducted in North America or Europe, 5 of the 7 studies published since Lin et al. [93] were conducted in China. Other than the global study that included over 250,000 children, the largest study is Norbäck et al. [19], which included over 39,000 children; the sample sizes of the other studies were between 100 and 29,000. The study population overlapped between Lin et al. [94] and Willers et al. [87] and between Norbäck et al. [19] and Norbäck et al. [20]. The study periods were between 1978 and 2018. The ages of children in these studies vary considerably, between 0 and 18 years. While some studies (e.g., Belanger et al. [53][, Casas et al. [57]) examined wide ranges of ages (≥10 years), other studies (e.g., Wong et al. [35], Belanger et al. [55]) focused on very specific ages (within 2 years). All studies included both boys and girls. The majority of the studies were conducted in the general population, except 2 that were conducted among children with preexisting conditions (i.e., high risk for asthma in Belanger et al. [55], and active asthma in Belanger et al. [53]).

Similar to the gas cooking and asthma literature, most of the studies measured gas cooking based on self-reported information that was typically collected at one point in time, and only 2 studies [50,70] used objective measurements (e.g., observed presence of gas stove). The definition of gas cooking exposure varied from ever use gas for cooking to generally/primarily use gas for cooking to presence of gas cooking stove. Few studies further specified whether "gas" was referring to natural gas (methane) or LPG (propane/butane). The prevalence of gas cooking exposure varied substantially across studies, ranging from 2.2% in Wong et al. [35] to 94.09% in Lin et al. [43]. Among studies conducted in the same country, the prevalence of gas cooking exposure also varied – from 2.2% to 94.09% in China, from 23.5% to 46.4% in USA, from 57.83% to 82.02% in UK, from 31.80% to 40.50% in Australia, from 10.90% to 48.70% in Germany, and from 77.90% to 86.50% in Netherlands. The measurements of all wheeze outcomes relied on self-reported information, and the definition of wheeze outcome varied substantially across studies. Other than "wheeze, past year" that was common across 12 studies, all other wheeze outcome definitions were unique to each study.

As shown in Table 4 and Supplemental Table 6, all studies examined gas cooking exposure as a binary variable, but the exposure contrast varied (e.g., gas vs. no gas, gas vs. electricity). Of the 30 studies, 21 examined any wheeze as the only outcome type, 4 studies examined persistent wheeze only, and 5 studies examined both any wheeze and persistent wheeze. Only a few studies examined very specific wheeze outcomes, such as duration of wheezing, speech-limiting wheeze, sleep disturbance due to wheeze, exercise-induced wheeze, wheeze with/without colds (results not shown). Overall, 23 of the 30 studies reported null results; 6 of the other 7 studies reported statistically significantly positive associations between gas cooking and wheeze [29,35,36,45,53,81], with point estimates ranging from 1.16 [81] to 2.27 [53], and 1 study reported statistically significantly inverse associations [88], with a point estimate of 0.55. All 7 studies that reported statistically significant results were cross-sectional by design and examined any wheeze as the health outcome type. None of the studies examining persistent wheeze reported statistically significant findings. Of the 6 studies that reported statistically significantly positive findings, 2 [35,36] were conducted in China, whereas the other 4 studies were conducted in USA [53], UK [29], Australia [81], and South Africa [45], respectively. All of these studies were captured in Lin et al. [93] except for Shirinde et al. [45], which was published since Lin et al. [93]. The newer studies generally adjusted for more potential confounders than the studies captured in Lin et al. [93]. Given the high heterogeneity across the studies in this literature, we do not consider a meta-analysis to be appropriate for evidence synthesis.

The quality of studies in this literature is generally low, as a large proportion of the studies are subject to multiple sources of biases. As shown in Table 4, 28 of the 30 studies have low quality with respect to gas cooking exposure assessment, 26 studies have low quality with respect to wheeze outcome assessment, 26 studies cannot establish the temporal link between gas cooking and wheeze, 18 studies have low quality with respect to confounding adjustment, and 12 studies are prone to selection bias (studies that examined multiple outcome types were each only counted once). The 7 newer studies are of similar quality as compared to the studies that were included in Lin et al. [93]. The distribution of study quality is also similar across wheeze outcome types.

Temporality is a key aspect in both study quality evaluation and causal inference guided by the Bradford Hill [4] considerations (discussed below). Among the 30 studies that this review focused on, only 3 used a cohort study design that could establish temporality, which is more reliable for making causal inference than a case-control design (used by 2 studies) or a cross-sectional design (used by the remaining 25 studies) [51,84]. These cohort studies all reported null results. Specifically, Lin et al. [94] examined the association between ever using gas for cooking (vs. never) and wheeze in the past 12 months, early transient wheeze, late onset wheeze, and persistent wheeze, respectively, in Dutch children aged 0–8 years and reported null results (OR = 1.10, 95% CI: 0.92–1.31; OR = 0.95, 95% CI: 0.74–1.23; OR = 0.75, 95% CI: 0.47–1.22; OR = 1.42, 95% CI: 0.93–2.17 respectively), after adjusting for all key confounders (ETS, family history, SES/home environment) as well as several other potential confounders. Samet et al. [50] examined the association between the presence of a gas cooking stove and wheezing during a respiratory illness episode in American children aged 1–1.5 years and reported null result (OR = 0.84, 95% CI: 0.64–1.09) after adjusting for all key confounders (ETS, family history, SES/home environment) as well as several other potential confounders. Yu et al. [40] examined the association between using gas for cooking and new onset wheeze in Chinese children aged 0–1.5 years and reported null result (HR = 3.24, 95% CI: 0.782–13.388), after adjusting for key confounders (family history and SES/home environment) as well as several other potential confounders. There is also large variability across the 3 cohort studies in terms of study region (three different countries), age of children (two different age ranges), gas cooking definition (use gas for cooking vs. presence of gas stove), and wheeze outcome indicator (three different indicators), indicating that the only few cohort studies available to date are not necessarily examining the same underlying gas cooking-wheeze relationship.

Guided by the Bradford Hill [4] considerations, we further discuss causal inference for the association between gas cooking and wheeze in detail in Table 7. Overall, most of the epidemiology studies, including all of those with positive findings, cannot establish temporality. There are no large and precise effect estimates, and the observed associations lack consistency and specificity. An exposure-response relationship was not observed in the one study that evaluated it. No study has examined whether or how wheeze symptom would change after removing or reducing gas cooking exposure. Experimental evidence is limited and does not sufficiently support any of the observed associations in epidemiology studies. We did not find a suitable analogy to address causality in this case. Taken together, we conclude that the evidence does not support causality.

In the meta-analysis by Lin et al. [93], all identified studies at the time were included, regardless of whether the studies reported adjusted effect estimates (vs. just performed crude comparisons), the study

Table 4
Epidemiology studies of gas cooking (exposed vs. unexposed) and wheeze.

Citation	Study design	Country	Age (Years)	Sample size	% Exposed	Measure of association	Effect estimate	95% CI	Quality				
									E	C	C	S[a]	T
Any wheeze													
Samet et al. [50]	Cohort	USA	0–1.5	1205	85[b]	OR	0.84	0.64–1.09	H	H	H	H	H
Lin et al. [94]	Cohort	Netherlands	0–8	3590	87	OR (Wheeze last year)	1.10	0.92–1.31	L	L	H	H	H
						OR (Early transient wheeze)	0.95	0.74–1.23					
						OR (Late onset wheeze)	0.75	0.47–1.22					
Yu et al. [40]	Cohort	China	0–1.5	544	91	HR	3.24	0.71–13.39	L	H	L	H	H
Behrens et al. [88]	Cross-sectional	Germany	6-7	2947	11	PR (Boys)	**0.55**	**0.31–0.98**	L	L	H	H	L
						PR (Girls)	1.52	0.93–2.47					
Belanger et al. [55] [c]	Cross-sectional	USA	0-1	849	34	OR (With maternal asthma)	1.03	0.59–1.79	L	H	H	L[4]	L
						OR (Without maternal asthma)	1.28	0.88–1.86					
Lin et al. [45]	Cross-sectional	China	5-13	2306	94	OR	NE	–	L	L	H	H	L
Norbäck et al. [19]	Cross-sectional	China	3-6	39,782	75	OR	1.02	0.93–1.13	L	L	H	H	L
Norbäck et al. [20]	Cross-sectional	China	3-6	17,679	75	OR (Baseline wheeze prevalence)	1.10	0.91–1.33	L	L	H	H	L
						OR (Wheeze onset)	1.03	0.86–1.22					
						OR (Wheeze remission)	0.95	0.65–1.41					
Spengler et al. [47]	Cross-sectional	Russia	8-12	5951	80	OR	1.06	0.86–1.31	L	L	H	H	L
Wong et al. [36]	Cross-sectional	China	2-6	3089	87	OR	**1.68**	**1.03–2.75**	L	L	L	H	H
Zacharasiewicz et al. [3]	Cross-sectional	Austria	6-9	28,747	NR	OR	1.16	0.92–1.46	L	L	H	H	L
Belanger et al. [53] [d]	Cross-sectional	USA	<12	728	55	OR (Multifamily housing)	**2.27**	**1.15–4.47**	L	H	L	L[4]	L
					24	OR (Single-family housing)	0.61	0.35–1.05					
Burr et al. [72]	Cross-sectional	UK	12-14	25,393	61	OR	1.03	0.97–1.10	L	L	L	H	L
Garrett et al. [70]	Cross-sectional	Australia	7-14	148	NR	OR	1.79	0.80–3.99	H	L	L	L[3]	L
Holscher et al. [10]	Cross-sectional	Germany	5-14	2061	49	OR	1.09	0.90–1.33	L	L	L	H	L
Huang et al. [86]	Cross-sectional	China	3-6	2214	88	OR	1.26	0.86–1.86	L	L	H	L[4]	L
Mitchell et al. [24]	Cross-sectional	New Zealand	6–7	10,810	16	OR	0.93	0.81–1.07	L	L	L	H	L
Shirinde et al. [45]	Cross-sectional	South Africa	13–14	1113	8	OR (Kempton Park)	**1.65**	**1.04–2.61**	L	L	L	H	L
						OR (Ever wheeze, Adelaide)	NS	Non sig					
Volkmer et al. [31]	Cross-sectional	Australia	4-5	8154	41	OR (Wheeze last year, Adelaide)	**1.16**	**1.01–1.32**	L	L	L	H	L
				2276		OR (Current wheeze)	0.99	0.74–1.32					
				2311		OR (Transient early wheeze)	0.91	0.73–1.14					
Willers et al. [87]	Cross-sectional	Netherlands	4-5	1718	78	OR (Late-onset wheeze)	0.89	0.48–1.67	L	L	H	L[4]	L
					2	OR (Exposed during infancy but not at present)	1.53	0.61–3.80[e]					
					19	OR (Exposed at present only)	1.40	0.85–2.31[e]					
Wong et al. [35]	Cross-sectional	China	10	8323	66	OR (Exposed at present and during infancy)	**2.00**	**1.29–3.09**[e]	L	L	L	H	L
Butland et al. [12]	Case-control	UK	7.5–8.5	791	75–82	OR (Infrequent wheeze)	1.34	0.93–1.95	L	L	L	L[2]	L
				949		OR (All wheeze)	1.34	0.95–1.89					
de Bilderling et al. [29]	Cross-sectional	UK	16–18	1868	58	OR (Childhood)	**1.47**	**1.05–2.04**	L	L	L	L[4]	L
					62	OR (Adolescence)	1.06	0.74–1.50					
Ekwo et al. [25]	Cross-sectional	USA	6-12	1138	31	OR	0.70	Non-sig	L	L	L	L[4]	L
Ponsonby et al. [8]	Cross-sectional	Australia	9-10	343	32	RR	1.08	0.75–1.55	L	L	L	L[4]	L
Wong et al. [34]	Cross-sectional	Global	6–7	97,726	74	OR	0.96	0.89–1.03	L	L	L	L[4]	L
		(47 countries)	13–14	154,287	66		0.99	0.92–1.07					
Persistent wheeze													
Lin et al. [94]	Cohort	Netherlands	0–8	3590	87	OR	1.42	0.93–2.17	L	L	H	H	H
Lin et al. [43]	Cross-sectional	China	5-13	2306	94	OR	1.76	0.22–14.01	L	L	H	H	L
Casas et al. [57]	Cross-sectional	Germany	0–10	3387	12	OR	1.09	0.76–1.57	L	L	H	L[4]	L
Dekker et al. [14]	Cross-sectional	Canada	5–8	10,185	5	OR	1.04	0.77–1.42	L	L	L	H	L
Strachan and Carey [23]	Case-control	UK	11-16	961	73–74	OR	0.86	0.61–1.23	L	L	L	H	L
Ware et al. [49]	Cross-sectional	USA	6-10	8237	46[f]	OR	0.93–1.07[g]	Non-sig	L	L	L	H	L
Willers et al. [87]	Cross-sectional	Netherlands	4-5	1923	78	OR	1.06	0.76–1.47	L	L	H	L[4]	L
Butland et al. [12]	Case-control	UK	7.5–8.5	541	75–79	OR	1.19	0.68–2.07	L	L	L	L[2]	L
de Bilderling et al. [29]	Cross-sectional	UK	16–18	1868	58	OR (Childhood)	1.02	0.77–1.36	L	L	L	L[4]	L
					62	OR (Adolescence)	0.78	0.58–1.06					

Notes
– = Not Applicable; C = Adjustment of Confounders; CI = Confidence Interval; E = Exposure Assessment; H = High; HR = Hazard Ratio; L = Low; NE = Not Estimable; NR = Not Reported; O = Outcome Assessment; OR = Odds Ratio; PR = Prevalence Ratio; RR = Relative Risk; S = Sample Selection; T = Temporality; UK = United Kingdom; USA = United States of America.
Statistically significant results are bolded.
[a] L^1 = cohort study, lost to follow-up (>25%); L^2 = case-control study, control selection; L^3 = cross-sectional study, inclusion/exclusion criteria; L^4 = cross-sectional study, missing data (>25%).
[b] Pre-determined per study design.
[c] Study was conducted among children at high-risk for asthma (having an asthmatic sibling).
[d] Study was conducted among children with active asthma.
[e] Overall $P = 0.004$ for global test across all categories.
[f] The prevalence was estimated based on numbers provided in the study.
[g] Only range was reported in the study.

design, or study quality. Summarizing across these studies, Lin et al. [93] reported no statistically significant association between gas cooking and wheeze (OR = 1.06, 95% CI: 0.99–1.13), consistent with our conclusion. It is worth noting that our systematic review shows that the literature to date, including the literature meta-analyzed by Lin et al. [93], is limited by the lack of reliable study designs (e.g., cohort), high heterogeneity across studies, and low study quality (primarily with respect to exposure assessment, outcome assessment, temporality, and confounding adjustment). Our detailed causal inference guided by the Bradford Hill [4] considerations also shows that the evidence does not support causality. These should be incorporated in the interpretation of any meta-analysis results for the literature.

Indoor NO_2 and asthma

We identified 20 studies that evaluated the association between indoor NO_2 and asthma. In 6 of these studies [5,28,39,41,76,78], no adjusted effect estimates were reported. Of the 6 studies, 5 compared the distribution of indoor NO_2 exposure between children with vs. without asthma, of which 3 [28,76,78] reported statistically significantly higher indoor NO_2 concentration among children with asthma and 2 [39,41] did not find statistically significant differences. The remaining study by Eghomwanre et al. [5] examined the correlation between indoor NO_2 and clinical asthma and diagnosed asthma, respectively, and in dry and wet season, respectively, and did not find a statistically significant correlation. These crude comparison results are not reliable for causal inference, so the following discussion focuses on the remaining 14 studies that generated confounder-adjusted effect estimates. Of the 14 studies, 8 were included in Lin et al. [93], 2 were published before 2013 but not included in Lin et al. [93], and 4 were published since Lin et al. [93] (Table 5, Supplemental Tables 7–8).

As shown in Table 5 and Supplemental Table 7, the 14 studies are highly heterogeneous. Specifically, 10 of the 14 studies used a cohort design, 2 used a cross-sectional design, and 2 used a case-control design. Of the 14 studies, 7 were conducted in the US, 2 in Australia, 2 in Netherlands, 1 in Canada, 1 in Japan, and 1 in the UK. The sample sizes of the studies were between 30 and 1600, generally much smaller than the gas cooking studies. The study periods were between 1983 and 2014. The ages of children in these studies vary considerably, from 0 to 17 years of age. While some studies (e.g., Tavernier et al. [32], Lu et al. [56]) examined wide ranges of ages (>10 years), other studies (e.g., Hoek et al. [30], Shima and Adachi [64]) focused on very specific ages (within 2 years). All studies included both boys and girls. Unlike the gas cooking studies, a large proportion (8 of 14) of the indoor NO_2 studies for asthma were conducted among children with preexisting conditions, such as at high risk for asthma, active asthma, and moderate/severe asthma; only 6 studies were conducted in the general population. Notably, all 4 studies published since Lin et al. [93] were cohort studies conducted in the USA among children with preexisting conditions.

All studies measured indoor NO_2 using passive samplers, with 6 studies using the tube type samplers, 7 studies using badge type samplers, and 1 study not specifying the sampler type. Of the 14, 7 studies measured indoor NO_2 at a single location within each household, 6 studies measured indoor NO_2 at multiple locations within each household, and 1 study did not specify the measurement location. As for the specific location, 9 of the 14 studies performed measurements in children's bedroom, 6 studies in the kitchen, 6 studies in the living room/main living area, and 2 studies in activity room/dayroom (i.e., room children spent the most time awake). The averaging time for NO_2 measurements ranged from 1 day to 1 year. The mean/median concentrations of indoor NO_2 varied substantially across, as well as between subgroups within, studies, ranging from 11.6 to 109.04 $\mu g/m^3$. Among studies conducted in the same country, indoor NO_2 concentrations also varied – from 16.17 to 109.04 $\mu g/m^3$ in the USA and from 20 to 67 $\mu g/m^3$ in Netherlands. The measurement of asthma outcome relied on physician diagnosis during study in only 1 study [13]. All 13 other

Table 5
Epidemiology studies of NO_2 and asthma.

Citation	Study design	Country	Age (Years)	Sample size	NO^2 exposure		Exposure contrast ($\mu g/m^3$)[a]	Measure of association	Effect estimate	95% CI[c]	Quality				
					Concentration ($\mu g/m^3$)[a]	Location					E	O	C	S[b]	T
Ever diagnosed asthma															
Neas et al. [61]	Cohort	USA	7–11	1567	16–44 (M)	B, D/A, K	per 28.20	OR	0.91	0.60–1.36	H	H	H	H	L
Garrett et al. [70]	Cross-sectional	Australia	7-14	148	12 (Mdn)	B, LR, K	per 10 (B)	OR	1.01	0.75–1.37	H	H	L	L[6]	L
							per 10 (Indoor mean)	OR (Overall)	1.00	0.75–1.31					
								OR (Winter)	0.99	0.84–1.16					
								OR (Summer)	2.52	0.99–6.42					
Hoek et al. [30]	Case-control	Netherlands	6	80	59–67 (M)	B, LR, K	per 10-fold	OR (K)	1.29	0.40–5.40[c]	H	H	L	L[2]	L
								OR (LR)	5.83	0.70–34.90[c]					
								OR (B)	2.68	0.40–16.20[c]					
Newly diagnosed asthma															
Shima and Adachi [64]	Cohort	Japan	9–10	842	35–61 (M)	LR	per 18.80	OR	0.87	0.51–1.43	H	H	H	H	L
Carlsten et al. [13] [d]	Cohort	Canada	0–7	155	21 (M)	B	>18.80 (vs. ≤18.80)	OR	1.80	0.70–4.80	H	H	L	L[1]	H
Tavernier et al. [32]	Case-control	UK	4-17	200	NR	B, LR	Not specified	OR (LR)	0.85	0.51–1.44	H	H	L	L[2]	L
								OR (B)	0.92	0.49–1.71					
Asthma exacerbation															
O'Connor et al. [35] [d]	Cohort	USA	0–7	442	38 (Mdn)	NR	Not specified	OR	0.97	0.75–1.26	H	H	L	H	H
Shima and Adachi [64]	Cohort	Japan	9–10	842	35–61 (M)	LR	per 18.80	OR (4th grade, males)	0.77	0.48–1.20	H	H	H	H	L
								OR (4th grade, females)	**1.63**	**1.06–2.54**					
								OR (5th grade, males)	0.92	0.60–1.39					
								OR (5th grade, females)	**1.67**	**1.06–2.66**					
								OR (6th grade, males)	0.78	0.45–1.30					
								OR (6th grade, females)	1.18	0.62–2.18					
Paulin et al. [62] [e]	Cohort	USA	5–12	30	109 (M)	K	per 10-fold	OR	0.63	0.08–4.72	H	H	L	H	L
Dijkstra et al. [58]	Cross-sectional	Netherlands	6-12	775	20–60 (M)[f]	B, LR, K	21–40 (vs. 0–20)	OR	0.67	0.32–1.41	H	L	L	H	L
							41–60 (vs. 0–20)		0.34	0.08–1.54					
							>60 (vs. 0–20)		0.56	0.15–2.06					
Garrett et al. [70]	Cross-sectional	Australia	7-14	148	12 (Mdn)	B, LR, K	per 10 (B)	OR	1.06	0.77–1.46	H	H	L	L[3]	L
Hansel et al. [73] [g,h]	Cohort	USA	2-6	150	56 (M)	B	per 37.60	RR	1.04	0.97–1.12	H	L	L	H	L
Lu et al. [56] [e]	Cohort	USA	5–17	142	39 (Mdn)	B	per 10-fold	OR (Normal weight)	1.34	0.83–2.14	H	L	L	H	L
								OR (Overweight)	1.42	0.65–3.11					
								OR (Obese)	1.93	0.77–4.82					
Nitschke et al. [63] [g,h]	Cohort	Australia	5-12	174	71 (M of Max)	K	per 18.80	RR (Daytime)	1.00	0.95–1.05	H	L	L	H	L
Schachter et al. [26] [i]	Cohort	USA	6–14	36	45–62 (M)	LR	Not specified	OR (Summer)	1.20	0.40–3.65	H	L	L	H	L
								OR (Winter)	**2.82**	**1.10–7.24**					
							11.32- ≤ 16.69 (vs. ≤11.32)		1.15	0.94–1.42					
							16.69- ≤ 26.88 (vs. ≤11.32)		**1.31**	**1.04–1.66**					
Belanger et al. [54] [e]	Cohort	USA	5–10	1342	20 (M)	B, D/A	>26.88 (vs. ≤11.32)	OR	**1.43**	**1.08–1.88**	H	L	L	L[1]	L
							per 5-fold (if >11.32 threshold)		**1.37**	**1.01–1.89**					

Notes
B = Bedroom; C = Adjustment of Confounders; CI = Confidence Interval; D/A = Dayroom/Activity Room; E = Exposure Assessment; H = High; K = Kitchen; L = Low; LR = Living Room; M = Mean; Max = Maximum; Mdn = Median; NO_2 = Nitrogen Dioxide; NR = Not Reported; O = Outcome Assessment; OR = Odds Ratio; ppb = Parts per Billion; RR = Relative Risk; S = Sample Selection; T = Temporality; UK = United Kingdom; USA = United States of America.
Statistically significant results are bolded.

[a] Converted if ppb was originally used as the unit in the paper.
[b] L^1 = cohort study, lost to follow-up (>25%); L^2 = case-control study, control selection; L^3 = cross-sectional study, inclusion/exclusion criteria; L^4 = cross-sectional study, missing data (>25%).
[c] 90% CI.
[d] Study was conducted among children at high-risk for asthma (i.e., having family history of asthma or allergies).
[e] Study was conducted among children with active asthma.
[f] Estimated from figure in the study.
[g] Published before but not included in Lin et al. [93].
[h] Study was conducted among children with physician-diagnosed asthma.
[i] Study was conducted among children with moderate/severe asthma.

studies relied on self-reported information, among which 4 relied on self-reported physician diagnosis and 3 relied on confirmed/validated clinically. The definition of asthma outcome varied substantially across studies, with 14 distinct definitions used.

As shown in Table 5 and Supplemental Table 8, the exposure contrast for which the effect estimates were calculated varied substantially across studies. Of the 14 studies, 6 calculated effect estimates from a set increment of indoor NO_2 concentration on the original scale, but the actual magnitude varied from 10 $\mu g/m^3$ to 37.6 $\mu g/m^3$; 5 studies examined indoor NO_2 concentration on the log scale; 3 studies categorized indoor NO_2 concentration, each in a unique way; and 3 studies did not specify the exposure contrast. Only 1 study [54] examined the operationalization of indoor NO_2 concentration in more than one way (categorical and continuous). Notably, only 1 study [63], which was captured by our literature search but not included in Lin et al. [93], focused on the examination of maximum rather than average indoor NO_2. In general, 3 of the 14 studies examined ever-diagnosed asthma, 3 studies examined newly diagnosed asthma, and 10 studies examined asthma exacerbation. Few studies examined very specific asthma outcome indicators (e.g., exercise-related asthma, medication use) (results not shown).

Overall, 11 of the 14 studies reported null results; 3 studies reported statistically significantly positive associations between indoor NO_2 and asthma [26,54,64], among which 2 [54,64] were included in Lin et al. [93] and 1 [26] was published since Lin et al. [93]. The magnitudes of the three statistically significant point estimates were not directly comparable with each other, given the different exposure contrasts used. Belanger et al. [54], a cohort study in the US, reported a 1.37 (95% CI: 1.01–1.89) times higher risk of asthma exacerbation per 5-fold increase in NO_2 if greater than a prespecified threshold of 11.32 $\mu g/m^3$. Shima and Adachi [64], a cohort study in Japan, reported a 1.63 (95% CI: 1.06–2.54) times and 1.67 (95% CI: 1.06–2.66) times higher risk of asthma exacerbation per 18.8 $\mu g/m^3$ increment of indoor NO_2 among girls in 4th and 5th grades, respectively. However, null result was observed for 6th grader girls or among boys. Schachter et al. [26], a cohort study in the US, reported a statistically significantly positive association between each interquartile range (not specified) increase of indoor NO_2 concentration and asthma exacerbation in winter (OR = 2.82, 95% CI: 1.10–7.24), but not in summer. Notably, all 3 studies were cohort by design and the statistically significant findings were all for asthma exacerbation as the outcome type. Compared to the studies included in Lin et al. [93], the newer studies generally adjusted for fewer potential confounders. Given the high heterogeneity across the studies in this literature, we do not consider a meta-analysis to be appropriate for evidence synthesis.

The quality of this literature is generally low. Although all studies have high quality with respect to exposure assessment, a large proportion of them are subject to multiple sources of biases. As shown in Table 5, 12 of the 14 studies have low quality with respect to confounding adjustment, 12 studies cannot establish temporality, 6 studies have low quality with respect to asthma outcome assessment, and 5 studies are prone to selection bias (studies that examined multiple outcome types were each only counted once). All 4 studies published since Lin et al. [93] have low quality with respect to confounding adjustment and high quality with respect to sample selection; although all 4 studies are cohort by design, three cannot establish temporality because the measurements of indoor NO_2 in the cohort were not necessarily taken prior to the measurements of asthma outcomes. The distribution of study quality is similar across asthma outcome types, except that, among studies of asthma exacerbation, a larger proportion are of low quality with respect to outcome assessment and a larger proportion are less prone to selection bias.

Temporality is a key aspect in both study quality evaluation and causal inference guided by the Bradford Hill [4] considerations (discussed below). Compared to the gas cooking studies, a much larger proportion (i.e., 10 of 14) of indoor NO_2 studies for asthma used a cohort design, which is in general more reliable for making causal inference than a case-control or cross-sectional design [51,84]. However, 8 of these 10 studies were of low quality with respect to temporality, despite the cohort design, due to the fact that the measurements of indoor NO_2 in the cohort were not necessarily taken prior to the measurements of asthma outcomes. Only the remaining 2 cohort studies [13,33] were of high quality with respect to temporality. Carlsten et al. [13] examined the association between indoor (bedroom) NO_2 and newly diagnosed asthma in Canadian children aged 0–7 years and reported null result after adjusting for key confounders (family history, SES/home environment, and outdoor NO_2) as well as several other potential confounders. O'Connor et al. [33] examined the association between indoor (location not specified) NO_2 and asthma exacerbation in American children aged 0–7 years and reported null result, after adjusting for the key confounder (family history) as well as several other potential confounders. While these 2 cohort studies both focused on children aged 0–7 years in North America region, their health outcomes varied, indicating that the only cohort studies that could establish temporality to date are not necessarily examining the same underlying indoor NO_2-asthma relationship.

Guided by the Bradford Hill [4] considerations, we further discuss causal inference for the association between indoor NO_2 and asthma in detail in Table 7. Overall, most of the epidemiology studies, including all of those with positive findings, cannot establish temporality. There are no large and precise effect estimates, and the observed associations lack consistency and specificity. An exposure-response relationship has not been well-characterized. No study has examined whether or how asthma risk or severity would change after reducing indoor NO2 exposure. Experimental evidence is limited and does not sufficiently support any of the observed associations in epidemiology studies. We did not find a suitable analogy to address causality in this case. Taken together, we conclude that the evidence does not support causality.

In the meta-analysis by Lin et al. [93], all identified studies at the time were included, regardless of whether the studies reported adjusted effect estimates (vs. crude comparisons), the study design, or study quality. Summarizing across these studies, Lin et al. [93] reported no statistically significant association between indoor NO_2 and asthma (OR = 1.09, 95% CI: 0.91–1.31 for a 15-ppb [i.e., 28.2 $\mu g/m^3$] increase in NO_2), consistent with our conclusion. It is worth noting that our systematic review shows that the literature to date, including the literature meta-analyzed by Lin et al. [93], is limited by the lack of consistent findings among studies with reliable study design (e.g., cohort), the high heterogeneity across studies, and the low study quality (primarily with respect to confounding adjustment and temporality). Our detailed causal inference guided by the Bradford Hill [4] considerations also shows that the evidence does not support causality. These should be incorporated in the interpretation of any meta-analysis results for the literature.

Indoor NO_2 and wheeze

We identified 16 studies that evaluated the association between indoor NO_2 and wheeze. In 2 of these studies [5,28], no adjusted effect estimates were reported. Eghomwanre et al. [5] examined the correlation between indoor NO_2 and wheeze in dry and wet season, respectively, and did not find a statistically significant correlation. Cibella et al. [28] compared indoor NO_2 concentrations in spring and winter, respectively, between children with vs. without wheeze and reported statistically significantly higher indoor NO_2 concentrations in each season among children with wheeze (vs. without wheeze, $P = 0.003$). These crude comparison results are not reliable for causal inference. As a result, the following discussion focuses on the remaining 14 studies that generated confounder-adjusted effect estimates. Of the 14 studies, 11 were included in Lin et al. [93], 1 was published before 2013 but not included in Lin et al. [93], and 2 were published since Lin et al. [93] (Table 6, Supplemental Tables 9–10).

Table 6
Epidemiology studies of NO_2 and wheeze.

Citation	Study design	Country	Age (Years)	Sample size	NO_2 Exposure		Exposure contrast ($\mu g/m^3$)[a]	Measure of association	Effect estimate	95% CI	Quality				
					Concentration ($\mu g/m^3$)[a]	Location					E	O	C	S[b]	T
Any wheeze															
Samet et al. [50]	Cohort	USA	0–1.5	1205	19–38 (Mdn)[c]	B, D/A, K	37.60–75.20 (vs. 0–37.60)	OR (Unlagged)	0.92	0.73–1.15	H	H	H	H	H
							>75.20 (vs. 0–37.60)		0.88	0.56–1.37					
							37.60–75.20 (vs. 0–37.60)	OR (Lagged)	0.95	0.75–1.19					
							>75.20 (vs. 0–37.60)		0.98	0.66–1.48					
Yu et al. [40]	Cohort	China	0–1.5	544	42 (M)	B	Not specified	HR	0.99	0.979–1.003	H	H	L	H	H
Belanger et al. [55] [d]	Cross-sectional	USA	0-1	849	>19 (46%)	LR	per 18.80	OR (With maternal asthma)	1.10	0.87–1.40	H	H	L	L[4]	H
								OR (Without maternal asthma)	1.10	0.96–1.25					
Li et al. [9]	Cohort	China	0–1.5	963	NR	NR	Not specified	HR	1.00	0.995–1.001	H	H	L	L[1]	H
Nitschke et al. [63] [e,f]	Cohort	Australia	5-12	174	71 (M of Max)	K	per 18.80	RR (Daytime)	0.98	0.92–1.04	H	H	L	H	L
Shima and Adachi [64]	Cohort	Japan	9–10	842	35–61 (M)	LR	per 18.80	OR (4th grade, males)	0.98	0.68–1.39	H	L	H	H	L
								OR (4th grade, females)	**1.90**	**1.30–2.83**					
								OR (5th grade, males)	0.88	0.59–1.29					
								OR (5th grade, females)	**1.60**	**1.06–2.44**					
								OR (6th grade, males)	0.91	0.58–1.41					
								OR (6th grade, females)	1.23	0.78–1.92					
								OR (Incident wheeze)	0.73	0.45–1.14					
Belanger et al. [53] [g]	Cross-sectional	USA	<12	728	19–43 (M)	LR	per 37.60	OR (Any wheeze, multifamily housing)	**1.52**	**1.04–2.21**	H	H	L	L[4]	L
								OR (Any wheeze, single-family housing)	0.99	0.71–1.38					
								RR (No. wheeze days, multifamily housing)	**1.33**	**1.05–1.68**					
								RR (No. wheeze days, single-family housing)	0.98	0.78–1.22					
Belanger et al. [54] [g]	Cohort	USA	5–10	1342	20 (M)	B, D/A	11.32- ≤ 16.69 (vs. ≤11.32)	OR	1.15	0.90–1.45	H	H	L	L[4]	L
							16.69- ≤ 26.88 (vs. ≤11.32)		**1.44**	**1.11–1.86**					
							>26.88 (vs. ≤11.32)		**1.53**	**1.16–2.02**					
							per 5-fold (if >11.32 threshold)		**1.49**	**1.09–2.03**					
Dijkstra et al. [58]	Cross-sectional	Netherlands	6-12	775	20–60 (M)[c]	B, LR, K	21–40 (vs. 0–20)	OR	0.75	0.40–1.41	H	L	L	H	L
							41–60 (vs. 0–20)		0.36	0.11–1.26					
							>60 (vs. 0–20)		0.94	0.37–2.40					
Esplugues et al. [2]	Cross-sectional	Spain	0-1	352	20 (M)	NR	per 10	OR	1.07	0.86–1.33	H	H	L	L[4]	L
Hoek et al. [30]	Case-control	Netherlands	6	124	59–63 (M)	B, LR, K	per 10-fold	OR (K)	1.45	0.50–4.00[h]	H	H	L	L[2]	L
								OR (LR)	1.57	0.38–6.20[h]					
								OR (B)	1.45	0.37–5.50[h]					
Garrett et al. [70]	Cross-sectional	Australia	7-14	148	12 (Mdn)[i]	B	per 10	OR	1.15	0.85–1.54	H	L	L	L[3]	L
Persistent wheeze															
Neas et al. [61]	Cohort	USA	7–11	1567	16–44 (M)	B, D/A, K	per 28.20	OR	1.16	0.89–1.52	H	L	H	H	L
Venn et al. [6]	Case-control	UK	9-11	416	21–50 (M)	K	22.1–34 (vs. 0–22)	OR	0.66	0.38–1.16[j]	H	L	L	H	L
							34.1–58 (vs. 0–22)		0.87	0.50–1.50[j]					
							>58 (vs. 0–22)		0.67	0.38–1.18[j]					

Notes
B = Bedroom; C = Adjustment of Confounders; CI = Confidence Interval; D/A = Dayroom/Activity Room; E = Exposure Assessment; H = High; HR = Hazard Ratio; K = Kitchen; L = Low; LR = Living Room; M = Mean; Max = Maximum; Mdn = Median; NO_2 = Nitrogen Dioxide; NR = Not Reported; O = Outcome Assessment; OR = Odds Ratio; ppb = Parts per Billion; RR = Relative Risk; S = Sample Selection; T = Temporality; UK = United Kingdom; USA = United States of America.
Statistically significant results are bolded.
[a] Converted if ppb was originally used as the unit in the paper.
[b] L^1 = cohort study, lost to follow-up (>25%); L^2 = case-control study, control selection; L^3 = cross-sectional study, inclusion/exclusion criteria; L^4 = cross-sectional study, missing data (>25%).
[c] Estimated from study figure.
[d] Study was conducted among children at high-risk for asthma (having an asthmatic sibling).
[e] Published before but not included in Lin et al. [93].
[f] Study was conducted among children with physician-diagnosed asthma.
[g] Study was conducted among children with active asthma.
[h] 90% CI.
[i] The median also takes into account concentrations in living room and kitchen.
[j] $P = 0.4$ for global test across all categories; P-trend = 0.3.

As shown in Table 6 and Supplemental Table 9, the 14 studies are highly heterogeneous. Specifically, 7 of the 14 studies used a cohort design, 5 used a cross-sectional design, and 2 used a case-control design. While the studies published prior to Lin et al. [93] used varied study designs, both of the studies published since Lin et al. [93] used a cohort design. Of the 14 studies, 5 were conducted in the US, 2 in Australia, 2 in China, 2 in Netherlands, 1 in Japan, 1 in Spain, and 1 in the UK. Notably, both of the studies published since Lin et al. [93] were conducted in China. The sample sizes of the studies were between 100 and 1600, generally much smaller than the gas cooking studies. The study periods were between 1983 and 2014. The ages of children in these studies vary considerably between 0 and 14 years. While some studies (e.g., Belanger et al. [53], Garrett et al. [70]) examined wide ranges of ages (>5 years), other studies (e.g., Hoek et al. [30], Shima and Adachi [64]) focused on very specific ages (within 2 years). All studies included both boys and girls. Most of the studies were conducted in the general population, except 4 that were conducted among children with preexisting conditions (i.e., high risk for asthma in Belanger et al. [55], active asthma in Belanger et al. [53,54], and physician-diagnosed asthma in Nitschke et al. [63]).

Similar to the indoor NO_2 and asthma literature, all studies measured indoor NO_2 using passive samplers, with 9 studies using tube type samplers, 2 studies using badge type samplers, and 3 studies not specifying the sampler type. Of the 14 studies, 7 measured indoor NO_2 at a single location within each household and 5 studies measured indoor NO_2 at multiple locations within each household; 2 studies did not specify the measurement location. As for the specific location, 7 of the 14 studies performed measurements in children's bedroom, 6 studies in the kitchen, 5 studies in the living room/main living area, and 3 studies in activity room/dayroom (i.e., room children spent the most time awake). The averaging time for NO_2 measurements ranged from 1 day to 1 year. The mean/median concentrations of indoor NO_2 varied substantially across, as well as between subgroups within, studies, ranging from 11.6 to 63 $\mu g/m^3$. Among studies conducted in the same country, indoor NO_2 concentrations also varied – from 16.17 to 44.18 $\mu g/m^3$ in the USA and from 20 to 63 $\mu g/m^3$ in Netherlands. Similar to the gas cooking studies, all of the wheeze outcomes relied on self-reported information, except that Hoek et al. [30] additionally relied on physician-reported information. The definition of wheeze outcome varied substantially across studies, with 12 distinct definitions used across the 14 studies.

As shown in Table 6 and Supplemental Table 10, the exposure contrasts for which the effect estimates were calculated varied substantially across studies. Of the 14 studies, 7 calculated effect estimates for a set increment of indoor NO_2 concentration on the original scale, but the actual magnitude varied from 10 $\mu g/m^3$ to 37.6 $\mu g/m^3$; 2 studies examined indoor NO_2 concentration on the log scale; 4 studies categorized indoor NO_2 concentration, each in a unique way; and 2 studies did not specify the exposure contrast. Only 1 study [54] examined the operationalization of indoor NO_2 concentration in more than one way (categorical and continuous). Notably, only 1 study [63], which was captured by our literature search but not included in Lin et al. [93], focused on the examination of maximum rather than average indoor NO_2. Of the 14 studies, 12 examined any wheeze as the only outcome type, and 2 studies examined persistent wheeze only. Few studies examined very specific wheeze outcomes (e.g., duration of wheezing) (results not shown).

Overall, 11 of the 14 studies reported null results; 3 studies reported statistically significantly positive associations between indoor NO_2 and wheeze [53,54,64], all of which were included in Lin et al. [93]. The magnitudes of the three statistically significant point estimates were not directly comparable with each other, given the different exposure contrasts used. Belanger et al. [53], a cross-sectional study in the US, reported a 1.52 (95% CI: 1.04–2.21) times higher risk of wheeze symptoms and a 1.33 (95% CI: 1.05–1.68) times greater number of days of wheeze symptoms per 37.6 $\mu g/m^3$ increment of indoor NO_2 for participants in

multi-family housing; however, null result was reported for participants in single-family housing. Belanger et al. [54], a cohort study in the US, reported a 1.49 (95% CI: 1.09–2.03) times higher risk of wheeze per 5-fold increase in NO_2 if greater than a prespecified threshold of 11.32 μg/m^3. Shima and Adachi [64], a cohort study in Japan, reported a 1.9 (95% CI: 1.30–2.83) times and 1.6 (95% CI: 1.06–2.44) times higher risk of wheeze per 18.8 μg/m^3 increment of indoor NO_2 among girls in 4th and 5th grades, respectively. However, null result was observed for 6th grader girls or among boys. All 3 studies examined any wheeze as the health outcome type. Compared to the studies included in Lin et al. [93], the newer studies generally adjusted for similar numbers of potential confounders. Given the high heterogeneity across the studies in this literature, we do not consider a meta-analysis to be appropriate for evidence synthesis.

The quality of this literature is generally low. Although all studies have high quality with respect to indoor NO_2 exposure assessment, a large proportion of them are subject to multiple sources of biases. As shown in Table 6, 11 of the 14 studies have low quality with respect to confounding adjustment, 10 studies cannot establish temporality, 7 studies are prone to selection bias, and 5 studies have low quality with respect to wheeze outcome assessment (studies that examined multiple outcome types were each only counted once). Both of the studies published since Lin et al. [93] have low quality with respect to confounding adjustment, high quality with respect to wheeze outcome assessment, and, as cohort studies, have high quality with respect to temporality. The distribution of study quality is similar across wheeze outcome types.

Temporality is a key aspect in both study quality evaluation and causal inference guided by the Bradford Hill [4] considerations (discussed below). Compared to the gas cooking studies, a larger proportion (i.e., 7 of 14) of indoor NO_2 studies for wheeze used a cohort study design, which is in general more reliable for making causal inference than a case-control or cross-sectional design [51,84]. However, 4 of the 7 studies [54,61,63,64] were of low quality with respect to temporality, despite the cohort design, due to the fact that the measurements of indoor NO_2 in the cohort were not necessarily taken prior to the measurements of wheeze outcomes. Only the remaining 3 cohort studies [9,40,50] were of high quality with respect to temporality. These 3 cohort studies all reported null results. Samet et al. [50] examined the association between indoor (bedroom) NO_2 and any wheezing during lower respiratory tract illness in American children under 1.5 years old and reported null results comparing across three exposure categories, after adjusting for all key confounders (ETS, family history, SES/home environment, and outdoor NO_2) as well as several other potential confounders. Li et al. [9] and Yu et al. [40] both examined the association between indoor NO_2 and new onset wheeze in Chinese children under 1.5 years old, and both studies reported null results (HR = 1.00, 95% CI: 0.995–1.001 and HR = 0.99, 95% CI: 0.979–1.003, respectively), after adjusting for key confounders (family history and SES/home environment) as well as several other potential confounders. While these 3 cohort studies all focused on children under 1.5 years old, their health outcome and study region varied, indicating that the only cohort studies that could establish temporality to date are not necessarily examining the same underlying indoor NO_2-wheeze relationship.

Guided by the Bradford Hill [4] considerations, we further discuss causal inference for the association between indoor NO_2 and wheeze in detail in Table 7. Overall, most of the epidemiology studies, including all of those with positive findings, cannot establish temporality. There are no large and precise effect estimates, and the observed associations lack consistency and specificity. An exposure-response relationship has not been well-characterized. No study has examined whether or how wheeze symptom would change after reducing indoor NO2 exposure. Experimental evidence is limited and does not sufficiently support any of the observed associations in epidemiology studies. We did not find a suitable analogy to address causality in this case. Taken together, we conclude that the evidence does not support causality.

In the meta-analysis by Lin et al. [93], all identified studies at the time were included, regardless of whether the studies reported adjusted effect estimates (vs. crude comparisons), the study design, or study quality. Summarizing across these studies, Lin et al. [93] reported a statistically significantly positive association between indoor NO_2 and wheeze (OR = 1.12, 95% CI: 1.04–1.21 for a 15 ppb [i.e., 28.2 μg/m^3] increase in NO_2). However, our systematic review shows that the literature to date, including the literature meta-analyzed by Lin et al. [93], is limited by the lack of consistent findings among studies with reliable study design (e.g., cohort), the high heterogeneity across studies, and the low study quality (primarily with respect to confounding adjustment, temporality, and sample selection). Our detailed causal inference guided by the Bradford Hill [4] considerations also shows that the evidence does not support causality. As a result, the effect estimates from Lin et al. [93] should be interpreted with caution.

Implications for meta-analyses

As discussed, we do not consider a meta-analysis to be appropriate for evidence synthesis in this review, due to the high heterogeneity across studies. A key source of heterogeneity is the differences in gas cooking practices (e.g., cooking methods, frequency, and duration; stove type and condition; ventilation; kitchen layout; natural gas vs. LPG) in different countries/regions. For example, we have discussed how a number of the post-2013 epidemiology studies have been conducted in China. As compared to typical USA cooking methods that consist of boiling, frying, roasting, and baking, traditional Chinese stir-frying/wok cooking methods rely on higher temperatures and gas combustion rates [59]. Ventilation practices can also differ between USA and Chinese residences; Chinese residences continue to heavily rely on natural ventilation modes (e.g., infiltration, windows) rather than mechanical ventilation systems [42]. It has also been shown that ventilation standards/regulations, as well as actual ventilation measurements in dwellings, vary across European countries [15]. Few studies in the current literature examined the details associated with gas cooking practices.

Changes in cooking practices and policy over time is another key source of heterogeneity. For example, from 1990 to 2020, when the majority of the studies in the present review were conducted, the percentage of the population mainly cooking with more polluting fuels (i.e., unprocessed biomass [wood, crop residues, and dung], charcoal, coal, and kerosene) dropped from over 75% to <50% in Central Asia and Southern Asia and from about 60% to about 30% in Eastern Asia and South-eastern Asia, indicating significant progress in transitioning towards universal use of clean fuels (i.e., gaseous fuels [LPG, natural gas, biogas], electricity, alcohol, and solar energy) as the main fuel for cooking; whereas during the same period, the percentage of population mainly cooking with polluting fuels only dropped from 90% to 84% in Sub-Saharan Africa and were consistently low (<10%) in North America and Europe [74]. Among the studies included in the present review, some defined gas cooking exposure as any or ever using gas for cooking in the home, whereas others defined it as primarily using gas for cooking; some compared children in homes with gas cooking to no gas cooking (but that could include other cooking fuels such as biomass cooking or electricity cooking, see Nantanda et al. [80] for example), whereas other studies compared children in homes with gas cooking strictly to electricity cooking. This further complicates the comparison and synthesis of study findings.

Studies in the current literature were also conducted among children of various age ranges for which the susceptibility and presentation of asthma vary [65]. Both global and USA analyses show that childhood asthma incidence rates were the highest among children under age 4, second highest among children aged 5–9 years, and lower among older children [17,21]. Pakkasela et al. [44] classified asthma into allergic (i.e., asthma with allergic rhinitis) vs. non-allergic types (asthma without allergic rhinitis) and showed that, throughout childhood, the incidence of non-allergic asthma remained low, whereas the incidence of allergic

Table 7
Causal inference guided by the Bradford Hill [4] considerations.

Definition	Evaluation			
	Gas – Asthma	Gas – Wheeze	NO_2 – Asthma	NO_2 – Wheeze
Consistency *Consistent associations (i.e., associations, especially statistically significant associations, mostly in the same direction [e.g., ORs > 1]) are observed by different authors, under different study designs, and in different study regions, populations, and time periods.*	The 22 reviewed studies were conducted by different authors in different countries/regions and time periods. The studies mostly used a cross-sectional design (n = 16), with a few using a cohort or case-control design (Main Table 3, Supplementary Tables 3 and 4). Overall, only a small proportion ($n = 7$) of the 22 studies reported statistically significantly positive associations, among which the majority ($n = 6$) used the same (cross-sectional) design. Some of the limited positive findings lack internal consistency (e.g., Ponsonby et al. [7], Spengler et al. [47]). The remaining 15 studies reported null results, with point estimates in both directions. When examined within more homogeneous study subgroups, the positive findings do not concentrate around a particular health outcome type, study region, or age group. Consistent associations were not observed in different study settings.	The 30 reviewed studies were conducted by different authors in different countries/regions and time periods. The studies mostly used a cross-sectional design (n = 25), with a few using a cohort or case-control design (Main Table 4, Supplementary Tables 5 and 6). Overall, only a small proportion (n = 6) of the 30 studies reported statistically significantly positive associations and 1 study reported a statistically significantly inverse association. All of these 7 studies used the same (cross-sectional) design. Some of the limited positive findings lack internal consistency (e.g., Belanger et al. [53], Volkmer et al. [81], Behrens et al. [88]). The remaining 23 studies reported null results, with point estimates in both directions. When examined within more homogeneous study subgroups, the positive findings do not concentrate around a particular study region or age group, although they are all for any wheeze as the health outcome type. Consistent associations were not observed in different study settings.	The 14 reviewed studies were conducted by different authors in different countries/regions and time periods. The studies mostly used a cohort design ($n = 10$), with a few using a cross-sectional or case-control design (Main Table 5, Supplementary Tables 7 and 8). Overall, only a small proportion ($n = 3$) of the 14 studies reported statistically significantly positive associations. All of these 3 studies used the same (cohort) design. Some of the limited positive findings lack internal consistency (e.g., Shima and Adachi [64]). The remaining 11 studies reported null results, with point estimates in both directions. When examined within more homogeneous study subgroups, the positive findings do not concentrate around a particular study region or age group, although they are all for asthma exacerbation as the health outcome type. Consistent associations were not observed in different study settings.	The 14 reviewed studies were conducted by different authors in different countries/regions and time periods. Half (n = 7) of the studies used a cohort design, 5 used a cross-sectional design, and 2 used a case-control design (Main Table 6, Supplementary Tables 9 and 10). Overall, only a small proportion (n = 3) of the 14 studies reported statistically significantly positive associations. Of these 3 studies, 2 used a cohort design and 1 used a cross-sectional design. Some of the limited positive findings lack internal consistency (e.g., Shima and Adachi [64]). The remaining 11 studies reported null results, with point estimates in both directions. When examined within more homogeneous study subgroups, the positive findings do not concentrate around a particular study region or age group, although they are all for any wheeze as the health outcome type. Consistent associations were not observed in different study settings.
Strength *The effect estimates are large and precise (e.g., narrow 95% CI). Small and imprecise effect estimates could be driven by bias, confounding, or chance.*[a]	The point estimates of the 7 studies that reported statistically significant associations ranged from 1.24 to 3.80, with all but one effect estimate being below 2.40 (Main Table 3, Supplementary Table 4). The larger the point estimate was, the less precise its 95% CI was, indicating that the point estimate was less stable. It is also notable that a large proportion of these studies (5 out of 7) did not fully adjust for key confounders. Many potential confounders (e.g., indoor factors such as dampness and mold) are positively associated with both gas cooking and asthma risk. Had these confounders been accounted for, magnitudes of the observed associations may have been attenuated. As for the 15 studies that reported null results, the lower 95% confidence limits ranged from 0.17 to 1.00, with all but 5 being 0.60 or greater; the upper 95% confidence limits ranged from 1.02 to 24.23, with all but 5 being 3.10 or lower. This indicates that if there were associations that were missed due to insufficient statistical power, they would have been relatively close to the null value (e.	The point estimates of the 6 studies that reported statistically significantly positive associations ranged from 1.16 to 2.27 (Main Table 4, Supplementary Table 6). The larger the point estimate was, the less precise its 95% CI was, indicating that the point estimate was less stable. It is also notable that none of these 6 studies fully adjusted for key confounders. Many potential confounders (e.g., indoor factors such as dampness and mold) are positively associated with both gas cooking and asthma-associated wheeze symptoms. Had these confounders been accounted for, magnitudes of the observed associations may have been attenuated. As for the 23 studies that reported null results, the lower 95% confidence limits ranged from 0.22 to 0.97, with all but 4 being 0.58 or greater; the upper 95% confidence limits ranged from 1.03 to 14.01, with all but 4 being 2.47 or lower. This indicates that if there were associations that were missed due to insufficient statistical power, they would have been relatively close to the null value (e.g., OR = 1).	The point estimates of the 3 studies that reported statistically significantly positive associations ranged from 1.31 to 2.82, with all but 1 effect estimate being below 1.70, although they are not directly comparable across studies given the different exposure contrasts used (Main Table 5, Supplementary Table 8). The larger the point estimate was, the less precise its 95% CI was, indicating that the point estimate was less stable. It is also notable that 2 of the 3 studies did not fully adjust for key confounders. Many potential confounders (e.g., indoor factors such as dampness and mold) are positively associated with both indoor NO_2 exposure and asthma risk. Had these confounders been accounted for, magnitudes of the observed associations may have been attenuated. As for the 11 studies that reported null results, the lower 95% confidence limits ranged from 0.08 to 0.99, with all but 4 being 0.40 or greater; the upper 95% confidence limits ranged from 1.05 to 34.9, with all but 4 being 4.82 or lower. This	The point estimates of the 3 studies that reported statistically significantly positive associations ranged from 1.33 to 1.90, with all but 1 effect estimate being 1.60 or lower, although they are not directly comparable across studies given the different exposure contrasts used (Main Table 6, Supplementary Table 10). The larger the point estimate was, the less precise its 95% CI was, indicating that the point estimate was less stable. It is also notable that 2 of these 3 studies did not fully adjust for key confounders. Many potential confounders (e.g., indoor factors such as dampness and mold) are positively associated with both indoor NO_2 exposure and asthma-associated wheeze symptoms. Had these confounders been accounted for, magnitudes of the observed associations may have been attenuated. As for the 11 studies that reported null results, the lower 95% confidence limits ranged from 0.11 to 0.995, with all but 1 being 0.37 or greater; the upper 95% confidence limits ranged from 1.001 to 6.20,

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Table 7 (*continued*)

Definition	Evaluation			
	Gas – Asthma	Gas – Wheeze	NO_2 – Asthma	NO_2 – Wheeze
	g., OR = 1). There are no large and precise effect estimates.	The only statistically significantly inverse association reported by Behrens et al. [88] is likely a chance finding. This finding lacked internal consistency, as the study reported a statistically significantly inverse association among boys (PR = 0.55, 95% CI: 0.31–0.98) but a null result (PR = 1.52, 95% CI: 0.93–2.47) among girls. Further, the point estimate (0.55) of the reported statistically significantly inverse association is below most of the lower 95% confidence limits reported by the 23 studies with null results. There are no large and precise effect estimates.	indicates that if there were associations that were missed due to insufficient statistical power, they would have been relatively close to the null value (e.g., OR = 1). There are no large and precise effect estimates.	with all but 3 being 2.40 or lower. This indicates that if there were associations that were missed due to insufficient statistical power, they would have been relatively close to the null value (e.g., OR = 1). There are no large and precise effect estimates.
Specificity *The observed associations are limited to a specific exposure and to a specific health outcome.*[b]	Asthma has multiple known risk factors and thus cannot be linked specifically to gas cooking exposure. For example, increased risk of asthma has been associated with family history of asthma, respiratory infections, ETS exposure, and indoor allergen exposure (e.g., dust mite, cat, dog, mouse, cockroach, and molds) [46,69]. Gas cooking in itself is not a very specific exposure, as the associated exposures to chemicals and their mixtures vary by a number of factors such as gas composition, stove type and condition, ventilation, cooking frequency and duration, as well as cooking methods. The health outcomes associated with each individual chemical also vary. As a result, gas cooking exposure cannot be linked specifically to asthma. The observed associations lack specificity.	In most of the reviewed studies, wheeze was examined as a symptom of asthma, which has multiple known risk factors, such as family history of asthma, respiratory infections, ETS exposure, and indoor allergen exposure (e.g., dust mite, cat, dog, mouse, cockroach, and molds) [46,69]. As a result, wheeze as a symptom of asthma cannot be linked specifically to gas cooking exposure. Gas cooking in itself is not a very specific exposure, as the associated exposures to chemicals and their mixtures vary by a number of factors such as gas composition, stove type and condition, ventilation, cooking frequency and duration, as well as cooking methods. The health outcomes associated with each individual chemical also vary. As a result, gas cooking exposure cannot be linked specifically to wheeze. The observed associations lack specificity.	Asthma has multiple known risk factors and thus cannot be linked specifically to indoor NO_2 exposure. For example, increased risk of asthma has been associated with family history of asthma, respiratory infections, ETS exposure, and indoor allergen exposure (e.g., dust mite, cat, dog, mouse, cockroach, and molds) [46,69]. While indoor NO_2 is a specific exposure, it cannot be linked specifically to asthma. For example, indoor NO_2 exposure has been associated with cardiovascular health outcomes such as coronary artery disease, arrhythmia, heart failure, and ischemic heart disease [22,89]. The observed associations lack specificity.	In most of the reviewed studies, wheeze was examined as a symptom of asthma, which has multiple known risk factors, such as family history of asthma, respiratory infections, ETS exposure, and indoor allergen exposure (e.g., dust mite, cat, dog, mouse, cockroach, and molds) [46,69]. As a result, wheeze as a symptom of asthma cannot be linked specifically to gas cooking exposure. While indoor NO_2 is a specific exposure, it cannot be linked specifically to wheeze. For example, indoor NO_2 exposure has been associated with cardiovascular health outcomes such as coronary artery disease, arrhythmia, heart failure, and ischemic heart disease [22,89]. The observed associations lack specificity.
Temporality *Causality can only exist if the exposure precedes the occurrence of the health outcome with a sufficient lag time, if any is expected.*	Of the 22 studies in this literature, 16 used a cross-sectional design, including 6 of the 7 studies that reported statistically significantly positive associations; only the remaining 1 study used a cohort design. As a result, temporality cannot be established in most of the studies in this literature, including the majority of the studies with positive findings. The lack of temporality prevents any causal inference.	Of the 30 studies in this literature, 25 used a cross-sectional design, including all 7 studies that reported statistically significant associations. As a result, temporality cannot be established in most of the studies in this literature, including all of the studies with positive findings. The lack of temporality prevents any causal inference.	Of the 14 studies in this literature, 10 used a cohort design, including all 3 studies that reported statistically significantly positive associations. However, the majority (n = 8) of these 10 cohort studies, including all 3 studies with positive findings, cannot establish temporality despite the cohort design. Temporality cannot be established in any of the 4 studies that used a cross-sectional or case-control design. The lack of temporality prevents any causal inference.	Of the 14 studies in this literature, 7 used a cohort design, including 2 of the 3 studies that reported statistically significantly positive associations. However, 4 of these 7 cohort studies, including both of the cohort studies with positive findings, cannot establish temporality despite the cohort design. Temporality cannot be established in either of the 2 case-control studies. Except for Belanger et al. [55], the cross-sectional studies, including that by Belanger et al. [53] with positive findings, cannot establish temporality. The lack of temporality prevents any causal inference.

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Table 7 (*continued*)

Definition	Evaluation			
	Gas – Asthma	Gas – Wheeze	NO_2 – Asthma	NO_2 – Wheeze
Dose-Response *There exists a well-characterized exposure-response relationship (e.g., increased disease risk at higher exposure level).*	All studies examined gas cooking exposure as a binary variable (e.g., gas vs. electricity, ever vs. never), preventing the characterization of potential exposure-response relationship. We only identified 1 study that explored potential exposure-response relationships. Specifically, Lin et al. [94] examined the association across different frequency levels of gas cooking exposure (never, intermittent, and always). However, the observed association was not statistically significant in either the intermittently exposed or the always exposed group (vs. never exposed), with the point estimate being very similar but slightly greater in the intermittently exposed group than the always exposed group (adjusted ORs both between 1 and 2 but not specified). An exposure-response relationship has been assessed only in 1 study and is not supported by its results.	All studies examined gas cooking exposure as a binary variable (e.g., gas vs. electricity, ever vs. never), preventing the characterization of potential exposure-response relationship. We only identified 1 study that explored potential exposure-response relationships. Specifically, Lin et al. [94] examined the association across different frequency levels of gas cooking exposure (never, intermittent, and always). However, the observed association was not statistically significant in either the intermittently exposed or the always exposed group (vs. never exposed), with the point estimate being greater in the intermittently exposed group than the always exposed group (adjusted ORs both between 1 and 2 but not specified). An exposure-response relationship has been assessed only in 1 study and is not supported by its results.	All but 1 study [13] addressed dose-response by examining indoor NO_2 exposure as a continuous variable or categorical variable with ≥3 levels. However, only 1 study [54] explored multiple potential shapes of the exposure-response relationship (log-scale vs. categorical); all other studies each examined one assumed shape and lag structure. Overall, only 3 studies observed a statistically significant exposure-response relationship, 2 of which [26,64] are linear and 1 [54] of which is linear on the log-scale or categorical. An exposure-response relationship has not been well-characterized.	All studies addressed dose-response by examining indoor NO_2 exposure as a continuous variable or categorical variable with ≥3 levels. However, only 1 study [54] explored multiple potential shapes of the exposure-response relationship (log-scale vs. categorical); 1 study [50] explored different lag structures (lagged vs. unlagged); and all other studies examined one assumed shape and lag structure. Overall, only 3 studies observed a statistically significant exposure-response relationship, 2 of which [53,64] are linear and 1 of which is linear on the log-scale or categorical. An exposure-response relationship has not been well-characterized.
Biological Plausibility *Causality is more scientifically defensible if there exists evidence for a plausible biological mechanism by which the exposure may lead to the health outcome, although the existing evidence may be limited.*	US EPA [91] concluded from experimental studies (e.g., animal and controlled human exposure studies) that short-term NO_2 exposure may induce asthma exacerbation via increased airway responsiveness or allergic inflammation, despite some mixed findings and that long-term NO_2 exposure may lead to asthma development via airway hyperresponsiveness or development of an allergic phenotype, although experimental evidence was limited. Notably, experimental studies evaluated high exposures (e.g., >100 ppb) that may not be relevant to those generally experienced by children in homes with gas cooking. It is currently not established that childhood NO_2 exposures associated with gas cooking are of sufficient duration, frequency, or concentration to cause or contribute to asthma (or associated wheeze symptom).			
Experiment *There exists experimental or semi-experimental evidence (e.g., reduced disease risk resulting from reduced exposure).*	We did not find interventional studies addressing asthma after removing or reducing gas cooking exposure.	We did not find interventional studies addressing wheeze after removing or reducing gas cooking exposure.	We did not find interventional studies addressing asthma after reducing indoor NO_2 exposure.	We did not find interventional studies addressing wheeze after reducing indoor NO_2 exposure.
Analogy *A similar exposure is an established causal factor for a similar health outcome.*	We did not find a suitable analogy in this case.	We did not find a suitable analogy in this case.	We did not find a suitable analogy in this case.	We did not find a suitable analogy in this case.
Coherence *The observed associations in epidemiology studies can be interpreted logically along other realms of evidence (e.g., animal studies).*[c]	Experimental evidence on the plausible biological mechanisms by which NO_2 exposure may lead to asthma (or associated wheeze symptom) is limited to high-level NO_2 exposures that may not be relevant to the exposure levels experienced by children in homes with gas cooking examined in epidemiology studies. The observed associations in epidemiology studies are not sufficiently supported by, although not contradicting, the experimental evidence.			
Causal Conclusion	Taken together, the evidence does not support causality.	Taken together, the evidence does not support causality.	Taken together, the evidence does not support causality.	Taken together, the evidence does not support causality.

Notes

CI = Confidence Interval; ETS = Environmental Tobacco Smoke; NO_2 = Nitrogen Dioxide; OR = Odds Ratio; ppb = Parts per Billion; PR = Prevalence Ratio; US EPA = United States Environmental Protection Agency.

[a] Small effect estimates in themselves do not undermine the likelihood of causality.

[b] The lack of specificity does not undermine the likelihood of causality.

[c] The lack of other realms of evidence does not undermine the likelihood of causality.

asthma was highest in early childhood and decreased towards older age.

Regarding the results of existing meta-analyses, it is crucial that the high heterogeneity and its sources are recognized in their interpretation. Of equal importance is the consideration of study quality. It is well-recognized that a meta-analysis performed using risk estimates from studies of low quality will be prone to bias and incorrect results [16,99]. Given our findings that show the relatively low quality of the epidemiology literature used in the Lin et al. [93] meta-analysis, we caution against over-interpretation of its results. Consistent with our words of caution, Vrijheid [66] stated in a contemporaneous commentary on the Lin et al. [93] meta-analysis concerns related to residual confounding by "asthma and wheeze risk factors such as dampness, mould, pets and environmental tobacco smoke" that "may be closely related to the use of gas cookers and indoor NO_2" and heterogeneity due to varying levels of adjustment for these confounders among studies.

Recently, Gruenwald et al. [90] relied on the North American- and European-specific risk estimates for gas cooking and current asthma that were reported in the Lin et al. [93] meta-analysis, among other data sources and a series of statistical assumptions, and estimated that "12.7% (95% CI = 6.3-19.3%) of current childhood asthma in the USA is attributable to gas stove use." This population attributable fraction (PAF) calculation used the quantitative evidence synthesis estimates from Lin et al. [93] at face value without considering the underlying high heterogeneity or low quality among the individual studies. More importantly, a key underlying assumption of any PAF calculation is that there is a clear causal relationship between the risk factor(s) and disease [11]; in this case, the Gruenwald et al. [90] PAF calculation is predicated on there being a clear causal association between gas stove use and current childhood asthma.[1] However, our in-depth evaluation of heterogeneity and study quality in the present review reveals that, although the quantitative evidence synthesis from Lin et al. [93] reported a statistically significantly positive association between gas cooking and asthma, the epidemiology literature is limited and a causal conclusion is not supported. The Gruenwald et al. [90] calculation is a clear example of over-interpretation of the Lin et al. [93] meta-analysis results, and the calculated PAF value is not valid. This echoes the conclusion of a recently published commentary by Cox Jr. [60] that "the projections of Gruenwald *et al.* that about 13% of childhood asthma in the US could be prevented by reducing or eliminating gas stove emissions have no known validity. They are not supported by the data and analyses performed."

Strengths and limitations

A major strength of this review is that we used transparent, systematic, rigorous methods. This included the fact that we registered our protocol before we began the review and that we only made minor changes during the review process, which we indicated in protocol amendments. We determined study eligibility based on PECOS elements and two reviewers were involved in selecting studies and extracting data to help ensure accuracy. We took study quality and heterogeneity across studies into account when synthesizing evidence across studies. We did not conduct a meta-analysis because of the heterogeneity of study designs, which would have produced meta-risk estimates that would have been difficult to interpret. We used Bradford Hill considerations to guide our overall evaluation, and followed PRISMA guidelines when reporting our results.

As noted by Goodman et al. [48], "When reviewing individual studies in the context of study quality, we found that it is most helpful to first determine what aspects of study quality are likely to have the most impact on the interpretation of results, instead of spending time and resources on sometimes up to dozens of aspects that ultimately may not have much impact." However, it is simply not possible to choose study quality criteria without some level of subjectivity. For example, we had to make decisions on what to classify as key confounders and what criteria needed to be satisfied for a study to be considered high quality with respect to confounding.

Other researchers may have made different choices than we did, but our choices are all fully transparent, and we also note that even though we categorized studies as high or low for each aspect, we discussed the results of all studies in the context of each aspect. That is, we did not merely check a box as overall high or low quality, and summarize results according to the boxes checked. Rather, we discussed study results in light of those individual aspects.

Future research

To better address the question of whether gas cooking exposure or indoor NO_2 can increase asthma or wheeze risk in children, the most reliable observational epidemiology study would be a cohort study that meets all key study quality criteria, including for sample selection, temporality, exposure assessment, outcome assessment, and confounder adjustment. We found that only 1 cohort study conducted over 30 years ago [50] was high quality in all of the major categories we consider to be important. A future study will require considerable resources to complete, but will be better able to address causation than any other study conducted to date.

With regard to sample selection, future cohort studies should be conducted with an adequate sample size to allow for the detection of any true underlying association and the adjustment of all potential confounders. Researchers should minimize loss to follow up, and determine whether any individuals who drop out of the study likely differ from those remaining.

With regard to gas cooking exposure assessment, researchers should confirm the type of stove in each home, and record information on the specific fuel used, the frequency and duration of use, and whether and what type of ventilation is used. They should also record information on when children are in the home, and their locations and activities in the home, particularly with respect to when gas stoves are used. For studies evaluating NO_2 exposure, NO_2 should be measured using a validated method (e.g., passive dosimeters) and over a sufficient period of time and over different seasons to ensure that measurements are representative of typical exposures. Ideally, personal NO_2 measurements should be made, with sensors capable of collecting time-resolved (e.g., minute-by-minute or hour-by-hour) data to capture both short-term peak exposure levels as well as time-averaged exposure levels [85]. In addition, the study should consider what the sources of NO_2 are in the home (e.g., gas stoves, gas heaters, other appliances, ETS) and children's NO_2 exposure outside of home (e.g., at school, in traffic).

With regard to outcome assessment, asthma can be particularly difficult to study, as both over-and underdiagnoses are common [1]. It can also be challenging to study wheeze since it is hard to define. Both asthma and wheeze should be clearly defined, and timing of events should be recorded. Health professionals associated with the study should confirm all diagnoses in the study to minimize misclassification.

With regard to confounder adjustment, researchers should make an effort to measure and adjust for all potential confounders. The present review considers as key confounders ETS, family history of asthma/allergies/atopy, SES/home environment (e.g., dust mite, cockroach, pets, mold, wood stove, dampness, heating fuels, crowdedness, pillow/quilt/mattress, form of cooling), and outdoor NO_2 (for NO_2 studies), all of which are known risk factors for asthma, and therefore asthma-associated wheeze symptoms [46,69]. ETS, some home environment factors (e.g., heating systems), and outdoor NO_2 are known sources of

[1] As further discussed in a commentary prepared by several of this paper's authors (Goodman et al., submitted), Gruenwald et al. (2023) also did not address other assumptions that must be met for the calculation of a PAF for gas stove use and current childhood asthma, including that having a gas stove is independent of other asthma risk factors, and that eliminating gas stoves would immediately reduce asthma risk.

indoor NO_2 [97]; ETS and SES/home environment (e.g., poverty/sub-standard housing) may be closely related to the use of gas cookers [52,66]. Having a family history of asthma/allergies/atopy may affect parents' choices of cooking appliances or other indoor factors that could affect indoor NO_2 levels. Other potential confounders include additional factors that may be associated with asthma, such as obesity, indoor and outdoor co-pollutants including ozone (O_3) and fine particulate matter ($PM_{2.5}$), and environmental parameters including temperature and relative humidity [46,69].

In addition to observational studies, interventional and experimental studies can be conducted to directly address causation. These studies are very resource-intensive, but can more directly address causation. These studies will need to involve families with similar home environments and other asthma or wheeze risk factors. Ideally, all residences will have similar gas stoves at the beginning of the study, and some (ideally, a random subgroup of) stoves will be replaced with other types of stoves (e.g., electric or induction). Ventilation should also be considered (e.g., enforce controlled ventilation, comparing with and without ventilation under the same gas stove use pattern). All of the study quality aspects discussed above for observational studies (e.g., exposures, health outcomes, potential confounders) apply to interventional studies. Further, researchers will need to record any noncompliance to the intended intervention and evaluate its potential impact on the study results. Only then would this type of study provide results that could be informative regarding causation.

Conclusion

We conducted the first systematic review of gas cooking or indoor NO_2 and asthma or wheeze in children that included an in-depth evaluation of study heterogeneity and study quality. We reviewed 66 relevant studies, including those in the most recent meta-analysis by Lin et al. [93]. We found that most of the studies are cross-sectional by design. The few cohort studies that could establish temporality largely reported null results. There is large variability across studies in terms of study region, age of children, gas cooking exposure definition, and asthma or wheeze outcome definition, precluding clear interpretations of meta-analysis estimates such as those reported in Lin et al. [93]. Furthermore, a large proportion of the studies are subject to multiple sources of bias and inaccuracy, primarily due to self-reported gas cooking exposure or respiratory outcomes, insufficient adjustment for key confounders and unestablished temporality. We conclude that the epidemiology literature is limited by high heterogeneity and low study quality and, therefore, it does not provide sufficient evidence regarding causal relationships between gas cooking or indoor NO_2 and asthma or wheeze. We caution against over-interpreting the quantitative evidence synthesis estimates from meta-analyses of these studies.

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Declaration of Competing Interest

Gradient has worked with several organizations in the past that have an interest in gas stoves and NO_2 science. None of these clients was involved with the conception or drafting of this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.gloepi.2023.100107.

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APPENDIX

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