# Summary of Results for the 2011 National-Scale Assessment

#### INTRODUCTION

NATA is a prioritization tool. Its purpose is to identify geographic areas, pollutants and emission sources that should be evaluated further to gain a better understanding of risks. EPA uses NATA in many ways, including:

- To set priorities for improving data in emissions inventories
- To work with communities in designing their own local-scale assessments, and
- To help direct priorities for expanding and improving air toxics monitoring.

NATA helps state, local and tribal air agencies focus resources on geographic areas, pollutants and types of emission sources for closer investigation. Once risks are further characterized, agencies can determine steps to reduce air toxics emissions where necessary. NATA provides broad estimates of risk over geographic areas of the country and not definitive risks to specific individuals. This is because NATA uses models to estimate risks; it is not designed to determine actual risks. NATA is designed to prioritize pollutants and areas for further study, not to compare one area of the country's risk to another. This is because the emissions data underlying the assessment can vary in level of detail from state to state.

Of the 180 air toxics plus diesel PM included in the 2011 national-scale assessment, the risk characterization considers the risk of both cancer and noncancer effects from inhalation of 138 of these air toxics -- the subset of pollutants with health data based on chronic exposure. The purpose of this national-scale assessment is to understand these cancer risks and noncancer health effects in order to help the EPA and others to identify pollutants and source categories of greatest potential concern, and to set priorities for the collection of additional information to improve future assessments. The assessment represents a "snapshot" in time for characterizing risks from exposure to air toxics. The national-scale assessment is not designed to characterize risks sufficiently for it to be the sole source for regulatory action.

The 2011 national-scale risk assessment is based on a 2011 inventory of air toxics emissions (the most complete and up-to-date available). It then assumes individuals spend their entire lifetimes exposed to these air toxics. Therefore, it does not account for the reductions in emissions that have occurred since 2011 or those that will happen in the near future due to regulations for mobile and industrial sources. This risk assessment represents an update and enhancement to EPA's 2005 national-scale assessment. The next assessment will focus on emissions for the year 2014.

Note that in this assessment, the potential carcinogenic risk from diesel PM is not addressed because there currently is no unit risk estimate available. However, there are noncancer results. Learn more about EPA's qualitative assessment of diesel PM.

Given its broad scope, this risk characterization is subject to a number of limitations due to gaps in data or in the state of the science for assessing risk. For example, the current assessment does not include results for dioxins, compounds that may contribute substantially to risks. In addition, the EPA is reassessing the health effects of many pollutants considered in this study. A status report for all EPA health effect assessments is available from EPA's <a href="Integrated Risk Information System">Integrated Risk Information System</a> (IRIS). For more details on the limitations of the 2011 NATA, refer to the <a href="Results">Results</a> section on the NATA Web site.

The risk characterization, which was limited to inhalation risk from outdoor sources, was designed to answer the following questions:

- 1. Which air toxics pose the greatest potential risk of cancer or adverse noncancer effects across the entire United States?
- 2. Which air toxics pose the greatest potential risk of cancer or adverse noncancer effects in some areas of the United States?
- 3. Which air toxics pose lesser, but still significant, potential risk of cancer or adverse noncancer effects across the entire United States?
- 4. When risks from all air toxics are combined, how many people have the potential for an upper-bound lifetime cancer risk greater than 10-in-1 million?
- 5. When potential adverse respiratory or neurological effects from all air toxics are combined, how many people have the potential for exposures that exceed reference levels intended to protect against adverse effects, i.e., a target organ-specific hazard index greater than 1.0?

For general background on risk characterization, see the discussion in <u>questions and answers</u> format on this topic.

#### **SUMMARY OF RESULTS**

Based on a comparison of the cancer and noncancer risks estimated for the 138 air toxics quantified by the 2011 national-scale assessment, it is possible to determine which air toxics pose the greatest potential risk in the United States. A summary of these findings are reported below. Cancer risks in this assessment are presented as lifetime risks, meaning the risk of developing cancer as a result of exposure to each air toxic compound over a normal lifetime of 70 years. Noncancer risks are presented in terms of the ratio between the exposure and a reference concentration. This ratio is called the hazard quotient. The risk characterization summary below focuses on results at the national level, where the EPA believes the results are most meaningful.

To help understand the results, it should be noted that:

- Concentration results (ambient and exposure) are provided for 180 air toxics plus diesel
  PM
- Cancer results are presented for 71 air toxics that have quantitative dose-response information

- Noncancer results are presented for 112 air toxics with quantitative dose-response information
- Many noncancer reference concentrations incorporate protective assumptions designed to provide a margin of safety. A hazard quotient greater than one does not necessarily suggest a likelihood of adverse effects. A hazard quotient equal to or less than one, however, suggests that exposures are likely to be without an appreciable risk of noncancer effects during a lifetime. Furthermore, the hazard quotient cannot be translated into a probability that an adverse effect will occur, and is not proportional to risk

The following conclusions on individual air toxics compounds were drawn from the risk characterization.

The following table presents the criteria for classifying the 2011 NATA air toxics and will be helpful in understanding the conclusions below. In general, drivers and contributors are defined as air toxics showing a particular level of risk or hazard for some number of people exposed.

2011 NATA Health Effects Drivers and Contributors Risk Characterization

Risk Characterization Category	Risk Exceeds (in 1 million) <sup>1</sup>	HI > 1.0 <sup>2</sup>	Number of People or Greater Exposed (in millions)
National Cancer Driver	10		25
Regional Cancer Driver	10		1
Regional Cancer Driver	100		0.01
National Cancer Contributor	1		25
Regional Cancer Contributor	1		1
National Noncancer Driver		1.0	25
Regional Noncancer Driver		1.0	0.01

<sup>1</sup>Cancer risks are upper-bound lifetime cancer risks (i.e., a plausible upper limit to the true probability that an individual will contract cancer over a 70 year lifetime as a result of a given hazard (such as exposure to a toxic chemical). This risk can be measured or estimated in numerical terms (e.g., one chance in one million).

<sup>2</sup>HI = the sum of hazard quotients for substances that affect the same target organ or organ system. Because different pollutants may cause similar adverse health effects, it is often appropriate to combine hazard quotients associated with different substances to understand the potential health risks associated with aggregate exposures to multiple pollutants.

• National cancer risk driver: Formaldehyde

• Regional cancer risk drivers: Benzene, Chloroprene, Coke Oven Emissions

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- National cancer risk contributors: 1,3-Butadiene, Acetaldehyde, Carbon tetrachloride, Chromium (VI), Ethylbenzene, Naphthalene
- **Regional cancer risk contributors**: 1,3-Dichloropropene, 1,4-Dichlorobenzene, Arsenic compounds, Ethylene oxide, Nickel compounds, PAH/POM
- National noncancer hazard drivers: Acrolein, Chlorine, Diesel PM
- Regional noncancer hazard drivers: Hexamethylene diisocyanate

## **Health Effects of National Air Toxic Drivers**

# **Cancer Risk Drivers**

**Formaldehyde** - Acute (short term) and chronic (long term) exposures have been shown to cause respiratory symptoms and irritation to the eyes, nose, and throat. Human studies have suggested an association between formaldehyde exposure and lung and nasopharyngeal cancer. Studies in animals have reported an increased incidence of nasal squamous cell cancer. EPA considers formaldehyde "likely to be carcinogenic to humans".

## **Noncancer Drivers**

**Acrolein -** It is toxic to humans following inhalation, oral or dermal exposures. Acute and chronic inhalation exposure may result in eye, nose and throat irritation and respiratory tract congestion. EPA considers the existing acrolein data to be inadequate for assessing human carcinogenic potential.

Chlorine – Acute (short term) and chronic (long term) exposure has been shown to cause irritation to the eyes, upper respiratory tract and lungs. Studies on workers in the chemical industry and experimental studies in animals have not reported evidence of carcinogenic effects from exposure to chlorine. EPA has not classified chlorine for potential carcinogenicity.

**Diesel exhaust (including diesel PM)** – Acute (short term) exposures can cause irritation (e.g., eye, throat), neurophysiological symptoms (e.g., lightheadedness, nausea), and respiratory symptoms (e.g., cough, phlegm). Chronic (long term) exposures may lead to inflammation and changes in the lung. EPA considers diesel exhaust "likely to be carcinogenic to humans by inhalation" but at this time does not have a quantitative characterization of cancer risk.

The following conclusions on simultaneous exposure to all air toxics compounds were drawn from the risk characterization.

#### Cumulative Cancer Risks:

NATA estimates that all 285 million people in the U.S. have an increased cancer risk of greater than 10 in one million. Half a million people (less than 1 percent of the total U.S. population based on the 2010 census) have an increased cancer risk of greater than 100 in a million. The average, national, cancer risk for 2011 is 40-in-1 million. This means that, on average, approximately 1 in every 25,000 people have an increased likelihood of contracting cancer as a

result of breathing air toxics from outdoor sources if they were exposed to 2011 emission levels over the course of their lifetime

#### Cumulative Noncancer Hazards:

Ideally, hazard quotients should be combined for pollutants that cause the same adverse effects by the same toxic mechanism. For the 2011 NATA assessment, we present results for HAP that act by similar modes of action, or (where this information is absent) that affect the same target organ. This process creates, for each target organ, a target-organ-specific hazard index, defined as the sum of hazard quotients for individual HAP that affect the same organ or organ system. For the 2011 NATA, the hazard indices for the respiratory system dominate the results.

The respiratory hazard index was dominated by a single substance, acrolein, which contributed about 70 percent of the nationwide average non-cancer hazard. The respiratory hazard index exceeded 1.0 for approximately 170 million people while the HI exceeded 10 for more than 75,000 people.

# 2011 NATA Web Application

Results can also be viewed on maps using <u>EPA's Web application</u>. Using the web app mapping tool, the user can generate maps showing geographic patterns of estimated cancer and non-cancer risks in 2011 from inhalation of air toxics. These maps represent a snapshot of conditions in 2011 and are not reflective of current conditions. EPA developed this GIS tool for the 2011 NATA to inform both national and more localized efforts to collect air toxics information and characterize emissions (e.g., prioritize pollutants/geographic areas of interest for more refined data collection such as monitoring). These maps are for screening purposes only. EPA suggests caution in interpreting the information displayed, as limitations and uncertainties of the assessment will vary from location to location as well as from pollutant to pollutant. In many cases more localized assessments, including monitoring and modeling, may be needed to better characterize local-level risk.