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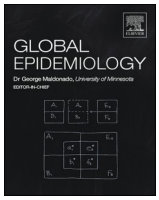

What constitutes valid evidence of causation? Gas stoves and childhood asthma revisited

1. Introduction

I have criticized the paper by Kashtan et al. [1] on the grounds that its conclusions depend on unverified modeling assumptions [2]. In their response letter, Nadeau et al. note that "The deleterious effect of outdoor NO_2 exposure on respiratory health has been well established for decades and confirmed by numerous large meta-analyses." They state that Government agencies including the U.S. Environmental Protection Agency, Health Canada, the European Environment Agency and the World Health Organization "all agree that NO_2 exposure is harmful generally to the respiratory system." They conclude that "Combining evidence from distinct types of studies is what gives the scientific community and regulatory agencies confidence that NO_2 causes pediatric asthma. In contrast with this comprehensive approach, Cox's rebuttal uses the inescapable limitations of certain types of studies to cast doubt on the field's findings entirely. This method of casting doubt on a scientific consensus is not new and has been used to challenge the science on smoking" and other exposure-response associations."

Preliminary Comments.

It is worth examining these arguments carefully. Some preliminary observations are as follows.

- The term "deleterious effect" leaves ambiguous whether the "effect" referred to is a statistical association or an interventional causal effect, or perhaps something else. As explained in book-length treatments of what "the effect" means, a statistical effect is often quite different from an interventional causal effect, and the former may well exist without the latter ([3,4]).
- The claim that "The deleterious effect of outdoor NO_2 exposure on respiratory health has been well established for decades and confirmed by numerous large meta-analyses" – whether accurate or not – neglects that gas stove emissions are *not* "outdoor NO_2 exposure." They do not necessarily occur under the same causally relevant conditions of concentration and duration, ambient temperature, co-exposures to other ambient pollutants, exercise and breathing rates, and so forth as exposures to outdoor (e.g., ambient or traffic-related) NO_2 exposures.
- The claim that the "deleterious effect" (or statistical association) of outdoor NO_2 with childhood asthma "has been well established for decades and confirmed by numerous large meta-analyses" also ignores the facts that (a) large meta-analyses have typically addressed association, not causation – and not the specific causal claim made by Nadeau et al. for gas stoves and childhood asthma; (b) these large meta-analyses typically do not claim that causally relevant confounders have been adequately controlled for, or that the associations that they report address causality; and (c) most do not claim that they have found clearly interventional causal relationships, e.g., that the crucial property of Invariant Causal Prediction (ICP) [5] holds for their reported exposure-response associations. Thus, contrary to Nadeau et al.'s suggestion, there is no "scientific consensus" on the extent to which NO_2 causes childhood asthma.
- To the contrary, reported associations in past meta-analyses are highly heterogeneous and confounding remains to be adequately controlled. For example, a systematic review and meta-analysis by Khreis et al. [6] of forty-one studies of whether children's exposure to traffic-related air pollution (TRAP) contributes to their development of asthma stated that "The question of whether children's exposure to traffic-related air pollution (TRAP) contributes to their development of asthma is unresolved. …There was notable variability in asthma definitions, TRAP exposure assessment methods and confounder adjustment. The overall random-effects risk estimates (95% CI) were… 1.05 (1.02, 1.07) per 4 $\mu g/m^3$ nitrogen dioxide (NO_2) [and]1.48 (0.89, 2.45) per 30 $\mu g/m^3$ nitrogen oxides (NO_x). Across the main analysis and age-specific analysis, the most heterogeneity [was seen] for the NO_2 and NO_x estimates. … Future meta-analyses would benefit from greater standardization of study methods including exposure assessment harmonization, outcome harmonization, confounders' harmonization and the inclusion of all important confounders in the individual analyses."
- To my objections that association and causation should be distinguished, Nadeau et al. reply that that "Merely pointing out an association between two phenomena would be insufficient to claim causality, but this is not what we did. Our causal claims are built on a decades-long body of research. Epidemiologists assess causality by reviewing the weight of evidence garnered from complementary fields, rather than by focusing only on a single type of experiment. This framework for causality was set out by Sir Bradford Hill in the mid-1960s, was refined and updated in the decades since, and has since been adopted by the U.S. EPA when setting the air quality standards cited in our original paper." But this defense simply compounds the original problem. The "effects" referred to by the US EPA, Sir Austin Bradford Hill, the WHO, and other authorities cited by Nadeau et al., are usually *associational* and are measured by associational methods such as relative risks, odds ratios, or regression coefficients. They are not *interventional*, and hence are not capable of predicting whether, or by how much, reducing exposure would reduce health outcomes such as childhood asthma. Treating measures of association as if they were measures of interventional causation is a fallacy [7]. This conflation of statistical associational "effects" with interventional causal "effects" is one of my chief criticisms of the original paper, and now also of the authors' letter defending it. The use of associational data to support interventional conclusions, such as that reducing exposure to NO_2 would reduce childhood asthma, is methodologically indefensible, as discussed by

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Pearl [7]. Moreover, Li et al. [8] examined the available literature on gas stoves and NO_2 and childhood asthma and concluded that there was not sufficient evidence to support causal conclusions under the Bradford Hill criteria, were they to be applied, and "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."

- The "basic distinction" [7] between *associational* and *interventional* causal "effects," while perhaps obscure to some epidemiologists in the 1960s when the Bradford Hill considerations were offered, has been greatly clarified since then. The advance of technical methods and concepts for defining and modeling interventional causality makes it both unnecessary and undesirable to continue conflating these distinct concepts ([7]; Huntington-Klein, 2022; [4]).
- Nadeau et al.'s claim that "Combining evidence from distinct types of studies is what gives the scientific community and regulatory agencies confidence that NO_2 causes pediatric asthma" improperly moves from a *general* claim that some exposures to NO_2 are associated with pediatric asthma or other adverse effects under some exposure conditions to a *particular* claim that emissions specifically from gas stoves cause pediatric asthma. This is not more logically sound than claiming that since some children have been observed to drown in some bodies of water, drinking a glass of water poses a risk of drowning to children. There is no license in logic or in epidemiology to move from general claims to particular claims in this way.
- Likewise, Nadeau et al.'s general appeal to authority, stating that various authorities "all agree that NO_2 exposure is harmful generally to the respiratory system," does not warrant the specific conclusion that NO_2 emissions from gas stoves increase risk of childhood asthma. This amounts to assuming what is to be proved.
- Similarly, their statement that "Based on a careful review of the evidence, the U.S. EPA's 2016 Integrated Science Assessment concludes that there is 'a causal relationship between respiratory effects and short-term NO_2 exposure, primarily based on evidence for asthma exacerbation'" is about US EPA views supporting National Ambient Air Quality Standards (NAAQS). It does not address the specific question of whether indoor emissions from gas stoves under real-world conditions increase risk of childhood asthma. The US EPA Integrated Science Assessments do not specifically address interventional causation, so this appeal to authority lacks relevance for the causal conclusions that Nadeau et al. draw for childhood asthma cases and indoor air pollution from gas stoves.
- I agree with Nadeau et al. that "Combining evidence from distinct types of studies" can lend support to the conclusion "that NO_2 causes pediatric asthma" *if* the combining is done correctly, the evidence being combined is relevant to the specific questions it is used to address, and if the results show that NO_2 emissions causes pediatric asthma under the conditions of the actual exposures of interest. Indeed, showing that evidence across multiple diverse study designs gives estimated interventional causal relationships for NO_2 and childhood asthma that satisfy ICP would be a major contribution to clarifying whether and by how much changing NO_2 exposure could change risk of asthma. But this is precisely the type of detailed quantitative analysis of relevant causal evidence that Nadeau et al., and the authorities they cite, have *not* done. Rather, their argument is that "By these standards, the evidence for causality between NO_2 exposure and asthma is very strong. We have… direct evidence: controlled laboratory studies have consistently observed adverse respiratory effects in human asthmatics when inhaling NO_2 for short time periods… observational studies consistently find associations between NO_2 and pediatric asthma on a population level, both outdoors and indoors… laboratory studies on lung cells and mice have directly observed cell death and asthma-like symptoms following NO_2 exposure… direct and mechanistic evidence for the effect of NO_2 on lung function comes from dozens of studies conducted over decades." Such mixed evidence that NO_2 has various effects in various systems (e.g., lung cells and mice) under various conditions stops well short of addressing ICP or of supporting (or even addressing) the specific causal claim that NO_2 emissions *from gas stoves under real exposure conditions* increases risk of childhood asthma. As noted by Khreis et al. [6], that question has yet to be resolved even for TRAP emissions. In other words, the leap from association to causation here lacks support.

Nadeau et al. also seem to me to also miss the point of my critique of their assumption that changes in NO_2 cause proportional changes in childhood asthma risk. They write "In plain English, Cox is claiming that we assumed without evidence that exposure to NO_2 is roughly proportional to pediatric asthma risk — the more exposure, the more risk — and that we ignored the implicit uncertainty built into this assumption. … Contrary to Cox's claim, we in fact cite three epidemiological studies to justify our assumption that NO_2 exposure is proportional to pediatric asthma risk." My main criticism was that this assumption has not been verified or empirically tested. The three references that Nadeau et al. cite (Achakulwisut et al. [9]; Anenberg et al. [10]; and Mohegh et al. [11], all of which share coauthors) address *attributable* risk (an associational measure), not whether changing exposure to gas stove emissions would cause childhood asthma to change [7]. Specifically,

- Achakulwisut et al. [9] state that "Globally, we estimated that 4.0 million (95% uncertainty interval [UI] 1.8-5.2) new paediatric asthma cases could be *attributable to* NO_2 pollution annually; 64% of these occur in urban centres" (emphasis added). This is clearly and explicitly an attributable risk calculation. (The authors also note that " it is possible that NO_2 is a proxy for other putative agents in the TRAP mixture, that the reported associations are sensitive to control for co-pollutants, or both. All these uncertainties mean that our NO_2-attributable asthma estimates might be overestimated or underestimated.")
- Similarly, Anenberg et al. [10] state that "We estimated that 1.85 million (95% uncertainty interval [UI] 0.93-2.80 million) new paediatric asthma cases were *attributable to* NO_2 globally in 2019, two thirds of which occurred in urban area… The proportion of paediatric asthma incidence that is *attributable to* NO_2 in urban areas declined from 19.8% (1.22 million attributable cases of 6.14 million total cases) in 2000 to 16.0% (1.24 million attributable cases of 7.73 million total cases) in 2019." (Emphases added.) Again, it is very clear that these are only attributable risk calculations based on population-level associations, not (interventional) causal modeling calculations.
- Finally, Mohegh et al. [11] state that "We use epidemiologically derived health impact functions to estimate *NO_2-attributable asthma incidence* for the U.S. and India, two countries with different degrees of urbanicity. … We used national-scale baseline asthma rates, though a large degree of heterogeneity in asthma rates exists between neighborhoods and different population subgroups. … Lastly, the LUR [land use regression] concentration data might not represent what individuals are actually exposed to, considering outdoor exposure misclassification and time spent in indoor environments. However, this issue also exists in the epidemiological studies that were used to assess exposure-response relationships, as well as other air pollution health impact assessments (Anenberg et al. 2018, [9])." (Emphasis added).

Thus, all three cited references address attributable risk based on aggregate exposure-response associations. Although the authors offered opinions and policy recommendations suggesting that reducing NO_2 or air pollution exposure would reduce childhood asthma, this is an interventional question that cannot logically be answered by appeals to

associations or to estimates of attributable risk based on associations [7]. The apparent lack of appreciation of this "basic distinction" (ibid) between associational and interventional causal concepts seems to me to be a fundamental flaw in the analysis and interpretation in Kashtan et al. [1]. The letter by Nadeau et al. doubles down on it by arguing that their citations showing that linear or log-linear models have been assumed before by the US EPA and others in making attributable risk calculations, suggesting that this somehow implies that the assumptions are also defensible for making interventional causal predictions and policy prescriptions. To me, this is a clear logical fallacy and methodological error. Attribution is not interventional causation. The reasons are well explained by Pearl [7] and in modern textbooks of causal analysis (Huntington-Klein, 2022; [4]).

Essential Points.

Putting technical details aside for the moment, what is the main argument about here? Nadeau et al. suggest that I am concerned with a quibble about *p*-values and statistical significance: "Cox is arguing here that there is uncertainty in statistical associations, that there is a norm of aiming for less than 5% uncertainty (a p-value of < 0.05), that the associations we cite have more than this 5% uncertainty, and that because of this the 'associations studied may not even exist.'" But I think the stakes are much higher and more interesting. They have to do with whether absence of evidence of a causal effect should be presented in publications such as Kashtan et al. [1] as if it were evidence of a causal effect.

To me, the discussion of *p*-values and statistical significance in Nadeau et al.'s letter is a red herring, as I fully agree with Nadeau et al. that nothing important turns on whether stated p-values are slightly above or slightly below 5%. Rather, I objected to Kashtan et al. citing Lin et al. as if it supported their causal claim because it does nothing of the sort. I stated that Lin et al. estimated a summary odds ratio for asthma from a 15-ppb increase in NO_2 as 1.09 (95 % CI 0.91–1.31), and that they acknowledged that "Residual confounding by (unmeasured) factors that are associated with gas cooking might be another explanation for our finding of an association between asthma and gas cooking, but not with indoor NO_2." I interpreted this as follows: "Thus, they [Lin et al.] report no significant statistical association specifically between NO_2 and pediatric asthma; moreover, *any associations, significant or not, are not necessarily causal*" (emphasis added). It is this italicized part that I think is most important. The real issue, as I see it, is that what Lin et al. found is that the statistical effect (or exposure-response association) for a 15 ppb increase in NO_2 could be anywhere between a reduced average asthma risk (lower 95 % CI = 0.91) to no effect to an increased risk (upper 95 % CI = 1.31), and that, in any case, this effect *has not been shown to be causal*. Yet, Kashtan et al. cite this ambiguous evidence as supporting their causal beliefs that NO_2 causes childhood asthma. I think this oversteps the evidence and misinterprets what was actually shown. That was the point I sought to emphasize.

Very similar remarks hold for the paper of Puzzolo et al. [12]. Nadeau et al.'s letter states that "the effect size Puzzolo et al. find is large. They find that children who live in a house with a gas stove are 9% more likely to develop asthma than those who live with an electric stove." But I think this misrepresents Puzzolo et al.'s finding, which they explain as follows (emphases added): "Compared with electricity using gas for cooking or heating *did not result in a higher risk estimate for asthma in children* (OR 1.09, 95% CI 0.99–1.19; p = 0·071; 20 studies… Sensitivity analyses… found use of gas to have a smaller, non-significant effect for studies with better adjustment for confounding… suggesting *the association between use of gas and asthma compared with electricity was at least partially explained by confounding* from exposure to tobacco smoke, ambient air pollution, or socioeconomic status." Kashtan et al. [1] cite this as supporting their causal conclusions, writing that "Combining data from the 2020 RECS with the 2024 meta-analysis of Puzzolo et al. of the association between pediatric asthma and gas stoves, we estimate that gas and propane stoves in the United States *are responsible for 200 [95% CI: −20, 410] thousand current cases* of pediatric asthma" (emphases added). I objected to the causal language ("responsible for") and to the suggestion that Puzzolo et al.'s meta-analysis supports an inference that gas stoves in the US cause (or "are responsible for") any cases of pediatric asthma, let alone 200,000, given that what the authors actually reported was that "using gas for cooking or heating did not result in a higher risk estimate for asthma in children." Puzzolo et al., far from supporting any causal conclusion, provide evidence that there is no sound statistical basis for making such a claim.

In discussing potential confounding, Nadeau et al. state that "neither Cox nor Lin et al. identify any uncontrolled confounding pathways that could produce a positive bias. Thus it is infeasible to assess the direction of any such confounding." However, a substantial literature addresses already such potential confounding pathways. As noted in Cox (2023), "For example, the first study cited by Lin et al., a study by Belanger and Triche, states that 'Gas stove use is particularly common in central cities; in the United States, inner cities are associated with high rates of poverty and substandard housing. In rural areas, the use of secondary heating devices (kerosene heaters, unvented gas) may be a means to reduce heating costs among low-income families. Thus*, it is possible that gas stoves and portable heating devices are markers for poverty and substandard housing, and this confounds any association with asthma*.' (Emphasis added.)" In addition, as just discussed, Puzzolo et al. [12] conclude that the evidence they present suggests that "the association between use of gas and asthma compared with electricity was at least partially explained by confounding from exposure to tobacco smoke, ambient air pollution, or socioeconomic status." Thus, while Nadeau et al. may be unaware of "any uncontrolled confounding pathways that could produce a positive bias," the literature reflected in their citations suggests poverty, substandard housing, environmental tobacco smoke, ambient air pollution, and socioeconomic status as some specific examples of such pathways. It is noteworthy that many other epidemiological studies do control for such obvious potential confounders.

Discussion and Conclusions.

Publications such as Kashtan et al. [1] raise the important practical question of what types of evidence – or perhaps whether any empirical evidence at all – are needed to support large risk estimates and calls for tighter regulation of activities such as use of gas stoves. On the one hand, "weight of evidence" enthusiasts may discern what they feel should be considered evidence of causation, reasons for concern, and needs for increased regulation in nearly any set of vaguely associated facts, such as the "evidence" cited by Nadeau et al. that NO_2 induces changes in mouse lung cells or in human lung function under certain conditions and has confidence intervals that include a relative risk of 1 for associations with childhood asthma in some observational studies that do not fully control for obvious confounders. On the other hand, a substantial discipline of rigorous data-driven causal modeling and testing now exists (Huntington-Klein, 2022; [4,7,5]) that shows how valid interventional causal conclusions can be drawn from observational data under certain conditions using appropriate technical methods such as conditional independence testing.

Yet Nadeau et al. do not verify the required conditions or apply these appropriate methods of causal analysis to support their causal conclusions. Rather, they rely on an argument, perhaps more political than scientific, that the US EPA and other authorities have long used similar models and assumptions and that their experts feel comfortable assigning a causal interpretation to the results. I believe that this begs the question of whether reducing NO_2 emissions from gas stoves in fact reduces childhood asthma. The question is worthwhile. A trustworthy answer requires more than the opinions of selected experts, especially since such expert opinions often have very uncertain validity [13]. Rather, a trustworthy answer requires the following steps:

- Collect relevant data, specifically on changes in exposures to NO_2 emissions from gas stoves and changes (or lack of them) in childhood asthma rates.

- Control for known, readily observed, potential confounders, such as poverty, substandard housing, environmental tobacco smoke, ambient air pollution, and socioeconomic status.
- Test for any hidden confounders [14] and for residual confounding.
- Test whether childhood asthma rates are conditionally independent of gas stove emissions given these confounders; and
- Acknowledge that, if confidence intervals contain substantial ranges of both positive and negative associations, then they do not provide strong statistical reason to conclude that there is a large positive effect.

These steps have not yet been taken in the work reported by Kashtan et al. [1] and defended by them in Nadeau et al. [15]. Instead, they state that "Cox's rebuttal uses the inescapable limitations of certain types of studies to cast doubt on the field's findings entirely. This method of casting doubt on a scientific consensus is not new and has been used to challenge the science on smoking…". However, this is a strawman fallacy, perhaps more suitable to inflame than to engage in a dispassionate, logical evaluation of the available evidence. I do not suggest that drawing sound and trustworthy conclusions requires overcoming "inescapable limitations," but rather that it requires taking such basic precautions as collecting relevant data, controlling for obvious potential confounders, and not misrepresenting absence of a statistical effect as presence of a causal one. The limitations engendered by failing to take these basic steps are self-imposed. They are entirely avoidable by scientists who are willing to undertake the hard, important work of learning from relevant data how the world actually works, rather than just assuming it [13].

Nadeau et al. suggest that, by pointing out the lack of empirical support for their conclusions, I am "casting doubt on a scientific consensus" in a way that "is not new and has been used to challenge the science on smoking." This highlights the absence of a scientific response (along the lines of "Here are the data sets we analyzed, the tests we used, and the results of those tests, which you may independently verify") and their reliance on essentially political and rhetorical devices, i.e., "scientific consensus," which many consider an oxymoron [16], to determine what shall be said to be true. This is the antithesis of sound science. In sound science, agreement of testable and falsifiable predictions with subsequent observations, not claimed "scientific consensus" (especially one with which the cited sources conspicuously disagree, as reviewed earlier), provides the warrant for provisional conclusions about the validity of causal claims [13].

Nadeau et al. end their letter by staying that "Our conclusions – that NO_2 exposure from gas stoves causes asthma – stand." It should be clear from our discussion that if they stand, it is not because they have been shown to be correct or plausible. They are not implied by the data cited, as the original authors of the cited studies make clear. If they stand at all, it is only in the sense that the authors feel that appeals to tradition and authority make a satisfactory substitute for sound science showing that the causal claims are implied by data in a way that others can independently confirm. I believe that it is both possible and important to do better.

CRediT authorship contribution statement

Louis Anthony Cox: Conceptualization, Writing – original draft, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Louis Anthony Cox Jr.

Cox Associates, Entanglement, University of Colorado at Denver, Denver, CO, USA

E-mail address: tcoxdenver@aol.com.