PUBLIC HEALTH

Nitrogen dioxide exposure, health outcomes, and associated demographic disparities due to gas and propane combustion by U.S. stoves

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Gas and propane stoves emit nitrogen dioxide (NO_2) pollution indoors, but the exposures of different U.S. demographic groups are unknown. We estimate NO_2 exposure and health consequences using emissions and concentration measurements from >100 homes, a room-specific indoor air quality model, epidemiological risk parameters, and statistical sampling of housing characteristics and occupant behavior. Gas and propane stoves increase long-term NO_2 exposure 4.0 parts per billion volume on average across the United States, 75% of the World Health Organization's exposure guideline. This increased exposure likely causes ~50,000 cases of current pediatric asthma from long-term NO_2 exposure alone. Short-term NO_2 exposure from typical gas stove use frequently exceeds both World Health Organization and U.S. Environmental Protection Agency benchmarks. People living in residences <800 ft^2 in size incur four times more long-term NO_2 exposure than people in residences >3000 ft^2 in size; American Indian/Alaska Native and Black and Hispanic/Latino households incur 60 and 20% more NO_2 exposure, respectively, than the national average.

INTRODUCTION

Gas stoves are used in approximately 50 million U.S. homes (*1*) and millions more worldwide (*2*, *3*). Gas and propane combustion in stoves emits hazardous air pollutants, including nitrogen dioxide (NO_2), benzene (C_6H_6), carbon monoxide (CO), formaldehyde (H_2CO), and ultrafine particles (*4*–*12*). Nitrogen dioxide and benzene emissions are of particular concern, as typical gas stove use can elevate indoor concentrations of these pollutants above health benchmarks (*5*, *6*, *8*, *10*, *11*). Long-term exposure (averaged over a year) to NO_2 has been linked to increased incidence and exacerbation of pediatric asthma (*13*–*16*), incidence and mortality from chronic obstructive pulmonary disease (COPD) (*17*–*19*), and incidences of lung cancer, preterm birth, and diabetes mellitus (*20*). Given the abundance of gas and propane stoves and the dangers of additional NO_2 exposure generally, quantifying the burden of NO_2 exposures and health outcomes from gas and propane combustion by stoves is needed for assessing public safety.

Previous studies estimating disease burdens attributable to combustion by gas stoves have limitations that reduce their abilities to assess exposure as a function of different behaviors or across different socioeconomic, racial, or ethnic groups. First, constraining estimates of indoor NO_2 exposure from direct measurements of indoor concentrations is challenging because the data are sparse and because observed concentrations can fluctuate with ventilation and stove use (*21*, *22*). Second, studies that correlate health outcomes with the presence or absence of a gas stove (rather than with direct pollutant exposure) typically do not capture large differences in housing size and layout, ventilation, or behavior that may substantively affect exposure across groups (*23*–*25*), hindering investigations of health disparities mediated through such factors. Existing meta-analyses calculating odds ratios (ORs) of specific health outcomes associated with gas stoves have relied either on correlations between measured indoor NO_2 concentrations and health outcomes, as opposed to directly quantifying NO_2 exposure, or have used the presence of gas stoves as a proxy for NO_2 exposure (*16*, *26*).

Gaps in current epidemiological knowledge can be addressed using detailed assessments of the population-wide distribution of long-term (year-averaged) and short-term (hour-averaged) NO_2 exposure attributable to combustion by natural gas (which is composed of >90% methane) and propane stoves. Previous studies have quantified NO_2 emission rates from gas stoves (*6*, *10*, *27*) but not, to our knowledge, from propane stoves. Previous studies (*5*, *8*, *10*, *28*–*31*) have also measured the resulting indoor NO_2 concentrations in a handful of residences representing a single building type or a local geography. Models have focused either on individual buildings (*32*–*34*) or on a specific, local geography (*35*) or, if encompassing a broader geography, have omitted variation in cooking and occupancy patterns (*36*). The two previous studies that assessed exposure in multiple building types relied on models that treat residences as open boxes that lacked interior walls rather than as homes with discrete rooms and hallways (*35*, *36*); hence, these single-zone models do not capture higher short-term exposures while people are cooking or spending additional time in the kitchen.

We constructed a population-level model of NO_2 exposure from combustion by gas and propane stoves using the National Institute of Standards and Technology's (NIST's) CONTAM multizone indoor air quality model (*37*) and our field measurements of NO_2 emission rates from gas and propane stoves. We ran the model on more than 31,100 combinations of input variables whose distributions and weights we obtained from our field measurements combined with published datasets for the U.S. housing stock and relevant behaviors of the U.S. population (see Materials and Methods). This model allowed us to estimate NO_2 exposure attributable to gas and

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propane stoves stratified by various environmental, behavioral, and demographic parameters, including residence size and layout, time spent with windows open, frequency of range hood use, range hood capture efficiency, and time spent in the kitchen and other rooms. We also updated previously published (*10*) NO_2 emission rates from gas stoves with field measurements of 50 additional homes (over 70 total homes when including measurements of propane and electric stoves), bedroom NO_2 concentrations in six houses for 8 hours during and following stove use, and NO_2 emission rates from propane stoves. We validated the CONTAM model by comparing our modeled and measured NO_2 concentrations in a set of 18 test houses of various sizes and floorplans, in each case directly measuring NO_2 concentrations in multiple rooms before, during, and––for several hours, at least––after gas and propane stove use ended.

Using the validated CONTAM model, we estimated long- and short-term NO_2 exposure attributable to combustion by gas and propane stoves for the U.S. population overall and in subpopulations, including various income levels and racial/ethnic groups (see Materials and Methods). We then used well-established epidemiological relationships (*26*, *38*) for NO_2 exposure to estimate the excess cases of pediatric asthma and adult mortality attributable to long-term NO_2 exposure from gas and propane stoves. We compare our results against estimates of pediatric asthma burden attributable to gas stoves overall (*16*).

RESULTS

Agreement between the CONTAM model and measured NO_2 concentrations

We assessed the performance of our indoor air model by comparing its outputs against NO_2 measurements in 18 test residences before, during, and after gas stove use. These residences ranged in size from 280 to 1650 ft^2 (25 to 150 m^2). They included both apartments and single- and multistory detached homes in the following locations: San Francisco Bay Area, CA; Los Angeles, CA; Bakersfield, CA; Denver, CO; Houston, TX; New York City, NY; and Washington, DC. We included scenarios with windows open and closed and with an outside-venting range hood both on and off. We observed close agreement between modeled and actual NO_2 concentrations ($r^2 = 0.64$, slope = 0.89, SE of slope = 0.11, $P \ll 0.01$; see Fig. 1 for plots of the largest and smallest test residences; see fig. S1A for a regression of modeled versus actual concentrations and fig. S1B for boxplots summarizing all measured residences). The model results showed no evidence of systematic bias relative to measurements as assessed statistically (fig. S1 and table S1). Without an outside-venting range hood on and with either one burner or one burner and one oven on, concentrations in over half of the kitchens, living rooms, and bedrooms tested exceeded the Environmental Protection Agency's (EPA's) 1-hour ambient exposure benchmark of 100 parts per billion volume (ppbv) (*39*) and the World Health Organization's (WHO's) 1-hour (i.e., short-term) exposure guideline of 200 $\mu g/m^3$ (~100 ppbv) (*40*). Surveys show that range hoods are used only 15 to 39% of the time (*41*, *42*).

Nitrogen dioxide emission factors from propane stoves and updated emission factors for gas stoves

Nitrogen dioxide emissions occurred only with fossil fuel use and scaled linearly with the amount of fuel burned, as assessed by CO_2 emissions, across 10 propane and 50 natural gas stoves (Fig. 2 and fig. S3A; $r^2 = 0.61$ for gas and 0.70 for propane; $P \ll 0.01$ for each). In contrast to the results for fossil fuel combustion, all 7 induction and 12 electric coil and radiant stoves that we measured had zero NO_2 emissions (i.e., emissions were statistically indistinguishable from zero; see Fig. 2 and table S3).

NO_2 emissions from propane and gas combustion were statistically identical when normalized per joule of fuel burned (table S2). Because NO_2 forms in flames as a function of temperature (*43*) and the adiabatic flame temperatures of methane and propane differ by less than 1% (*44*), it is unsurprising that gas and propane burners emitted the same amount of NO_2 per joule of fuel burned.

Estimates of NO_2 emission factors calculated from the 50 gas stoves measured newly in this work were statistically identical to the emission factors measured previously for 32 gas stoves by Lebel *et al.* (*10*). Combining data from this work with the data from Lebel *et al.*, we calculated median NO_2 emission factors for gas stoves to be 8.7 [95% confidence interval (CI): 8.2, 9.3] ng J^{-1} for gas burners on high and 8.2 [95% CI: 7.5, 9.2] ng J^{-1} for burners on low. See fig. S3B and table S3 for emission rates and emission factors per joule for all burner and oven types measured. We used results from the pooled set of 82 burners in our modeling.

NO_2 concentrations in bedrooms and with range hoods on

Bedroom NO_2 concentrations tested during oven use, with interior doors open and the range hood off exceeded the U.S. EPA's 1-hour ambient benchmark (*39*) and the WHO's 1-hour exposure guideline (*40*) within 25 min in half the homes we tested (three of six homes). In two test cases, bedroom NO_2 levels remained above health-based guidelines for 2 to 3 hours after the oven was turned off (houses 1 and 2; Fig. 3A). We found that an outside-venting range hood reduced peak NO_2 concentrations in some cases (Fig. 3B) but that some outside-venting range hoods are ineffective at reducing NO_2 concentrations (Fig. 3C). Across a subset of five randomly selected homes, we found that outside-venting hoods reduced hour-averaged kitchen NO_2 concentrations by between 10 and 70% (mean reduction in concentration = 35%, $n = 5$; fig. S15). This result is consistent with prior work assessing the efficacy of installed range hoods, which found that most hoods operating in homes have capture efficiencies well below 70% (*45*, *46*). Our measurements both with and without hoods on further support our model's finding that gas and propane stove use increases long- and short-term NO_2 exposures (see below).

Modeled long- and short-term NO_2 exposure

We estimated long- and short-term NO_2 exposure attributable to gas and propane stoves by combining our measured NO_2 emissions data with published housing characteristics and with published statistical distributions of resident use patterns. These variables included how much time a person spent cooking, how much gas was burned (i.e., how many burners/ovens were used plus cooking duration), time spent with windows open, percentage of cooking time with the range hood on, and capture efficiency of the range hood used—all applied in a multizone indoor air quality model (see Materials and Methods). We calculated CIs using a Monte Carlo method (see Materials and Methods). See the "Definitions" section for more details on how we calculated long-term (year-averaged) and short-term (hour-averaged) exposures.

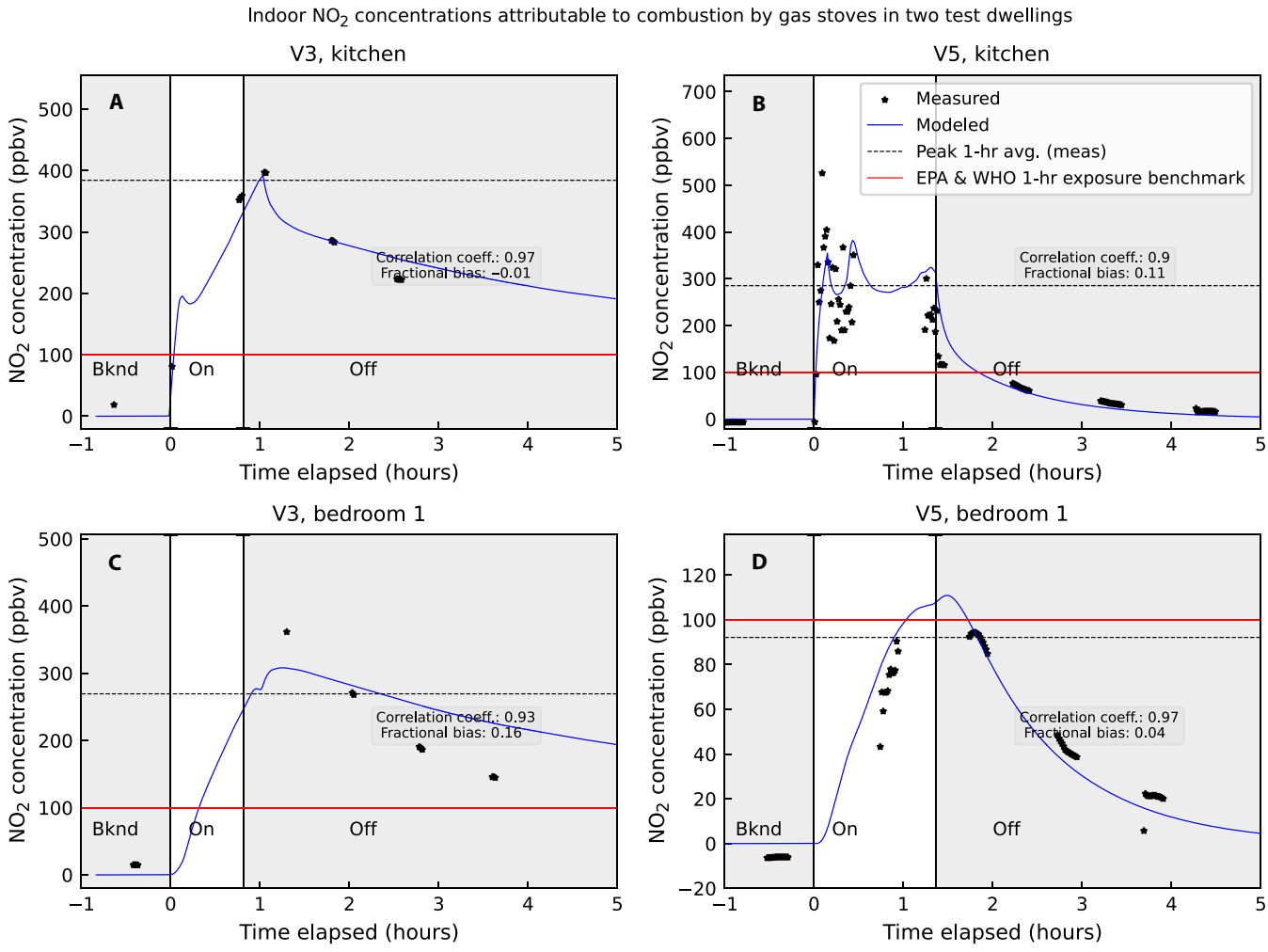

Fig. 1. Measured and modeled NO_2 concentrations in two test residences. ("V3" on the left and "V5" on the right). NO_2 concentrations measured (black points) and modeled (blue lines) in the (**A**) kitchen and (**C**) bedroom (farthest from the kitchen) of a 900-ft^2 (85-m^2) house, "V3," and in the (**B**) kitchen and (**D**) bedroom of a 1500-ft^2 (140-m^2) house, "V5" (see table S2 for metrics for each house). "Background" ("Bknd") represents NO_2 concentrations in a given room before lighting the stove, "On" represents concentrations with one stove burner on high and the oven set to 350°F (175°C), and "Off" represents concentrations after turning off the stove burner and oven. "Time elapsed" represents hours since lighting the burner and oven. The white background demarcates the interval in which the burner and oven were in use. The horizontal black line represents the highest concentrations measured in each room, time-averaged over 1 hour. The solid horizontal red line represents the U.S. Environmental Protection Agency's (EPA's) 1-hour ambient exposure benchmark of 100 ppbv (*39*) and the World Health Organization's (WHO's) 1-hour exposure guideline of 200 μg/m^3 (~100 ppbv) (*40*). A range hood was not used in the tests shown here, though we tested the CONTAM model with a range hood on and window open. See fig. S2 for analogous plots and fig. S1 for summary plots of all tests including tests with range hoods on and windows open. The correlation coefficients (*r*) and fractional biases of the modeled concentrations are reported for each room and for all rooms together (see table S1).

Based on our results, gas and propane stoves in the United States elevate long- and short-term NO_2 exposure substantially. We estimate that U.S. median gas and propane stove use increases long-term NO_2 exposure by 4.0 [95% CI: 2.4, 6.1] ppbv. Maximum daily hour-averaged exposure to NO_2 from median gas and propane stove use in the United States exceeds 200 μg/m^3 (~100 ppbv), the WHO's 1-hour indoor exposure guideline (*40*), on 12 [95% CI: 4, 24] days of the year (3.3% of days), averaged across the population. Home cooks who are in the 95th percentile of stove use encounter ~110 days per year exceeding 200 μg/m^2 (see below).

These long- and short-term stove-attributable exposures are large relative to common health benchmarks. For instance, the 4.0 ppbv of stove-attributable long-term exposure comprises 75% of the WHO's annual NO_2 exposure guideline (*47*) (Fig. 4) and 50% of the U.S.-averaged outdoor NO_2 concentration (*48*) in 2021. In addition to exceeding the WHO 1-hour exposure guideline, stove-attributable exposures would also exceed the U.S. EPA's outdoor standard of no more than 2% of days with maximum NO_2 concentrations exceeding 100 ppbv (*39*) if this standard were applied indoors (fig. S5). Although the U.S. EPA does not currently regulate indoor air pollution (*39*), Canada does—its hour-averaged indoor NO_2 standard is 170 μg/m^3 [~90-ppbv NO_2], slightly lower than the WHO standard (*40*, *49*).

Long- and short-term exposure burdens from combustion by gas and propane stoves are unequally distributed across the U.S. population. Gas and propane stoves increase the long-term

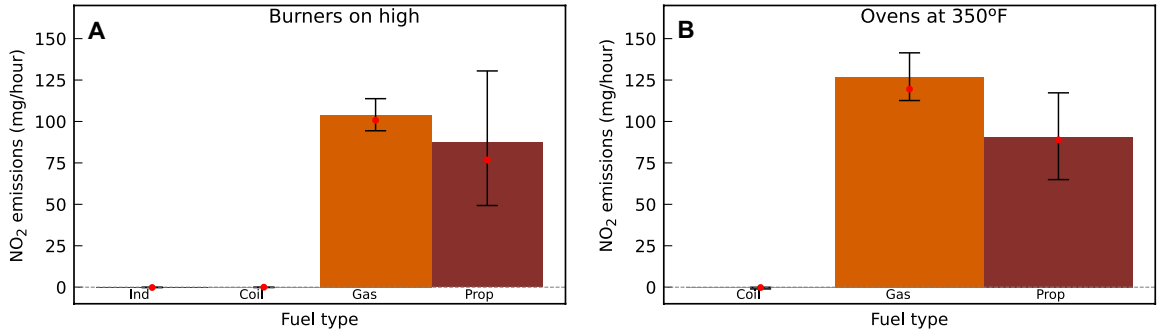

Fig. 2. Mean and median NO_2 emissions by fuel type. Emissions reported in milligrams of NO_2 per hour by fuel type (electric induction, electric coil/radiant, gas, and propane) for burners on high (**A**) and for ovens set to 350°F (175°C) (**B**). The red points are median values, the bar heights are mean values, and the black error bars are the 95% CIs of the mean (calculated using a bootstrap method as described in the methods).

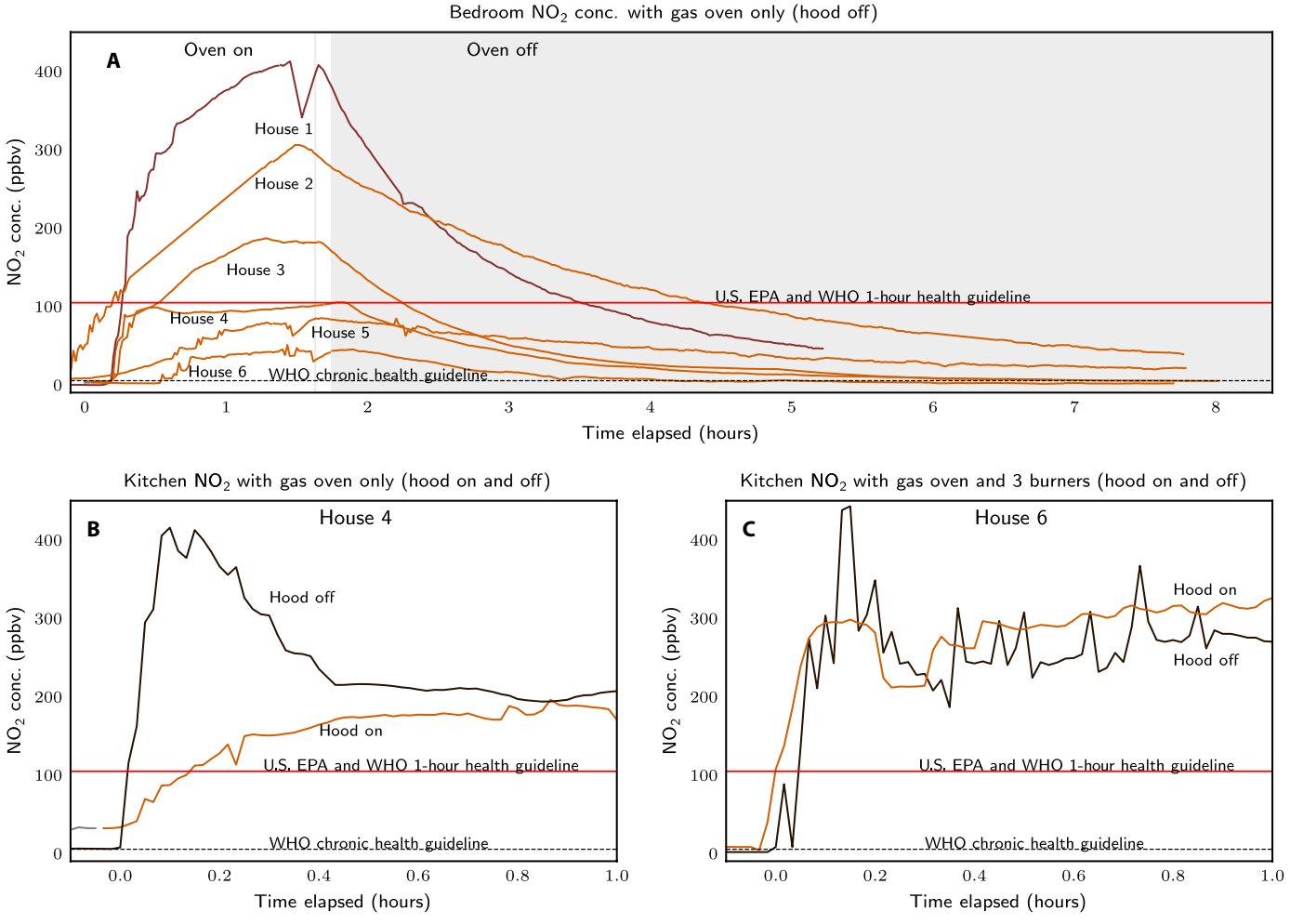

Fig. 3. Timecourses of NO_2 concentrations in bedrooms during and after oven use. (**A**) NO_2 concentrations (parts per billion volume) measured in bedrooms furthest from kitchens with the oven set to 475°F (245°C) for 1.5 hours and then turned off for 3.5 to 6.5 hours to mimic a common "bread-baking" scenario. Air-sampling hoses in houses 2, 3, 4, 5, and 6 were placed in the farthest bedrooms ~8 m down the hall from the kitchen; the sampling hose in house 1 was also in the farthest bedroom, but it was only ~4 m from the kitchen. (**B**) NO_2 concentrations measured in the kitchen of house 4 with the oven set to 475°F for 1 hour with the house's outside-venting hood off (dark brown) and on (orange), and (**C**) NO_2 concentrations measured in the kitchen of house 6 with three burners on high and the oven set to 350°F (175°C), corresponding with a higher-use scenario in the RECS database, for 1 hour with the house's outside-venting hood on (orange) and, for comparison, off (dark brown). The red horizontal line represents the U.S. EPA's 1-hour ambient concentration benchmark of 100 ppbv (*39*) and the WHO's 1-hour exposure guideline of 200 μg/m^3 (~100 ppbv) (*40*); the horizontal dashed line near the bottom of the plot represents the WHO's long-term exposure guideline of 10 μg/m^3 (*60*).

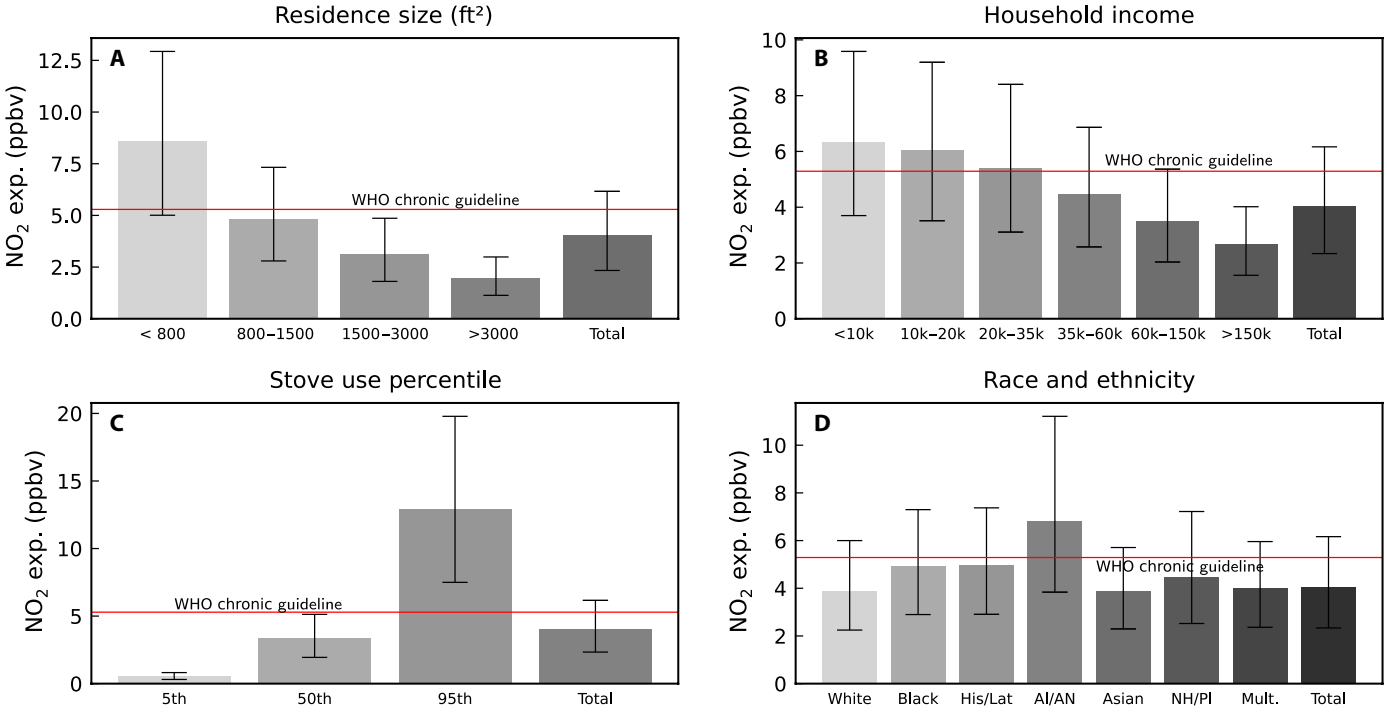

Fig. 4. Additional long-term NO_2 exposure attributable to combustion by gas and propane stoves in the United States. NO_2 exposure stratified by: (**A**) Home size (ft^2). 800 ft^2, 75 m^2; 800–1500 ft^2, 75–140 m^2; 1500–3000 ft^2, 140–280 m^2; >3000 ft^2, >280 m^2. (**B**) RECS respondent household income (US$ per year; k, $1000). (**C**) Amount of gas or propane stove use as a percentile in the population as measured by burner minutes and oven minutes (proportional to total enthalpy of gas or propane burned; see Materials and Methods). (**D**) Race/ethnicity self-reported to RECS. The racial and ethnic categories identified by RECS include (*1*): His/Lat, Hispanic or Latino; AI/AN, American Indian or Alaska Native; NH/PI, Native Hawaiian or Pacific Islander; Mult., multiracial. In each panel, "Total" refers to the average across the U.S. population. The red horizontal line in each plot is the WHO's annual NO_2 guideline (*47*). Error bars represent 95% CIs calculated using a Monte Carlo method (see Materials and Methods).

NO_2 burden of people living in residences <800 ft^2 (75 m^2) in size by 8.6 [95% CI: 5.1, 13] ppbv, more than four times the exposure of people living in residences >3000 ft^2 (280 m^2) in size (2.0 [95% CI: 1.2, 3.0] ppbv; Fig. 4A). Short-term exposures are also substantially higher for people living in smaller residences; people living in residences <800 ft^2 experience more than nine times as many days with 100-ppbv exceedances as people living in residences >3000 ft^2 in size (fig. S5A).

The relationship between increased exposure and smaller residence size also drives disparities in exposure burden borne by different U.S. subpopulations. For instance, people in households making <$10,000 per year, as recorded by the Residential Energy Consumption Survey (RECS) (*1*), are exposed to 6.3 [95% CI: 3.7, 9.5]–ppbv long-term NO_2 from gas and propane stoves. This additional exposure is more than twice that of people in households making >$150,000 per year (Fig. 4B). Short-term exposures follow the same pattern of increased risk for poorer residents; people in households making <$10,000 per year are exposed to three times as many days with 100-ppbv exceedances as people in households making >$150,000 per year (fig. S5B).

Racial and ethnic disparities arise in exposures attributable to gas and propane combustion from stoves, as well. Using RECS' racial/ethnic categories, we found that people in households with American Indian/Alaska Native respondents experience the highest stove-attributable long-term NO_2 exposure (6.8 [95% CI: 4.0, 10.0] ppbv), followed by Hispanic/Latino and Black households (5.0 [95% CI: 2.9, 7.3] and 4.9 [95% CI: 2.9, 7.2] ppbv, respectively). These exposure levels represent 60, 20, and 20% more than the average U.S. stove-attributable exposure, respectively, and, for all three groups, exceed WHO's total annual exposure benchmark just from using a gas stove—before including any contribution from outdoor air pollution (*47*). Households with white (3.9 [95% CI: 2.3, 5.9] ppbv) and Asian (3.9 [95% CI: 2.3, 5.7] ppbv) respondents experienced the lowest stove-attributable long-term NO_2 exposures of all racial/ethnic groups identified in the RECS database (Fig. 4C; $P < 0.01$ for pairwise group comparison of white and Asian exposures versus Black, Hispanic/Latino, and American Indian/Alaska Native exposures). Short-term exposures follow the same outcomes as long-term exposures; people in households with Hispanic/Latino, Black, and American Indian/Alaska Native respondents experience between 40 and 100% more days with 100-ppbv exceedances than the national average (fig. S5C). The racial and ethnic disparities we observed are primarily influenced by differences in average residence sizes: 29, 23, and 23% of American Indian/Alaska Native, Hispanic/Latino, and Black respondents, respectively, live in residences <800 ft^2 in size, whereas only 12 and 20% of white and Asian respondents do, respectively (*1*).

Both long- and short-term NO_2 stove-attributable exposures are strongly affected by behavioral factors, including the duration and intensity of stove use (i.e., how much gas is burned), range hood use and window opening, and time spent in the kitchen. The dominant factor predicting NO_2 exposure in our analysis was the total amount

of gas or propane burned. People in residences in the 95th percentile of duration of burner and oven use (corresponding with two burners on medium for 30 min daily in the morning, and four on medium for 30 min and the oven set to 350°F (175°C) for roughly 2.25 hours in the evening) (*41*) were exposed to 3 times more than the average long-term stove-attributable NO_2 and 25 times more than people in households in the 5th percentile of burner and oven use (i.e., one burner on medium for 5 min daily in the morning and two on for 5 min in the evening with no oven used; Fig. 4D). Short-term exposures follow the same trend: People in houses in the 95th percentile of duration of burner and oven use experience 110 [95% CI: 50, 170] days per year with a 100-ppbv exceedance while people in the 5th percentile typically experience zero (fig. S5D). See data S1 for numerical values and Materials and Methods for a more detailed description of the distribution of burner and oven use.

Model sensitivity

We assessed the sensitivity of the CONTAM model combined with our exposure calculations by altering parameters systematically and estimating the effects of parameter changes on long- and short-term NO_2 exposure risk. We varied the levels of each input parameter from the 5th to 95th percentiles (or minimum to maximum values, if distribution statistics were unavailable), holding all other parameters constant at their default values (see table S7). Our analysis showed that long-term NO_2 exposure was most sensitive to duration and intensity of gas stove use (as measured by total burner minutes and oven minutes, which are proportional to enthalpy of gas or propane burned; see Materials and Methods).

The next-most important behavioral factors in determining exposure (after the amount of fuel burned) were mechanical and natural ventilation and time spent in the kitchen. People who lack an outside-venting hood or who do not use their hoods are exposed to 25% more long-term stove-attributable NO_2 than average. Meanwhile, people with a 75% capture efficiency outside-venting hood who use it every time they cook are exposed to 70% less long-term stove-attributable NO_2 than average. The effect on long-term stove-attributable NO_2 of opening and closing windows (one window modeled in the kitchen and at least three additional windows, opened or closed all at once; see Materials and Methods) was comparable to the average benefit of using a range hood (Fig. 5).

However, opening a window was far less helpful in reducing short-term exposures, measured by days per year with a 1-hour averaged NO_2 exposure >100 ppbv. Whereas people who leave their windows closed are exposed to nine times more long-term NO_2 than people who leave their windows open, people who leave their windows closed are exposed to only 1.5 times more days per year with a 100-ppbv exceedance (compare the relative sizes of the burnt orange bars in Fig. 5, A and B).

Prevalence and economic burden of stove-attributable adverse health outcomes

Combining data from the 2020 RECS (*1*) with the 2024 meta-analysis of Puzzolo *et al.* (*26*) 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. Our central estimate represents roughly 10% the number of pediatric asthma cases attributable to pollution from all road traffic (*50*). Following the calculation of Nurmagambetov *et al.* (*51*), applying the EPA's value of a statistical life (VSL) to asthma-induced deaths and combining this cost with asthma-related medical costs yields an annual societal cost of gas and propane stoves of $1 (0, 2) billion. Our estimate of pediatric asthma population-attributable fraction (PAF) (see Table 1) is smaller than but statistically indistinguishable from that reported by Gruenwald *et al.* (*24*), who relied on an older meta-analysis (*16*).

Our estimate of long-term NO_2 exposure attributable to gas stoves allows us to address what portion of pediatric asthma attributed to gas stoves overall may be due specifically to long-term NO_2 exposure. Our central estimate for pediatric asthma attributable to long-term NO_2 exposure from stoves (Table 1) is ~25% of the estimate for stoves overall. This discrepancy may be due to several potential factors. These may include (i) that the majority of stove-attributable pediatric

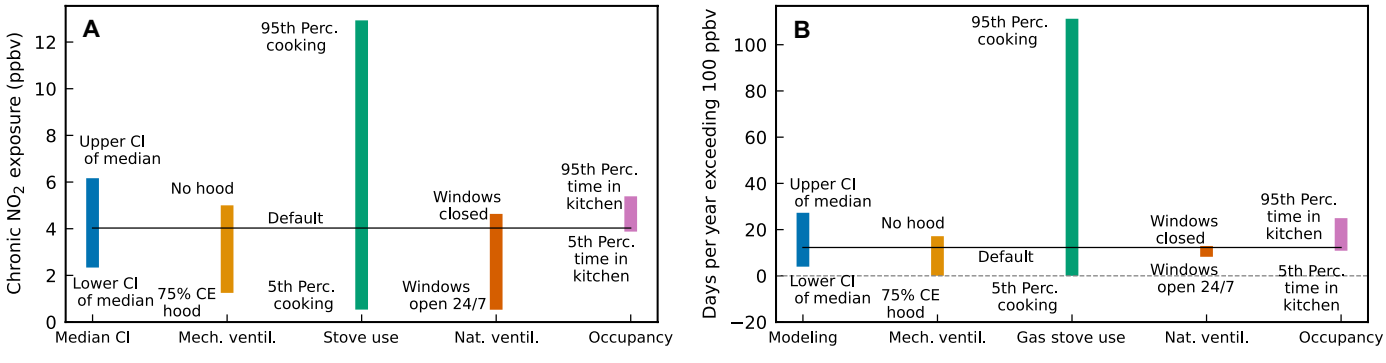

Fig. 5. Sensitivity analysis of exposure estimates. Modeled increase in (**A**) long-term NO_2 exposure (parts per billion volume) and (**B**) days with 1-hour 100-ppbv exceedances attributable to gas and propane stoves at median values of model input parameters and at high and low ends of the observed distributions. The black horizontal line in each panel indicates the modeled exposure (A) and exceedances (B) assuming default input values of all parameters (see table S7). The blue vertical range at the left of each panel spans the 90% CI of the median (from 5th to 95th percentiles; see Materials and Methods). Light orange ranges span central estimates of exposure corresponding with maximum and minimum reasonable use cases for mechanical ventilation (based on surveys and direct measurements; see Materials and Methods). Green ranges span central estimates of exposure corresponding with values modeled between the 5th and 95th percentiles of joules of gas or propane burned. Burnt orange ranges span central estimates of exposures corresponding with the maximum and minimum reasonable use cases for window opening (based on surveys, see Materials and Methods). Lilac ranges to the right span values modeled for 5th to 95th percentiles of kitchen occupancy. "CI" and "CE" stand for confidence interval and capture efficiency, respectively.

Table 1. Pediatric asthma outcomes attributable to long-term exposure to NO_2 pollution from gas and propane stoves. Modeled population attributable fraction (PAF) and annual burden attributable to the presence of gas and propane stoves (i.e., as a function of having a gas stove independent of any exposure estimate) and to long-term NO_2 exposure from gas and propane stoves in the United States. The right-most column reports the odds ratio (OR) used to calculate the asthma burden attributable to gas stoves overall (*26*) and the relative risk (RR) per 15-ppb increase in annual indoor NO_2 exposure used to calculate the asthma burden attributable to NO_2 exposure (*16*). Parenthetical numbers indicate 95% CIs calculated using a Monte Carlo method (see Materials and Methods) or, for OR and RRs, come from values reported in the literature (*16*, *26*). See data S2 and S3 and fig. S6 for a breakdown by individual U.S. states and for gas and propane stove prevalence by state (*1*).

Attribution	Pediatric asthma PAF	Modeled current pediatric asthma burden (thousand cases)	Pediatric asthma OR or RR
Presence or absence of gas stove	3.8 [0.0, 8.0] %	180 [−20, 380]	1.09 [0.99, 1.19] (OR)
Presence or absence of propane stove	0.36 [0.00, 0.75] %	17 [−2, 36]	1.09 [0.99, 1.19] (OR)
Long-term NO_2 from gas stoves	0.91 [−1.33, 3.0] %	43 [−63, 142]	1.09 [0.91, 1.31] (RR per 15 ppb)
Long-term NO_2 from propane stoves	0.09 [−0.13, 0.0.29] %	4 [−6, 14]	1.09 [0.91, 1.31] (RR per 15 ppb)

asthma cases are due to additional factors such as short-term NO_2 exposure and other gas stove pollutants; (ii) that estimates of pediatric asthma attributable to stoves do not fully account for confounding variables and could be too high; and (iii) that our model underestimated the true long-term NO_2 exposure attributable to gas and propane stoves (see limitations discussed in our conclusion and in our methods).

Exposure to NO_2 outdoors has been associated with statistically significant increases in all-cause adult mortality, though quantifying its direct effect is challenging because of potential confounding with exposure to co-occurring outdoor pollutants such as particulate matter in automobile exhaust and other combustion sources (*38*, *52*, *53*). Assuming that meta-analyses of outdoor NO_2 and all-cause adult mortality may be applied to long-term exposure to indoor NO_2, our analysis suggests that long-term NO_2 exposure from gas and propane stoves in the United States may be responsible for up to 19,000 [95% CI: 8500, 34,000] deaths annually—0.67 (0.29, 1.2)% of total U.S. adult deaths—or roughly 40% the number of deaths attributable to secondhand smoke (*54*). Applying the U.S. EPA's VSL (*55*) to each death yields an annual societal cost of gas and propane stoves of $250 (75, 480) billion (data S2), or approximately $4500 per year per U.S. household with a gas or propane stove, based on 2020 RECS data (*1*). These estimates likely overestimate the health and cost burdens attributable to NO_2 because of additional pollutants found in traffic-related air pollution. However, they also underestimate health and cost burdens because our estimates account for only long-term NO_2 exposures and not short-term exposures to high concentrations, which routinely exceeded 100 ppbv in our measurements (see Figs. 1 and 3). Better disambiguation between the effects of NO_2 and $PM_{2.5}$ as well as more studies on short-term NO_2 effects are needed to constrain NO_2 mortality estimates.

DISCUSSION

Gas stoves are common globally and in U.S. homes. Emissions from gas and propane stove combustion degrade indoor air quality (*3*, *10*, *40*, *56*, *57*) and are associated with adverse health outcomes that include pediatric asthma and hospitalizations (*23*–*25*, *52*, *58*, *59*). We report updated field estimates of NO_2 emission rates for gas stoves and previously unreported emission rates of NO_2 from propane stoves. We combined our emissions measurements with the NIST's CONTAM indoor air model and other published datasets; in doing so, we produced a population-level estimate of the distribution of NO_2 exposure and health outcomes attributable to gas and propane stoves as a function of specific behaviors and for different income and race and ethnicity groups (see Materials and Methods).

Consistent with previous research (*10*, *24*, *25*), we find that combustion from gas and propane stoves represents a major source of long- and short-term NO_2 exposure that can exceed U.S. and WHO guidelines just by using a stove, independent of any outdoor NO_2 exposures. Demographic detail provided by the RECS (*1*) and the precision afforded by the multizone CONTAM model (*37*) allowed us to extend previous research to the entire United States and to exposures in specific rooms, estimating both short- and long-term exposure as a function of behavioral and demographic variables such as gas and propane stove use, time spent in the kitchen and other rooms, and the income and race of occupants. Our findings also quantified the importance of gas and propane stove use (i.e., joules burned) compared with ventilation and occupancy in determining indoor NO_2 exposure. We also found that housing size greatly influences exposure. Differences in housing size partly drive income-based and racial disparities in stove-attributable NO_2 exposure, though there are other potentially relevant factors not captured by our model, such as social differences in cooking and ventilation behavior and differences in the time spent indoors and outdoors.

Our estimated health consequences of gas and propane stove use are large. We found that gas and propane stoves may contribute up to 19,000 adult deaths annually in the United States. We also estimated that long-term NO_2 exposure from gas and propane stoves is responsible for approximately 50,000 current cases of pediatric asthma. In addition, the total number of current pediatric asthma cases attributable to pollution from gas and propane stoves is likely closer to 200,000. That number of cases is approximately 10% of pediatric asthma attributable to pollution from road traffic and corresponds with a societal cost of roughly $1 billion annually.

Additional research could enhance future estimates of adverse health outcomes associated with gas and propane stoves. First, we assessed only one pollutant, NO_2, in this exposure assessment. Because gas stoves also emit carbon monoxide (CO), benzene, formaldehyde, and ultrafine particles (*7*, *9*–*11*), which are linked to adverse health outcomes beyond asthma (*40*), our estimates of disease burden and societal cost almost certainly underestimate the full health consequences of combustion from gas and propane stoves. Second, the granularity of our modeling was limited by the availability of

data on burner and range hood use for different geographies and demographic groups. Gathering and incorporating these data into an exposure model may produce a more precise estimate of socioeconomic and racial disparities in gas stove-attributable NO_2 exposure. Third, our study's quantification of all-cause mortality was limited by potential confounding of NO_2 with other coproduced pollutants outdoors. Future work focusing on the effect of indoor NO_2 on mortality, COPD, or other adverse health outcomes would enable modeling of the stove-attributable component of these diseases. Fourth, our study did not estimate the health effects of our measured short-term exposure to high NO_2 concentrations. High-quality epidemiological studies assessing the health effects of short-term NO_2 exposure would allow the results of this study and future work to be used to model adverse health outcomes associated with short-term stove-attributable NO_2. Fifth, our study relied on data for the United States only; incorporating behavioral and housing stock data from outside the United States would expand the scope of exposure estimates to other countries and continents. To address this shortcoming, we are undertaking indoor pollution measurements associated with gas stoves in countries that include Australia, the United Kingdom, the Netherlands, Italy, and China.

Although successful policies have reduced sources of air pollutants such as NO_2 in the United States (*48*), indoor air quality remains largely unmeasured and unregulated (*39*). Our research shows that pollution from gas and propane stoves disproportionately affects lower-income people and racial and ethnic minorities and that gas and propane stoves are responsible for substantial pediatric asthma and adult mortality. Our results also highlight the importance of including indoor sources of air pollution in future policies designed to protect people from pollutants such as NO_2, benzene, and carbon monoxide.

MATERIALS AND METHODS

Definitions

We defined a "cooktop" as a flat surface with two to six individual cooking elements and "burners" as cooking elements using a gas or propane flame. We defined gas, propane, and electric ovens as enclosed spaces heated by gas, propane, or electricity, respectively. We defined a "stove" (also called a "range") as a freestanding unit that contains both a cooktop and an oven. We defined an "outside-venting range hood" as an exhaust fan located directly above a stove, cooktop, or oven that sends kitchen air outdoors. We defined a "recirculating range hood" as a range hood that returns exhaust air to the kitchen rather than venting it outside.

Throughout the paper we used the term "concentration" for its accessibility in place of the more strictly correct term "molar mixing ratios." We assumed a temperature of 25°C and a pressure of 1 atm when converting between true concentrations and molar mixing ratios, which yields the conversion 1-ppbv NO_2 = 1.89 μg m^{-3} NO_2. We also used a conversion of 1-ppbv NO_2 = 1.89 μg m^{-3} to convert relative risks (RR) reported in units of micrograms per cubic meter to units of parts per billion volume.

We used CONTAM, a multizone indoor air model developed by the NIST, to model indoor NO_2 concentrations (*37*). We validated the model by comparing its predictions against measured NO_2 concentrations in a set of 18 test residences referred to as "validation residences" or "validation homes." We then used the validated model to estimate NO_2 concentrations in a set of other residence footprints (detached houses, duplexes and multiplexes, mobile homes, and apartments) for which we lacked measured NO_2 data. We refer to these as the "model residences" or "model homes."

We define "long-term" exposures in terms of parts per billion volume of NO_2 exposure averaged over a year and "short-term" exposures in terms of the number of days per year on which daily maximum hour-averaged NO_2 exposure exceeds 200 μg/m^3 (~100 ppbv), the WHO hour-averaged NO_2 standard (*60*).

We used commonly accepted definitions of epidemiological terms: (*38*, *61*) "population attributable fraction" (PAF) is the fraction of cases of a health outcome in a population attributable to an exposure; "odds ratio" (OR) is the ratio of the odds that a health outcome will occur given an exposure to the odds that the same health outcome will occur without the exposure; "relative risk" (RR) is the ratio of the probability of a health outcome in the exposed population divided by the probability in the unexposed population; "incidence" is the number of new cases of a disease per time interval (1 year, unless stated otherwise); "prevalence" is the total number of active cases of the disease in the population at a given time; and "burden" is the number of health outcomes (either at a given time or per unit time) attributable to an exposure.

Instrumentation

We measured NO_2 concentrations using a Teledyne T200P analyzer. We measured CO_2 and N_2O concentrations using an Aeris carbon dioxide/nitrous oxide MIRA Ultra Mobile LDS analyzer and measured CH_4 and C_2H_6 using an Aeris methane/ethane MIRA Ultra Mobile LDS analyzer. The calibrations of the analyzers were checked weekly and whenever transported.

Site selection

We measured NO_2 emission rates from 50 gas, 11 propane, and 14 electric stoves in 20 counties in California, Colorado, Texas, New York, and Washington, D.C. between January 2022 and July 2023 (fig. S7). Our measurements included 24 gas, 9 propane, and 14 electric stoves for which we previously reported benzene emission factors (*11*) (see table S4 for a summary of the characteristics of stoves we sampled and table S3 for a comparison against other published emission rates). Our set of sample residences also included a range of kitchen sizes (15 to 150 m^3) in private houses, apartments, and several Airbnb rentals, where we could measure longer uninterrupted time courses. We selected residences through online participant sign-up pages and neighborhood and community associations. We performed CONTAM validations tests (see below) on an 18-home subset of our sample, whose open floor area (excluding closed-off rooms and garages) varied in size from 250 to 1650 ft^2 and included 10 detached houses and eight apartments in three U.S. states (table S3).

Emission rate calculations and statistics

We calculated NO_2 and CO_2 emission rates from gas and propane combustion by measuring the increase in NO_2 concentration through time in sealed kitchens of known volumes, an approach analogous to that used in our previous work to measure NO_x, methane, and benzene emission factors from stoves (*10*, *11*). We converted measured concentrations into emission rates using Eq. 1

$$f = \frac{V_k}{t - t_0}(C_t - C_o + \Sigma_{i=1}^{n}(C_{t_i} - C_{bkg})(e^{-\lambda(t_i - t_{(i-1)})} - 1) \quad (1)$$

where t_i is the timestamp of the ith datapoint, t_0 is the initial time, f is the mean gas emission rate over the course of a measurement (in volume per time), V_k is the kitchen volume, λ is the kitchen chamber's air exchange constant (in reciprocal time), n is the number of gas concentration datapoints (typically 12) collected in a given measurement period, C_{ti} is the gas concentration at time t_i (in parts per billion volume), and C_0 is the concentration inside the chamber at the start of the measurement, and C_{bkg} is the background gas concentration outside the kitchen chamber (see the "Correction for air exchange" section, below). We assume that C_b is equal to the gas concentration we measure inside the kitchen immediately after airing it out with fresh outdoor air. We converted volumetric emission rates into gravimetric emission rates using the temperature measured in the kitchen.

We measured the energy output from gas burners and ovens using the flow rate of CO_2 and the enthalpy of combustion of methane to calculate the joules (J) of energy emitted per unit time.

We calculated the kitchen volume (V_k) and air exchange constant (λ) using 300- to 500-ml injections of a known volume of either ethane (C_2H_6) or nitrous oxide (N_2O) as a dilution tracer, using fans to mix the kitchen air, a method validated by Lebel *et al.* (the slope of laser-measured versus tracer gas–measured room volume is 1.1 [95% CI: 0.9, 1.3]; adjusted $r^2 = 0.91$) (*10*). The estimated kitchen volume is the injected tracer gas volume divided by the peak tracer gas concentration immediately following injection, and the air exchange constant is the tracer gas's decay constant through time after the peak concentration (see the "Correction for air exchange" section, below).

We calculated mean and median 95% CIs of emission rates from a 25,000 replicate bootstrap sample set (*62*, *63*) using a method described by Lebel *et al.* and by Kashtan *et al.* (*10*, *11*): We generated each replicate sample in the bootstrap by randomly sampling with replacement the set of emission factors in question to form a random sample of equal size to the set of emission factors in question. For instance, the bootstrap gas burners on high consisted of 25,000 replicates of size 50, generated by randomly sampling (with replacement) the set of 50 emission rates for gas burners on high. We then calculated 95% CIs for the means and medians of these bootstraps (*62*, *63*). We calculated statistical significance between gas and propane stove emissions using the two-sided Mann-Whitney *U* test (also known as the Wilcoxon rank sum test).

Our method for measuring NO_2 emission factors was the same as that used in Kashtan *et al.* to measure benzene, and similar to that used by Lebel *et al.*, to measure methane and NO_2. We sampled kitchen air approximately 1.5 m off the ground using a 7 liter min^{-1} pump drawing air through a polytetrafluoroethylene (PTFE) hose attached to our analyzers outside the sampling area. Where necessary to estimate emissions factors (but never when measuring concentrations), we created enclosed kitchen partitions by closing the kitchen's doors and windows, closing off open spaces with plastic, and placing fans in each kitchen to mix the air (being careful not to disturb the flame). We measured NO_2 concentration in the closed chamber for at least 15 min before igniting the stove to verify that no other sources or sinks were present (see fig. S8A).

The 5- to 8-hour time course NO_2 concentrations reported in the "NO_2 concentrations in bedrooms and with range hoods on" section were measured using no plastic partitions anywhere and with all interior doors open in six different houses. To avoid mixing the air, we did not use fans or other means of active air circulation during these concentration-based time courses. In all six houses for which we did the 8-hour time courses, we set the oven to 475°F (245°C), a temperature commonly used to bake bread, for 1.5 hours with the hood off. We continued monitoring NO_2 concentrations for 3.5 to 6.5 additional hours in the farthest bedroom from the kitchen after turning the oven off.

Correction for air exchange

Because it is impossible to seal kitchens perfectly, we corrected for air exchange between the chamber and the air outside the chamber. We calculated the air exchange constant for each kitchen by injecting 300- to 500-ml volumes of ethane or nitrous oxide and measuring changes in concentration through time as described in Materials and Methods and in Lebel *et al.* (*10*).

Kitchen volume is calculated using Eq. 2

$$V_k = \frac{V_i}{C_{tracer\_peak}} \tag{2}$$

where V_k is the kitchen volume, V_i is the volume of injected tracer (ethane or nitrous oxide), and C_{tracer_peak} is the peak ethane concentration following injection.

The concentration of the tracer, ethane or nitrous oxide, after injection follows an exponential decay attributable to air exchange and is described by Eq. 3

$$C_{tracer,t} - C_{tracer,b} = C_{tracer,0}e^{-\lambda t} \tag{3}$$

where $C_{tracer,t}$ is the concentration of the tracer at time t, $C_{tracer,b}$ is the background concentration of the tracer, $C_{tracer,0}$ is the concentration of the tracer in the kitchen before injection (typically very close to background), t is time, and λ is the air exchange constant.

Rearranging, we can calculate the air exchange constant λ using Eq. 4

$$\lambda = \frac{\ln\left(\frac{C_{tracer,0}}{C_{tracer,t} - C_{tracer,b}}\right)}{t} \tag{4}$$

Then, the corrected gas concentration $\hat{C}_{g,t}$ for the ith datapoint collected is given by Eq. 5

$$\hat{C}_{t_i} = C_{t_i} + \Sigma_{i=1}^{n}(C_{t_i} - C_{bkg})(e^{-\lambda(t_i - t_{(i-1)})} - 1) \tag{5}$$

where t_i is the time of the ith datapoint, $\hat{C}_{t_i}$ is the corrected gas concentration at time t_i, C_{bkg} is the background gas concentration outside the kitchen chamber, C_{t_i} is the true gas concentration at time t_i, and C_0 is the initial gas concentration in the chamber (usually almost the same as C_{bkg}).

The flow rate of the gas can then by calculated using the linear model given by Eq. 6

$$f = \frac{V_k(\hat{C}_t - C_0)}{t - t_0} = \frac{V_k}{t - t_0}(C_t - C_0 + \Sigma_{i=1}^{n}(C_{t_i} - C_{bkg})(e^{-\lambda(t_i - t_{(i-1)})} - 1) \tag{6}$$

where f is the gas flow rate (expressed as volume per time) and t_0 is the initial time.

We used Eq. 6 to calculate flow rates for CH_4, and CO_2. This method is the same as that used by Lebel *et al.* (*10*).

Modeling population-level exposures and health risks

We used CONTAM to model indoor NO_2 concentrations resulting from gas and propane stove use under different conditions. All quantities not measured, such as wall and door leakage areas and wind pressure profiles, used the default values in the ASHRAE Handbook-Fundamentals based on a house's age and height (*64*). We assumed a previously reported (*32*) NO_2 decay rate of -2.4×10^{-4} s^{-1}.

Validation of the CONTAM multizone model

We validated our CONTAM model by comparing measured and modeled NO_2 concentrations in 18 test houses. In each test house, we measured the NO_2 emission rate of a specific combination of burners and oven on known settings using the methods described above (see table S2). After fully airing out the house, we then measured the NO_2 concentrations in one to four different rooms (including at least the kitchen and another room or, in studio-style apartments, a single zone representing a combined kitchen/living space) for 40 min to an hour with the given burner/oven combination on and for another 1 to 3 hours with all burners and oven off. Note that the 18 1-hour–and–40-min– to 4-hour CONTAM validation measurements are distinct from the 6 8-hour bedroom measurements.

For validation measurements, all internal doors in residences were left open. In several instances, we measured both with windows open and closed and with an outside-venting range hood on or off. Because we had only one Teledyne NO_2 analyzer, we sampled sequentially from each room in question using a valve system to cycle between rooms so as not to disturb the sampling hoses or to have to enter the rooms (see fig. S8B).

After obtaining time-resolved concentration measurements, we modeled NO_2 concentrations in each residence using CONTAM. We constructed each residence in CONTAM based on laser distance measurements (see table S2 for a summary of home sizes and characteristics). We modeled each room as a single zone, except for hallways which we represented using two to four zones, depending on length. In each validation house, we inputted into CONTAM the NO_2 emission rate we measured from the burner/oven combination being tested. We also inputted into CONTAM weather profiles [obtained from OpenWeather Marketplace; (*65*)], corresponding with the address and time-period of each validation measurement.

Because interior airflow is strongly influenced by temperature gradients, time-resolved temperature gradients in multiple zones of each residence were required to accurately model NO_2 concentrations attributable to stoves. We measured temperature profiles of each room in 11 of the 18 validation homes (see fig. S9 for CONTAM floorplans of each of the 11 homes) and observed strong overall agreement between modeled and measured NO_2 concentrations in the 11 houses for which we have temperature data (see Results).

We could not scale such a time-resolved model to the entire U.S. housing stock because internal temperature profiles are variable and we thus could not use house-specific time-resolved temperature gradients as inputs in our nationwide model. To extend our model, we also verified that we could replace airflow due to temperature variation with a fixed-bidirectional airflow at each open door and still accurately predict kitchen, living room, and bedroom NO_2 concentrations averaged across several residences.

We considered a range of different bidirectional flow values between 300 and 1300 m^3/hour and found empirically that setting bidirectional flow to 1000 m^3/hour [590 cfm; within the range of turbulent flow rates observed in real interior doorways; (*66*)] minimized the error between averaged measured and modeled hour-averaged and day-averaged concentrations (see table S5; figs. S4 and S10 show results for different bidirectional flow rates; day-averaged concentrations extrapolated from measured and modeled values at the end of the measurement period using a decay rate of -2.4×10^{-4} s^{-1}). We calculated error as $100\,\% \times \frac{\text{Measured} - \text{Modeled}}{\text{Measured}}$.

NO_2 exposure calculations using CONTAM

We used CONTAM to model time-resolved NO_2 concentrations under different scenarios in each of the 7632 residences with gas, propane, and mixed-fuel stoves included in the United States Energy Information Administration's 2020 RECS. The 2020 RECS is a survey of 18,500 households in all 50 states and the District of Columbia that reports information on participants' energy use, housing characteristics, and demographics, and which assigns each participating household a representation weight such that results may be extrapolated to the entire U.S. population (*1*).

We combined our CONTAM modeling with the RECS data by assigning each RECS household to 1 of 24 distinct CONTAM floorplans that most resembled the RECS household in question based on housing type, floor area, number of stories, presence or absence of a forced air system, and home age. We selected the 24 floorplans from a pool of 209 residences constructed in CONTAM by Persily *et al.* (*67*) to represent the U.S. housing stock. The 24 floor plans represent diverse yet common types of homes that encompass the characteristics of the 7632 RECS residences (see fig. S11 for an assignment flowchart and table S6 for a summary of the 24 floorplans). We represented with a single floorplan homes that only differed from one another in factors out of the scope of this study (e.g., foundation type and room types). We assigned RECS residences to our set of floorplans according to, in order of decreasing prioritization, type of home, floor area, number of stories, presence or absence of a forced air system, and home age.

We left each model floorplan unchanged except for the following: because windows were not included in the default floorplans in CONTAM, we added one National Fenestration Research Council standard window (1.2 m × 1.5 m or 4 ft × 5 ft) to an exterior wall in every bedroom, living room, and kitchen (*68*); as discussed above, to simulate indoor air transport we replaced each modeled open door with bidirectional flow at a rate of 1000 m^3/hour (590 cfm); because we adjust the modeled NO_2 emission rate to account for outside-venting range hoods (for instance, we model a 50% capture efficiency outside-venting hood by reducing the emission rate by 50%), we removed modeled range hoods in floorplans that had them so as not to double-count range hoods. We left central forced-air systems unchanged and assigned their schedule on the basis of modeled ambient temperature (see table S7).

We captured a range of behaviors and environments by assigning a weighted distribution of scenarios to each RECS household. We used prior surveys and direct measurement studies to select three or four distinct values for each of six parameters: range hood use, stove use, window use, ambient temperature, windspeed, and occupancy, for a total of $4 \times 3 \times 3 \times 4 \times 3 \times 3 = 1296$ combined scenarios for each floorplan. We modeled each scenario for each of the 24 CONTAM floorplans and then calculated peak 1-hour–averaged and day-averaged NO_2 concentrations associated with each scenario. On the basis of existing data on each parameter and on the number of heating degree days and cooling degree days expected for the specific

location of each RECS residence, we assigned relative weights for each of the 1296 modeled scenarios for each RECS residence. The weights of each value are presented in table S7. We also performed a sensitivity analysis on all six parameters (see Results).

We calculated the distribution of national long-term NO_2 exposure burden by iterating through all 7632 RECS representative residences with gas or propane stoves and for each residence calculating the weighted mean of the 1296 modeled day-averaged NO_2 exposures. We then multiplied this mean by the residence's weight in the RECS database and normalized for total number of residences represented in the RECS database. We calculated the distribution of the percentage of days with 1-hour–averaged NO_2 exposure exceeding 100 ppbv in an analogous fashion: for each RECS residence, we calculated the percentage of modeled exposure days with a 100-ppbv exceedance, then multiplied the value by the given residence's weight and normalized for total number of residences represented by RECS.

Estimation of pediatric asthma attributable to gas and propane stoves overall and to NO_2 exposure from gas and propane stoves

We estimated the PAF of asthma incidence attributable to the use of gas and propane stoves compared with the use of electric stoves using ORs reported by Puzzolo *et al.* (*26*), who conducted a meta-analysis featuring multiyear, multicohort, geographically diverse studies. Because Puzzolo *et al.* did not report ORs or RRs per unit of indoor NO_2, we estimated the PAF of asthma incidence due to long-term NO_2 exposure from gas and propane stoves using Lin *et al.*'s 2013 estimate (*16*), which also featured multiyear, multicohort, geographically diverse studies focusing exclusively on indoor NO_2. Following Gruenwald *et al.* (*24*), we used ORs in place of RRs, as pediatric asthma affects less than 10% of the U.S. child population (*69*).

To our knowledge, there are no meta-analyses assessing the effect of indoor NO_2 on mortality. To estimate the PAF of deaths attributable to long-term NO_2 exposure from gas and propane stoves, we thus used the RRs reported by a meta-analysis featuring multiyear, multicohort, geographically diverse studies of ambient NO_2 (*38*). Potential confounding with other coproduced ambient pollutants, such as particulate matter, increases the uncertainty of our mortality calculations. While Buonocore *et al.* used the RR for all-cause mortality associated with NO_2 reported by Faustini *et al.*'s 2014 meta-analysis (*70*), we instead opted for an RR reported in a newer meta-analysis by Atkinson *et al.* (*38*), which exclusively incorporated cohort studies (1.02 [95% CI: 1.01, 1.03] per 10 μg/m^3 increment in long-term NO_2 concentration, smaller than Faustini *et al.*'s value of 1.04 [95% CI: 1.02, 1.06] per 10 μg/m^3).

Consistent with prior epidemiological work assessing the influence of long-term NO_2 exposure on respiratory diseases (*15*, *71*, *72*), we assumed a log-linear concentration-response function and calculated health outcome burdens as

$$\text{Burden} = Inc_g \Sigma_n P_n \times W_n \times (1 - e^{-\beta \Delta c_n}) \quad (7)$$

where values are summed over all $n = 7632$ RECS residence types with gas or propane ranges or cooktops in the RECS database, Burden is the number of adverse health outcomes (death or pediatric asthma) attributable to NO_2 from stoves, Inc_g is the current incidence rate of the adverse health outcome in question in the geography in question, P_n is the number of people living in the nth household, W_n is the number of households the nth RECS household represents in the U.S. housing stock, β is the concentration response factor (calculated as $\frac{\ln(RR)}{\Delta c}$), which is assumed to be constant, and Δc_n is the median year-averaged gas-stove-attributable NO_2 exposure in the nth residence. We calculated burden separately for pediatric asthma and adult mortality using the appropriate P_n, based on child and adult occupancy of RECS houses.

We calculated pediatric asthma burden using a U.S. pediatric asthma incidence rate averaged between 2006 and 2008 (12.5 cases per 1000 children) (*69*) multiplied by the current U.S. population under 18. We used state-by-state incidence of pediatric asthma from the U.S. Centers for Disease Control and Prevention from 2021 (*73*), the most recent date for which data stratified by state were available. We calculated state-stratified asthma rates only in states for which the incidence rate was reported.

Because the total population covered by the RECs database (62 million children, 239 million adults) is slightly lower than today's U.S. population (258 million adults and 73 million children) (*74*), likely because of occupancy data that is older than the most recent U.S. census, we proportionally adjusted our calculated burdens to match the current U.S. adult and child population

$$\text{Burden}_{adj} = \text{Burden}_{orig} \times \frac{Pop_{current}}{Pop_{RECS}} \quad (8)$$

Following Mansournia (*75*), we calculated PAFs from calculated burdens and baseline incidence of deaths and pediatric asthma as

$$\text{PAF} = \frac{\text{Burden}}{\text{Incidence}} \quad (9)$$

where Incidence is the number of new deaths or pediatric asthma cases per year in the geography in question. When calculating PAFs for individual states, we used state-specific incidence estimates, and when calculating national PAFs, we used national incidence estimates.

Because the uncertainty in RRs may be correlated with uncertainty in exposure, we conservatively assumed a wide uncertainty in PAFs. Rather than add fractional errors of RR and Δc_n in quadrature, we calculated our lower uncertainty bound using the lower bound of each reported RR and of each Δc_n estimate we calculated. We performed the analogous calculation for upper bounds.

Following Buonocore *et al.* (*61*), we calculated the valuation of excess deaths using the VSL from the EPA's BenMAP (*55*), $13.1 ($7.9, $18.5) million per death when adjusted to U.S. $ 2023, and the valuation of asthma incidence from an analysis by Nurmagambetov *et al.* (*51*), $5300 ($4120, $6490) per year per pediatric asthma case. We assumed that uncertainties in health outcome burdens and valuations were independent.

Uncertainty

We calculated uncertainty in exposure and health outcomes using a Monte Carlo method. To calculate uncertainty in exposure, we computed a Monte Carlo distribution resulting from the combination of three input distributions representing three key elements of the model: (i) measured NO_2 emission factors, (ii) estimated burner intensities used, and (iii) the distribution of NO_2 exposures modeled in different CONTAM scenarios, normalized to amount of gas burned and weighted according to the prevalence of each scenario, according to Eq. 10A and 10B

$$MC_{mean} = \text{mean}(EF * BI * CE * WE) \quad (10A)$$

$$MC_{\text{median}} = \text{median}(EF * BI * CE * WE) \quad (10B)$$

where *EF*, *BI*, and *CE* are randomly sampled (with replacement) values from the distributions of emission factors, burner intensities, and ratios of CONTAM-modeled exposures (normalized to gas burned), respectively, and *WE* is the weight corresponding to the particular CONTAM-modeled exposure *CE* selected. Because we have sparse data on the frequency with which people cook with burners on low, medium, and high (*41*), we assumed a normal distribution for burner intensity expressed as a fraction of emissions on high (mean = 0.5; SD = 0.1). See the "Limitations" section for further discussion.

Each sample *EF* and *CE* contained a number of elements equal to the minimum number of elements in each of the included distributions. For gas, this was 390 (number of gas emission factors) and for propane it was 20. Because *BI* was drawn from a known normal distribution, each sample *BI* contained only one element. MC_{mean} and MC_{median} are the mean and median, respectively, of the product of a single set of *X*-element input samples, where $X = 390$ for gas and 20 for propane. We calculated MC_{mean} and MC_{median} over 10,000 iterations to form a Monte Carlo distribution of the mean and median and calculated 95% CIs for mean and median exposure based on the Monte Carlo distributions.

We calculated central-estimate, lower-bound, and upper-bound long-term exposure for each floorplan as the average of the central estimate, lower bound, and upper bound of modeled day exposures, respectively. We calculated short-term exposure exceedances as the fraction of modeled days with a maximum 1-hour average exposure exceeding 100 ppb and converted the fraction to days per year by multiplying by 365. We used the central estimate, lower bound, and upper bound of modeled day exposures to calculate our CI in an analogous fashion to long-term exposures.

We used the same method to calculate uncertainties in our estimates of health outcomes and costs associated with exposure to NO_2. This time, we combined the distribution of concentrations calculated by Eq. 10A with a normal distribution of RRs reported for asthma (*16*) or mortality (*38*), according to Eq. 11

$$MC_{\text{health}_{\text{mean}}} = \text{mean}(1 - e^{\frac{\ln(RR)}{\Delta c} \times MC_{\text{conc}}}) \quad (11)$$

where $MC_{\text{healthmean}}$ is the mean of a single result in the Monte Carlo distribution of health outcome risks (pediatric asthma or adult mortality), *RR* is a relative risk value randomly sampled (with replacement) from a normal distribution, according to 95% CIs provided by Lin *et al.* and Atkinson *et al.* (*16*, *38*) for asthma and mortality, respectively, and MC_{conc} is a long-term exposure value randomly sampled (with replacement) from the 10,000-element normal distribution produced by running Eq. 10A 10,000 times. We then calculated the 95% CI of this Monte Carlo distribution. We calculated uncertainties in costs using the same method, this time drawing values from our calculated distribution of predicted health outcome burdens and from a distribution of per-case costs derived from uncertainties provided in the literature (see above).

We performed an analysis of the sensitivity of our calculated adverse health outcome burdens to the choice of concentration-response model. We did so by calculating the estimated burden of pediatric asthma and adult all-cause mortality attributable to long-term NO_2 from stoves using a log-linear model, a linear model, and a log-linear model with a 2-ppbv no-effect threshold, three models mentioned in recent long-term NO_2 literature (*13*, *15*, *71*, *72*). In each case, we calculated 95% CIs using a Monte Carlo method analogous to that described above for the calculation of log-linear adverse health outcome burden. The observed differences between models were modest and statistically indistinguishable (fig. S13).

Limitations

Our study assessed only one pollutant from gas stoves, NO_2. Because gas stoves also emit CO, benzene, formaldehyde, and ultrafine particulate matter (*7*, *9*–*12*, *76*), our estimates of disease burden and societal cost almost certainly underestimate the full health consequences of gas and propane stoves.

Results are based on measurements and assumptions throughout the modeling chain. We can partition the modeling chain, and associated uncertainties and limitations, as follows:

1) NO_2 emission rates of gas and propane burners and ovens.

To our knowledge, before Lebel *et al.*, NO_2 emission rates from gas stoves were last systematically quantified in 1985, but summary statistics were not reported. Other researchers have since reported emission factors per joule but not emission rates per time (see table S3 for a comparison of our results and previously reported emission rates and emission factors). Our modeling thus used emission rates measured in this work and by Lebel *et al.* (*10*). Our work and results from Lebel *et al.* (*10*) have constrained median NO_2 emission rates from gas burners on high and ovens to ±15% (see table S3). Larger uncertainties (up to ±50% for burners on high) remain for propane burner and propane oven emissions (see table S3). Moreover, our independent estimates in this paper for NO_2 emissions were statistically indistinguishable from those in Lebel *et al.* (see fig. S4 and table S3 for emission rates and table S8 for a summary of uncertainties).

2) Modeled indoor NO_2 concentrations from CONTAM, given a set of known input parameters.

We constrained uncertainties in modeled concentrations at ±18% (with positive values indicating an underestimate; see table S5). Our set of validation measurements included 18 homes and spanned a range of characteristics and measurement conditions. However, given time constraints it was impossible to validate the model on every combination of input parameters.

3) The population-wide distribution of burner minutes and oven minutes used per day, range hood use and capture efficiency, window opening schedules, outdoor temperatures, and outdoor windspeeds (the input parameters in step 2).

As demonstrated in our sensitivity analyses, there is a broad range in how frequently people use their stoves and their range hoods (and range hoods' efficacy) and open their windows, and these input parameters can alter estimated exposure (see Fig. 4). Of these inputs, stove use (i.e., length of time and number and intensity of burner and oven usage) had the largest spread and effect on exposure estimates. To our knowledge, no U.S.-wide study of burner and oven use exists, so we relied on a direct-measurement study by Zhao *et al.* (*41*) of stove use in 54 single family houses and 17 low-income apartments in California.

4) Other behavioral assumptions.

Apart from the behaviors listed above, there were other behaviors for which we lacked information. Two such behaviors that affect exposure are burner intensity and interior door opening. We inferred burner intensity based on reported cooking behaviors (see table S7), but because we lack measured data, our assumption that "on" burners are set to 50% of "high" may be an over- or underestimate of NO_2 emissions. We attempted to account for this uncertainty by adding

a ± 10% normally distributed uncertainty to our exposure and health outcome estimates. We assumed that all interior doors of houses remain open. Compared with an assumption of more closed doors, our open-door assumption results in lower exposure to people in and near the kitchen and higher exposure to others in the house. While this should not substantively affect our population-averaged estimates of long-term exposure, it may lead us to underestimate exposure for primary cooks in a given household and to overestimate exposure for people who spend little time in the kitchen. This issue applies to both long-term and short-term exposure estimates but is more relevant to short-term exposures.

5) Assignment of each residence in the RECS database to a specific floorplan in CONTAM.

We assigned each residence in the RECS database to 1 of 24 floorplans and thus were unable to perfectly match each RECS residence to a given floorplan. For example, we split apartments into >1000 ft^2 (>90 m^2) and <1000 ft^2, representing the 65th percentile of floorspace for apartments with gas or propane stoves [the median is 900 ft^2 (80 m^2)] (*1*). This assignment (and analogous assignments for other variables) thus result in the floorspace of roughly equal numbers of RECS residences being over- and underestimated and thus should not substantively affect our population-averaged exposure estimates. However, it may underestimate differences in exposure due to housing size and other characteristics.

6) Conversion of exposure estimates to estimates of disease burden.

We relied on RR values from recent meta-analyses to calculate stove-attributable pediatric asthma and mortality (*16*, *26*, *38*). For pediatric asthma, we relied on meta-analysis assessing indoor NO_2 only (*16*). Because no meta-analysis has assessed indoor NO_2 and mortality, to our knowledge, we calculated mortality using a meta-analysis on outdoor NO_2. Our estimate of mortality is thus limited in its precision by potential confounding pollutants that co-occur with outdoor NO_2, such as particulate matter, and by the variability in RRs reported by different studies. We applied a mortality RR of 1.02 (1.01, 1.03)) per 10 $\mu g/m^3$ of long-term NO_2 exposure derived from meta-analysis by Atkinson *et al.* (*38*) that included only long-term, cohort studies. Other meta-analyses (*70*, *77*) have estimated higher RRs than what we used. For instance, a 2014 meta-analysis (*70*) on NO_2 exposure and mortality used by Buonocore *et al.* to calculate premature deaths from upstream oil and gas production (*61*) calculated $RR = 1.04$ (1.02, 1.06) per 10 $\mu g/m^3$ increase in long-term NO_2. We propagated uncertainty bounds provided by Atkinson *et al.* but recognize that there is additional uncertainty and that choosing meta-analyses with broader inclusion criteria than used by Atkinson *et al.* may have resulted in higher mortality estimates than those we calculated.

Supplementary Materials

This PDF file includes:

Figs. S1 to S15
Tables S1 to S8
Legends for data S1 to S3
References

Other Supplementary Material for this manuscript includes the following:

Data S1 to S3

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