Dorothy Holasek
[redacted]

August 4, 2016

Re: Open comment
Scoping Processing
4FRI Rim Country

Coconino National Forest
Attention: 4FRI
1824 Thompson Street
Flagstaff AZ 86001

Gentlemen:

There can be no justification for the increased suffering and death from smoke pollution generated by or preventable by the USFS when there are viable alternative methods to managing our forests. Averting behavior is costly and impractical for many, making it ineffectual. Averting behavior is inaccessible to low income families in downwind populations with high poverty rates. Reminiscent of "let them eat cake"; let the poor turn on their air conditioning or take a vacation to San Diego".

The human body does not distinguish between dying from PM 2.5 for the sake of a "perceived" noble or ignoble cause.

Sincerely,

Dorothy Holasek

Dorothy Holasek

encl: references + hard copies

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Air Pollution Increases Death Risk in People with Diabetes, Heart Failure, COPD or Rheumatoid Arthritis

SAN DIEGO—People with diabetes, heart failure, chronic obstructive pulmonary disease and inflammatory diseases such as rheumatoid arthritis are at increased risk of death when they are exposed to particulate air pollution, or soot, for one or more years, according to a study to be presented at the American Thoracic Society International Conference on May 22nd.

The study looked at hospital discharges for people with these four types of diseases living in 34 cities between 1985 and 1999. The researchers compared this information with 12-month averages of PM10, a type of particulate matter air pollution that includes particles with a diameter of 10 micrometers or less than 0.0004 inches or one-seventh the width of a human hair.

The study found that for an increase of 10 micrograms/per cubic meter of PM10 over two years, the risk of dying was increased by:

- 32% for people with diabetes
- 28% for people with COPD
- 27% in people with congestive heart failure
- 22% for people with inflammatory disease such as rheumatoid arthritis or lupus

"The study significantly strengthens evidence that breathing in particulate matter is associated with dying sooner," said researcher Joel Schwartz, Ph.D., Professor of Environmental Epidemiology at the Harvard School of Public Health in Boston.

"While previous studies have found that long-term exposure to air pollution is associated with increased risk of death, we looked at risk of death in the first three years after patients were discharged from the hospital, and saw that the risk increased in the first couple of years. That means if we can lower air pollution levels, people will start living longer right away—we don't have to wait many years to see health improvements. That wasn't clear from previous air pollution studies."

Technology that can reduce particulate matter levels already exists, Dr. Schwartz noted. "For instance, we know how to put scrubbers on coal-burning power plants, which are a major source of particulate matter. There is no safe level of particulate air pollution, so we need to get the levels as low as reasonably possible."

While previous studies have linked exposure to PM10 to harmful effects on breathing and respiratory systems, damage to lung tissue, cancer, and premature death, this is the first study to follow people with specific diseases to determine their risk of death in response to particle exposure, Dr. Schwartz said.

He noted an important difference between this new study and past air pollution studies. "Past studies have compared average air pollution conditions in one city to other cities, and you need to worry about potential confounders, or other factors that could affect the differences between the cities," he said. "In this study, we looked at air pollution and deaths within each city—we didn't compare across cities, so we didn't need to worry about confounding factors."

The study helps validate the findings of previous studies that have shown that long-term exposure to air pollution is associated with shortened survival in the general population, said Dr. Schwartz, a co-author of the Six Cities Study, which evaluated the effects of pollution on adults in the 1970s and 1980s. The results of that study found a strong, positive correlation between levels of air pollution and mortality. The study led to a revision of existing air quality standards by the U.S. Environmental Protection Agency. An eight-year follow up study found an association between people living longer and cities reducing the amount of fine particulate matter in their air. That study was published in the March 15, 2006 issue of the American Thoracic Society journal, *The American Journal of Respiratory and Critical Care Medicine*.

Occup Environ Med. 2009 Mar;66(3):189-97. Epub 2008 Nov 18.

New Zealand Air Gets Cleaner

Fairfax Stuff, stuff.co.nz, May 16, 2014

Lives are being saved as the quality of air in New Zealand improves through technology that reduces vehicle pollutants, and as fewer homes burn wood or coal. A report published May 16th, 2014, said there was an 8% fall in the concentration of PM10 — very small airborne particles 10 micrometers or less in diameter — between 2006 and 2012.

PM10 particles are associated with health problems ranging from respiratory irritation to cancer. A model estimated that deaths from exposure to man-made PM10 were down 14% because of the drop in PM10 concentrations.

Hospital admissions were down an estimated 15%, and days with restricted activity were down an estimated 9%. The figures were in the 2014 air domain report produced by the Ministry for the Environment and Statistics New Zealand. It is the first report in a new environmental reporting series. Other aspects of the environment to be covered will be atmosphere and climate, fresh water, land and the marine environment.

"Good outdoor air quality is fundamental to our wellbeing. On average, a person inhales about 14,000 liters of air every day, and the presence of contaminants in this air can adversely affect people's health," the report said. "People with pre-existing respiratory and heart conditions, diabetes, the young and older people are particularly vulnerable."

The report said estimated emissions from road transport fell by between 25 and 49% for a range of pollutants from 2001 to 2012. That was mainly because of technological advances in vehicles and fuel, with vehicle use up 11%. The decrease in estimated emissions may have contributed to the fall in PM10 concentrations.

Despite the improvement, road transport continued to be a problem, with high levels of nitrogen dioxide and benzene in some places during peak traffic, the report said. From 1996 to 2013, there was a 25% fall in the number of households burning wood or coal for heating, which was also likely to have contributed to the fall in PM10 concentrations.

Burning wood and coal was still associated with air-quality issues, including high levels in some places of the finer PM2.5 particles, arsenic from burning treated timber, and the hydrocarbon benzo(a)pyrene.

New Zealand's average national PM10 concentration was the seventh lowest of 32 Organization for Economic Co-operation and Development countries in 2011, while 48 out of 55 monitoring sites met World Health Organization long-term guidelines in 2012, the report said.

8% reduction in fine particulates => 15% reduction in hospital admissions.

» Print

Heart experts warn of air pollution dangers

Thu, Dec 18 2014

PM2.5 => Heart disease

By Janice Neumann

(Reuters Health) - Air pollution should be one of the avoidable heart risk factors - just like smoking and excess fat - that doctors warn patients to steer clear of, according to a new statement from 20 heart experts.

Citing pollution's heavy toll on cardiovascular health, the panel urges people to take steps to protect themselves from breathing heavy traffic fumes or industrial air pollution whenever possible, and public officials to pass laws to reduce air pollution.

"Cardiovascular disease is a huge global problem, causing immense suffering and premature death, as well as placing severe strain on national healthcare budgets and/or family finances," said Dr. Robert Storey, a professor of cardiology at the University of Sheffield in the UK and senior author of the new position paper.

Air pollution causes more than 3 million deaths worldwide each year and causes 3.1 percent of all cases of disability, Storey and his coauthors write in the European Heart Journal.

Air pollution is also ninth most important on a list of modifiable heart-disease risk factors - ranking above low physical activity, high-salt diet, high cholesterol and drug use, the authors point out.

Although gaseous air pollutants can be dangerous too, Storey said, airborne particles are the biggest contributor to cardiovascular disease because they cause inflammation of the lungs and enter the circulation, inflaming blood vessels, provoking clots and causing heart rhythm disturbances.

Particulate matter includes coarse particles from road dust, construction work and industrial emissions and fine particles from traffic, power plants and industrial and residential burning of oil, coal or wood for heating.

The bulk of particulate air pollution is made up of these fine particles, known as PM2.5, that are less than 2.5 micrometers - about one fifth the size of visible dust.

The World Health Organization sets the safe outdoor exposure limit for PM2.5 at an average of 25 micrograms, or 25 millionths of a gram, per cubic meter of air over a 24-hour period, or average annual levels of 10 micrograms per cubic meter. In 2013, the U.S. Environmental Protection Agency lowered the 24-hour exposure limit to an average of 12 micrograms.

European studies have found that PM2.5 levels are often markedly higher near heavy traffic zones compared to elsewhere in the same city, and that the levels can more than double during rush hours, according to the position statement.

Some of the authors' advice for people to protect themselves is as simple as walking, cycling and using public transportation instead of driving cars, and exercising in parks or gardens, rather than near busy roads.

And everyone should avoid being outside when pollution is highest, though this is especially important for infants, elderly and people with heart problems, the authors say.

People who live in heavily polluted areas should also consider ventilation systems with filtration in their homes, since a large portion of outdoor pollution can penetrate buildings.

The use of fossil fuels for heating and energy should also be decreased, according to the statement.

"Many countries have made good progress towards reducing risk factors such as smoking, high cholesterol and high blood pressure but much less effort has been extended on reducing exposure to air pollution," Storey said in an email to Reuters Health.

Studies have shown even short-term exposure to high PM2.5 levels increases deaths from heart disease and respiratory disease, and that people living in places with high PM2.5 have an 11 percent greater risk of dying from heart attacks, strokes and heart failure than those who live in cleaner areas.

Dr. Robert Brook, a cardiologist at the University of Michigan Health System and another author of the policy statement, said many people don't realize the dangerous effects of air pollution on the heart.

"While most people can readily observe and believe that air pollution may cause lung diseases, it is in fact cardiovascular diseases that are the largest adverse health effect of fine particulate matter exposure," Brook said in an email.

Dr. Alan Abelsohn of the Dalla Lana School of Public Health at the University of Toronto in Canada, called the statement an important reminder. Too few cardiologists and primary care doctors advise their patients of pollution's risks, he said.

"It's a very important and neglected area of prevention," he said.

Abelsohn, who was not involved in the position statement, noted that national-level guidelines on allowable amounts of pollution can only do so much. He said individuals should always pay attention to the local Air Quality Index, which rates the level of air pollution according to health risk, and reduce their exposure accordingly.

Brook said that while the U.S. has made great strides reducing air pollution since the 1970s or even 2000, the efforts should continue.

"What we should not do is lessen our regulations and pose a threat to the cardiovascular health of the nation in the name of expediency or supposed economic growth or stimulus," Brook said.

SOURCE: bit.ly/1zt1lw6 European Heart Journal, online December 9, 2014.

Air Pollution May Shrink the Brain, Study Suggests

by Agata Blaszczak-Boxe, Contributing Writer | April 23, 2015 07:02pm ET

Smog was a term originally describing a fusion of smoke and fog.
Credit: Viktor Fiker | Dreamstime
View full size image

Breathing polluted air every day may change a person's brain in ways that end up leading to cognitive impairment, according to a new study.

In the study, researchers examined 943 adults who were at least 60 years old and lived the New England region. The investigators used magnetic resonance imaging (MRI) to look at the participants' brain structures, and compared the images with the air pollution levels in the places where the participants lived.

The researchers found that an increase of 2 micrograms per cubic meter in fine-particle pollution — a range that can be observed across an average city — was linked to a 0.32 percent reduction in brain volume. (Fine-particle pollution is a common type of pollution that comes from car exhaust, among other sources.)

That amount of change in brain volume "is equivalent to about one year of brain aging," said study author Elissa H. Wilker, a researcher in the cardiovascular epidemiology research unit at Beth Israel Deaconess Medical Center in Boston.

In general, a smaller brain volume is caused by the loss of neurons that comes with aging, the researchers said. [10 Things You Didn't Know About the Brain]

That same increase of 2 micrograms per cubic meter of fine-particle pollution was also linked with a 46 percent increase in the participants' risk of having what researchers call "silent strokes," which can be seen on brain scans but don't usually cause symptoms. Such strokes have been associated with poorer cognitive function and dementia, the researchers said.

The researchers found that the people in the study who lived in areas with higher levels of pollution had smaller brain volumes and were also at higher risk of silent strokes compared with the people who lived in areas where the air was less polluted. However, the study was done at one point in time, and Wilker noted that it does not prove there is a cause-and-effect relationship between air pollution and brain changes.

This is the first study to examine a link between air pollution, brain volume and the risk of silent strokes in a population of older adults, the researchers said. Previous studies have examined the relationship between air pollution and the brain in children, but not in older adults, they said.

It is not clear exactly how air pollution may change people's brains, the researchers said. They suspect that air pollution may cause increased inflammation, but the researchers are still trying to understand the link, Wilker said. Previous research has linked markers of inflammation to smaller brain volume, according to the study. The new results may help

the researchers understand "what could be going on between air pollution and serious outcomes like stroke and cognitive impairment," Wilker told Live Science.

The study was published today (April 23) in the American Heart Association journal Stroke.

Follow Agata Blaszczak-Boxe on Twitter. Follow Live Science @livescience, Facebook & Google+. Originally published on Live Science

Long-term exposure to air pollution may harm your brain

American Heart Association Rapid Access Journal Report

April 23, 2015 Categories: Stroke News

Study Highlights

- Long-term exposure to air pollution is linked with brain shrinkage.
- A small increase in fine particulate matter pollution was associated with hidden brain damage linked to impaired cognitive function.

Embargoed until 3 p.m. CT/4 p.m. ET Thursday, April 23, 2015

DALLAS, April 23, 2015 — Long-term exposure to fine particle air pollution may cause subtle structural changes in the brain that could precede cognitive impairment and hidden brain damage, according to research in the American Heart Association journal *Stroke*.

Fine particle air pollution – smaller than 2.5 micrometers in diameter ($PM_{2.5}$) – may be the most common and hazardous type of air pollution. It comes from burning wood or coal, car exhaust and other sources.

"Long-term exposure to air pollution showed harmful effects on the brain in this study, even at low levels, particularly with older people and even those who are relatively healthy," said Elissa H. Wilker, Sc.D., study lead author and researcher in the Cardiovascular Epidemiology Research Unit at Beth Israel Deaconess Medical Center and the Harvard T.H. Chan School of Public Health in Boston.

Researchers analyzed 943 adults in the Framingham Offspring Study, who were relatively healthy and free of dementia and stroke. The participants lived in the greater Boston area and throughout New England and New York — regions where air pollution levels are low compared to other parts of the nation and the world.

During 1995-2005, researchers used magnetic resonance imaging (MRI) to determine the effect of long-term exposure to air pollution on markers of brain structure. They found a 2 microgram per cubic meter of air ($\mu g/m^3$) increase in $PM_{2.5}$, a range commonly observed across a metropolitan region, was associated with a 0.32 percent smaller total cerebral brain volume and a 46 percent higher risk of covert brain infarcts, a type of silent stroke.

"The magnitude of association that we observed for brain volume was similar to approximately one year of brain aging," Wilker said.

Fundamental changes in the structure of the cerebral brain volume and smaller brain size are markers of age-associated brain atrophy.

"We found that people who live in areas where there are higher levels of air pollution had smaller total cerebral brain volume and were more likely to have evidence of covert brain infarcts," said Wilker, who is also an instructor of medicine in the Harvard Medical School.

The small infarcts, typically located in deep regions of the brain, have been associated with neurological abnormalities, poorer cognitive function, dementia, and are thought to reflect small vessel disease, she said.

Fine particulate matter affects more people than any other pollutant, with chronic

A small increase in 2.5PM "was associated with hidden Brain Damage Linked to impaired Cognitive function".

exposure causing the most deaths from serious disease, according to the World Health Organization (WHO). $PM_{2.5}$ may trigger disease because the particles penetrate into the alveoli of the lungs. Fine particulate matter can also contribute to the narrowing of arteries that supply blood to the brain.

These findings are consistent with prior studies that have shown an association between long-term pollution exposure and living close to major roads and first-time stroke and poorer cognitive function in older adults.

To educate the public about daily air quality levels, including ozone and particulate matter levels, the EPA provides daily updates at www.epa.gov/airnow and in many newspapers across the country.

Co-authors are Sarah Preis, Sc.D.; Alexa Beiser, Ph.D.; Philip Wolf, M.D.; Rhoda Au, Ph.D.; Itai Kloog, Ph.D.; Wenyuan Li, M.S.; Joel Schwartz, Ph.D.; Petros Koutrakis, Ph.D., Charles DiCarli, M.D.; Sudha Seshadri, M.D.; and Murray Mittleman, M.D.

The National Institutes of Health and the Environmental Protection Agency funded the study.

Additional Resources:

- Researcher photo, stroke animation, brain image, and b-roll available on the right column of the release link http://newsroom.heart.org/news/long-term-exposure-to-air-pollution-may-harm-your-brain?preview=953cec8522fc97ef38b2db045f2e1cbc
- Spanish release
- Air Pollution and Heart Disease, Stroke
- Follow AHA/ASA news on Twitter @HeartNews.
- For stroke science, follow *Stroke* at @StrokeAHA_ASA.

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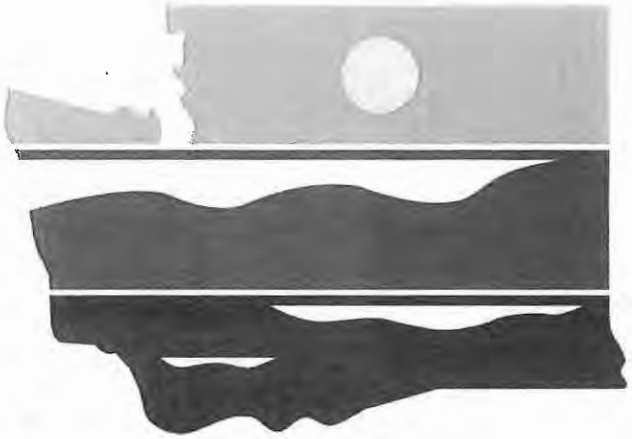
heart.org and strokeassociation.org



DEPARTMENT OF
ECOLOGY
State of Washington

Health Effects and Economic Impacts of Fine Particle Pollution in Washington

Washington State Department of Ecology

Air Quality Program

December 15, 2009

Publication number: 09-02-021

Executive Summary

Since 1990, a large number of scientific studies have documented the negative health effects of air pollution on people, especially the health effects of particle and fine particle pollution - particles smaller than 2.5 microns in size. Just as these health effect studies have been used to determine the health and financial consequences of air pollution for the national population, they can be used to better understand the consequences for citizens and communities in Washington State. This report describes a Department of Ecology (Ecology) analysis to quantify the health and monetary impacts of fine particle pollution in the state. The analysis was done by comparing estimates of the impacts at existing pollution levels to estimates of health effects at a "clean air" or background level.

Estimating costs/benefits

Ecology used a tool developed by the US Environmental Protection Agency (EPA) to analyze and estimate the benefits of regulating air pollution. This tool, called Environmental Benefits Mapping and Analysis Program (BenMAP), estimates the health effects and health care costs of disease resulting from air pollution. To make these estimates, BenMAP uses:

- population data,
- air quality measurement information,
- information on the relationship between $PM_{2.5}$ exposure health effects from a variety of epidemiological studies of air pollution,
- disease incidence and prevalence rates, and
- information from economic studies of the monetary benefits of reducing the incidence of these health impacts .

Ecology limited its analysis to the health effects of fine particle air pollution - also called $PM_{2.5}$. Ecology focused on $PM_{2.5}$ because it is a pollution problem in many Washington communities, has received the largest volume of national and international research, and has been associated with significant adverse human health effects. $PM_{2.5}$ has been strongly associated with increases in heart, circulatory and lung diseases that sometimes result in death.

Results of analysis

Numerous national and international studies show an association between increases in $PM_{2.5}$ and increases in premature death. Ecology selected five different studies to model the relationship between $PM_{2.5}$ and an increase in premature death in Washington. These studies estimate between 300 and 4,300 additional deaths in Washington each year. Using one of the most widely-cited studies in the health effects literature (Pope, et al. 2002), Ecology estimates conservatively that approximately 1,100 people die each year in Washington due to $PM_{2.5}$. Numerous other studies show an association

between increases in $PM_{2.5}$ and the incidence of serious diseases. Based on studies included in the BenMAP model, Ecology estimates that, every year in Washington, $PM_{2.5}$ contributes to approximately:

- 1,500 nonfatal heart attacks,
- 450 incidents of different heart diseases not resulting in heart attacks,
- 1,900 incidents of acute bronchitis,
- 100 cases of chronic lung disease,
- 250 incidents of pneumonia,
- 400 emergency room visits for asthma, and
- thousands of incidents of worsened asthma.

Ecology estimates that the direct and indirect costs of these diseases for citizens, businesses and state health care institutions approach $190 million each year.

Health Effects and Economic Impacts of Fine Particle Pollution in Washington

Since 1990, there has been a significant increase in information about the health effects of air pollution on people, especially the health effects of particle and fine particle pollution. Hundreds of such studies have been conducted in the U.S. and worldwide. The results of these studies have been used to inform decisions by the World Health Organization and national environmental organizations about clean air guidelines and standards. These studies have consistently shown that fine particle pollution is more dangerous to human health than originally thought. Exposure to levels previously believed to be safe can result in a range of diseases and, in some cases, death. In 2007, the Environmental Protection Agency (EPA) used the results of these studies to establish more protective pollution limits for fine particles in the United States.

Just as the health effect studies have been used to determine the health and financial consequences of air pollution for the national population, they can be used to better understand the same consequences for citizens and communities of Washington State. Ecology has undertaken an effort to better understand the health and economic effects of air pollution in Washington. Understanding these effects can help inform state and local government policy decisions about air quality.

Ecology's analysis seeks to answer the question "What are the health effects and economic costs of the existing levels of fine particle air pollution in Washington?" We did this by comparing estimates of the impacts at existing pollution levels to estimates of health effects at a "clean air" or background level.

Initial Analysis

Ecology limited its initial analysis to the health effects of fine particle air pollution - also called $PM_{2.5}$. $PM_{2.5}$ comes from burning fossil fuels and organic matter. It is a pollution problem in many communities in Washington. It is also the pollutant that has received the bulk of academic study over the past two decades. Fine particles are easily inhaled and, because of their small size, are not filtered by the nose. The smaller these particles are, the more deeply they penetrate into the lungs, where they can cause damage. Breathing fine particles is associated with most types of respiratory illness, cardiovascular disease (heart disease and strokes), and even death. Extensive research studies consistently show that exposure to elevated fine particle concentrations is associated with:

- Death and mortality:
    - Increased total number of deaths
    - Increased number of deaths from respiratory illnesses
    - Increased cardiovascular deaths
    - Increased lung cancer
    - Increased risk of premature births and infant mortality

- Hospitalization:
  - Increased hospital admissions and emergency room visits
  - Increased hospital admissions, emergency room visits for respiratory and cardiovascular conditions
- Respiratory illness:
  - Increased risk of pneumonia
  - Increased risk of post-neonatal mortality from respiratory disease
  - Increased pneumonia, bronchitis and chronic obstructive pulmonary disease
  - Increased respiratory symptoms in both the lower and upper respiratory tract
  - Decreased lung function
  - Increased incidence of rhinitis (respiratory allergic reactions) and sinusitis.
- Asthma:
  - Worsened asthma attacks
  - Increased bronchodilator use
  - Increased hospital admissions due to asthma attacks
- Lost work days and quality of life:
  - Increased absences from work or school
  - Increased number of days of restricted activity

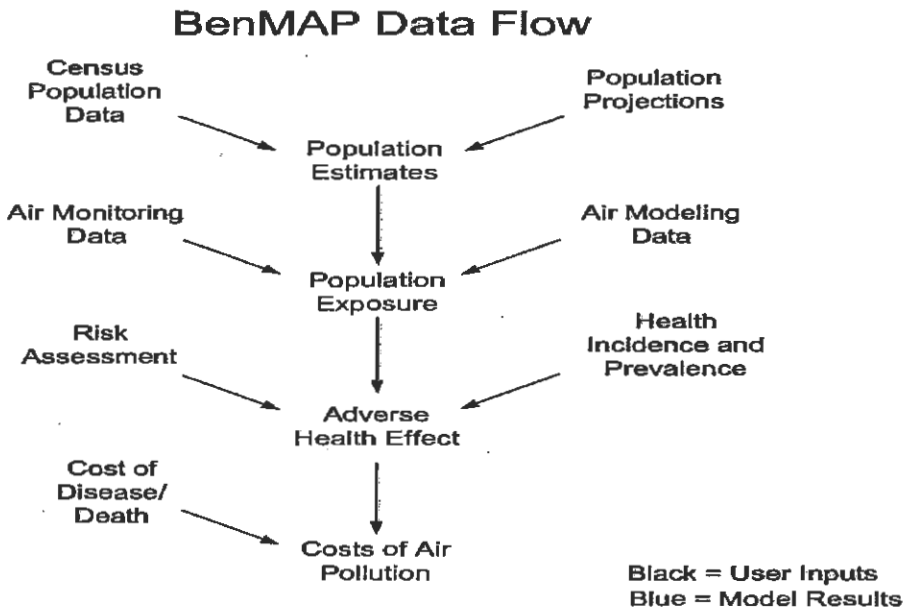
In Ecology's initial analysis, we estimated the likely number of some of these effects in Washington. We used information from epidemiological studies, along with monitored and modeled $PM_{2.5}$ data, to develop our estimates.

Regulatory benefit analysis

Ecology used a tool developed by EPA to analyze the benefits of national air quality regulations. This tool, called Environmental Benefits Mapping and Analysis Program (BenMAP), uses:

- geographic population data
- air quality measurement information
- information on health effects from a variety of epidemiological studies of certain types of air pollution, and
- information from economic studies of the costs of disease.

BenMAP combines this information to estimate the health effects, health care costs and economic impacts of disease resulting from air pollution within geographic areas.



Using BenMAP

Team

Ecology assembled a technical team of specialists to select appropriate data for the model and learn how to use the tool. The agency sought initial guidance from the developer of BenMAP. We developed a team of specialists in air quality monitoring and modeling, toxicology, epidemiology, emissions inventory, policy analysis, information technology, and economics. The team included relevant Ecology Air Quality Program staff and representatives of the state Department of Health and the Puget Sound Clean Air Agency, as well as a research professor from the University of Washington.

Data

We have run the model using Washington-specific population and air pollution information, as well as Washington health care cost data, when available.

For purposes of estimating health effects, we believe it is acceptable to apply national- or regional-level health data to Washington's population and air quality conditions (Hubbell et. al. 2009). Several of the national-level studies, while not specific to Washington, nevertheless included Washington cities in the analyses of health effects. To help verify the results, we have compared the estimates generated by the model to the number of hospitalizations and deaths in Washington. This provides an indication of how reasonable the modeled estimates were compared to the total number of recorded health effects. Therefore, we believe our results reflect reasonable estimates of the health effects of fine particle pollution.

Methods

EPA developed the BenMAP tool specifically to compare the health impacts and economic benefits of implementing particular policy choices. Therefore, the models used in the tool are set up to compare current air pollution conditions to changed air pollution conditions to estimate a change in health and cost outcomes. The team estimated $PM_{2.5}$'s health and corresponding monetary costs to Washington by essentially comparing existing air pollution levels to "clean" air quality conditions. We defined "clean air" as the average of the cleanest 20% of days per monitored area.

Conditions that affect human exposure to $PM_{2.5}$, such as weather patterns, can vary considerably from year to year. In an effort to minimize this variability, we used calendar day median values from five recent, continuous years of air pollution readings to create a "representative year" for this analysis.

Population

The team used 2007 county census population projections from the state Office of Financial Management. We separated these data into 18 different age groups. Note that BenMAP uses these age groups to more closely match the age groups targeted by each health study. This improves the accuracy of the health effect impact estimates.

Monitoring

Ecology has a network of over 50 continuous $PM_{2.5}$ monitors statewide. The data from these monitors meets all the EPA guidelines for the collection and use of such data. We loaded five years of Washington State $PM_{2.5}$ monitoring data (2004 through 2008) into BenMAP for this study. In order to align monitoring data with health effects information from the epidemiological studies, we calculated daily $PM_{2.5}$ averages (midnight to midnight). From the five years of daily data, we then calculated median values for each calendar day, effectively creating a complete "representative" year for each location.

Modeling

BenMAP uses modeled data to estimate $PM_{2.5}$ concentrations in areas between air quality monitoring sites. Washington State University models the statewide $PM_{2.5}$ concentrations on a daily basis. We obtained three years of model data (2006-2008) and calculated the daily $PM_{2.5}$ averages at each 12 kilometer grid location of the state. Medians at each grid point were then computed for each calendar day of the "representative" year. We think the resulting dataset of the "representative year" is a robust estimate of $PM_{2.5}$ in Washington State, and is not unduly influenced by year-to-year variations.

Health functions

A major branch of environmental epidemiology concerns the effect of air pollution on humans. This research examines whether there are significant associations between exposure to certain air pollutants and certain diseases or death, and how strong such associations are. To different degrees, studies also

account for other factors that may influence the results. The BenMAP tool comes pre-loaded with information from a number of studies that show the adverse health effects of air pollutants.

The technical team evaluated the studies in BenMAP to determine which appeared to be the most relevant to Washington. We selected several studies and their related health impact functions (also called concentration-response functions, or CRFs) from the hundreds preloaded in BenMAP. We also reviewed the most recent epidemiology literature to make sure we included any important studies not yet in BenMAP. As a result of this review, we added data based on Jerrett et. al. 2009. All of the studies used have been published in major scientific journals following peer review. Our intent was to choose the most relevant and appropriate studies to predict outcomes in Washington. Including confidence intervals in BenMAP allows results to be expressed as ranges for each possible health outcome among populations exposed to $PM_{2.5}$. Note that the epidemiological studies differ in their conclusions, and that the estimates we report are on the low end of the scale. In other words, the incidence rates and associated costs may be considerably higher than we estimate.

BenMap also includes disease incidence and prevalence rates. Pre-loaded rates within the tool were used for all analyses. Mortality (death) rates were available by counties in each state. BenMap used regional rates for hospitalization for four regions of the United States: northeast, mid-west, south, and west. The west coast regional rates used in our analyses include combined rates from Montana, Idaho, Wyoming, Colorado, New Mexico, Arizona, Utah, Nevada, Washington, Oregon, California, Alaska, and Hawaii. BenMap obtained prevalence or incidence rates of chronic and acute diseases and respiratory symptoms from national databases, surveys, and research investigations.

Health care costs and economic impacts

In addition to health effect studies, the BenMAP model has a number of pre-loaded economic and health care cost studies. The technical team's economist evaluated those studies and selected the ones that were most appropriate for the health effects we estimated. In addition, where possible, the economist looked for Washington-specific health care costs and updated all costs to reflect 2009 dollars.

Costs of death and illness

Studies in BenMAP include estimates of direct costs of illness and indirect costs associated with pollution-caused illness:

- Direct costs are those tangible costs for hospitalization, medical tests and services, medicines, doctor visits, etc. Direct costs are estimated using actual charges billed by hospitals and medical establishments.
- Indirect costs are sometimes less tangible, and can include lost work days due to illness or caring for a sick child, lost productivity or education, pain and suffering, and the cost of death. Indirect costs can also be costs to society – not just the sick individual – when they impact families, employers and production, or long-term educational attainments. To estimate indirect costs (or combined direct and indirect costs) economists use estimates of willingness to pay – the amount an individual would be willing to pay to avoid illness or death.

- Willingness to pay is estimated using widely accepted survey techniques that reveal willingness to pay through experiments, or by asking people directly. Willingness to pay estimates can include various direct, indirect, and societal costs at the same time, because they are based on the set of costs perceived by respondents.

Ecology included the most appropriate estimates of costs based on available data, and gave preference to direct costs where available, since they are easier to accurately quantify and provide a conservative base estimate of the costs of illness. However, it should be noted that some of the valuation studies include both direct costs and indirect/willingness to pay estimations.

Uncertainty

Models are tools developed and used to indirectly estimate outcomes when direct measurements are not possible. BenMAP is a model that relies on a variety of inputs and the inputs are subject to varying degrees of uncertainty: Population data is a forecast of people living within geographic areas; 2) monitors provide highly accurate, direct measurement of air pollution at individual monitoring sites – however, in between monitoring sites and at the community scale monitored and modeled data provide only estimates of air pollution levels; health-effect data are estimates with ranges of uncertainty based on studies that estimate the link between certain health end points and exposure to air pollution; rates of disease and hospitalizations are often not state or county specific; and health care cost data is the estimated value of treatment and impact for symptoms and disease which could be expected to vary between patients and communities. Even given these uncertainties, BenMAP is an internationally recognized and accepted tool for estimating health effects and health care costs of exposure to air pollution. Ecology recognizes these uncertainties in the model's outputs and has chosen to use reliable, conservative inputs to the model to generate conservative estimates of outcomes.

Results

General

Because $PM_{2.5}$ is created mostly from combustion in industrial, commercial and residential activities near population centers, higher pollution levels and therefore greater health impacts will occur in and around those areas. Wildfires that occurred in eastern Washington during our study period also generate high $PM_{2.5}$ concentrations; however, the health impacts are smaller than in urban areas since these areas are usually less populated (see mapped results in Appendices).

Incidence of mortality/death

Numerous national and international studies show an association between increases in $PM_{2.5}$ and increases in premature death. Ecology selected five different studies to investigate the relationship between $PM_{2.5}$ and an increase in premature death in Washington. Study findings (at the 50^{th} percentile) ranged from 900 additional deaths (Jerrett, et al. 2009) to 2,800 additional deaths (Laden, et al. 2006) each year. Using one of the most widely-cited studies contained in BenMAP (Pope, et al. 2002), Ecology estimates that approximately 1,100 people die each year in Washington due to $PM_{2.5}$.

Study	Ages	Number of Deaths 50th Percentile	Range
Pope et. al. (2002)	30-99	1,100	300-1,900
Laden et. al. (2006)	25-99	2,800	1,300-4,300
Jerrett et. al. (2009) PM	30-99	900	500-1,300
Jerrett et. al. (2009) PM/O_3	30-99	1,500	900-2,000
Woodruff et. al. (2006)	0-1 (Infant)	5	4-14

Rounded to two significant figures

Of note, a late breaking study by Krewski et. al. published by the Health Effects Institute in June 2009, re-evaluated the data used in the 2002 Pope study. Those results strengthen Pope's conclusions.

Incidence of morbidity/disease

$PM_{2.5}$ has been strongly associated with increases in heart, circulatory and lung diseases. Some of these health impacts are serious enough to result in the premature deaths reflected above. Estimates of nonfatal health impacts for more serious diseases are shown in the table below. The table below shows disease, study used for the CRF, ages of population included, and the midrange of the number of people affected. Different studies result in different findings, therefore there may be some difference or overlap between the studies in this table of results. The BenMAP model allows the user to choose, compare, and/or pool relevant studies.

Disease	Study	Ages of Patients	Incidence (50th Percentile)
Heart Attack (Nonfatal)	Peters et al. 2001	18-99	1,500
All Cardiovascular (less Heart Attack)*	Moolgavkar et al. 2000	18-64	160
All Cardiovascular (less Heart Attack)*	Moolgavkar et al. 2000	65-99	280
Congestive Heart Failure*	Ito et al. 2003	65-99	150
Dysrhythmia*	Ito et al. 2003	65-99	43
Ischemic Heart Disease*	Ito et al. 2003	65-99	87
Chronic Lung Disease (Less Asthma)*	Moolgavkar et al. 2000	18-64	39

Chronic Lung Disease*	Moolgavkar et al. 2003	65-99	66
Chronic Lung Disease*	Ito et al. 2003	65-99	41
Pneumonia*	Ito et al. 2003	65-99	260
Acute Bronchitis*	Dockery et al. 1996	8-12	1,900
Asthma Hospitalization*	Sheppard et al. 2003	0-64	100
Asthma, Emerg. Rm. Visits	Norris et al. 1999	0-17	400
Asthma Exacerbation, Cough	Ostro et al. 2001	6-18	13,000
Asthma Exacerbation, Cough	Vedal et al. 1998	6-18	63,000
Asthma Exacrb, Short of Breath	Ostro et al. 2001	6-18	18,000
Asthma Exacrb, Wheeze	Ostro et al. 2001	6-18	29,000

Rounded to two significant figures
*Hospital admissions

Monetary benefits (Ecology-selected estimates)

Ecology estimated Washington State-specific values whenever possible, and otherwise maintained consistency with the Environmental Protection Agency's (EPA, 2003) methodology and sources, when a more appropriate or precise state-specific methodology was not available. This means some estimates are entirely Washington State-specific, others are based on national valuations, and some combine national and state-level data.

Overall, Ecology's goal in developing these numbers was to calculate the most state-appropriate values, while maintaining a high degree of confidence in the representativeness and reliability of the results. All dollar valuations are in 2009 inflation-adjusted dollars.

The table below shows the disease, the study used for the valuation, the mid-range of the number of people affected, and cost estimates based on Ecology's estimates of costs in Washington State.

Disease	Valuation Function	Incidence (50^{th} %)	Estimated Cost
Heart Attack (Nonfatal)	Russel, et al. (1998)	1,500	$70,000,000
All Cardiovascular (less Heart Attack)*†	CHARS 2006 – 2008Q3 (WA-DOH, 2008)	450	$17,000,000
Chronic Lung Disease*	CHARS 2006 – 2008Q3 (WA-DOH, 2008)	100	$ 2,000,000
Pneumonia*	CHARS 2006 – 2008Q3 (WA-DOH, 2008)	250	$ 5,000,000
Acute Bronchitis	Dickie, et al. (1987), Tolley, et al. (1986), and Loehman, et al. (1979)	1,900	$ 850,000
Asthma*	CHARS 2006 – 2008Q3 (WA-DOH, 2008), U.S. Bureau of Labor and Statistics, (2009b)	100	$ 1,200,000
Asthma, Emerg. Rm. Visits	Stanford, et al. (1999)	400	$ 150,000
Work Loss Days	US Bureau of Labor Statistics (BLS, 2007)	174,000	$31,000,000
Minor Restricted Activity Days	IEc (1993)[i] and Tolley, et al. (1986)	1,000,000	$64,000,000
Total Costs			$190,000,000

Rounded to two significant figures

* Hospital admissions

†The above table combines incidence estimates from studies of similar disease and non-overlapping age groups. Because the combined estimates of incidence listed above were derived using pre-rounded numbers, the number of incidents may vary slightly from those listed in the previous table.

Appendices

Figures

BenMAP uses a 12-kilometer grid scale to represent spatial information. The following figures show population, estimated annual fine particle concentrations and the estimated number of annual deaths attributable to fine particles within the state.

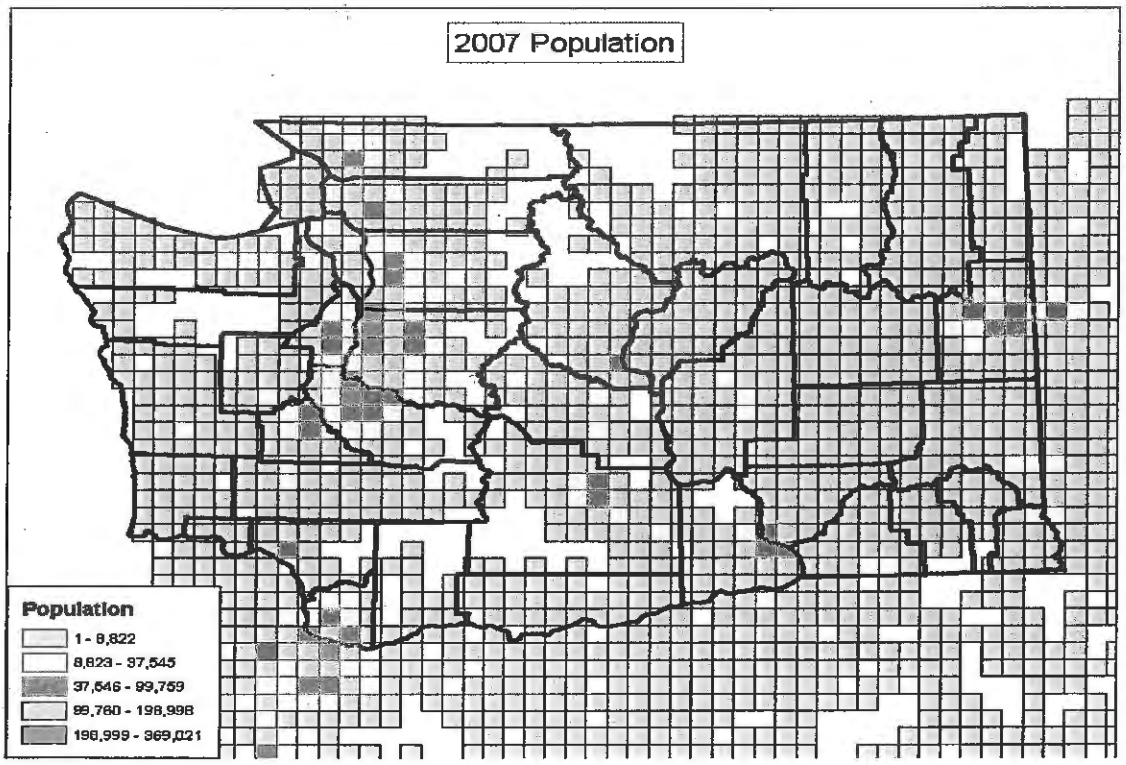


Figure 1: Estimated 2007 Washington State population (data from Washington State Office of Financial Management forecasts)

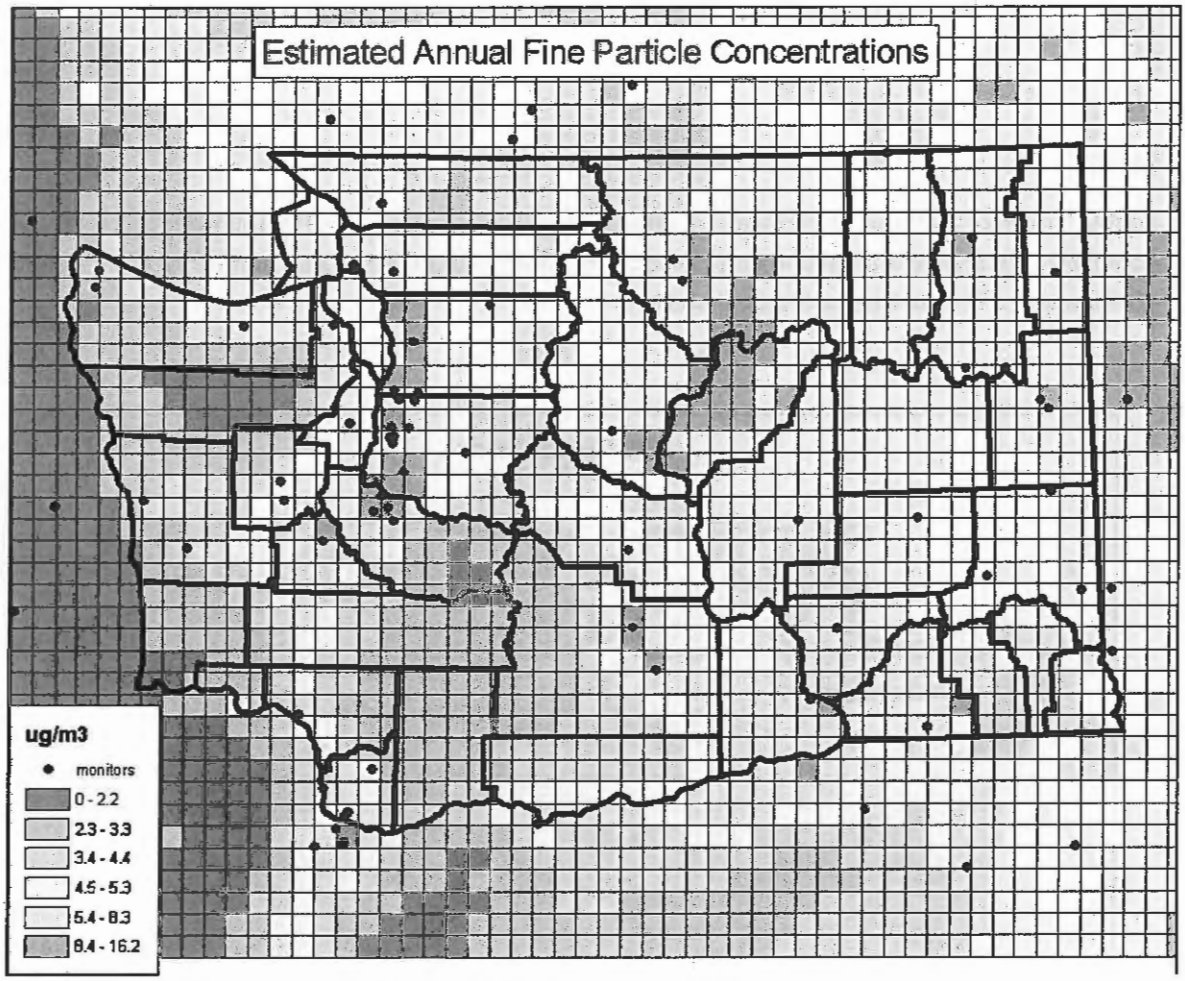


Figure 2: Estimated annual fine particle concentrations (average of 2004-2008 monitored data daily medians scaled with 2006-2008 modeled data daily medians)

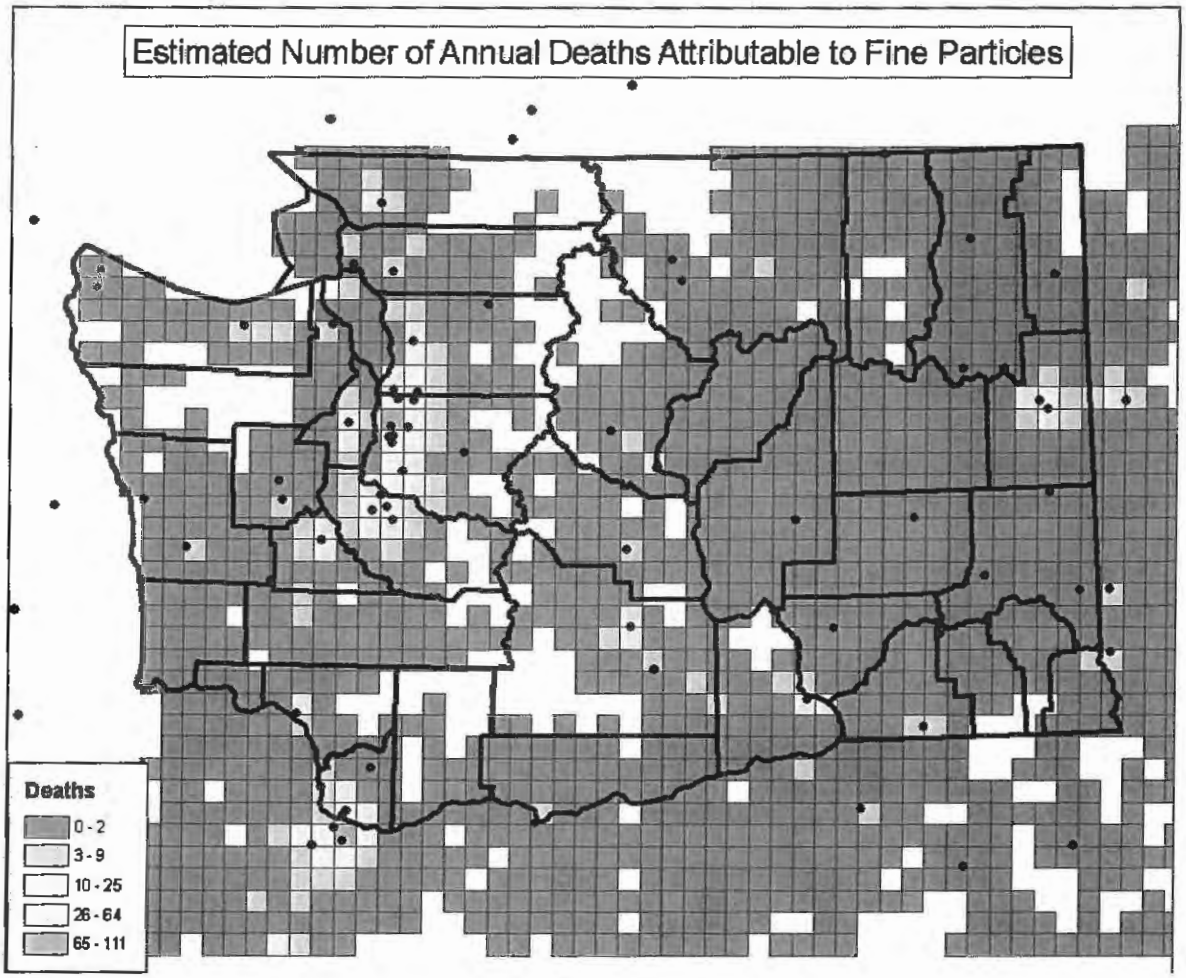


Figure 3: Estimated number of annual deaths of people between the ages of 30 and 99 attributable to fine particle pollution (Pope, et al. 2002)

For more information

On BenMAP:
Go to http://www.epa.gov/air/benmap/

On health effects studies:
For descriptions of individual studies contained in BenMAP, see BenMAP User Manual Appendices E & F. The User Manual Appendices is at:
http://www.epa.gov/air/benmap/models/BenMAPappendicesSept08.pdf

Other studies include:

"Long-Term Ozone Exposure and Mortality," Dr. Michael Jerrett et al., New England Journal of Medicine, March 12, 2009.

"Extended Follow-up and Spatial Analysis of the of the American Cancer Society Study Linking Particulate Air Pollution and Mortality," Dr. Daniel Krewski et al., Health Effects Institute, June 2009

On economic studies and evaluation:

Please request a copy of Ecology's Technical Report for details on the economic analyses by emailing Ecology at BenMap@ecy.wa.gov.

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Contact us

If you have comments or questions about this report, please contact our technical team through the following e-mail address. Your thoughts or questions will be forwarded to the appropriate staff for response. Thank you. E-mail: BenMap@ecy.wa.gov

If you need this publication in another format, please contact the Air Quality Program at (360) 407-6800. If you have a hearing loss, call 711 for Washington Relay Service. If you have a speech disability, call 877-833-6341.

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[i] Industrial Economics Incorporated (IEc) (1993). Memorandum to Jim DeMocker, US EPA, Office of Air and Radiation, Office of Policy Analysis and Review. 30 September.

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Isocyanic acid in the atmosphere and its possible link to smoke-related health effects

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Isocyanic acid & atherosclerosis, cataracts and Rheumatoid Arthritis

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Abstract

We measured isocyanic acid (HNCO) in laboratory biomass fires at levels up to 600 parts per billion by volume (ppbv), demonstrating that it has a significant source from pyrolysis/combustion of biomass. We also measured HNCO at mixing ratios up to 200 pptv (parts-per-trillion by volume) in ambient air in urban Los Angeles, CA, and in Boulder, CO, during the recent 2010 Fourmile Canyon fire. Further, our measurements of aqueous solubility show that HNCO is highly soluble, as it dissociates at physiological pH. Exposure levels > 1 ppbv provide a direct source of isocyanic acid and cyanate ion (NCO^-) to humans at levels that have recognized health effects: atherosclerosis, cataracts, and rheumatoid arthritis, through the mechanism of protein carbamylation. In addition to the wildland fire and urban sources, we observed HNCO in tobacco smoke. HNCO has been reported from the low-temperature combustion of coal, and as a by-product of urea-selective catalytic reduction (SCR) systems that are being phased-in to control on-road diesel NO_x emissions in the United States and the European Union. Given the current levels of exposure in populations that burn biomass or use tobacco, the expected growth in biomass burning emissions with warmer, drier regional climates, and planned increase in diesel SCR controls, it is imperative that we understand the extent and effects of this HNCO exposure.

troposphere heterogeneous chemistry

Every day billions of people are exposed to smoke: from tobacco, from biomass, or low-temperature coal combustion used for cooking and heating, and from wildfires (1, 2). Extensive research on the pyrolysis of biomaterials has shown that numerous volatile and semivolatile organic compounds are produced (3–5). It is important to understand the impacts of these emissions from the global scale down to the personal level. Significant human health effects have been associated with smoke exposure, including cataracts, cardiovascular impairment, and chronic diseases such as rheumatoid arthritis (2, 6). A number of studies have shown that protein carbamylation and associated inflammatory response is a common biochemical pathway causing these effects (7–9). In vivo isocyanic acid (HNCO, H–N=C=O) and its aqueous anion, cyanate (NCO^-), have been identified as biochemical intermediates in this protein carbamylation (8). In this work we show that smoke from biomass, including tobacco, contains HNCO at concentrations that cause carbamylation at physiologically significant levels. Thus smoke-related HNCO exposure is strongly linked to several classes of major negative health effects.

Isocyanic acid has been known since Liebig and Wöhler (10), but it has not previously been measured in the atmosphere. The compound is moderately acidic ($pK_a = 3.7$) and unstable in pure form as it readily polymerizes (11). However, it is volatile (BP = 23.5 °C estimated) and relatively stable at dilute (several ppmv) concentrations in the gas-phase (12). Isocyanates are toxic at high concentrations, as was demonstrated after the accidental release of methyl isocyanate, CH_3NCO, in Bhopal, India, when thousands of people suffered injury and death (13). The work-place exposure to isocyanates is of concern and has been linked to a number of health effects. Consequently, quite low limits for occupational exposure, 0.5 ppbv for methyl isocyanate, and 5 ppbv for total isocyanates have been established in some jurisdictions (14, 15).

We have recently developed a negative-ion proton-transfer chemical ionization mass spectrometer (NI-PT-CIMS) for sensitive [5 pptv (parts-per-trillion by volume) detection limit] and fast response (1 sec) measurement of HNCO and other acids in air (12) (see *Materials and Methods* below). The NI-PT-CIMS was used to measure HNCO in the emissions from laboratory biomass fires (4, 12, 16), in the Los Angeles (LA) urban area during May and June, 2010 (17), and during a period when Boulder, CO was impacted by emissions from the 2010 Fourmile Canyon wildfire. In addition we measured the Henry's Law solubility of HNCO at pH = 3, and using the expression for solubility of a weak acid, estimate HNCO to be highly soluble at physiologic conditions, pH = 7.4. Finally we show that the HNCO levels observed in our combustion/pyrolysis measurements, combined with its solubility and reported in vitro biochemical studies, imply that HNCO makes a significant contribution to smoke-related health impacts that are a major societal concern.

Results and Discussion

Sources. An example emission-time profile from measurements made at the US Forest Service Fire Sciences Laboratory in Missoula, MT (hereafter referred to as the Firelab), Fig. 1*A*, shows that HNCO and CO are highly correlated during flaming combustion with an HNCO/CO ratio between 0.1% and 0.6%. Smoldering combustion usually produced a second peak of CO emissions, with values of HNCO/CO that were factors of 5–10 lower. The two emission regimes are shown roughly as colored regions in Fig. 1*B* along with data from LA and the Fourmile Canyon wildfire. HNCO had an ambient "background" concentration, on the order of 10 pptv or less, and ranged up to 100 pptv at the LA site, and up to 200 pptv during the Boulder measurements. The laboratory biomass burning smoke was measured close to the source hence had much higher levels of HNCO and CO with up to 600 ppbv of HNCO. Laboratory biomass burning ratios of HNCO to other well known CN-containing biomass burning marker species ranged from 1 to 1.6 for HNCO/acetonitrile (CH_3CN), and from 0.33 to 0.5 for HNCO/hydrogen cyanide (HCN) with one fuel as low as 0.16 (4).

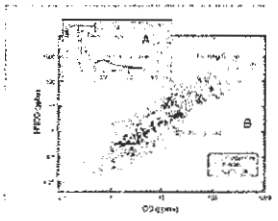

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Fig. 1. (*A*) Time line for HNCO and CO emissions from a laboratory burn of California sage brush. (*B*) Measured HNCO vs. CO for the Fire Lab emissions measurements (red circles), CalNex LA ground site (black diamonds), and the Boulder Fourmile Canyon fire (blue triangles), with the general flaming stage and smoldering stage relationships highlighted.

Other laboratory studies have reported HNCO in concentrated emissions from pyrolysis and low-temperature combustion of biomass (18) and coal (19), and pyrolysis of tobacco ingredients (20). HNCO has also been observed as a by-product of urea-selective catalytic reduction systems that are being instituted to control NO_x (nitric oxide and nitrogen dioxide) emissions from diesel engines (21). A number of studies show that biomass combustion and pyrolysis produce HNCO from amide or polyamide functionalities (18, 22, 23) {-HN-C(O)-}. Reduced nitrogen coproducts include HCN, CH_3CN, and NH_3, and the oxidized nitrogen coproducts include N_2O, NO, HNO_2, NO_2, and HNO_3. The temperatures involved in natural convection combustion and pyrolysis are low enough that NO_x is not formed from N_2 and O_2, so the emitted nitrogen comes solely from the fuel (24).

Coal is a common fuel used directly for cooking and heating especially in rural areas of developing countries (25). Low-temperature combustion of coal having sufficient nitrogen, has been shown to be a source of HNCO (19). In this case the precursors are likely to be nitrogen heterocyclic compounds, although specific precursors have not been identified. Further work on the combustion of coal char at 600 °C measured the HNCO emission as 12 ± 4.5% of original fuel nitrogen (26).

Tobacco smoking presents an obvious source because, in this case, HNCO is produced from both the plant material, mostly from proteins (polyamides) (18) as well as from pyrolysis of urea, an additive in some cigarettes:

$$H_2N\text{-}C(O)\text{-}NH_2 \Rightarrow HNCO + NH_3. \quad [1]$$

Apparently there are no reports of HNCO measurements in actual tobacco smoke, but a surrogate pyrolysis study found that 93% of added urea (4 mg/g tobacco) was pyrolyzed to HNCO, resulting in an estimated emission rate of 1.9 mg/g tobacco smoked (20). The amount of HNCO delivered to the average smoker, from this source alone, can be estimated using the parameters given in Baker and Bishop (20). If the lower ranges of values are assumed for: cigarette size (0.7 g), fraction of tobacco burned in puffing (0.3), fraction of HNCO transferred to mainstream smoke (0.3), and the fraction transmitted through the filter (0.3), then a smoker consumes 36 μg (filtered) to 108 μg (unfiltered) of HNCO per cigarette. The mixing ratio of HNCO in mainstream cigarette smoke can be estimated from these factors and an average volume of mainstream smoke of 470 mL (27), to be 40 to 140 ppmv. We observed HNCO in cigarette smoke in a brief laboratory test, however the levels were too high for us to quantify with the CIMS instrument configured in the ambient measurement mode. A more detailed study of tobacco smoke was beyond the scope of the current work. We conclude that tobacco-derived HNCO needs to be measured more extensively and potential exposure to it quantified. This measurement is especially important because HNCO is not currently included in the FDA "Proposed Initial List of Harmful/Potentially Harmful Constituents in Tobacco Products, including Tobacco Smoke" (28).

Diesel urea-selective catalytic reduction (SCR) exhaust systems represent a source of emerging interest, because HNCO is a recognized intermediate in this chemistry. These systems work by injecting a small flow (1–3% by volume urea/fuel ratios) of urea solution (32% by weight) into a catalyst system. Conditions and materials are optimized to induce not just reaction [1], but also provide for the complete catalytic hydrolysis of HNCO:

$$HNCO + H_2O \Rightarrow NH_3 + CO_2. \quad [2]$$

While extensive measurements of HNCO emissions from actual working diesel systems are lacking, there are reports of HNCO emissions from model systems that show that up to 5–10% of injected urea N is measured in exhaust as HNCO, mostly at lower temperatures and with older catalysts (> 1,000 h operation). The observed HNCO mixing ratios in exhaust streams ranged up to 50 ppmv (21). Understanding this source must be given a high priority considering the expected growth in SCR-controlled diesel engines in the European Union (EU) and United States.

Ambient Measurements. The impact of HNCO emitted by a wildfire on the ambient air of an urban area can be seen in the close correlation of the HNCO and CO levels we measured in Boulder, Colorado during the recent 2010 Fourmile Canyon fire (Fig. 2*A*). In this case there is essentially minute-by-minute correlation in the levels of the two species. The ratio of HNCO/CO during the three enhancement events is consistent with smoldering in the laboratory biomass fires (Fig. 1*B*). Concurrent measurements of CH_3CN made by gas chromatography/mass spectrometry (GC/MS) (see *Materials and Methods* below), are also shown in Fig. 2*A*, and also show close correlation with HNCO and CO, given that the GC/MS sampling time was the first 5 min of each $\frac{1}{2}$ h period. The HNCO/CH_3CN ratios in these measurements were quite a bit lower than those observed in the laboratory burns described above. This lower ratio implies either very different source ratios for the fuels involved in this fire, or a much shorter lifetime for HNCO.

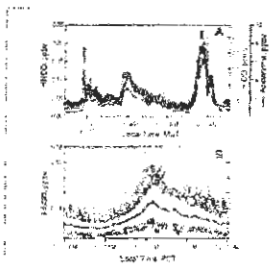

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Fig. 2. (*A*) Mixing ratios of HNCO, CO, and acetonitrile from measurements made in Boulder, CO. during the 2010 Fourmile Canyon fire, with HNCO 30-s averages shown in green circles, CO 1-min averages shown by the red line, and CH_3CN shown as blue bars representing the 5-min sample time. (*B*) shows the individual 20-s HNCO measurements (blue dots), and the 5-min averaged HNCO (blue line) and ± 1σ (light blue) vs. time of day for the measurements at the CalNex LA ground site.

There are no other reports of measurements of HNCO in the ambient atmosphere with which to compare, but we can contrast our HNCO observations with those of other reduced nitrogen species known to have strong biomass burning sources: CH_3CN and HCN (29). The lowest HNCO values we observe were below 10 pptv, much lower than corresponding background levels of HCN and CH_3CN, which are in the range of 50 to 100 pptv. Because biomass burning is a far larger source of trace gases than diesel engines or tobacco smoke (5), it is almost certainly the largest global source of HNCO, thus the disparity in background values implies a shorter lifetime for HNCO relative to the other species.

A clear diurnal variation in HNCO was observed in the LA basin (Fig. 2*B*) at the Pasadena ground site during the CalNex 2010, a study with combined air quality and climate goals (30). Wildfires were not observed during this period and are therefore not responsible for the observed HNCO. As the diurnal variation of HNCO coincides with those of other photochemical products, such as ozone and oxygenated VOCs, which peaked midday when processed air from the downtown LA area arrived at the site, we suggest that HNCO in LA is also formed photo-chemically, potentially from monomethyl amine, formamide, and acetamide (31, 32) a source that has not been previously considered in urban areas. Moreover, a surface or vehicle source of HNCO would show low midday values when the boundary layer mixing is maximized and higher values during rush hours, particularly in the morning. Our LA measurements give a baseline urban value, in the absence of local wildfires or a possible future increase from vehicle sources.

Atmospheric Removal Processes. Hydroxyl radicals are generally responsible for removing trace organic species from the atmosphere (33). HNCO is relatively stable against reaction with OH radical [$k \approx 10^{-15}$ cm^3/molec-s extrapolated from high temperature data (34)], yielding an atmospheric lifetime of several decades for this process. It is possible that there are mechanisms, such as adduct formation, that are important at low temperatures, but we are not aware of any studies of that phenomenon. HNCO is expected to have a very low absorption at near-UV to visible wavelengths that constitute the solar actinic region based on measurements at wavelengths shorter than 280 nm (35, 36). The major dissociation channel (HNCO ⇒ H + NCO) has a threshold of 261 nm which corresponds to a bond energy D_0(H-NCO) = 109.6 ± 0.4 kcal/mole (37), and a channel that forms triplet NH has a 332 nm threshold (38), however the absorption cross-section in that region is quite low. These absorption features result in an HNCO lifetime against photolysis of months.

The major loss processes of HNCO in the lower troposphere are likely the heterogeneous uptake to aerosols or liquid water (fog, clouds, precipitation, and the ocean) and subsequent reactions. HNCO is a moderately weak acid in aqueous solution ($pK_a = 3.7$) (11) and exhibits relatively slow hydrolysis that is pH-dependent (39). The partitioning of HNCO to aqueous solution at low-concentration, i.e., Henry's Law solubility, H (M/atm), has apparently not been measured previously. We have measured H for isocyanic acid in an aqueous buffer at pH = 3.0 ± 0.1, and room temperature ($t = 25 \pm 1$ °C). The decrease in gas-phase HNCO, exiting a saturated liquid sample, was measured for a range of volume flow-rate to liquid volumes (Fig. 3*A*). For a system in which the mass transfer between the liquid and gas phases is rapid, the following relationship holds (40):

$$\ln(C_t/C_0) = -[\varphi/(\mathrm{HRTV}) + k_l]t. \quad [3]$$

Where C_t/C_o is the relative concentration in the gas phase exiting the reactor, φ is the volumetric flow rate, V is the liquid volume, R is the ideal gas constant, T is temperature, k_l is the first order loss rate in solution, and t is the time. The slopes of these exponential curves vs. the flow-rate/liq.-volume yielded the Henry's Coefficient (Fig. 3*B*). The resulting value is given in Table 1 and is commensurate with Henry's Coefficients of some related species (41). HNCO is only slightly soluble at pH = 3, however, it is a weak acid, $pK_a = 3.7 \pm 0.2$, hence its effective Henry's Constant can be calculated as a function of pH with the following relationship (42):

$$H_{\mathrm{eff}} = H^*(1 + K_a/[H^+]), \quad [4]$$

where H^* is the intrinsic Henry's Coefficient independent of any liquid-phase equilibria. This relationship is plotted in Fig. 4 for HNCO using H^* derived from the H_{eff} measured at pH = 3, and the known pK_a.

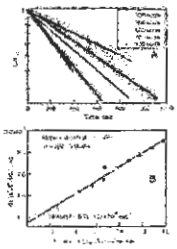

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Fig. 3. (*A*) shows the concentration decay curves data for some of the individual equilibration experiments at the flow rates shown, and (*B*) shows the resulting fit to Eq. 3 and the points for the individual experiments from (*A*) along with points from additional experiments (solid dots), which are not shown in (*A*) for the sake of clarity.

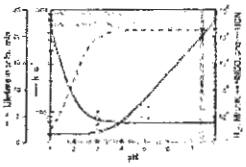

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Fig. 4. The Henry's Law constant (blue) from our measurement at pH = 3 and the weak acid equilibrium relationship Eq. 4, first-order loss rate due to hydrolysis from the rate constants measured by Jensen (39) (solid red), and corresponding aqueous phase lifetime (dashed red) of HNCO vs. pH. The open red circle is our measurement of the first-order loss rate at pH = 3 (± 1σ) and the open blue square is our measurement of H_{eff} at pH = 3. Also shown is the Henry's Law constant for HCN (green). The yellow band indicates the range of pHs most characteristic of ambient aerosol, and the pink band indicates physiological pH, and the error bar at pH = 7.4, is the estimated uncertainty based on the uncertainties in H_{eff} measured at pH = 3, (± 3 M/atm) and, pK_a (± 0.2 pH units).

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Table 1. Henry's Law constants of HNCO and related compounds

The rate of hydrolysis of HNCO has been measured as a function of pH and found to have several mechanisms, one set that is direct (i.e., first order), and one that is acid-catalyzed (39):

$$\mathrm{HNCO} + \mathrm{H_2O} \Rightarrow \mathrm{NH_3} + \mathrm{CO_2} \quad [5a]$$

and

$$\mathrm{NCO^-} + 2\mathrm{H_2O} \Rightarrow \mathrm{NH_3} + \mathrm{HCO_3^-} \quad [5b]$$

$$k^{\mathrm{I}} = 7.8 \times 10^{-4}\ \mathrm{sec^{-1}}$$

$$\mathrm{HNCO} + \mathrm{H_3O^+} \Rightarrow \mathrm{NH_4^+} + \mathrm{CO_2} \quad [6]$$

$$k^{\mathrm{II}} = 6.0 \times 10^{-2}\ \mathrm{M^{-1}\ sec^{-1}}.$$

This hydrolysis rate is also shown in Fig. 4 as a function of pH. A point of comparison is available from the solubility experiment described above, in which $8.7 \pm 1.3 \times 10^{-4}$ $\mathrm{sec^{-1}}$ was measured, a value that is in good agreement with the previous measurement (39). HNCO hydrolyzes slowly enough ($t \approx 20$ min) at physiological pHs that there is ample time for carbamylation chemistry, but just fast enough that tradition analytical methods, e.g. aqueous acid/base extractions etc., will underestimate its concentration. While detailed kinetic parameters are lacking, there are a number of studies that show that carbamylation is rapid relative to hydrolysis (8, 43, 44).

Given the above solubility data and the absence of fast gas-phase loss processes, uptake of HNCO on aerosol and cloud droplets, or natural surfaces, will likely be the relevant loss process. The rate of this process can be estimated from knowledge of the Henry's Law solubility and liquid-phase reaction rate. The loss of a reactive species to a liquid surface of an aerosol particle or droplet can be thought of as a network of resistances, in series and in parallel, that represent diffusion and reaction processes (45). Often one process is limiting, simplifying the representation. The time scale for gas-phase diffusion to particles is on the order of seconds to a few minutes depending on particle size and number. The time scales for loss of HNCO in liquid aerosol or cloud droplets are much longer and can be estimated by the following equation (46);

$$t = 1/HRTF_l k_{\mathrm{hyd}}. \quad [7]$$

where F_l is the volume fraction of liquid water, which ranges from 10^{-12} for an aerosol of total surface area of 200 $\mu m^2/cm^3$, and 0.2 μm mean diameter, and up to 10^{-6} for fogs or clouds, and k_{hyd} is k^l. The estimated atmospheric lifetimes for HNCO then range from > 10^4 years for reaction on an atmospheric aerosol at pH 3, to approximately 0.5 d for reaction in cloud or fog water of pH 5.5 and the above liquid water content. Clearly, the lifetime of HNCO against uptake on aerosols and clouds is limited by liquid-phase reactions.

Slightly different considerations govern the uptake of HNCO on ground or natural water surfaces (lakes, oceans). In these cases it can be assumed that the solubility of HNCO is the dominant factor. Because those surfaces are essentially at neutral pH or above, HNCO should behave similarly to other highly soluble species, such as nitric acid (HNO_3). Nitric acid has lifetimes against deposition in the planetary boundary layer in the range of an hour to half a day (33). A comparison of the above lifetimes for HNCO can be made to the other biomass-derived CN compounds, HCN, and CH_3CN, which are not very soluble at environmental pHs, and have lifetimes of 5 mo (HCN), and 6.6 mo (CH_3CN) against loss, mainly due to deposition to the ocean (29).

Potential Health Impacts. The chronic and acute health effects of smoke are well documented (2, 6, 8, 47), however detailed causal biochemical pathways are not completely understood and are the subject of current research. The eyes, respiratory, and cardiovascular systems are the areas of the human body that show chronic effects from smoke exposure (2). The potential for health impacts due to HNCO in smoke can be traced to two features of its chemistry: high solubility at physiologic pH as noted above, and the reaction of HNCO with amine, hydroxyl, and sulfhydryl groups, by addition across the N-C bond, to form a carbamyl group, $-H_2NC(O)-$, in a process termed carbamylation (43):

$$HNCO + H_2N\text{-}R \Rightarrow H_2N\text{-}C(O)\text{-}NH\text{-}R \quad [8]$$

$$HNCO + HOR \Rightarrow H_2N\text{-}C(O)\text{-}O\text{-}R \quad [9]$$

$$HNCO + HSR \Rightarrow H_2N\text{-}C(O)\text{-}S\text{-}R. \quad [10]$$

Recent work has shown that carbamylation of proteins is a key step in the inflammatory response that links smoking to cardiovascular disease (8) and rheumatoid arthritis (6), and the link between carbamylation and cataracts has been recognized for some time (7). Wang et al. (8) identified isocyanic acid/cyanate ion as a key intermediate in this reaction. However, the source of cyanate in this chemistry was postulated to be the enzymatic oxidation of thiocyanate (NCS^-) by hydrogen peroxide. Our work shows that smoke provides a route to uptake and absorption of HNCO into the blood stream that will drive protein carbamylation directly. The effective Henry's Law solubility of 10^3 M/atm at pH 7.4 means that a 1 ppbv (10^{-9} atm) mixing ratio in inhaled breath will produce an equilibrium aqueous concentration of 100 μM, a concentration that mimics carbamylation in vitro (8). The transport of HNCO into the blood stream and into the sensitive tissues of the eye depends on membrane transport, the precise aspects depending on its pK_a and oil/water partition coefficient (48), which is apparently not known for HNCO. However, we would expect HNCO to have similar behavior to formic acid, because of its similar size, polarity, and acidity ($pK_a = 3.75$), which has moderate permeability in lipid bilayer membranes (49). As a result, transport of HNCO should be rapid enough that solution concentrations will be close to those calculated from Henry's Law equilibrium.

Conclusions. Our work shows that there is potential for significant exposure of humans to HNCO as a consequence of biomass burning, biofuel usage, cooking, and tobacco usage, and perhaps by the use of new diesel SCR emission control systems. Studies of indoor CO in rural areas of China, where biomass or coal is burned in open indoor fires for cooking and heating, report average concentrations in the range 4–12 ppmv (25). If our data from Fig. 1 are typical of biomass cooking fuels, then HNCO levels up to 10 ppbv or higher could exist in those homes and result in blood NCO^- levels far in excess of those shown to produce protein carbamylation. The tobacco source of HNCO has apparently not been quantified directly, but pyrolysis of urea, (a major tobacco additive) produces HNCO directly (20), implying that this source could be considerable. HNCO mixing ratios in the 1 ppbv range were not measured in urban areas, but the possibility that new diesel SCR sources could increase ambient HNCO is a real concern. Wildfire impacts on populated areas and firefighters could also be significant based on our Firelab measurements (Fig. 1*B*).

These results suggest several issues that require further study. Accurate HNCO emission factors are needed for the different biofuels and coals used for cooking, and the fuels that burn in wildfires and tropical deforestation. Emission ratios for diesel SCR vehicles need to be determined. Human exposure to HNCO needs to be studied in depth, including lung and membrane transport, and their relation to blood and tissue levels of NCO^-. Increased biomass burning emissions are expected with warmer, drier regional climates (50), and diesel SCR controls are being phased-in by the EU and some states in the United States. Extensive source characterization and targeted toxicological and exposure studies are needed to better understand and mitigate this potentially harmful HNCO exposure.

Materials and Methods

Acid CIMS Instrument. Our HNCO method is described by Veres et al. (51) and Roberts et al. (12) and involves [1] selective ionization of HNCO by reaction with acetate ion via proton transfer in the gas phase and [2] detection of the NCO^- ion at 42 amu by a quadrupole mass spectrometer. Contributions to mass 42 from other compounds such as HN_3 and other H C N O isomers, were considered and rejected for reasons discussed by Roberts et al. (12). The possibility that mass 42 is $C_3H_6^-$ produced by electron transfer from O_2^- can be ruled out by the fact that O_2^- had at most a 0.1% abundance relative to acetate ions in our Firelab and ambient studies, so even if the reaction occurred at the gas kinetic limit, sensitivity to propene would be about 0.3%, because acetate-acid reactions occur at about 1/3 the gas kinetic limit (16). The detection limit of this measurement is 5 pptv for a 1 sec measurement, and the uncertainties are ± (25%+5 pptv) for ambient measurements. Biomass burning emissions measurements were conducted at the US Department of Agriculture, Fire Science Laboratory in Missoula, MT (4). The LA measurements were made at the CalNex-LA Pasadena ground site (34.140582 N, 118.122455 W) (30), and the Boulder measurements were made right outside the NOAA/ESRL laboratory (39.991431 N 105.261032 W). The inlet time constant was a few seconds in the case of LA and Firelab measurements and was estimated to be 10 to 15 s during the Boulder Fourmile Canyon measurements.

HNCO Calibration. Calibration of the HNCO signal was accomplished by FTIR measurement of a gas-phase diffusion source of HNCO. A gas stream of HNCO at mixing ratios in the range of 1–2 ppmv in 50 sccm (standard cubic centimeters per minute) zero air was produced by the thermal decomposition (210°–230 °C) of cyanuric acid, the trimer of HNCO. Limiting the HNCO mixing ratio to a few ppmv was found to be essential to avoid polymerization as the gas stream cooled to room temperature down stream of the source. The concentration of HNCO in the diffusion source was measured by FTIR in a room pressure (620 Torr) multipass cell (4.8 m length). The standard HNCO absorption cross-section from the database described by Sharpe et al. (52) and Johnson et al. (53) was used to calculate mixing ratios. The HNCO source was diluted in several stages by larger flows of zero air or nitrogen to the ranges appropriate for the Firelab study and the LA study.

Acetonitrile Measurement. The gas chromatographic/mass spectrometric method used for the measurement of acetonitrile in Boulder during the Fourmile Canyon fire, was described by Gilman et al. (54). The instrument is a custom built two channel GC coupled to an Agilent 5973 quadrupole mass spectrometer. Air was sampled from the west side of the laboratory building at 7 L/ min through a short (several meter) Teflon PFA sample tube. A small subflow of 70 sccm was sampled off of this flow for 5 min through a series of traps designed to reduce water, carbon dioxide, and O_3 interferences (55). Acetonitrile was separated on a semipolar (Restek-MXT-624) capillary column [20 m × 0.18 mmID (inner diameter)] and detected at *m/z* 41. The GC separation required 25 min, permitting a 5 min sample to be analyzed every 30 min. Calibrations were accomplished by dynamic dilution of gravimetrically prepared gas-phase standards.

Henry's Constant Measurement. Henry's Law describes the equilibration between a gas-phase chemical species and a liquid-phase, at infinite dilution. At equilibrium, the partial pressure of a species in the gas phase is proportional to its concentration in the liquid phase:

$$C_l = \mathbf{H} \times C_g, \quad [11]$$

where the Henry's Law constant, H is typically given in units of M/atm. Measurement of H is relatively simple if the compound of interest is stable in both phases. However, HNCO hydrolyzes at appreciable rates in a pH-dependent way. Therefore a dynamic method was used in this work, following the description by Kames and Schurath (40), and utilizing the apparatus described by Roberts (56), and the HNCO sources described by Roberts et al. (12). The apparatus consisted of two fritted bubblers (Aldrich 250 mL Gas Reducing Flask) placed in series: the first bubbler contained de-ionized (DI) water to humidify the gas stream, and the second contained the sample of DI water buffered to pH = 3 ± 0.1 with a citric

acid/NaOH/NaCl buffer (Fluka Chemicals), through which a flow of zero air, in the flow range 100 to 1,000 sccm, was directed. The HNCO source was connected to the gas stream in between the two bubblers by means of a 3-way valve, so that the source could be directed into the bubbler stream, or to vent, without perturbing the main flow. The outlet of the bubbler stream was teed into the inlet of the NI-PT-CIMS so that the pressure at the outlet remained at room pressure. Equilibration experiments were conducted by placing a sample of pH = 3.0 solution (25 ± 0.25 mL) into the bubbler and measuring the HNCO at the outlet at a series of flow rates, as the HNCO source was switched in-line and equilibrated with the solution, and then switched out of line and observed to decay due to a combination of hydrolysis and loss to the gas phase. The exponential decays were then fit to the relationship given in Eq. 3, as shown in Fig. 3. Measurement of H at physiologic pH is not possible with this method as it is too high to yield decay curves on a meaningful time scale, rather we rely on the well demonstrated relationship in Eq. 4 to calculate H at pH = 7.4.

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Footnotes

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Author contributions: J.M.R., R.J.Y., and J.d.G. designed research; J.M.R., P.R.V., A.K.C., C.W., I.R.B., B.L., J.B.G., and W.C.K. performed research; J.M.R., P.R.V., A.K.C., C.W., I.R.B., B.L., and J.B.G. analyzed data; and J.M.R., R.F., and J.d.G. wrote the paper.

The authors declare no conflict of interest.

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Gene Altering Chemicals Found In Forest Fire Smoke

Date:
May 2, 2009
Source:
DOE/Pacific Northwest National Laboratory
Summary:
Researchers have detected common plant toxins that affect human health and ecosystems in smoke from forest fires. The results from the new study also suggest that smoldering fires may produce more toxins than wildfires - a reason to keep human exposures to a minimum during controlled burns. Finding these toxins -- known as alkaloids -- helps researchers understand how they cycle through earth and air.

Smoldering ponderosa pine fires contain alkaloids.
Credit: Image courtesy of DOE/Pacific Northwest National Laboratory
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Researchers have detected common plant toxins that affect human health and ecosystems in smoke from forest fires. The results from the new study also suggest that smoldering fires may produce more toxins than wildfires - a reason to keep human exposures to a minimum during controlled burns.

Finding these toxins -- known as alkaloids -- helps researchers understand how they cycle through earth and air. Smoke-related alkaloids in the environment can change aquatic and terrestrial ecosystems, as well as where and when clouds form. The study, which was of Ponderosa pines, by scientists at the Department of Energy's Pacific Northwest National Laboratory will appear June 1 in *Environmental Science and Technology.*

"Ponderosa pines are widespread in areas that are prone to forest fires," said PNNL physical chemist Julia Laskin, one of the coauthors. "This study shows us which molecules are in smoke so we can better understand smoke's environmental impact."

As trees and underbrush burn, billowing smoke made up of tiny particles drifts away. The tiny particles contain a variety of natural compounds released from the plant matter. Researchers have long suspected the presence of alkaloids in smoke or detected them in air during fire season, but no one had directly measured them coming off a fire. The PNNL researchers had recently developed the technology to pick out alkaloids from the background of similar molecules.

To investigate chemicals given off by fires, the team captured some smoke from test fires organized by Colorado State University researchers. These researchers were doing controlled burns of ponderosa pines, underbrush and other fuels at the Forest Service Fire Science Laboratory in Missoula, Mont.