

Methods to Assess Co-Benefits of California Climate Investments

Asthma/Respiratory Disease Incidence

Center for Resource Efficient Communities, UC-Berkeley
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I. Background

Under California's Cap-and-Trade program, the State's portion of the proceeds from Cap-and-Trade auctions is deposited in the Greenhouse Gas Reduction Fund (GGRF). The Legislature and Governor enact budget appropriations from the GGRF for State agencies to invest in projects that help achieve the State's climate goals. These investments are collectively called California Climate Investments (CCI).

Senate Bill 862 requires the California Air Resources Board (CARB) to develop guidance on reporting and quantification methods for all State agencies that receive appropriations from the GGRF. Guidance includes developing quantification methodologies for greenhouse gas (GHG) emission reductions and other social, economic, and environmental benefits of projects, referred to as "co-benefits."

This document is one of a series that reviews the available methodologies for assessing selected co-benefits for CCI projects at two phases: estimating potential project-level co-benefits prior to project implementation (i.e., forecasting of co-benefits), and measuring actual co-benefits after projects have been implemented (i.e. tracking of co-benefits). The assessment methodology at each of these phases may be either quantitative or qualitative. As with CARB's existing GHG reduction methodologies, these co-benefit assessment methods will be developed to meet the following standards:

- Apply at the project level
- Align with the project types proposed for funding for each program
- Provide uniform methods to be applied statewide, and be accessible by all applicants
- Use existing and proven tools or methods where available
- Use project level data, where available and appropriate
- Reflect empirical literature

CARB, in consultation with the State agencies and departments that administer CCIs, has selected ten co-benefits to undergo methodology assessment and development. This document reviews available empirical literature on the ***asthma/respiratory disease incidence*** co-benefit and identifies:

- the direction and magnitude of the co-benefit,
- the limitations of existing empirical literature,

- the existing assessment methods and tools,
- knowledge gaps and other issues to consider in developing co-benefit assessment methods
- a proposed assessment method for further development, and
- an estimation of the level of effort and delivery schedule for a fully developed method

II. Co-benefit description

An extensive body of research demonstrates that decreased emissions of criteria air pollutants lead to reductions of respiratory-related exacerbations and health care visits. As decreased emissions of criteria air pollutants are often concomitant with decreased greenhouse gas emissions, reductions in respiratory-related exacerbations and health care visits could also be co-benefits of decreased greenhouse gas emissions. The State of California has funded key efforts to document the relationship between air pollution and respiratory diseases.¹⁻¹⁰ Criteria air pollutants—including ozone, nitrogen oxides (NO_x), and particulate matter (PM)—are directly linked to 1) the onset and exacerbation of asthma, 2) decreased lung function, and 3) increased asthma-related hospitalizations and emergency department (ED) visits.¹¹ In addition, the National Institutes of Health's National Institute of Environmental Health Sciences reports that climate change affects respiratory disease through increased frequency of droughts, which in turn increases particulate matter, and increased precipitation in some regions, which may lead to the proliferation of asthma-triggering mold spores.¹²

Table 1 illustrates the California Climate Investment (CCI) programs that may be able to document asthma- and respiratory disease-related co-benefits:

Table 1: CCI Programs Affected by Co-Benefit

Program	Project	Likely direction of co-benefit (+ = beneficial change)
Transportation and Sustainable Communities		
HSRA	<i>High Speed Rail</i>	+
CalSTA	<i>Transit and Intercity Rail Capital Program (TIRCP)</i>	+
Caltrans	<i>Low Carbon Transit Operations (LCTOP)</i>	+
	<i>Active Transportation</i>	+
CARB	<i>Low Carbon Transportation</i>	+
SGC	<i>Affordable Housing and Sustainable Communities Program (AHSC)</i>	+
	<i>Sustainable Agricultural Lands Conservation Program (SALC)</i>	+
	<i>Transformative Climate Communities (TCC)</i>	+
Clean Energy and Energy Efficiency		
CSD	<i>Low Income Weatherization Program (LIWP)</i>	+
	<i>Community Solar</i>	+
CDFA	<i>State Water Efficiency and Enhancement Program (SWEEP)</i>	+
DWR	<i>Water-Energy Grant Program</i>	+
CARB	<i>Woodsmoke Reduction Program</i>	+
Natural Resources and Waste Diversion		
CNRA	<i>Urban Greening Program</i>	+
CAL FIRE	<i>Urban and Community Forestry (UCF)</i>	+

These CCI programs may produce asthma or respiratory health co-benefits through one of the following pathways:

- Reducing vehicle miles traveled (VMT) and the associated GHG and criteria air pollutant emissions (High Speed Rail, TIRCP, Active Transportation, AHSC, SALC, TCC)
- Reducing the GHG and criteria air pollutant emissions of transportation through introduction of low- or zero-emission vehicles (LCTOP, Low Carbon Transportation, TCC)
- Reducing electricity use and the associated GHG and criteria air pollutant emissions from power plants through equipment upgrades (SWEEP, LIWP, Water-Energy Efficiency) or the shading of buildings (Urban Greening, Urban Forestry)

- Reducing the GHG and criteria air pollutant emissions of electricity generation through installation of renewable energy generating capacity (Community Solar)
- Reducing natural gas use in buildings (LIWP)
- Directly reducing criteria air pollutant emissions through technology upgrades (Woodsmoke Reduction, SWEEP)

III. Directionality of the co-benefits

Research indicates that projects that reduce greenhouse gas emissions will generally also reduce emissions of criteria air pollutants and reduce respiratory exacerbations, a *positive co-benefit*. However, projects that result in increases of emissions of air criteria pollution could potentially increase asthma and respiratory exacerbations. For example, areas surrounding new transit stations could experience higher local emissions as passengers travel to and park their vehicles at the station.

IV. Magnitude of the co-benefit

The literature demonstrates that criteria air pollution emissions increase exacerbations of respiratory conditions.¹¹ In particular, many epidemiologic studies have quantified the effect of PM_{2.5} emissions on respiratory-related hospital admissions, ED visits for asthma, episodes of acute bronchitis, lower respiratory symptoms, upper respiratory symptoms, and asthma exacerbations.¹³ For example, Mar et al. (2010) report that a 7 µg/m³ increase in PM_{2.5} concentration in Tacoma, Washington was associated with a 4% increase in risk of an asthma ED visit on the second day after the change in PM_{2.5} levels.¹⁴ This study included ED visit data for all ages. Slaughter et al. (2004) also describe a 3% increase in risk of an asthma ED visit after an increase of 10 µg/m³ in PM_{2.5} concentration in Spokane, Washington.¹⁵ This study also included ED visit data for all ages. In addition, long-term exposure to PM_{2.5} is associated with uncontrolled asthma in children and adults.¹¹ Uncontrolled asthma can be defined by symptoms (e.g., diurnal symptoms more than once a week over the course of three months), exacerbations (e.g., 12 asthma attacks over the course of three months), or lung function (e.g., forced expiratory volume in one second that is less than 80% predicted function).¹⁶ In addition, uncontrolled asthma may be characterized by an asthma-related hospital or emergency admission in the past year or the use of oral corticosteroids in the past year.¹⁶

Long-term exposure to ozone also contributes to uncontrolled asthma among children and adults.¹⁶ Even at relatively low ambient concentrations, ozone is associated with ED visits for pediatric asthma or wheeze.¹⁷ One study reports a 9.6% increase in the risk of asthma ED visits per 22.6 ppb increase in ozone concentration.¹⁸ The study also reports a smaller, but significant association between NO₂ and asthma ED visits. In addition, one meta-analysis found a “small but real association” between NO₂ and increased asthma prevalence in children.¹⁹

Research also indicates that NO_x and sulfur dioxide (SO₂) impact respiratory exacerbations. One study found that a 17.9 ppb increase in near-roadway NO_x was

associated with a 1.6% decrease forced vital capacity (the amount of air exhaled during a forced-breath test) and a 1.1% decrease in forced expiratory volume among children in Southern California.²⁰ Increases in SO₂ concentration are also associated with pediatric asthma ED visits during the warm season in the northern hemisphere (May through October).^{17,21} Byers et al. (2016) report that a 10.6 ppb increase in SO₂ concentration is associated with a 3.3% increase in risk of an asthma ED visit.²¹ This study included ED visit data for all ages.

The literature suggests that residential proximity to major roadways is an important factor in examining the relationship between air pollution and respiratory exacerbations because vehicles release emissions of criteria air pollutants near ground level.²² Research suggests that living within 50 to 200 meters of major roadways can trigger asthma symptoms among adults and children and contribute to the development of asthma in children.^{3-7,9,19,20,22-37} Among adults, traffic exposure is also associated with decreased lung function.²⁴ Research indicates that near-roadway traffic exposure can even exacerbate respiratory conditions among children who live in areas with good regional air quality.³⁸ The Children's Health Study has provided critical information about the relationship between ambient air pollution, near-roadway pollution, and respiratory conditions among children in Southern California since 1992. Table 2 describes key findings from the Children's Health study related to traffic and respiratory exacerbations.

Table 2: Key Findings from the Children's Health Study Related to Traffic and Respiratory Exacerbations

Author	Description
McConnell et al., 1999 ³⁹	Increased prevalence of phlegm was significantly associated with increasing exposure to ambient concentrations of PM ₁₀ , PM _{2.5} , NO ₂ , and acid vapor. In the 12 study communities in Southern California, children with asthma were more likely to develop persistent lower respiratory tract symptoms when exposed to air pollution.
McConnell et al., 2006 ⁵	Among children with no parental history of asthma who lived within 75 meters of a major road, the odds of lifetime asthma were 29% greater, the odds of prevalent asthma were 50% greater, and the odds of wheeze were 40% greater when compared with children who lived more than 300 meters from a major road.
Gauderman et al., 2007 ⁴⁰	Children who lived within 500 meters of a freeway had "substantial deficits in 8-year growth of forced expiratory volume" in one second in addition to substantial deficits in maximum midexpiratory flow rate when compared with children who lived at least 1500 meters from a freeway.
McConnell et al., 2010 ⁶	The risk of developing asthma was 51% higher among children who lived within 150 meters of major roadways and 45% higher among children who attended school within 150 meters of major roadways, when compared to children who lived more than 150

	meters away. Exposure to higher levels of ambient NO ₂ was also significantly associated with the onset of asthma.
Perez et al., 2012 ⁹	Eight percent of all cases of childhood asthma (n= 27,100) in Los Angeles County were at least partly attributable to pollution associated with living within 75 meters of a major road.
Brandt et al., 2012 ²	Yearly childhood asthma-related costs attributable to air pollution for Riverside and Long Beach were estimated to be \$18 million. ¹
Brandt et al., 2014 ¹	The cost of air pollution-related asthma in Los Angeles County in 2007 was an estimated \$441 million for ozone and \$202 million for NO ₂ .
Urman et al., 2014 ²⁰	An increase in near-roadway NO _x of 17.9 ppb was associated with deficits of 1.6% in forced vital capacity and 1.1% in forced expiratory volume in one second, when compared to children who lived more than 1500 meters away from a freeway and 300 meters away from a major road.
Gauderman et al., 2015 ⁴¹	Over the 13 years and three cohorts of the Children's Health Study, improvements in four-year growth of both forced expiratory volume in one second and forced vital capacity were associated with declining levels of criteria air pollutants (NO ₂ , PM _{2.5} , and PM ₁₀).
Berhane et al., 2016 ⁴²	Decreased ambient pollution levels of NO ₂ , ozone, PM _{2.5} , and PM ₁₀ were associated with statistically significant decreases in bronchitis symptoms among children over the 13 years and three cohorts of the Children's Health Study.

V. Limitations of current studies

Local assessments of air pollution effects often use concentration-response functions (CRFs) that were developed from large epidemiologic studies. Hubbell et al. (2009) outline several issues with treating CRFs as “off-the-shelf” estimates for the relationship between the concentration of a pollutant and health outcomes. First, CRFs could be influenced by the health care systems or overall health of the population in the city where the original study took place. In addition, biological and environmental factors such as genetics or socioeconomic status influence the extent to which individuals are exposed to pollutants and their susceptibility to the exposure. The concentration of co-pollutants in the environment may also influence CRFs. For example, a study that measured the association between ozone and PM_{2.5} concentrations and mortality found that CRFs were attenuated in regions with higher prevalence of air conditioning. Finally, program applicants will likely serve small groups of people. If the characteristics of the

¹ Costs included “the indirect and direct costs of health care utilization due to asthma exacerbations linked to traffic-related pollution” and “the costs of health care for asthma cases attributable to local traffic-related pollution exposure.” Authors used peer-reviewed literature and surveys such as the Medical Expenditure Panel Survey to estimate costs.

subpopulation affected by a proposed project is substantially different from the sample population used to calculate the CRF, the CRF may not be applicable to the project population.

In addition, the studies cited in this review generally associate the health effects related to a single pollutant. Oakes et al. (2014) identify a “lack of an existing ‘gold standard’ for multipollutant health effects and exposure.”⁴³ It could potentially be difficult to determine whether the health effects of exposure to multiple pollutants are additive.

There are also knowledge gaps associated with predictive approaches that estimate the impact of decreased pollutant levels on the health of a population. For example, there are inherent uncertainties in making assumptions about the time required to achieve a decrease of the concentration of pollutant levels following a decrease in source emissions.⁴² The lag between the decrease in pollutant levels and the occurrence of health benefits can also be difficult to calculate.⁴⁴ In addition, the seasonality of the exacerbation of respiratory diseases may make it difficult to link changes in decreased pollutant levels with decreased exacerbations. One study found that ED visits and hospital admissions for asthma increased in the summer among children who were exposed to higher levels of PM_{2.5} and ozone, and in the winter among children exposed to higher levels of carbon monoxide, NO₂, and NO_x.⁴³ As respiratory diseases are more common in winter months, this may confound air pollutant-related asthma exacerbations and ED visits.

VI. Existing quantification methods/tools

Modeling the Health Effects of Air Pollution

The majority of studies used some form of modeling to estimate the health effects of changes in air pollutant concentrations. Researchers used models to control for seasonal trends and daily temperature; analyze data from air quality monitoring sources; compare daily averages of pollutant concentrations with daily counts of respiratory disease measures. Proprietary software for dispersion modeling include three platforms from the U.S. EPA: Community Multiscale Air Quality (CMAQ) Modeling System; R-LINE; and AERMOD. Proprietary software for mapping roadways and calculating the number of people affected by traffic pollution include Tele Atlas and Esri's ArcGIS. Modeling methods may allow applicants to both estimate potential co-benefits at the onset of the project, although the feasibility of modeling approaches may be limited for smaller spatial resolutions. In addition, modeling would allow applicants to estimate changes in concentration of emissions and associated health effects for a range of CCI programs, including projects related to energy efficiency, clean energy, and natural resources.

Health Effects Estimates

Several studies estimate the risks of air pollution.^{15,45–51} These estimates can be used to inform the decision-making process for environmental policies and estimate the impact of proposed transportation programs. Figure 1 illustrates the basic process for measuring changes in asthma exacerbations associated with changes in PM_{2.5} levels.

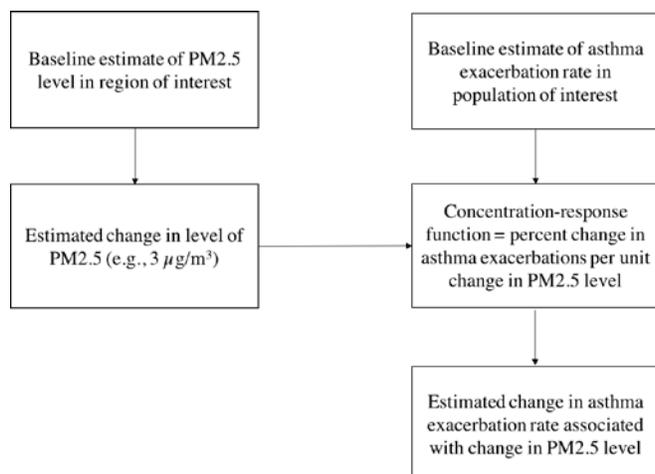


Figure 1: Basic process for measuring changes in asthma exacerbations associated with changes in PM_{2.5} levels, adapted from Pascal et al., 2011

Similar to modeling methods, Health effects estimates are concerned with changes in emissions-related portion of ambient air concentrations, but do not specify the source of the emissions. Therefore, the health effect estimate model could be applicable to a wide range of CCI programs, including projects related to energy efficiency, clean energy, and natural resources.

Typically health outcomes are calculated with the use of a health impact function, $\Delta y = y_0(1 - e^{-\beta\Delta x})$. Researchers use local or regional surveillance data to calculate y_0 , the baseline respiratory disease rate. Ambient air monitoring and grid-based air modeling are used to calculate Δx , the change in the concentration of the pollutant. The CRF, β , is defined as a percent change in the number of respiratory disease cases per unit change in the pollutant concentration. Some studies define CRFs in terms of relative risks (RR), where the RR per 10 µg/m³ = $e^{(10*\beta)}$.

The U.S. EPA offers two publicly available tools that use health effects estimates to estimate the co-benefits of reduced emissions, the Environmental Benefits Mapping and Analysis Program - Community Edition (BenMAP-CE) and Co-Benefits Risk Assessment (COBRA).

The **BenMAP-CE program** uses health impact functions that incorporate modeled or monitored air quality changes, an estimate of the affected population, baseline incidence rates, and effect estimates. To calculate air quality changes, BenMAP-CE requires users to either input modeling data about air quality changes or generate estimates from air pollution monitoring data. The program uses data from epidemiologic studies to apply a relationship between pollution and health effects, such as asthma-related exacerbations, hospitalizations, or emergency department visits. Finally, BenMAP-CE requires information about the number of people affected by the air pollution reduction and an estimate of the baseline number of people who experience

an adverse health effect, such as asthma, in a given population over a given period of time.

COBRA is a simplified version of BenMAP-CE that contains baseline estimates of emissions—including PM_{2.5}, SO₂, NO_x, NH₃, and VOCs—developed by the U.S. EPA. Users create scenarios for states or counties by specifying increases or decreases to the baseline emission estimates. While COBRA requires users to input emissions reductions in tons or percentages for their selected region, the program offers preloaded data on health effects and the number of people who would be affected by the change. Figure 2 illustrates a sample scenario where the user would input information about reductions in tons of PM_{2.5}, SO₂, and NO_x emitted by highway vehicles. COBRA allows users to estimate reductions in emissions not only due to highway and off-highway traffic, but also due to fuel combustion of electric utilities and industrial processes. Therefore, it could potentially apply to many CCI programs.

As COBRA asks users to input annual reductions in emissions, the model could be used to estimate the effect of decreased emissions at the beginning of the project. At the end of each project year or project term, CCI program applicants could revise estimates of emissions in order to calculate a more accurate assessment of potential health impacts. Monthly or quarterly tracking would likely not be feasible.

Figure 2: Specifying Changes in Emissions in the COBRA Model

COBRA allows users to input changes in emissions from several sources. Some sources that may be relevant to CCI programs include:

- Chemical and allied product manufacturing
- Fuel combustion from electric utilities
 - Coal, gas, internal combustion, oil, and other
- Fuel combustion from industrial activities
 - Coal, gas, internal combustion, oil, and other
- Highway vehicles

- Diesels, heavy-duty gas vehicles, light-duty gas trucks, light-duty gas vehicles and motorcycles
- Metals processing
- Agriculture and forestry
- Health services
- Natural sources
- Off-highway
 - Aircraft, marine vessels, non-road diesel, non-road gasoline, railroads
- Waste disposal and recycling

Estimating the Effect of Near-Roadway Emissions

Several studies focused on calculating the impact of near-roadway emissions on the exacerbation and development of respiratory conditions.^{4-7,9} Kunzli et. al (2008), Perez et. al (2012), and McConnell et. al (2010) have described a method for quantifying the number of asthma cases that can be attributed to near-roadway traffic pollution. Table 3 describes the calculations that are involved in this quantification method.

Table 3. Attributable Cases of Asthma Due to Traffic Pollution

Description	Equation	Information Required
The attributable fraction of asthma due to traffic pollution among the population of children in a defined area	$AF_{chron} = \frac{p_p(RR-1)}{p_p(RR-1)+1}$	p_p = the proportion of children living within 75 meters of busy roads RR = CRF determined by Children's Health Study data
The attributable number of prevalent asthma cases due to traffic pollution	$AN_{chron} = Pop_{tot} * P_{chron} * AF_{chron}$	P_{chron} = the prevalence of asthma in a defined area Pop_{tot} the total population of children in a defined area
The attributable number of cases of asthma in this population that are due to causes other than traffic pollution	$AN_{(1-chron)} = Pop_{tot} * P_{chron} * (1 - AF_{chron})$	Calculations from previous equation
The attributable fraction of acute annual exacerbations of symptoms attributable to air pollution	$AF_{acute} = \frac{RR_{\Delta}-1}{RR_{\Delta}}$	RR_{Δ} = CRF for the change (Δ) in the ambient concentration of a specified pollutant
The attributable number of acute exacerbations	$AN_{acute} = P_{chron} * Pop_{tot} * P_{acute} * AF_{acute}$	Calculations from previous equation

This series of equations can be combined and rearranged to calculate the total number of asthma exacerbations (Tot AN_{acute}) that are attributed to children who live within 75

meters of a major road and annual mean concentrations of a pollutant that is above a hypothetical reference value. The final equation for Tot AN_{acute} is:

$$\text{Pop}_{\text{tot}} * P_{\text{chron}} [AF_{\text{chron}} * P_{\text{acute}} * AF_{\text{acute}} * (AF_{\text{acute}}^{-1} + AF_{\text{chron}}^{-1} - 1)]$$

After using this method to determine the attributable number of asthma cases due to near-roadway traffic pollution, researchers then apply different reduction scenarios to estimate how decreasing ambient concentrations of pollutants would decrease respiratory exacerbations. For example, in a study of near-roadway and regional air pollution in Los Angeles County, Perez et. al (2012) found that an increase in the proportion of children living near major roadways would increase the number of asthma ED visits despite a 20% reduction in regional ambient concentrations of NO₂.

Near-roadway emissions calculations would likely only apply to CCI program applicants that are focused on sustainable communities and clean transportation. While program applicants may use this method to estimate health effects of a proposed project, tracking regular changes in the ambient concentration of a pollutant attributable to the project may be a challenge.

VII. Knowledge gaps and other issues to consider in developing co-benefit quantification methods

COBRA Screening Model

The U.S. EPA identifies several limitations of the COBRA model and suggests that it should primarily serve as a screening tool for program planners to be used in conjunction with an air quality analysis and a full health impact assessment.⁵² For example, COBRA relies on preloaded baseline estimates for states and counties based on different sources and does not allow users to import their own baseline data. The U.S. EPA also cautions that assumptions about statewide percentage reductions in pollutants may be an “oversimplification.”

Creating simplified tools from software programs such as COBRA could also pose challenges. For example, to calculate changes in concentration, the COBRA program uses preloaded meteorological data from the selected state or county to calculate an appropriate wind layer. This calculation involves “using the centroid of the diffusing plume: σ_z for a ground-based plume that has not yet mixed uniformly in the vertical, H for an elevated source, and $h_m/2$ for a uniformly mixed plume.”⁵² The program also uses a model to run preloaded emissions data through a source-receptor matrix and atmospheric chemistry calculations. In addition, COBRA uses discharge data from the Healthcare Cost and Utilization Project to calculate hospitalization and ED visit counts. The model uses an adjustment factor that accounts for missing age or Federal Information Processing Standards codes. The model also uses a forecasting model to calculate changes in population exposure to air pollution. Therefore, the method of accurately assessing changes in respiratory disease exacerbation linked to reductions in air pollution is complex.

Estimating Changes in Pollutant Concentration

Health effect estimates require users to estimate changes in concentration of pollutants. Changes in emissions depend on changes in the amount of air pollutants that are released from a source into the atmosphere, while changes in concentration depend on “the competition between the rates of emission of the gas into the atmosphere and the rates of processes that remove it from the atmosphere.”⁵³ In addition, one case of asthma may have multiple causes. For example, if an individual with asthma has been exposed to chronic air pollution and early-life viral infections, their asthma exacerbation may be attributable to both air pollution and viral infection. This could make it difficult to link asthma exacerbations with increases or decreases in criteria pollutant emissions on the project level.

For traffic-related pollutants, determining changes in concentration will likely involve the use of modeling software and may require calculations that are beyond the scope of requirements for CCI program applicants. For instance, many studies used the U.S. EPA’s CMAQ, a powerful but complex tool that produces air quality management scenarios. In addition, Fann et al. (2009) note that ambient changes in PM_{2.5} can be difficult to calculate because the concentration of the pollutant is influenced by a range of factors, including complex chemical interactions, meteorological conditions, and baseline levels.⁵⁴ The “complex nonlinear chemistry governing PM formation” indicates that the benefit of reducing greenhouse gas emissions could vary significantly according to unique regional environmental factors.

It may also be difficult to estimate concentration reductions due to programs not related to traffic, such as urban greening or forestry efforts. For example, to estimate reductions in concentrations due to tree planting, program applicants will need to use a tool like the i-Tree Eco model to calculate air pollution removal. Factors like tree size and the number of trees surviving will also impact yearly pollution reductions. It may be difficult to understand how newly planted trees will affect the concentration of emissions in an area without dispersion modeling.⁵⁵ In addition, health impact functions require a baseline rate of asthma exacerbations, ED visits, hospitalizations, or another measure of respiratory disease. If changes in concentration affect a wide area, it could be challenging to develop a simple method to estimate a baseline rate of respiratory disease. If changes affect a small subpopulation that differs in composition from study populations on which the baseline respiratory disease rates were based, then calculating a baseline rate of respiratory disease will be even more difficult.

Near Roadway

Determining the number of people affected by changes to traffic may require the use of complex tools. Studies that examined the effects of near-roadway emissions used software to determine the number of people living within a determined number of meters of major roadways.^{1-4,8,9,18-34,36-39,56-59} Several studies also used Tele Atlas to map roadways of interest and geographic information systems such as ArcGIS to geocode residential addresses and assign distances to roadways. Program applicants may be able to use public mapping platforms to estimate numbers of people affected by near-roadway emissions. The US EPA’s EnviroAtlas offers a map with the estimated number of residents within each census block who live within 300 meters of a busy roadway,

defined as “interstates, arterial roads, and collector roads.”⁶⁰ However, the literature indicates that near-roadway emissions generally return to background concentrations after a distance of 200 meters.²² Capturing those who live within 300 meters of busy roadways may overestimate the effect of decreased pollution levels.

In addition, as described above, the specific demographics (e.g., age, gender, race/ethnicity, and health status) of the population affected by the project will impact the magnitude of the health effect. If the population of interests’ characteristics are substantially different from those of the population from which the CRFs were derived from, application of the CRF to the project’s population may not be appropriate.

Kunzli et. al’s attributable risk model also requires some complex calculations that may be difficult to scale down for CCI program applicants. For example, to analyze a scenario that reduced levels of NO_x by 20%, one study “used modeled NO_x to represent the incremental contribution of local traffic to a more homogeneous community background concentration of NO_x that included both primary and secondary pollution resulting from long-range transport and regional atmospheric photochemistry.”⁹ In addition, the authors used the CALifornia LINE (CALINE) Source Dispersion Model, the TeleAtlas MultiNet Roadway network, and the EMFAC model to estimate yearly averages of traffic-related NO_x concentrations. It could be difficult to develop a simpler method of estimating a change in pollutant concentration for individual CCI projects.

Even relatively large changes in pollutant concentrations may have modest health effects if the number of people who experience near-roadway emissions increases in the same time period. In a scenario where concentrations of NO₂ decreased by 20% and the proportion of children living within 75 meters of a major road increased by 3.6%, Perez et al. (2012) report a net increase in asthma ED visits. This indicates that CCI program applicants may not be able to significantly decrease respiratory exacerbations through project activities. In addition, it may be difficult for program applicants to track changes in the proportion of people living near roadways on a year-to-year basis. However, in the scenario described by Perez et al. (2012), there was no change in the assumed vehicle fleet make-up, traffic density, fuel mixture or vehicle technology from the baseline to alternate scenario.

In discussing their attributable risk model, Kunzli et al. note that estimates of attributable cases do not provide ranges of uncertainties.⁴ The authors state that they are using Monte Carlo simulations to create risk assessment applications that integrate a range of uncertainty calculations, a method of analysis that is too complex for CCI program applicants. However, the authors also acknowledge that all risk assessments face challenges in accounting for uncertainties.

Qualitative measures

Given the complexities involved in calculating changes in concentration of air pollutants associated with changes in source emissions, a qualitative assessment of decreased respiratory exacerbations may not be possible. For example, decreased greenhouse gas emissions may not affect the community in which the project is taking place. A

project that focuses on decreased energy consumption may impact greenhouse gas emissions at the power plant level, instead of at the project location. Therefore, asking a question such as, “did asthma-related hospital admissions decrease in this location in this year?” could be difficult to assess. In addition, due to the multi-causal nature of asthma and asthma exacerbations, a year-over-year increase or decrease in asthma-related hospitalizations could be unrelated to air pollution or near-roadway exposure. Studies may average asthma outcomes over multiple years to calculate a more stable estimate of hospitalizations. Looking up data about hospital utilization and assessing changes may also place an undue burden on project applicants. It would be difficult to estimate what changes in ED visits or health care utilization could be attributed to the project, and it may also be difficult to identify where affected individuals are seeking health care in a certain area. In addition, given the complexities in calculating changes in concentration that are associated with changes in emissions, it may not be valid to simply ask about decreased emissions and assume that these changes will be sufficient to impact respiratory health outcomes in a certain area.

Overall significance

CCI programs vary widely in their potential impact on asthma and respiratory disease. As noted in Section II, CCI programs may produce asthma or respiratory health co-benefits through one of the following pathways:

- Reducing vehicle miles traveled (VMT) and the associated GHG and criteria air pollutant emissions (High Speed Rail, TIRCP, Active Transportation, AHSC, SALC, TCC)
- Reducing the GHG and criteria air pollutant emissions of transportation through introduction of low- or zero-emission vehicles (LCTOP, Low Carbon Transportation) or the introduction of low-emitting fuels (Alternative and Renewable Fuels program)
- Reducing electricity use and the associated GHG and criteria air pollutant emissions from power plants through equipment upgrades (SWEEP, Water-Energy Efficiency) or the shading of buildings (Urban Greening, Urban Forestry)
- Reducing the GHG and criteria air pollutant emissions of electricity generation through installation of renewable energy generating capacity (Community Solar)
- Reducing natural gas use in buildings (LIWP)
- Directly reducing criteria air pollutant emissions through technology upgrades (Woodsmoke Reduction)

There may be asthma and respiratory disease co-benefits at both the project and program levels for the large transportation CCIs, including the High Speed Rail, TIRCP, LCTOP, and Low Carbon Transportation programs because criteria air pollution emission reductions will be proportional to the VMT reductions and emissions-intensity reductions achieved by projects in those programs. In addition, the avoided emissions are concentrated in urban areas.

The potential significance of asthma and respiratory disease co-benefits from the Active Transportation, AHSC and SALC programs will also be proportional to avoided VMT and reduced emissions intensity, but it is not clear if projects in these programs will produce such reductions at a large enough scale to result in significant respiratory health co-benefits.

Asthma and respiratory disease incidence reduction from other CCIs are not likely to be significant at the project or program level. Projects funded by the SWEEP, Water-Energy Efficiency, Urban Greening, Urban Forestry, and Community Solar programs collectively may slightly reduce criteria air pollution emissions from certain power plants, but these reductions would be very small and the plants are generally located far from population centers (in California) where respiratory effects would occur.

Reducing natural gas use in buildings through projects funded by the LIWP program could potentially reduce criteria air pollutant emissions and associated respiratory disease. One retrospective Australian study found that children exposed to gas heaters in infancy were more likely to develop asthma in childhood.^{61,62} Another cross-sectional study that used data from the National Health and Nutrition Examination Survey (NHANES) found that children who lived in homes with heating provided by gas stoves or ovens were more likely to have a diagnosis of asthma.^{62,63} A 2015 update to the 2000 Institute of Medicine review of indoor environmental exposures and exacerbation of asthma discussed the association between NO₂ indoor pollution caused by indoor combustion sources and asthma.⁶⁴ The review found “sufficient evidence of an association between brief high-level exposures to NO₂ and increased airway responses to nonspecific chemical irritants and inhaled allergens among asthmatic subjects.”⁶⁴ However, the authors found insufficient evidence of an association between gas stove use and asthma exacerbation.

Similarly, emissions reductions from projects funded by the Woodsmoke Reduction program could decrease respiratory disease incidence. One literature review of the relationship between woodsmoke and respiratory conditions found that use of a woodstove was associated with shortness of breath, cough, and chest tightness among adults and increased asthma-related ED visits for people under 65 years of age.⁶⁵

VIII. Proposed method/tool for use or further development

Given these findings, we offer the following recommendations for methods to assess asthma and respiratory disease incidence co-benefits, schedule for development of guidance documents, and applicant data needs:

Methods for estimation prior to award of CCI funds:

- Estimation of avoided asthma incidence and associated hospitalizations, avoided lost work days, and avoided mortality using the U.S. EPA’s COBRA model for

any CCI program using CARB guidance to estimate criteria air pollution emission reduction co-benefits²

Methods for tracking after award of CCI funds:

- Estimation of avoided asthma incidence and associated hospitalizations, avoided lost work days, and avoided mortality using the U.S. EPA's COBRA model for any CCI program using CARB guidance to track criteria air pollution emission reduction co-benefits³

We do not recommend development of any guidance to assess asthma and respiratory disease incidence co-benefits for any CCI projects that will not estimate or track criteria air pollution emission reduction of at least one ton of a given pollutant per year within a given county (the smallest input amount that COBRA will accept). Emissions reductions smaller than this amount are unlikely to result in appreciable co-benefits at the project level. However, if the emissions reduction effects of numerous similar projects within a CCI program can be aggregated for a given county (such as for the Low Carbon Transportation program, potentially), it may be feasible to use COBRA to estimate the asthma and respiratory disease incidence co-benefits. COBRA also provides users with estimates of effects on every county in the state, although impacts on other counties for project-level reductions in pollution are likely to be negligible.

If CARB develops a feasible method to estimate changes in concentration of a pollutant based on changes in emissions, then a modified version of the health impact function, $\Delta y = y_0(1 - e^{-\beta\Delta x})$, could potentially be developed to estimate associated health effects. However, changes in concentration are dependent on several factors that may be difficult to calculate on a project level without modeling, as discussed in more detail above. However, it could require a large amount of effort for applicants to determine baseline rates of hospitalizations, exacerbations, and asthma emergency department visits. For example, program applicants may not be able to determine exactly where people affected by changes in emissions are receiving care for their asthma, and whether any changes in asthma care are related to their program or to seasonal changes or other air quality programs in the area.

Transit programs could potentially use a modified version of Kunzli et al.'s method to determine decreased exacerbations of respiratory conditions associated with reduced traffic on major roadways. However, this quantification method could only be used if program applicants were planning to decrease traffic on major roadways, could easily estimate the number of people living near major roadways, and could measure changes in the concentration of pollutants that would result from program activities. Using this method would likely require a high level of effort on the part of the Berkeley team.

² Methods to assess criteria air pollution co-benefits are to be developed by CARB simultaneous to the development of this co-benefit assessment method

³ Methods to assess criteria air pollution co-benefits are to be developed by CARB simultaneous to the development of this co-benefit assessment method

Schedule

Because application of the COBRA model is generally straightforward in the context of CCI programs, we anticipate that we could develop draft co-benefit assessment methodology guidance within two months of CARB's instruction to proceed.

Data needs

To use the COBRA model, users must provide the following inputs:

- Decrease in emissions of PM_{2.5}, SO₂, NO_x, NH₃, and/or VOC, in tons per year

The COBRA model is available to the public free of charge⁴ and requires minimal inputs from the user to estimate health effects associated with decreases in pollution. The countywide rate of emissions reduction needs to be at least one ton per year in order for any changes to be calculated. If the baseline rate is already close to zero, COBRA will not allow users to input reductions. Table 4 includes a sample of the information that the COBRA model can provide.

Table 4. Sample Output from COBRA

Source (individual)	Decrease in in tons/year	Avoided number of asthma exacerbations per person per year	Avoided asthma ED visits per 100 people per year	Avoided average yearly work-loss-day rate per 100 people
Agricultural crops (Sacramento County)	1 ton PM _{2.5}	0.91 (compared to national incidence of 27.74 number of cases of asthma wheeze per person per year)	0.015 (compared to national rate of 0.573 for the 18-44 age group)	4.01 (compared to national average yearly work-loss-day rate of 217 per 100 people)
Highway vehicles (Sacramento County)	1 ton PM _{2.5} 1 ton SO ₂	1.10	0.018	4.84
Agricultural crops (Fresno County)	1 ton PM _{2.5}	0.16	0.003	0.60
Highway vehicles (Fresno County)	1 ton PM _{2.5} 1 ton SO ₂	0.22	0.004	0.81

⁴ Changes to the US EPA's website may limit the availability of COBRA after May 2017

References

1. Brandt, S. *et al.* Cost of near-roadway and regional air pollution–attributable childhood asthma in Los Angeles County. *J. Allergy Clin. Immunol.* **134**, 1028–1035 (2014).
2. Brandt, S. J., Perez, L., Künzli, N., Lurmann, F. & McConnell, R. Costs of childhood asthma due to traffic-related pollution in two California communities. *Eur. Respir. J.* **40**, 363–370 (2012).
3. Künzli, N. *et al.* Traffic-related air pollution correlates with adult-onset asthma among never-smokers. *Thorax* **64**, 664–670 (2009).
4. Künzli, N. *et al.* An attributable risk model for exposures assumed to cause both chronic disease and its exacerbations. *Epidemiology* **19**, 179–185 (2008).
5. McConnell, R. *et al.* traffic, susceptibility, and childhood asthma. *Environ. Health Perspect.* **114**, 766–772 (2006).
6. McConnell, R. *et al.* Childhood incident asthma and traffic-related air pollution at home and school. *Environ. Health Perspect.* **118**, 1021–1026 (2010).
7. McConnell, R. *et al.* Asthma and School commuting time. *J. Occup. Environ. Med.* **52**, 827–828 (2010).
8. Perez, L. *et al.* Global goods movement and the local burden of childhood asthma in southern California. *Am. J. Public Health* **99**, S622–S628 (2009).
9. Perez, L. *et al.* Near-roadway pollution and childhood asthma: implications for developing ‘win–win’ compact urban development and clean vehicle strategies. *Environ. Health Perspect.* **120**, 1619–1626 (2012).
10. California Environmental Protection Agency & Air Resources Board. Asthma and Air Pollution. Available at: <https://www.arb.ca.gov/research/asthma/asthma.htm>. (Accessed: 28th February 2017)
11. Guarnieri, M. & Balmes, J. R. Outdoor air pollution and asthma. *The Lancet* **383**, 1581–1592 (2014).
12. National Institutes of Health & National Institute of Environmental Health Sciences. Asthma, Respiratory Allergies and Airway Diseases. Available at: https://www.niehs.nih.gov/research/programs/geh/climatechange/health_impacts/asthma/index.cfm. (Accessed: 28th February 2017)
13. United States Environmental Protection Agency. *Technical Support Document Estimating the Benefit per Ton of Reducing PM_{2.5} Precursors from 17 Sectors*. (2013).
14. Mar, T. F., Koenig, J. Q. & Primomo, J. Associations between asthma emergency visits and particulate matter sources, including diesel emissions from stationary generators in Tacoma, Washington. *Inhal. Toxicol.* **22**, 445–448 (2010).
15. Slaughter, J. C. *et al.* Association between particulate matter and emergency room visits, hospital admissions and mortality in Spokane, Washington. *J. Expo. Sci. Environ. Epidemiol.* **15**, 153–159 (2004).
16. Jacquemin, B. *et al.* Air pollution and asthma control in the Epidemiological study on the Genetics and Environment of Asthma. *J. Epidemiol. Community Health* **66**, 796–802 (2012).

17. Strickland, M. J. *et al.* Short-term associations between ambient air pollutants and pediatric asthma emergency department visits. *Am. J. Respir. Crit. Care Med.* **182**, 307–316 (2010).
18. Noh, J. *et al.* Short-term effects of ambient air pollution on emergency department visits for asthma: an assessment of effect modification by prior allergic disease history. *J. Prev. Med. Pub. Health* **49**, 329–341 (2016).
19. Favarato, G. *et al.* Traffic-related pollution and asthma prevalence in children. Quantification of associations with nitrogen dioxide. *Air Qual. Atmosphere Health* **7**, 459–466 (2014).
20. Urman, R. *et al.* Associations of children’s lung function with ambient air pollution: joint effects of regional and near-roadway pollutants. *Thorax* **69**, 540–547 (2014).
21. Byers, N., Ritchey, M., Vaidyanathan, A., Brandt, A. J. & Yip, F. Short-term effects of ambient air pollutants on asthma-related emergency department visits in Indianapolis, Indiana, 2007–2011. *J. Asthma* **53**, 245–252 (2016).
22. Batterman, S. *et al.* Dispersion modeling of traffic-related air pollutant exposures and health effects among children with asthma in Detroit, Michigan. *Transp. Res. Rec. J. Transp. Res. Board* **2452**, 105–113 (2014).
23. Andersson, M., Modig, L., Hedman, L., Forsberg, B. & Rönmark, E. Heavy vehicle traffic is related to wheeze among schoolchildren: a population-based study in an area with low traffic flows. *Environ. Health* **10**, 91 (2011).
24. Balmes, J. R. *et al.* Exposure to traffic: Lung function and health status in adults with asthma. *J. Allergy Clin. Immunol.* **123**, 626–631 (2009).
25. Brown, M. S. *et al.* Residential proximity to a major roadway is associated with features of asthma control in children. *PLoS ONE* **7**, e37044 (2012).
26. Carlsten, C., Dybuncio, A., Becker, A., Chan-Yeung, M. & Brauer, M. Traffic-related air pollution and incident asthma in a high-risk birth cohort. *Occup. Environ. Med.* **68**, 291–295 (2011).
27. Chang, J. *et al.* Repeated respiratory hospital encounters among children with asthma and residential proximity to traffic. *Occup. Environ. Med.* **66**, 90–98 (2009).
28. Cook, A. G., Annemarie, J. B. M., Pereira, G., Jardine, A. & Weinstein, P. Use of a total traffic count metric to investigate the impact of roadways on asthma severity: a case-control study. *Environ. Health* **10**, 52 (2011).
29. Dales, R., Wheeler, A. J., Mahmud, M., Frescura, A.-M. & Liu, L. The influence of neighborhood roadways on respiratory symptoms among elementary schoolchildren. *J. Occup. Environ. Med.* **51**, 654–660 (2009).
30. Delfino, R. J. *et al.* Repeated hospital encounters for asthma in children and exposure to traffic-related air pollution near the home. *Ann. Allergy. Asthma. Immunol.* **102**, 138–144 (2009).
31. Eckel, S. P. *et al.* Residential Traffic-Related pollution exposures and exhaled nitric oxide in the Children’s Health Study. *Environ. Health Perspect.* **119**, 1472–1477 (2011).
32. Ghosh, R. *et al.* Near-roadway air pollution and coronary heart disease: burden of disease and potential impact of a greenhouse gas reduction strategy in southern California. *Environ. Health Perspect.* **124**, (2015).

33. Longley, I., Somervell, E. & Gray, S. Roadside increments in PM₁₀, NO_x and NO₂ concentrations observed over 2 months at a major highway in New Zealand. *Air Qual. Atmosphere Health* **8**, 591–602 (2015).
34. Margolis, H. G. *et al.* Altered pulmonary function in children with asthma associated with highway traffic near residence. *Int. J. Environ. Health Res.* **19**, 139–155 (2009).
35. Porebski, G., Woźniak, M. & Czarnobilska, E. Residential proximity to major roadways is associated with increased prevalence of allergic respiratory symptoms in children. *Ann. Agric. Environ. Med.* **21**, 760–766 (2014).
36. Rice, M. B. *et al.* Long-term exposure to traffic emissions and fine particulate matter and lung function decline in the Framingham Heart Study. *Am. J. Respir. Crit. Care Med.* **191**, 656–664 (2015).
37. Salam, M. T., Islam, T. & Gilliland, F. D. Recent evidence for adverse effects of residential proximity to traffic sources on asthma: *Curr. Opin. Pulm. Med.* **14**, 3–8 (2008).
38. Ostro, B. Traffic pollution and children's health: refining estimates of exposure for the East Bay children's respiratory health study. (2008).
39. McConnell, R. *et al.* Air pollution and bronchitic symptoms in Southern California children with asthma. *Environ. Health Perspect.* **107**, 757–760 (1999).
40. Gauderman, W. J. *et al.* Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. *Lancet Lond. Engl.* **369**, 571–577 (2007).
41. Gauderman, W. J. *et al.* Association of improved air quality with lung development in children. *N. Engl. J. Med.* **372**, 905–913 (2015).
42. Berhane, K. *et al.* Association of changes in air quality with bronchitic symptoms in children in California, 1993-2012. *JAMA* **315**, 1491–1501 (2016).
43. Oakes, M., Baxter, L. & Long, T. C. Evaluating the application of multipollutant exposure metrics in air pollution health studies. *Environ. Int.* **69**, 90–99 (2014).
44. Miller, B. & Hurley, J. Life table methods for quantitative impact assessments in chronic mortality. *J. Epidemiol. Community Health* **57**, 200–206 (2003).
45. Abe, K. & Miraglia, S. Health impact assessment of air pollution in São Paulo, Brazil. *Int. J. Environ. Res. Public. Health* **13**, 694 (2016).
46. Dannenberg, A. L. *et al.* Use of health impact assessment in the U.S. *Am. J. Prev. Med.* **34**, 241–256 (2008).
47. James, P., Ito, K., Buonocore, J., Levy, J. & Arcaya, M. A health impact assessment of proposed public transportation service cuts and fare increases in Boston, Massachusetts (U.S.A.). *Int. J. Environ. Res. Public. Health* **11**, 8010–8024 (2014).
48. Joffe, M. & Mindell, J. A framework for the evidence base to support Health Impact Assessment. *J. Epidemiol. Community Health* **56**, 132–138 (2002).
49. Kersten, E., Rausa, J., Schuchter, J. & Van Erp, B. *Health Impact Assessment: California High Speed Rail San Jose To Merced Corridor.* (2011).
50. Martenies, S. E., Wilkins, D. & Batterman, S. A. Health impact metrics for air pollution management strategies. *Environ. Int.* **85**, 84–95 (2015).
51. Pascal, M. *et al.* Assessing the public health impacts of urban air pollution in 25 European cities: Results of the Aphekom project. *Sci. Total Environ.* **449**, 390–400 (2013).

52. United States Environmental Protection Agency. *User's Manual for the Co - Benefits Risk Assessment (COBRA) Screening Model*. Available at: <https://www.epa.gov/statelocalclimate/users-manual-co-benefits-risk-assessment-cobra-screening-model>. (Accessed: 25th February 2017)
53. IPCC. *IPCC, 2007: Climate Change 2007: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*. (Cambridge University Press, 2007).
54. Fann, N., Fulcher, C. M. & Hubbell, B. J. The influence of location, source, and emission type in estimates of the human health benefits of reducing a ton of air pollution. *Air Qual. Atmosphere Health* **2**, 169–176 (2009).
55. Morani, A., Nowak, D. J., Hirabayashi, S. & Calfapietra, C. How to select the best tree planting locations to enhance air pollution removal in the MillionTreesNYC initiative. *Environ. Pollut.* **159**, 1040–1047 (2011).
56. Gass, K. *et al.* Associations between ambient air pollutant mixtures and pediatric asthma emergency department visits in three cities: a classification and regression tree approach. *Environ. Health* **14**, (2015).
57. Leclercq, B. *et al.* Differential responses of healthy and chronic obstructive pulmonary diseased human bronchial epithelial cells repeatedly exposed to air pollution-derived PM₄. *Environ. Pollut.* **218**, 1074–1088 (2016).
58. Newcomb, P. & Li, J. Predicting admissions for childhood asthma based on proximity to major roadways. *J. Nurs. Scholarsh.* **40**, 319–325 (2008).
59. Taj, T., Stroh, E., Åström, D. O., Jakobsson, K. & Oudin, A. Short-term fluctuations in air pollution and asthma in Scania, Sweden. Is the association modified by long-term concentrations? *PLOS ONE* **11**, e0166614 (2016).
60. Daniel, J., Sears, A. & Jackson, L. *Residential Population within 300m of Busy Roadway*. (United States Environmental Protection Agency).
61. Ponsonby, A. L. *et al.* The relation between infant indoor environment and subsequent asthma. *Epidemiol. Camb. Mass* **11**, 128–135 (2000).
62. Belanger, K. & Triche, E. W. Indoor combustion and asthma. *Immunol. Allergy Clin. North Am.* **28**, 507–vii (2008).
63. Lanphear, B. P., Aligne, C. A., Auinger, P., Weitzman, M. & Byrd, R. S. Residential exposures associated with asthma in US children. *Pediatrics* **107**, 505–511 (2001).
64. Kanchongkittiphon, W., Mendell, M. J., Gaffin, J. M., Wang, G. & Phipatanakul, W. Indoor environmental exposures and exacerbation of asthma: an update to the 2000 review by the Institute of Medicine. *Environ. Health Perspect.* **123**, 6–20 (2015).
65. Naeher, L. P. *et al.* Woodsmoke health effects: a review. *Inhal. Toxicol.* **19**, 67–106 (2007).