Centers for Disease Control and Prevention Standards for Nationally Consistent Data and Measures within the Environmental Public Health Tracking Network

Version 3.0
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Environmental Health Tracking Branch
Division of Environmental Hazards and Health Effects
National Center for Environmental Health
Centers for Disease Control and Prevention

Foreword

This document was first published in March, 2008, setting the standards for the first Nationally Consistent Data and Measures (NCDMs) for the National Environmental Health Tracking Program. The purpose of these NCDMS was to ensure compatibility and comparability of data and measures useful for understanding the impact of our environment on our health. Version 2.0

- reflect the lessons learned in implementing the first NCDMs across local, state, and national tracking networks
- improve the utility of specific measures
- identify recommended temporal and spatial resolution, specifically for health outcomes, based on confidentiality protection needs and data steward requests

Specific updates included in version 2 include:

- Clarified description of process for creating and adopting the first set of NCDMs
- Clarified the meaning of indicator, measure, and data within the Tracking Network
- Added columns to the table summarizing the indicators and measures in order to identify
    - minimum temporal and geographic resolution
    - data source
    - grantee requirements
- Updated indicator templates to reflect minimum temporal and geographic resolution at which measures are to be displayed on public portals

Version 3.0 includes a change from required to optional for the Fertility indicator and documentation for NCDMs adopted since the release of version 2 in August 2011.

- Hospitalizations and ED visits for heat
- ED visits for asthma
- Blood lead levels by birth cohort and annual blood lead levels
- Updates to drinking water NCDMs

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Introduction

Environmental Public Health Tracking is the ongoing collection, integration, analysis, interpretation, and dissemination of data from environmental hazard monitoring, human exposure, and health effects surveillance. In financial year 2002, Congress appropriated funds to the Centers for Disease Control and Prevention (CDC) to develop a national environmental public health tracking network and to improve environmental health capacity at the state and local level.

CDC established its National Environmental Public Health Tracking Program with the following goals:

1. Build a sustainable national environmental public health tracking network (Tracking Network);
2. Enhance environmental public health tracking workforce and infrastructure;
3. Disseminate information to guide policy, practice, and other actions to improve the Nation's health;
4. Advance environmental public health science and research;
5. Foster collaboration among health and environmental programs.

In 2006, CDC transitioned from a piloting and planning phase to implementation. The network was envisioned as a web-based, secure, distributed network of standardized electronic health and environmental data. Sixteen states and New York City were funded in August 2006 to construct state-wide (city-wide) networks that will be components of the national network and to participate in a collaborative process to develop network standards development process. Additional funding from Congress allowed CDC to add 6 more states in 2009 and 1 in 2010.

As part of the implementation process, CDC established a Content Work Group (CWG) to:

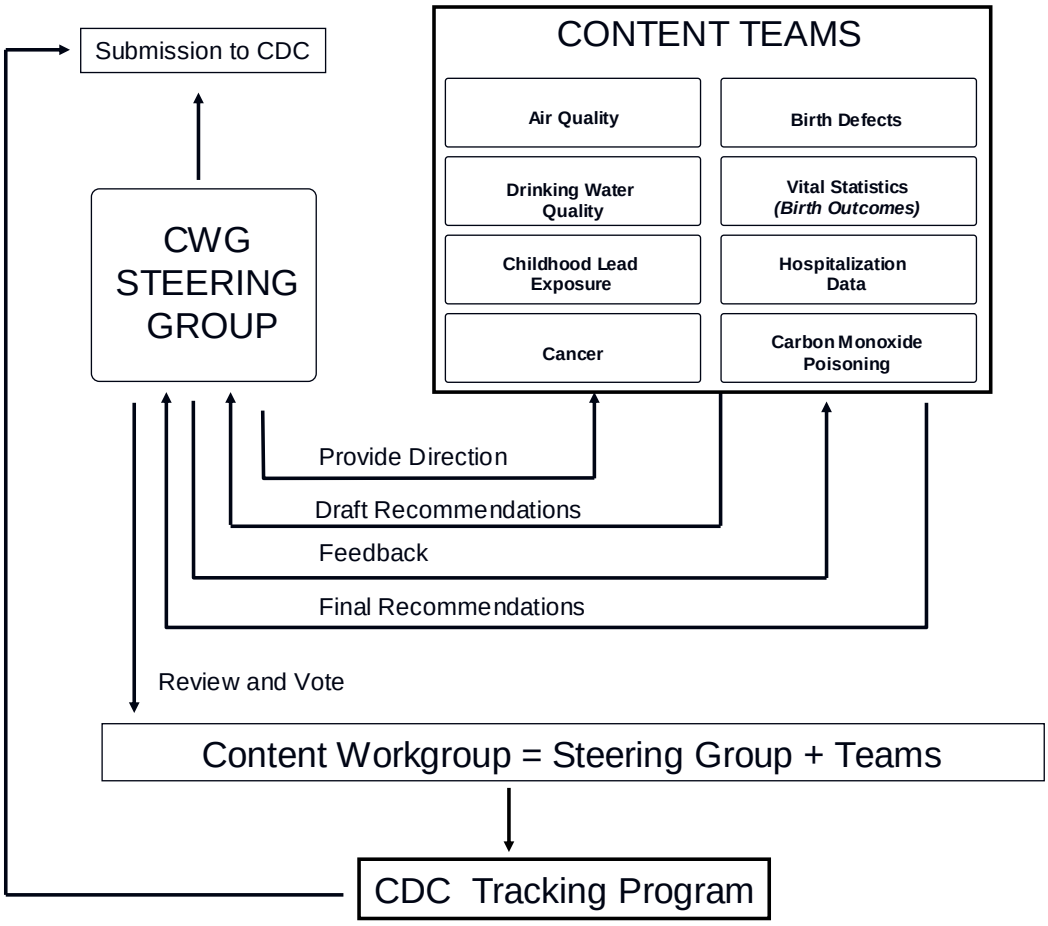
1. Identify and recommend core measures for the Tracking Network;
2. Examine the availability and applicability of existing data and identify approaches for deriving or collecting needed data;
3. Identify and adapt standards and guidelines to facilitate nationally consistent data collection and ensure compatibility with existing standards efforts;
4. Recommend metadata elements to describe data quality;
5. Identify and recommend methods and tools for data integration, analysis and presentation.

The CWG structure included a steering group made up of the principal investigators for grantee health departments and academic partners. Content-specific teams advised the steering group These teams included content experts from: grantee states, cities and academic partners; non-funded states and cities; CDC; other government agencies including the Environmental Protection Agency (EPA), the National Aeronautics and Space Administration (NASA), the US Geological Survey (USGS) and the National Institutes of Health (NIH); and non-governmental organizations including the American Association of Poison Control Centers (AAPCC), the National Birth Defects Prevention Network (NBDPN), the National Association of Health Data Organizations (NAHDO), the National Association for Public Health Statistics and Information

Systems (NAPHSIS) and the North American Association of Central Cancer Registries (NAACCR).

Eight content teams were established, and each provided recommendations to CDC via the steering group for an initial set of Nationally Consistent Data and Measures (NCDMs)(Figure 1). NCDMs consist of measures, grouped by indicators, and the data required to generate them. A measure is a summary characteristic or statistic, such as a sum, percentage, or rate. There may be several measures of a specific indicator which when considered in conjunction fully describe the indicator. An indicator is one or more items, characteristics or other things that will be assessed and that provide information about a population's health status, their environment, and other factors with the goal allowing us to monitor trends, compare situations, and better understand the link between environment and health. It is assessed through direct and indirect measures (e.g. levels of a pollutant in the environment as a measure of possible exposure) that describe health or a factor associated with health (i.e., environmental hazard, age) in a specified population. In general, content teams focused on developing measures specific to one of these areas, but they also considered both proven and potential linkages to the other areas.

Figure 1: Content Work Group (CWG) Structure and Process, 2006 - 2010



Recommendations from content teams were separated into two parts; the first part concerned indicators, measures, and how-to-guides which described the methods for extracting necessary data and generating the measures. The second part was a data dictionary which described the data to be shared with CDC. Recommendations were reviewed by the CWG Steering Group for scientific rigor, utility for Tracking, and feasibility of each grantee generating the measures and where specified providing data to CDC for use on the National Tracking Portal.

This document provides an updated summary of the NCDMs adopted by CDC as Tracking standards. Section One of this document includes tables that summarize the indicators and measures and identify the requirements of Tracking grantees for creating measures and providing data to CDC. These Tracking standards incorporate discussions among the CWG steering group as well as the recommendations of content teams concerning the use of existing national datasets, where relevant.

Section Two includes the indicator templates originally developed by the teams and updated by CDC. An indicator template describes the indicator's measures and their deviations, uses, and limitations. Although teams generally adhered to the template there was some minor variation in

the submitted documents. In creating this document original recommendations were modified to ensure compatibility with the National Network and consistency across NCDMs.

Details regarding the data needed to generate the measures are provided in the how-to-guides, data dictionaries, and schemas available from the CDC Tracking Program. Each set of documentation represents a data feed needed to generate one or more measures.

SECTION ONE: SUMMARY OF NATIONALLY CONSISTENT DATA AND MEASURES

This section lists all NCDMs for the Tracking Network by indicator and measure name. The minimum temporal and geographic resolutions are provided for the display of each required measure. These resolutions were selected to provide the most granular view of the measure possible while considering the rarity of the outcome being measured and data steward requirements. Grantees able to publish more temporally or geographically resolved measures are encouraged to do so. Grantees unable to publish at least the minimum temporal and geographic resolutions should provide written documentation to CDC Tracking Program. **The temporal and geographic resolutions of the measures in this document are not necessarily the temporal and spatial resolution of the data requirements. Information about the required fields and resolution of the data to generate the measures are provided in the how-to-guides and data dictionaries.** The source of the data required to generate each measure at the national level is provided in the summary table. Some data are provided by state and local grantees while other data are provided by national partners. Each measure is also listed as either required or optional for Tracking Grantees. Required means the grantees must (1) provide the data to CDC Tracking Program if the data are not available nationally and (2) publish the measure on their state or local portals.

Content Domain: Heart Attacks or Acute Myocardial Infarction (AMI)

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Heart Attacks	Number of hospitalizations for heart attack	Annual	State and county	Grantee Provided	Required
	Average daily number of hospitalizations for heart attack, by month	Annual	State and county	Grantee Provided	Optional
	Maximum daily number of hospitalizations for heart attack by month	Annual	State and county		
	Minimum daily number of hospitalizations for heart attack by month	Annual	State and county		
	Rate of hospitalization for heart attack among persons 35 and over by age group (total, 35-64, 65+) per 10,000 population	Annual	State and county	Grantee Provided	Required
	Age-adjusted rate of hospitalization for heart attack persons 35 and over per 10,000 population	Annual	State and county		

Content Domain: Air Quality

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Ozone—Days Above Regulatory Standard	Number of days with maximum 8-hour average ozone concentration over the National Ambient Air Quality Standard	Annual	County	Nationally Derived	Required
	Number of person-days with maximum 8-hour average ozone concentration over the National Ambient Air Quality Standard	Annual	County		
Fine Particle (PM2.5)—Days Above Regulatory Standard	Percent of days with PM2.5 levels over the National Ambient Air Quality Standard (NAAQS)	Annual	County	Nationally Derived	Required
	Number of person-days with PM2.5 over the National Ambient Air Quality Standard (NAAQS)	Annual	County		
Annual PM2.5 Level	Average ambient concentrations of PM 2.5 in micrograms per cubic meter (based on seasonal averages and daily measurement)	Annual	County	Nationally Derived	Required
	Percent of population living in counties exceeding the National Ambient Air Quality Standard (compared to percent of population living in counties that meet the standard and percent of population living in counties without PM2.5 monitoring)	Annual	State		

Content Domain: Asthma

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Hospitalizations for Asthma	Number of hospitalizations for asthma	Annual	State and county	Grantee Provided	Required
	Average daily number of hospitalizations for asthma, by month	Annual	State and county	Grantee Provided	Optional
	Maximum daily number of hospitalizations for asthma by month	Annual	State and county		
	Minimum daily number of hospitalizations for asthma by month	Annual	State and county		
	Rate of hospitalization for asthma by age group (total, 0-4, 5-14, 15-34, 35-64, and 65+) per 10,000 population	Annual	State and county	Grantee Provided	Required
	Age-adjusted rate of hospitalization for asthma per 10,000 population	Annual	State and county		
Emergency Department Visits for Asthma	Annual number of emergency department visits for asthma	Annual	State and county	Grantee Provided	Required
	Average number of emergency department visits for asthma as primary diagnosis per month	Annual	State and county		
	Annual crude rate of emergency department visits for asthma by age group (total, 0–4, 5–14, 15–34, 35–64, and 65+) per 10,000 population by age group	Annual	State and county		
	Annual age-adjusted rate of emergency department visits for asthma by age groups (total, 0–4, 5–14, 15–34, 35–64, and 65+) per 10,000 population	Annual	State and county		

Content Domain: Birth Defects

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Prevalence of Birth Defects	Prevalence of Anencephaly per 10,000 live births	5 year	State and county	Grantee Provided	Required
	Prevalence of Spina Bifida (without Anencephaly) per 10,000 live births over	5 year	State and county		
	Prevalence of Hypoplastic Left Heart Syndrome per 10,000 live births	5 year	State and county		
	Prevalence of Tetralogy of Fallot per 10,000 live births	5 year	State and county		
	Prevalence of Transposition of the Great Arteries (vessels) per 10,000 live births	5 year	State and county		
	Prevalence of Cleft Lip with or without Cleft Palate per 10,000 live births	5 year	State and county		
	Prevalence of Cleft Palate without Cleft Lip per 10,000 live births	5 year	State and county		
	Prevalence of Hypospadias per 10,000 live male births	5 year	State and county		
	Prevalence of Gastroschisis per 10,000 live births	5 year	State and county		
	Prevalence of Upper Limb Deficiencies per 10,000 live births	5 year	State and county		
	Prevalence of Lower Limb Deficiencies per 10,000 live births	5 year	State and county		
	Prevalence of Trisomy 21 per 10,000 live births by maternal age at delivery (<35 and >/=35)	5 year	State and county		

Content Domain: Cancer

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Incidence of Selected Cancers	Number of cases of Mesothelioma	5 year	State	Nationally Derived	Required
	Age-adjusted incidence rate of Mesothelioma per 100,000 population	5 year	State		
	Number of cases of Melanoma of the Skin	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Melanoma of the Skin per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Liver and Intrahepatic Bile Duct Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Liver and Intrahepatic Bile Duct Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Kidney and Renal Pelvis Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Kidney and Renal Pelvis Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Breast Cancer in females by Age group (<50, ≥50, total)	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Breast Cancer in females per 100,000 population by Age group (<50, ≥50, total)	Annual	State		

		5 year	State and county		
	Number of cases of Lung and Bronchus Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Lung and Bronchus Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Bladder Cancer (including in situ)	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Bladder Cancer (including in situ) per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Brain and other nervous systems Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Brain and other nervous systems Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Brain and Central Nervous System Cancer in children (<15 years and <20 years)	Annual	State		
	Age-adjusted incidence rate of Brain and Central Nervous System Cancer in children (<15 years and <20 years) per 1,000,000 population	Annual	State		
	Number of cases of Thyroid Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Thyroid Cancer per 100,000	Annual	State		

	population	5 year	State and county		
	Number of cases of Non-Hodgkin's Lymphoma	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Non-Hodgkin's Lymphoma per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Leukemia	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Leukemia per 100,000 population	Annual	State		
		5 year	State and county		
	Number of Leukemia in children (<15 years and <20 years)	Annual	State		
	Age-adjusted incidence rate of Leukemia in children (<15 years and <20 years) per 1,000,000 population	Annual	State		
	Number of cases of Chronic Lymphocytic Leukemia	Annual	State		
	Age-adjusted incidence rate of Chronic Lymphocytic Leukemia per 100,000 population	Annual	State		
	Number of cases of Acute Myeloid Leukemia	Annual	State		
	Age-adjusted incidence rate of Acute Myeloid Leukemia per 100,000 population	Annual	State		
	Number of Acute Myeloid Leukemia in children (<15 years and <20 years)	Annual	State		

	Age-adjusted incidence rate of Acute Myeloid Leukemia in children (<15 years and <20 years) per 1,000,000 population	Annual	State		
	Number of cases of Acute Lymphocytic Leukemia in children (<15 years and <20 years)	Annual	State		
	Age-adjusted incidence rate of Acute Lymphocytic Leukemia in children (<15 years and <20 years) per 1,000,000 population	Annual	State		
Incidence of Selected Cancers	Number of cases of Oral Cavity and Pharynx Cancer	Annual	State	Nationally Derived	Optional
		5 year	State and county		
	Age-adjusted incidence rate of Oral Cavity and Pharynx Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Larynx Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Larynx Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Esophagus Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Esophagus Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Pancreas Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Pancreas Cancer per 100,000 population	Annual	State		
		5 year	State and county		

Content Domain: Carbon Monoxide

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Hospitalizations for Carbon Monoxide (CO) Poisoning	Number of hospitalizations for CO poisoning by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State	Grantee Provided	Required
	Crude rate of hospitalization for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State		
	Age-adjusted rate of hospitalization for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State		
Emergency Department Visits for CO Poisoning	Number of emergency department visits for CO Poisoning by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State	Grantee Provided	Optional
	Crude rate of emergency department visits for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State		
	Age-adjusted rate of emergency department visits for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional	Annual	State		

	non-fire related, and unknown intent)				
CO Poisoning Mortality	Number of deaths from CO poisoning by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State	Nationally Derived	Required
	Crude rate of death from CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State		
	Age-adjusted rate of death from CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State		
Reported Exposure to CO	Number of unintentional CO exposures reported to poison control centers by resulting health effect and treatment in a healthcare facility	Annual	State	Nationally Derived	Optional
	Crude rate of unintentional CO exposures reported to poison control centers per 100,000 population by resulting health effect and treatment in a healthcare facility	Annual	State		
Home CO Detector Coverage	Percent of Behavioral Risk Factor Surveillance System (BRFSS) respondents reporting at least one CO detector in their household	Annual	State	Nationally Derived	Optional

Content Domain: Childhood Lead Poisoning

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Testing and Housing Age	Number of children born in the same year and tested	Annual	State and county	Nationally Derived	Required
	Percent of children born in the same year and tested	Annual	State and county		
	Number of homes built before 1950 (as measured in the 2000 Census)	Annual	State and county		
	Percent of homes built before 1950 (as measured in the 2000 Census)	Annual	State and county		
	Number of children younger than 5 years living in poverty (as measured in 2000 census)	Annual	State and county		Optional
	Percent of children younger than 5 years living in poverty (as measured in 2000 census)	Annual	State and county		
Blood Lead Levels by Birth Cohort	Number of children born in the same year and tested	Annual	State and county	Nationally Derived	Required
	Percent of children born in the same year and tested	Annual	State and county		
	Number of children born in the same year and tested with confirmed blood lead levels ≥ 10 µg/dL	Annual	State and county		
	Percent of children born in the same year and tested with confirmed blood lead levels ≥ 10 µg/dL	Annual	State and county		
	Number of children born in the same year and tested with confirmed blood lead levels ≥ 10 µg/dL, by blood lead level category	Annual	State		
	Percent of children born in the same	Annual	State		

	year and tested with confirmed blood lead levels ≥ 10 µg/dL, by blood lead level category				
	PROPOSED *Number of children born in the same year and tested with blood lead levels between 5 and <10 µg/dL	Annual	State and county		
	PROPOSED*Percent of children born in the same year and tested with blood lead levels between 5 and <10 µg/dL	Annual	State and county		
Annual Blood Lead Levels	Number of children tested, by age group	Annual	State and county	Nationally Derived	Required
	Percent of children tested, by age group	Annual	State and county		
	Number of children tested with confirmed blood lead levels ≥ 10 µg/dL, by age group	Annual	State and county		
	Percent of children tested with confirmed blood lead levels ≥ 10 µg/dL, by age group	Annual	State and county		
	Number of children tested with confirmed blood lead levels ≥ 10 µg/dL by blood lead level category, by age group	Annual	State		
	Percent of children tested with confirmed blood lead levels ≥ 10 µg/dL, by blood lead level category, by age group	Annual	State		
	PROPOSED *Number of children tested with blood lead levels between 5 and <10 µg/dL	Annual	State and county		
	PROPOSED*Percent of children tested with blood lead levels between 5 and <10 µg/dL	Annual	State and county		

Content Domain: Climate Change

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Heat Stress Hospitalizations	Number of hospitalizations for heat stress	Annual from May–September	State and national	Grantee Provided	Required
	Crude rate of hospitalization for heat stress by age groups (total, 0–4, 5–14, 15–34, 35–64, and 65+) per 100,000 population	Annual from May–September	State and national		
	Age-adjusted rate of hospitalization for heat stress (by age groups 0–4, 5–14, 15–34, 35–64, and 65+) per 100,000 population	Annual from May–September	State and national		
Heat Stress Emergency Department Visits for Heat Stress	Annual number of emergency department visits for heat stress	Annual from May–September	State and county	Grantee Provided	Required
	Annual crude rate of emergency department visits for heat stress by age group (total, 0–4, 5–14, 15–34, 35–64, and 65+) per 100,000	Annual from May–September	State and county		
	Age-adjusted rate of emergency department visits for heat stress by age groups (total, 0–4, 5–14, 15–34, 35–64, and 65+) per 100,000 population	Annual from May–September	State and county		

Content Domain: Drinking Water

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Atrazine Level and Potential Population Exposures	Distribution of number of Community Water Systems (CWS) by mean atrazine concentration (micrograms per liter)	Quarterly	County	Grantee Provided	Required
	Distribution of number of CWS by maximum atrazine concentration (micrograms per liter)	Annual	County		
	Distribution of number of CWS by mean atrazine concentration (micrograms per liter)	Annual	County		
	Mean concentration of atrazine (micrograms per liter) at CWS-level	Annual	County		
	Distribution of number of people served by CWS by mean atrazine concentration (micrograms per liter)	Quarterly	County		
	Distribution of number of people served by CWS by maximum atrazine concentration (micrograms per liter)	Annual	County		
	Distribution of number of people served by CWS by mean atrazine concentration (micrograms per liter)	Annual	County		
Arsenic Level and Potential Population Exposures	Distribution of number of community water systems by mean arsenic concentrations (micrograms per liter)	Annual	State	Grantee Provided	Required
	Distribution of number of people served by community water systems by mean arsenic concentrations (micrograms per liter)	Annual	State		
	Distribution of number of community water systems by maximum arsenic concentrations (micrograms per liter)	Annual	State		

	Distribution of number of people served by community water systems by maximum arsenic concentrations (micrograms per liter)	Annual	State		
	Mean concentration of Arsenic (micrograms per liter) at CWS-level	Annual	State		
Di (2-Ethylhexyl) phthalate (DEHP) Level and Potential Population Exposures	Distribution of number of Community Water Systems (CWS) by maximum DEHP concentration (micrograms per liter)	Annual	County	Grantee Provided	Required
	Distribution of number of CWS by mean DEHP concentration (micrograms per liter)	Annual	County		
	Mean concentration of DEHP (micrograms per liter) at CWS-level	Annual	County		
	Distribution of number of people served by CWS by maximum DEHP concentration (micrograms per liter)	Annual	County		
	Distribution of number of people served by CWS by mean DEHP concentration (micrograms per liter)	Annual	County		
Nitrate Level and Potential Population Exposures	Distribution of number of community water systems by mean nitrate concentrations (milligrams per liter)	Annual	State	Grantee Provided	Required
	Distribution of number of people served by community water systems by mean nitrate concentrations (milligrams per liter)	Annual	State		
	Distribution of number of community water systems by maximum nitrate concentrations (milligrams per liter)	Annual	State		
	Distribution of number of people served by community water systems by maximum nitrate concentrations (milligrams per liter)	Annual	State		

	Distribution of number of community water systems by mean nitrate concentrations (milligrams per liter)	Quarterly	State		
	Distribution of number of people served by community water systems by mean nitrate concentrations (milligrams per liter)	Quarterly	State		
	Mean concentration of nitrate (milligrams per liter) at CWS-level	Annual	State		
Disinfection Byproducts (DBP) Level and Potential Population Exposure (TTHM)	Distribution of number of community water systems by mean trihalomethane (THM) concentrations (micrograms per liter)	Annual	State	Grantee Provided	Required
	Distribution of number of people served by community water systems by mean trihalomethane (THM) concentrations (micrograms per liter)	Annual	State		
	Distribution of number of community water systems by maximum trihalomethane (THM) concentrations (micrograms per liter)	Annual	State		
	Distribution of number of people served by community water systems by maximum trihalomethane (THM) concentrations (micrograms per liter)	Annual	State		
	Distribution of number of community water systems by mean trihalomethane concentrations (micrograms per liter)	Quarterly	State		
	Distribution of number of people served by community water systems by mean trihalomethane (THM) concentrations (micrograms per liter)	Quarterly	State		

Disinfection Byproduct: Levels and Potential Population Exposures (HAA5)	Distribution of number of community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter)	Annual	State	Grantee Provided	Required
	Mean concentration of HAA5 (micrograms per liter) at CWS-level	Annual	State		
	Distribution of number of community water systems by maximum haloacetic acids (HAA5) concentrations (micrograms per liter)	Annual	State		
	Distribution of number of CWS by maximum TTHM concentration (micrograms per liter)	Annual	State		
	Distribution of number of people served by community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter)	Quarterly	State		
	Distribution of number of CWS by mean TTHM concentrations (micrograms per liter)	Quarterly	State		
	Distribution of number of CWS by mean TTHM concentration (micrograms per liter)	Annual	State		
	Mean concentration (micrograms per liter) of TTHM at CWS-level	Annual	State		
Public Water Use	Number of people receiving water from community water systems	Annual	State	Grantee Provided	Required
Combined Radium-226 and -228 Levels and Potential Population	Distribution of number of Community Water Systems (CWS) by maximum Radium concentration picoCuries per Liter	Annual	County	Grantee Provided	Required
	Distribution of number of CWS by mean Radium concentration picoCuries per Liter	Annual	County		

Exposure	Mean concentration of Radium picoCuries per Liter at CWS-level	Annual	County		
	Distribution of number of people served by CWS by maximum Radium concentration picoCuries per Liter	Annual	County		
	Distribution of number of people served by CWS by mean Radium concentration picoCuries per Liter	Annual	County		
Tetrachloroethene (PCE) Levels and Potential Population Exposure	Distribution of number of Community Water Systems (CWS) by maximum PCE concentration (micrograms per liter)	Annual	County	Grantee Provided	Required
	Distribution of number of CWS by mean PCE concentration (micrograms per liter)	Annual	County		
	Mean concentration of PCE (micrograms per liter) at CWS-level	Annual	County		
	Distribution of number of people served by CWS by maximum PCE concentration (micrograms per liter)	Annual	County		
	Distribution of number of people served by CWS by mean PCE concentration (micrograms per liter)	Annual	County		
Trichloroethene (TCE) Levels and Potential Population Exposure	Distribution of number of CWS by maximum TCE concentration (micrograms per liter)	Annual	County	Grantee Provided	Required
	Distribution of number of CWS by mean TCE concentration (micrograms per liter)	Annual	County		
	Mean concentration of TCE (micrograms per liter) at CWS-level	Annual	County		
	Distribution of number of people served by CWS by maximum TCE concentration (micrograms per liter)	Annual	County		
	Distribution of number of people served by CWS by mean TCE concentration (micrograms per liter)	Annual	County		

Uranium Levels and Potential Population Exposure	Distribution of number of Community Water Systems (CWS) by maximum Uranium concentration (micrograms per liter)	Annual	County	Grantee Provided	Required
	Distribution of number of CWS by mean Uranium concentration (micrograms per liter)	Annual	County		
	Mean concentration of Uranium (micrograms per liter) at CWS-level	Annual	County		
	Distribution of number of people served by CWS by maximum Uranium concentration (micrograms per liter)	Annual	County		
	Distribution of number of people served by CWS by mean Uranium concentration (micrograms per liter)	Annual	County		

Content Domain: Reproductive Health Outcomes

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Prematurity	Percent of preterm (less than 37 weeks gestation) live singleton births	Annual	State and county	Nationally Derived	Required
	Percent of very preterm (less than 32 weeks gestation) live singleton births	5 year Annual Average	State and county		
Low Birthweight	Percent of low birthweight (less than 2500 grams) live term singleton births	Annual	State and county	Nationally Derived	Required
	Percent of very low birthweight (less than 1500 grams) live singleton births	5 year Annual Average	State and county		
Mortality	Average Infant (less than 1 year of age) Mortality Rate per 1000 live births	5 year Annual Average	State and county	Nationally Derived	Required
	Average Neonatal (less than 28 days of age) Mortality Rate per 1000 live births	5 year Annual Average	State and county		
	Average Perinatal (equal to or greater than 28 weeks gestation to less than 7 days of age) Mortality Rate per 1000 live births (plus fetal deaths equal to or greater than 28 weeks gestation)	5 year Annual Average	State and county		
	Average Postneonatal (equal to or greater than 28 days to less than 1 year of age) Mortality Rate per 1000 live births	5 year Annual Average	State and county		
Fertility	Total Fertility Rate per 1000 women of reproductive age	Annual	State and county	Nationally Derived	Optional
Sex Ratio at Birth	Male to Female sex ratio at birth (term singletons only)	Annual	State and county	Nationally Derived	Required

SECTION TWO: INDICATOR TEMPLATES

This section contains an indicator template for each indicator and corresponding measures listed in section one. The indicator template provides basic information about the indicator including:

1. Measures
2. Derivations of the measures
3. Units
4. Geographic Scope
5. Geographic Scale
6. Time Period
7. Time Scale
8. Rationale
9. Use of the Measure
10. Limitations of the Measure
11. Data Sources
12. Limitations of Data Sources
13. References

Additional information about the underlying data needed for the indicator and steps for extracting the data and generating the measures can be found in the how-to-guides and data dictionaries.

CONTENT DOMAIN: HEART ATTACK
INDICATOR: HOSPITALIZATIONS FOR HEART ATTACK

Type of EPHT Indicator	**Health Outcome**
Measures	1. Number of hospitalizations for acute myocardial infarction (AMI) 2. Minimum daily number of hospitalizations for AMI by month 3. Maximum daily number of hospitalizations for AMI by month 4. Average daily number of hospitalizations for AMI by month 5. Crude rate of hospitalizations for AMI among persons 35 and older by age group (total, 35-64, 65+) per 10,000 population 6. Annual age-adjusted rate of hospitalizations for AMI among persons 35 and older per 10,000 population When supported by sufficient data volume, the measures may also be reported stratified by sex, race, and ethnicity.
Derivation of Measures	**Numerator:** Resident hospitalizations for AMI, ICD-9-CM: 410.00–410.92 by gender and total for state and by county **Denominator:** Midyear resident population by gender, for state and by county **Adjustment:** Age-adjustment by the direct method to Year 2000 U.S. Standard population
Unit	Hospital admission (categorized by discharge diagnosis)
Geographic Scope	State and national (tracking network states)
Geographic Scale	State and county
Time Period	Hospital admissions from January 1 through December 31 for each year, 2000–current
Time Scale	Daily, monthly, and annually (as appropriate for the measure)
Rationale	There currently is no single AMI surveillance system is in place in the United States, nor does such a system exist for coronary heart disease (CHD) in general. Mortality is the sole descriptor for national data for AMI. Estimates of incidence and prevalence of AMI and CHD are largely based on survey samples (e.g., NHANES) or large cohort studies such as the Atherosclerosis Risk in Communities (ARIC) study. In 2007, the American Heart Association estimated 565,000 new attacks and 300,000 recurrent attacks of MI annually (National Heart, Lung, and Blood Institute: based on unpublished data from the ARIC study and the Cardiovascular Health Study [CHS]). Among

	Americans aged ≥20 years, new and recurrent MI prevalence for both men and women represented 3.7% of the U.S. population, or 7,900,000 (4.9 million men and 3.0 million women). Corresponding prevalence by race and ethnicity is 5.4% for white men, 2.5% for white women, 3.9% for black men, and 3.3% for black women. The well-documented risk factors for AMI include diabetes, hypertension, obesity, hypercholesterolemia, and cigarette smoking. Increasingly, investigators both in the United States and abroad have shown significant relationships between air pollutants and increased risk of AMI and other forms of CHD. Studies have often focused on persons aged >65 years. A number of epidemiologic studies have reported associations between air pollution (ozone, PM_{10}, CO, PM 2.5, SO_2) and hospitalizations for AMI and other forms of heart disease. Models have demonstrated increases in AMI hospitalization rate in relation to fine particles (PM2.5), particularly in sensitive subpopulations such as the elderly, patients with pre-existing heart disease, and particularly persons who are survivors of MI or persons with COPD. An increase of 10 ug/m^3 in PM 2.5 was associated with a 4.5% elevation in risk of acute ischemic coronary events (unstable angina and AMI) (95% CI, 1.1–8.0). Mortality statistics have been linked for a 16-year period to chronic exposure of multiple air pollutants in 500,000 adults residing throughout the United States. Each 10 ug/m3 in annual PM2.5 was related to a 12% increased mortality risk.
Use of the Measures	Developing a standardized analytic method for AMI hospital admissions among residents in each state will provide more uniform information for multiple users at the national, state, and local levels. These measures will allow monitoring of trends over time, identify high risk groups, and inform prevention, evaluation, and program planning efforts. These measures will address the following surveillance functions: • Examination of time trends in AMI hospitalizations. • Identification of seasonal trends. • Assessment of geographic differences in hospitalizations. • Evaluation of differences in AMI hospitalizations by age, gender, and race/ethnicity. • With further analysis … evaluation of disparities in AMI hospitalizations by factors such as age, race/ethnicity, gender, education, and/or income. • Determination of populations in need of targeted interventions. • Identification of possible environmental relationships that warrant further investigation or environmental public health action when AMI data are linked with environmental variables.

Limitations of the Measures	Hospitalization data for AMIs omit persons who do not receive medical care or who are not hospitalized, including those who die in emergency rooms, in nursing homes, or at home without being admitted to a hospital, and those treated in outpatient settings. Differences in rates by time or area may reflect differences or changes in diagnostic techniques and criteria and in the coding of AMI or in medical care access. Differences in rates by area may be due to different sociodemographic characteristics and associated behaviors. When rates across geographic areas are compared, a variety on non-environmental factors, such as access to medical care and diet, can affect the likelihood of persons hospitalized for AMI. Reporting rates at the state and/or county level will not show the true AMI burden at a more local level (i.e., neighborhood). Reporting rates at the state and/or county level will not be resolved geographically enough to be linked with many types of environmental data. When looking at small geographic levels (e.g., ZIP code), users must consider appropriate cell suppression rules imposed by the data providers or individual state programs. Although duplicate records and transfers from one hospital to another are excluded, the measures are based upon events, not individuals, because no unique identifier is always available. When multiple admissions are not identified, the true prevalence will be overestimated. Even at the county level, the measures generated will often be based upon numbers too small to report or present without violating state and federal privacy guidelines and regulations. Careful adherence to cell suppression rules in cross tabulations is necessary, and methods to increase cell sizes by combining data across time (e.g., months, years) and geographic areas may be appropriate.
Data Sources	**Numerator:** State inpatient hospitalization data (using admission date) **Denominator:** U.S. Census Bureau population data
Limitations of Data Sources	**State hospital discharge data:** Using a measure of all AMI hospitalizations will include some

	transfers between hospitals for the same person for the same AMI event. Variations in the percentage of transfers or readmissions for the same AMI event may vary by geographic area and impact rates. However, efforts were made to identify and exclude transfers based on unique identifiers consisting of date of birth, zip code, gender, and encrypted social security number when available. Without reciprocal reporting agreements with abutting states, statewide measures and measures for geographic areas (e.g., counties) bordering other states may be underestimated because of health care utilization patterns. Each state must individually obtain permission to access and, in some states, provide payment to obtain the data. Veterans Affairs, Indian Health Services, and institutionalized (prison) populations are not usually included in hospitalization datasets. Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers to hospitalize patients Street address is not available in many states. Sometimes mailing address of patient is listed as the residence address of the patient. Patients may be exposed to environmental triggers in multiple locations, but hospital discharge geographic information is limited to residence. Since the data capture hospital discharges (rather than admissions), patients admitted toward the end of the year and discharged the following year will be omitted from the current year dataset. Data will need to be de-duplicated (i.e., remove duplicate records for the same event). There is usually a two-year lag period before data are available from the data owner. **Census data:** Available only every 10 years; thus, postcensal data will be estimated for calculating rates for years following the census year. Postcensal estimates at the ZIP code level are not available from the Census Bureau. These estimates should be extrapolated or purchased

	from a vendor.
References	**1.** Rosamond, W., et al., Heart disease and stroke statistics--2007 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation, 2007. 115(5): p. e69–171. **2.** Boland, L.L., et al., Occurrence of unrecognized myocardial infarction in subjects aged 45 to 65 years (the ARIC study). Am J Cardiol, 2002. 90(9): p. 927–31. **3.** Thom, T., et al., Cardiovascular disease in the United States and preventive approaches, in Hurst's The Heart, Arteries and Veins, V. Fuster, R. Alexander, and R. O'Rourke, Editors. 2001, McGraw-Hill: New York, NY. **4.** Jones, D.W., et al., Risk factors for coronary heart disease in African Americans: the atherosclerosis risk in communities study, 1987–1997. Arch Intern Med, 2002. 162(22): p. 2565–71. **5.** Kannel, W.B., et al., Menopause and risk of cardiovascular disease: the Framingham study. Ann Intern Med, 1976. 85(4): p. 447–52. **6.** Pope, C.A., 3rd, et al., Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. Circulation, 2004. 109(1): p. 71–7. **7.** Vermylen, J., et al., Ambient air pollution and acute myocardial infarction. J Thromb Haemost, 2005. 3(9): p. 1955–61. **8.** Pope, C.A., 3rd, et al., Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. Circulation, 2006. 114(23): p. 2443–8 **9.** von Klot, S., et al., Ambient air pollution is associated with increased risk of hospital cardiac readmissions of myocardial infarction survivors in five European cities. Circulation, 2005. 112(20): p. 3073–9.

CONTENT DOMAIN: AIR QUALITY
INDICATOR: OZONE-DAYS ABOVE REGULATORY STANDARD

Type of EPHT Indicator	Hazard
Measures	1. Number of days with maximum 8-hour average ozone concentration over the National Ambient Air Quality Standard (NAAQS) 2. Number of person-days with maximum 8-hour average ozone concentration over the National Ambient Air Quality Standard (NAAQS)
Derivation of Measures	This overview provides the key technical points in how EPA and CDC processed EPA's air quality data for use in the EPHT air indicators. **Processing raw data** First, EPA extracts the air quality data from the Air Quality System (AQS). EPA uses the following steps in developing the air data and measures for EPHT air quality indicators. Step 1: EPA accesses daily maximum 8-hour average ozone concentrations (ppm) (parameter code '44201' and duration code 'W') and supplemental data fields (e.g. latitude, longitude, elevation) for all the monitoring sites across the US from the EPA's Data Mart. The data are obtained only from monitors that are designated as Federal Reference Methods or equivalent. The data include any flagged values associated with exceptional events (high winds, fires, construction, etc) regardless of concurrence by the EPA Regional Office. EPA retains data from monitors that meet the minimum data completeness criteria set forth in the national air quality standard (i.e. if valid 8-hour averages are available for at least 75% of possible hours in a day or the maximum 8-hour average is above ozone 8-hr NAAQS). Step 2: For each monitoring site, retain the maximum concentration at the site for each monitored day. The pollutant occurrence code (poc) which distinguishes multiple monitors at a single site is listed in the output data set. Step 3: Site-level daily monitoring data are used to create ozone 8-hr maximum daily county-level dataset. Daily county-level dataset is created by retaining the maximum concentration among all monitors within the county for each monitored day. The county-level daily dataset is used to create number of days and number of person-days with ozone levels over the daily NAAQS measures.

	Creating Measures Step 3: Ozone levels decrease significantly in the colder parts of the year in many areas, ozone is required to be monitored at monitoring sites only during the ozone season, which is defined on a state by state basis. Only counties that have at least 75% of the days monitored during the ozone seasons are considered complete. The measures are computed only for counties that satisfy the completeness criteria. ***Number of days with Ozone levels over the NAAQS:*** Step 4: Select counties which pass the completeness criteria mentioned in Step 3. Step 5: To calculate the annual number of days over the daily NAAQS, sum the number of days with ozone levels over the daily 8-hr NAAQS for the entire year. ***Number of person-days with ozone levels over the NAAQS:*** Step 4: To calculate Person-days with ozone levels over the daily 8-hr NAAQS, multiply the number of days over the daily NAAQS by the total population of the county.
Units	1. Exceedance days 2. Population-weighted exceedance days
Geographic Scope	United States
Geographic Scale	County (where monitors exist)
Time Period	2001-current
Time Scale	Calendar year
Rationale	According to the published literature, air pollution is associated with premature death, increased rates of hospitalization for respiratory and cardiovascular conditions, adverse birth outcomes, and lung cancer (2, 3). Air pollution places a large economic burden on the country. In a report prepared for the American Lung Association,(2) estimated that air pollution related illness was estimated to cost approximately $100 billion annually (2) (1988 dollars) in the United States, with an estimated number of excess deaths ranging from 50,000 to 100,000 annually (3). More than half of the U.S. population, approximately 159 million persons, live in counties with unhealthy levels of air pollution in the form of either ozone or particulate matter (1). Elevated pollution levels depend on sources, transport, season geography, and atmospheric conditions. Each part of the country has its own level of pollution concentrations that can be exacerbated by many conditions, including stagnation, fire, or wind. The seasons for peak concentrations also vary between geographical regions. (4) The Clean Air Act, which was last amended in 1990, requires EPA to set NAAQS for widespread pollutants from numerous and diverse sources

	considered harmful to public health and the environment. The Clean Air Act established two types of national air quality standards. Primary standards set limits to protect public health, including the health of "sensitive" populations such as asthmatics, children, and the elderly. Secondary standards set limits to protect public welfare, including visibility impairment and damage to animals, crops, vegetation, and buildings. (5) Our indicator is based on comparing measured levels of ozone by county to the primary ozone 8-hr NAAQS, which is set at 75 ppb The Clean Air Act requires periodic review of the science upon which the standards are based and the standards themselves. Primary air quality standards indicate the acceptable level of substances in the air before harm will occur based on proven scientific and medical research. State governments also set air quality standards. In several cases, California's standards or other benchmarks are more stringent than the EPA NAAQS.
Use of Measure	The indicator for the number of days with maximum 8-hour average ozone concentration over the standard is similar to EPA's analyses on number of days with air quality index (AQI) levels higher than 100 (for ozone) – see www.epa.gov/airtrends/aqi_info.html. This measure is consistent with the EPA and state AQI program efforts to communicate an area's air quality levels to the public. In addition, this indicator can be used to inform policy makers and the public of the degree of hazard within a state (by county or MSAs with monitors) during a year. For example, the number of days per year that ozone is higher than the NAAQS can be used to communicate to sensitive populations (such as asthmatics) the number of days that they may be exposed to unhealthy levels of ozone; this is the same level used in the air quality alerts that inform these sensitive populations when and how to reduce exposure. See http://www.epa.gov/air/airtrends/2007/report/groundlevelozone.pdf and http://www.epa.gov/air/airtrends/aqtrnd00/pdffiles/aqioz.pdf. In the use of the measure, it is important to explain that not all counties have monitors although most populated areas are monitored.

Limitations of The Measure	Since ozone levels decrease significantly in the colder parts of the year in many areas, ozone is required to be monitored only during the ozone season., which are designated on a State by State basis.(6) The number of high ozone days per year varies, which makes tracking trends over time difficult to analyze or interpret. The variability results from the following: a) the number of high ozone days is related to temperature; there will be more high days in hotter summers; and b) there are a small number of events per year, so for statistical reasons this type of measure will bounce around more than an average. c) When creating measures, we only consider monitors with 75% completeness during the ozone season and ozone seasons are designated on a state by state basis. Variation within counties may exist but will not be captured in this measure. Within these areas, the monitor with the highest reading on any day is used in the measure. Larger areas will have a broader range of pollution values and perhaps more monitors that may measure a high value on a given day. Thus, day and person-day estimates for larger areas may be biased higher than estimates for smaller areas. The relative variation among county populations in many states may be large enough relative to the variation in the number of days greater than the ozone NAAQS that the population component can dominate the calculation of the number of person-days. Thus, careful investigation of the underlying data to properly identify changes in population and air quality is needed when comparing person-days in space and time. The data for this indicator represent only counties that have air monitors; thus the data tend to reflect urban air quality (where most people live). Although populations in areas without monitors also may be exposed to ozone that exceeds the standard, they are not counted. The number of days that exceed the EPA NAAQS or other health benchmarks does not provide information regarding the severity (max concentrations) of potential exposures. The relationship between ambient concentrations and personal exposure is largely unknown and variable depending upon pollutant, activity patterns, and microenvironments. This indicator is not for use compliance determination with NAAQS or reasonable further progress toward attaining compliance.
Data Sources	Air quality data: EPA Air Explorer http://epa.gov/mxplorer/index.htm
Limitations of Data Sources	The AQS monitoring data, which are used in the calculation of measures, are not present for all counties and days.
References	1. American Lung Association. State of the Air 2004; 2004 [cited 2008 Dec 4]. Available from: http://lungaction.org/reports/sota04_full.html 2. Cannon J. The Health Costs of Air Pollution: A Survey of

	Studies Published 1984– 1989. New York: American Lung Association; 1990. 3. Dockery DW and Pope CA. Acute respiratory effects of particulate air pollution. Annu Rev Public Health 1994;15:107–132. 4. US Environmental Protection Agency. US EPA general site on ozone effects. Available from: http://www.epa.gov/air/ozonepollution/health.html 5. Criteria document for ozone NAAQS: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=149923 6. Ozone Season definition by state: http://www.epa.gov/ttn/naaqs/ozone/ozonetech/40cfr58d.htm

CONTENT DOMAIN: AIR QUALITY
INDICATOR: $PM_{2.5}$—DAYS ABOVE REGULATORY STANDARD

Type of EPHT Indicator	Hazard
Measures	1. Percent of days with $PM_{2.5}$ levels over the National Ambient Air Quality Standard (NAAQS) 2. Number of person-days with $PM_{2.5}$ over the National Ambient Air Quality Standard (NAAQS)
Derivation of Measures	This overview provides the key technical points in how EPA and CDC processed EPA's air quality data for use in the EPHT air indicators. **Processing raw data:** First, EPA extracts the air quality data from the Air Quality System (AQS). EPA uses the following steps in developing the air data and measures for EPHT air quality indicators. Step 1: EPA accesses $PM_{2.5}$ daily concentrations ($\mu g/m^3$) (parameter code '88101' and duration code '7') and daily maximum 8-hour average ozone concentrations (ppm) (parameter code '44201' and duration code 'W') and supplemental data fields (e.g. latitude, longitude, elevation) for all the monitoring sites across the US from the EPA's Data Mart. The data are obtained only from monitors that are designated as Federal Reference Methods or equivalent. The data include any flagged values associated with exceptional events (high winds, fires, construction, etc) regardless of concurrence by the EPA Regional Office. Step 2: For each monitoring site, retain the maximum concentration at the site for each monitored day. The pollutant occurrence code (poc) which distinguishes multiple monitors at a single site is listed in the output data set. Step 3: Site-level daily monitoring data are used to create 24-hr maximum daily county-level $PM_{2.5}$ dataset. Daily county-level dataset is created by retaining the maximum concentration among all monitors within the county for each monitored day. The county-level daily dataset is used to create percent of days and number of person-days with $PM_{2.5}$ levels over the daily NAAQS measures. **Creating Measures** ***Percent of days with $PM_{2.5}$ levels over the NAAQS:*** Step 4: To calculate the annual percent of days over the daily NAAQS, sum the number of days with $PM_{2.5}$ levels over the daily NAAQS and

	divide by the total number of monitored days. Multiply this exceedance fraction by 100 to get percent of days. ***Number of person-days with $PM_{2.5}$ levels over the NAAQS:*** Step 5: To calculate person-days with $PM_{2.5}$ levels over the NAAQS multiply the exceedance fraction from Step 4 by 365 to get the annual days and then multiply by the total population of the county. For $PM_{2.5}$ - days above regulatory standard indicator, tracking portal only displays counties that have year-round monitoring.
Unit	1. Exceedance days 2. Population weighted exceedance days
Geographic Scope	Contiguous United States
Geographic Scale	County (where monitors exist)
Time Period	2001-current
Time Scale	Calendar year
Rationale	According to the published literature, air pollution is associated with premature death, increased rates of hospitalization for respiratory and cardiovascular conditions, adverse birth outcomes, and lung cancer (2,3,4). Air pollution places a large economic burden on the country. In a report prepared for the American Lung Association, (2) estimated that air pollution related illness was estimated to cost approximately $100 billion annually (2) (1988 dollars) in the United States, with an estimated number of excess deaths ranging from 50,000 to 100,000 annually (3). More than half of the U.S. population, approximately 159 million persons, live in counties with unhealthy levels of air pollution in the form of either ozone or particulate matter (1). Elevated pollution levels depend on sources, transport, season geography, and atmospheric conditions. Each part of the country has its own level of pollution concentrations that can be exacerbated by many conditions, including stagnation, fire, or wind. The seasons for peak concentrations also vary between geographical regions. The Clean Air Act, which was last amended in 1990, requires EPA to set NAAQS for widespread pollutants from numerous and diverse sources considered harmful to public health and the environment. The Clean Air Act established two types of national air quality standards. Primary standards set limits to protect public health, including the health of "sensitive" populations such as asthmatics, children, and the elderly. Secondary standards set limits to protect public welfare, including visibility impairment and damage to animals, crops, vegetation, and buildings. Our indicator is based on comparing measured levels of $PM_{2.5}$ by county to the 24-hr NAAQS for $PM_{2.5,}$ which is set at 35 $\mu g/m^3$. The

	Clean Air Act requires periodic review of the science upon which the standards are based and the standards themselves. Primary air quality standards indicate the acceptable level of substances in the air before harm will occur based on proven scientific and medical research. State governments also set air quality standards. In several cases, California's standards or other benchmarks are more stringent than the EPA NAAQS. (5)
Use of the Measure	This indicator can be used to inform the public and policy makers of the degree of potential exposures within a state (for counties with monitors) during a year. For example, the percentage of days per year that $PM_{2.5}$ is higher than the NAAQS can be used to communicate to sensitive populations (such as asthmatics) the percentage of days that they may be exposed to unhealthy levels of $PM_{2.5}$; this is similar to the level used in the Air Quality Alerts that inform these sensitive populations when and how to reduce exposure. The number of person-days may be directed toward policy makers who are interested in roughly comparing population exposure between areas, to determine the areas most in need of prevention and pollution control activities.
Limitations of the Measure	The data for this indicator represent highly populated counties that have $PM_{2.5}$ monitors. As a result, the data tend to reflect urban air quality and longer-term average air quality levels. Populations in counties without monitors may also be exposed to concentrations that exceed a standard. The percentage of days during which the EPA NAAQS or other health benchmarks are exceeded does not provide information regarding the severity (maximum concentrations) of potential exposures. Even with these limitations, trends in $PM_{2.5}$ levels are a useful measure to describe public health concerns within these areas. We identify several limitations with this indicator below. This indicator is based on the percentage of high days rather than the total number of high days to highlight the fact that $PM_{2.5}$ monitors follow different operating schedules. Most operate on a once-every-third day schedule, but a small proportion operates on a daily or once-every-sixth day schedule. Because most of the monitors do not take measurements every day, the number of short-term events (e.g., days in which the NAAQS is exceeded) is uncertain, and except where $PM_{2.5}$ levels vary uniformly throughout the year, estimating short-term measures that are representative of short-term exposures over a year is complex. To address this limitation, the measure can be based on the percentage of monitored days. It should be noted that state air programs will be evaluating the daily $PM_{2.5}$ NAAQS by using a frequency-based analysis to determine whether areas within the state

	attain this NAAQS. Populations in counties without monitors may be exposed to concentrations that exceed a standard. Person-day estimates for larger, highly populated counties may be biased higher than estimates for smaller and lower populated counties. The indicator uses the highest value of all monitors in the area so that larger counties with more monitors may have a broader range of pollution values and greater potential to measure a high day than smaller counties with fewer monitors The relationship between ambient concentrations and personal exposure is largely unknown, and it varies depending upon pollutant, activity patterns, and microenvironments. Because the number of high $PM_{2.5}$ days per year can vary considerably, tracking trends over time needs to be done carefully. The variability results because: the number of high $PM_{2.5}$ days is related to meteorological factors (e.g., temperature and mixing heights), and few events occur per year, so that this type of extreme value measure will vary considerably for statistical reasons. When creating measures, we only consider monitors, which have atleast 11 observations per calendar quarter.
Data Sources	Air–quality data: EPA Air Explorer http://epa.gov/mxplorer/index.htm Population data: county population data can be found at http://www.census.gov/popest/counties/CO-EST2006-01.html
Limitations of Data Sources	Air–monitoring data provides information regarding concentrations around the specific location of each monitor. For $PM_{2.5}$ this can be a rather large area, except when unusual local emissions (agricultural fires) occur. Within-county variation in concentrations will likely exist but will not be captured in this measure. Many $PM_{2.5}$ monitors operate once-every third day (some once-every-sixth day); a few monitors operate every day.
References	1. American Lung Association. State of the Air 2004; 2004 [cited 2008 Dec 4]. Available from: http://lungaction.org/reports/sota04_full.html 2. Cannon J. The Health Costs of Air Pollution: A Survey of Studies Published 1984– 1989. New York: American Lung Association; 1990. 3. Dockery DW and Pope CA. Acute respiratory effects of particulate air pollution. Annu Rev Public Health 1994;15:107–132.

	4. Schwartz, J. Air pollution and hospital admissions for heart disease in eight U.S. counties. Epidemiology 1999;10:17–22. 5. U.S. Environmental Protection Agency. U.S. EPA Criteria Document for PM. Available from: Volume 1 VOL_I_FINAL_PM_AQCD_OCT2004.PDF and Volume 2 VOL_II_FINAL_PM_AQCD_OCT2004.PDF

CONTENT DOMAIN: AIR QUALITY
INDICATOR: ANNUAL $PM_{2.5}$ LEVEL

Type of EPHT Indicator	Hazard
Measure	1. Annual average ambient concentrations of $PM_{2.5}$ in micrograms per cubic meter (based on seasonal averages and daily measurement) 2. Annual percent of population living in counties exceeding the National Ambient Air Quality Standard (compared to percent of population living in counties that meet the standard and percent of population living in counties without $PM_{2.5}$ monitoring)
Derivation of Measure	First, EPA extracts the air quality data from the Air Quality System (AQS). EPA uses the following steps in developing the air data and measures for EPHT air quality indicators. **Processing raw data** Step 1: EPA accesses $PM_{2.5}$ daily concentrations (mcg/m^3) (parameter code '88101' and duration code '7') and supplemental data fields (e.g. latitude, longitude, elevation) for all the monitoring sites across the US from the EPA's Data Mart. The data are obtained only from monitors that are designated as Federal Reference Methods or equivalent. The data include any flagged values associated with exceptional events (high winds, fires, construction, etc) regardless of concurrence by the EPA Regional Office. Step 2: For each monitoring site, retain the maximum concentration at the site for each monitored day. The pollutant occurrence code (poc) which distinguishes multiple monitors at a single site is listed in the output data set. **Creating Measures** Step 3: The annual average measures of $PM_{2.5}$ are created using the site-level daily monitoring data. Only monitors that have at least 11 observations for each of the four calendar quarters are considered complete. The annual averages are computed only for monitors that satisfy the completeness criteria. ***Annual average ambient concentrations of $PM_{2.5}$ measure:*** Step 4: Select monitors with complete quarterly and annual data using the site-level monitoring data. Step 5: Calculate the quarterly average for each calendar quarter and then compute the annual average for each monitor with four valid quarters by averaging the quarterly averages. If a county has more than one monitor then the maximum annual average among monitors with complete (4 valid quarters) data is assigned as the annual average for that county.

	Annual percent of population living in counties exceeding the NAAQS (compared to percent of population living in counties that meet the standard and percent of population living in counties without $PM_{2.5}$ monitoring) measure: Step 6a: This is a state-level measure and uses the county-level annual average concentrations calculated in step 3. Step 6b: To calculate the annual percent of population living in counties that exceed the annual NAAQS, sum the population of all counties that exceed the annual NAAQS and divide by the total population of the state. Multiply this fraction by 100 to get percent. Step 6c: To calculate the annual percent of population living in counties that meet the annual NAAQS, sum the population of all counties that meet the annual NAAQS and divide by the total population of the state. Multiply this fraction by 100 to get percent. Step 6d: To calculate the annual percent of population living in counties that do not have complete monitors, sum the population of all counties that do not have complete monitors and divide by the total population of the state. Multiply this fraction by 100 to get percent.
Unit	1. Microgram per cubic meter (μg/m3) 2. Population proportion by hazard level
Geographic Scope	Contiguous United States
Geographic Scale	County (where monitors exist)
Time Period	2001- current
Time scale	Calendar year
Rationale	According to work conducted by Pope et al. (1), long-term exposure to $PM_{2.5}$ is related to many adverse health conditions. Each 10 ug/m^3 elevation in $PM_{2.5}$ is related to an 8% increase in lung cancer mortality, a 6% increase in cardiopulmonary mortality, and a 4% increase in death from general causes.(2) The annual average provides an indication of the long-term trends in overall $PM_{2.5}$ burden, relevant to its long-term effects. The percent of the population living in counties that exceed the standard provides an indication of the population at risk for long-term exposure. Note: these indicators are similar to indicators developed by EPA and state air quality agencies for use in air quality stats and trends analyses and reports (see www.epa.gov/airtrends**)**
Use of The Measure	This indicator can be used to inform policy makers and the public about the degree of potential exposures to fine particles within a state during a year and over time (trends). This is appropriate, as many existing health studies have found the strongest association with health outcomes based on long-

	term studies; thus, EPA developed the annual NAAQS at 15 ug/m^3. The indicator (annual average $PM_{2.5}$ concentrations) can be compared to the National Ambient Air Quality Standard (NAAQS) level of 15 ug/m^3 or other health-based standards (although not in a regulatory manner) to communicate the degree of public health concern to policy makers and the general public. (3)
Limitations of the Measure	This measure provides a general indication of the overall trend in annual $PM_{2.5}$ concentrations. It may be affected by density and placement of monitors, and coverage will vary across the country and within states. It does not directly reflect exposure. Certain geographic areas, such as those near busy roads, are likely to have higher values. When creating measures we only consider monitors that have at least 11 observations per calendar quarter. It is important to understand that this indicator is not for use–compliance determination with NAAQS or reasonable further progress toward attaining compliance. The relationship between ambient concentrations and personal exposure is largely unknown, and it varies depending upon pollutant, activity patterns, and microenvironments. The percent of state population living in counties with no $PM_{2.5}$ measurements must always be considered when attempting to estimate the proportion of population at risk.
Data Sources	EPA Air Quality System Monitoring Data, State Air Monitoring Data. http://www.epa.gov/air/data/aqsdb.html
Limitations of Data Sources	Air monitoring data provides information regarding concentrations around the specific location of each monitor. For $PM_{2.5}$ this can be a rather large area, except when unusual local emissions (agricultural fires) occur. Within-county variation in concentrations will likely exist but will not be captured in this measure. Many $PM_{2.5}$ monitors operate once-every-third day (some once-every-sixth day) and a few measure every day
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CONTENT DOMAIN: ASTHMA
INDICATOR: HOSPITALIZATIONS FOR ASTHMA

Type of EPHT Indicator	**Health Outcome**
Measures	1. Number of hospitalizations for asthma 2. Minimum daily number of hospitalizations for asthma by month 3. Maximum daily number of hospitalizations for asthma by month 4. Average daily number of hospitalizations for asthma by month 5. Crude rate of hospitalization for asthma by age group (total, 0-4, 5-14, 15-34, 35-64, and 65+) per 10,000 population 6. Age-adjusted rate hospitalizations for asthma per 10,000 population (all ages) When supported by sufficient data volume, the measures may also be reported stratified by sex, race, and/or ethnicity.
Derivation of Measures	**Numerator:** Resident hospitalizations for asthma, ICD-9-CM: 493.XX. **Denominator:** Midyear resident population. **Adjustment:** Age-adjustment by the direct method to Year 2000 U.S. Standard population
Unit	Hospital admission (categorized by discharge diagnosis)
Geographic Scope	State and national (tracking network states)
Geographic Scale	State and county
Time Period	Hospital admissions from January 1 through December 31 for each year, 2000–current
Time Scale	Daily, monthly, and annually (as appropriate for the measure)
Rationale	In 2004, 20.5 million people in the United States reported having asthma. In 2003, there were more than 574,000 hospitalizations for asthma. In 2002, there were more than 4,200 deaths in which asthma was the underlying cause. Asthma is the leading chronic health condition among children. There are also large racial, income, and geographic disparities in poor asthma outcomes. Asthma causes lower quality of life, preventable undesirable health outcomes, and large direct and indirect economic costs. Environment attributable fractions of the 1988–1994 economic costs for asthma were 39.2% for children aged <6 years and 44.4% for children aged 6–16 year, costing more than $400 million for each age group. A number of epidemiologic studies have reported associations between air pollution exposures and asthma. The association between ambient

	air particulate matter (PM) concentrations and asthma, including increased hospital admissions, is well documented. Models demonstrate 5–20% increases in respiratory-related hospital admissions per 50μg/m^3 of PM_{10} and 5–15% per 25μg/m^3 of $PM_{2.5}$, with the largest effect on asthma admissions. In the eastern United States, summer ozone pollution was associated with more than 50,000 hospital admissions per year for asthma and other respiratory emergencies. Large multi-city and individual city studies found a positive association between ozone and total respiratory hospital admissions, including asthma, especially during the warm season. Among U.S. and Canadian studies, the ozone-associated increase in respiratory hospital admissions ranged from 2-30% per 20 ppb (24 hour), 30 ppb (8-hour) or 40 ppb (1-hour) increment of ozone in warm seasons**.** In 2000, the **Institute of Medicine** concluded that allergens produced by cats, cockroaches, and house dust mites exacerbates asthma, as does exposure to environmental tobacco smoke (ETS) in pre-school aged children. A 2005 California Air Resources Board report concluded that ETS exacerbates asthma in children and adults (CARB, 2005). That report also estimated 202,300 childhood asthma episodes occur each year in the United States as a result of exposure to ETS.
Use of the Measures	Developing a standardized analytic method for asthma hospital admissions among residents in each state will provide more uniform information for multiple users at the national, state, and local levels. These measures will allow monitoring of trends over time, identify high risk groups, and inform prevention, evaluation, and program planning efforts. These measures will address the following surveillance functions: • How many hospitalizations for asthma occur in every month? • Is there a seasonal or temporal trend of asthma hospitalizations? • What's the distribution of asthma hospitalizations by place of residence? • How do hospitalizations for asthma differ between geographic areas (e.g., ZIP code, county, state, region)? • With further analysis … Are there disparities in asthma hospitalizations by factors such as age, race, ethnicity, gender, education, and/or income?

	• Which populations need targeted interventions? • When asthma data are linked with environmental variables, do the linked measures identify environmental relationships that warrant further investigation or environmental public health action?
Limitations of the Measures	Hospitalization data, by definition, do not include asthma among individuals who do not receive medical care or who are not hospitalized, including those who die in emergency rooms, in nursing homes, or at home without being admitted to a hospital, and those treated in outpatient settings. Differences in rates by time or area may reflect differences or changes in diagnostic techniques and criteria and in the coding of asthma. Reporting rates at the state and/or county level will not show the true asthma burden at a more local level (i.e., neighborhood). Differences in rates by area may be due to different sociodemographic characteristics and associated behaviors. When rates across geographic areas are compared, many non-environmental factors, such as access to medical care and diet, can affect the likelihood of a person being hospitalized for asthma. Reporting rates at the state and/or county level will not be resolved geographically enough to be linked with many types of environmental data. When looking at small geographic levels (e.g., ZIP code), users must consider appropriate cell suppression rules imposed by the data providers or individual state programs. Although duplicate records and transfers from one hospital to another are excluded, the measures are based upon events, not individuals, because no unique identifier is always available. When multiple admissions are not identified, the true prevalence will be overestimated. Even at the county level, the measures generated will often be based upon numbers too small to report or present without violating state and federal privacy guidelines and regulations. Careful adherence to cell suppression rules in cross tabulations is necessary, and methods to increase cell sizes by combining data across time (e.g., months, years) and geographic areas may be appropriate.
Data Sources	**Numerator:** State inpatient hospitalization data (using admission date)

	Denominator: US Census Bureau population data
Limitations of Data Sources	**State hospital discharge data:** The use of a measure of all asthma hospitalizations will include some transfers between hospitals for the same person for the same asthma event. Variations in the percentage of transfers or readmissions for the same asthma event may vary by geographic area and impact rates. However, efforts were made to identify and exclude transfers based on unique identifiers consisting of date of birth, zip code, gender, and encrypted social security number when available. Without reciprocal reporting agreements with abutting states, statewide measures and measures for geographic areas (e.g., counties) bordering other states may be underestimated because of health care utilization patterns. Each state must individually obtain permission to access and, in some states, provide payment to obtain the data. Veterans Affairs, Indian Health Services, and institutionalized (prison) populations are excluded. Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers to hospitalize patients Street address is not available in many states. Sometimes mailing address of patient is listed as the residence address of the patient. Patients may be exposed to environmental triggers in multiple locations, but hospital discharge geographic information is limited to residence. Since the data capture hospital discharges (rather than admissions), patients admitted toward the end of the year and discharged the following year will be omitted from the current year dataset. Data will need to be de-duplicated (i.e., remove duplicate records for the same event). There is usually a two-year lag period before data are available from the data owner.

	Census data: Available only every 10 years; thus, postcensal data must be estimated when rates for years following the census year are calculated. Postcensal estimates at the ZIP code level are not available from the Census Bureau. These need to be extrapolated or purchased from a vendor.
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Indicator Template Content Area: Asthma Indicator: Emergency Department Visits for Asthma Environmental Public Health Tracking	
Type of EPHT Indicator	Health outcome
Measures	1. Annual age-adjusted rate of emergency department visits for asthma per 10,000 population 2. Annual crude rate of emergency department visits for asthma per 10,000 population 3. Annual number of emergency department visits for asthma 4. Average Number of emergency department visits for asthma as primary diagnosis per month
Derivation of Measure(s)	*Numerator*: • Emergency Department Visits during a calendar year with asthma (ICD-9-CM 493) as the primary diagnosis (includes records for ED Visits resulting in a hospitalization) • Both inpatient and outpatient records with duplicates removed and transfers to other hospitals included *Denominator*: • Annual population estimates for state and county from U.S. Census Bureau *Adjustment*: • Age-adjustment by the direct method to the Year 2000 US Standard population • U.S. 2000 standard population by age categories from Surveillance Epidemiology and End Results (SEER), National Cancer Institute
Unit	1. Age-adjusted rate per 10,000 population 2. Rate per 10,000 population 3. Number 4. Number
Geographic Scope	State and national
Geographic Scale	Residents of jurisdiction – State, County
Time Period	Hospital admissions between January 1 to December 31, inclusive, for each year, 2000–
Time Scale	Daily, monthly, and annually (as appropriate for the measure)
Rationale	Asthma continues to be a serious public health problem that affects over 23 million people including 7 million children in the United States. In 2008,

	there were 456,000 hospitalizations and 1.8 million emergency department visits (ED) for asthma.[3] Asthma is the leading chronic health condition among children.[4] There are also large racial, income, and geographic disparities in poor asthma outcomes.[5] Asthma causes lower quality of life, preventable undesirable health outcomes, and large direct and indirect economic costs. As a chronic respiratory disease, asthma attacks interfere with everyday activities According to NCHS National Health Interview Survey, there were 10.5 million missed school days among children age 5–17 years and over 14.5 million missed work days in adult's age 18 years or over in 2008. In 2007, there were over 3,400 deaths in which asthma was the underlying cause. Environment Attributable Fractions of the 1988-1994 economic costs for asthma were 39.2% for children <6 years of age and 44.4% for 6- to 16-year-olds, costing more than $400 million for each age group. According to a more recent estimation 30% of asthma exacerbations among children were related to the environment. This was associated with an annual cost of $2.0 billion. Despite the availability of effective prevention measures, asthma associated costs are increasing. Associations between environmental exposures and asthma have been consistently demonstrated. Many outdoor air pollutants have been associated with increased asthma ED visits. There is strong scientific evidence for direct associations between increased ozone concentrations and increases in asthma ED visits, in children and adults. In one study, asthma ED visits increased by 33 percent when daily 1-hour maximum ozone concentrations exceeded 75 ppb. Associations between asthma-related ED visits and ambient air particulate matter—both PM_{10} and $PM_{2.5}$—have been repeatedly confirmed, and are especially robust for children. Other pollutants related to higher asthma ED visit totals include carbon monoxide (CO), nitrogen dioxide (NO2), and pollution from coal and petrochemical sources. Other outdoor environmental triggers for asthma ED visits in children include weed and tree pollen, and ambient temperature. Increased asthma ED visits has also been associated with environmental tobacco smoke (ETS). Asthma ED visits in children are consistently higher in the fall, co-occurring with the start of the school year; increases in asthma ED visits in children have been shown to be related to increased respiratory viral infections. The state emergency department visit data is electronically maintained and is available in almost every state in the U.S. Data stewards for 18 grantees maintain ED data. The data has comparable basic information about each visit and can provide a better tracking measure of asthma burden than inpatient hospitalization data on its own. These measures can be used to evaluate the impact of ambient air pollution on respiratory health of children and adults. Also, the measures can be used for better resource management to further reduce the asthma related

	expenditures. Combined with inpatient asthma data, emergency department data will provide more complete spatial and temporal trends for asthma. Additionally, emergency department visits are believed to be largely preventable if managed properly through the use of Asthma Action Plans and avoiding environmental triggers. This offers an outcome that may be a more measurable indicator of environmental events and of public health intervention
Use of the Measure	The development of a single analytic method for asthma emergency department visits among persons living in state will inform multiple users: *State*: • May be linked with other risk factors such as air pollution to identify susceptible populations and explore ecologic relationships • Allows for a better understanding of what the asthma surveillance data represents when interpreting number of inpatient hospitalizations • Permits the monitoring of trends temporally and spatially *National*: • It will allow for comparison across states which can be used to target interventions (especially for CDC and EPA). *Public*: • Public and concerned community members will be able to view the Tracking Network webpage and learn the annual rate of asthma emergency department visits and burden of asthma is high in their community from.
Limitations of the Measure	• Numbers may be too small in rural areas to calculate stable rates. • These measures do not account for other causes (triggers) of asthma or other reasons for visiting the ED. • The timing of the exposure may not correspond with the timing of the asthma exacerbation leading to the ED visit. • Individuals may have asthma exacerbations due to exposure to an environmental risk factor that does not result in an ED visit and thus are not captured in this measure. • Cannot combine counts from asthma ED visit measure with counts from asthma hospitalization measure because records for ED patients who are subsequently hospitalized are already counted as hospitalizations (i.e., would result in double-counting of events). • Differences in rates by time or area may reflect differences or changes in diagnostic techniques and criteria and in the coding of asthma. • Reporting rates at the state and/or county level will not show the true asthma burden at a more local level (i.e. neighborhood).

	• Differences in rates by area may be due to different socio-demographic characteristics and associated behaviors. • When comparing rates across geographic areas, a variety on non-environmental factors, such as access to medical care and diet, can impact the likelihood of persons hospitalized for asthma. • Reporting rates at the state and/or county level will not be geographically resolved enough to be linked with many types of environmental data. • When looking at small geographic levels (e.g. ZIP code), users must take into consideration appropriate cell suppression rules imposed by the data providers or individual state programs. • Although duplicate records and transfers from one hospital to another are excluded, the measures are based upon events, not individuals, because no unique identifier is always available. When multiple admissions are not identified, the true prevalence will be overestimated. • Even at the county level it can be expected that the measures generated will often be based upon numbers too small to report or present without violating state and federal privacy guidelines and regulations. Careful adherence to cell suppression rules in cross tabulations is necessary and methods to increase cell sizes by combining data across time (e.g., months, years) and geographic areas may be appropriate.
Data Sources	*Numerator:* State inpatient emergency department data *Denominator:* US Census Bureau population data
Limitations of Data Sources	*State emergency department data*: • State emergency department data • Need to obtain permission to use; not publicly available • ED visits for asthma are only one piece of a larger picture that describes asthma burden. • Veteran's Administration, Indian Health Service and institutionalized (e.g. prison) populations are excluded • In-state residents who visit in surrounding states would not be included unless states have emergency department data sharing agreements. • Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers. • Do not have a zip code for all patients. • Sometimes mailing address of patient (e.g., P.O. Box) is listed as the residence address of the patient • Patients may be exposed to environmental triggers in multiple locations, but ED geographic information is limited to residence. • Data will need to be de-duplicated using a standardized method. *Census data:* • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the

	Census Bureau. These need to be extrapolated or purchased from a vendor.
Related Indicators	• Hospitalizations for Asthma • Asthma Prevalence among Adults and Children
References	1. Summary Health Statistics for U.S. Adults: National Health Interview Survey, 2008, Tables 3 and 4. **http://www.cdc.gov/nchs/data/series/sr_10/sr10_242.pdf** 2. Summary Health Statistics for U.S. Children: National Health Interview Survey, 2008, Table 1. **http://www.cdc.gov/nchs/data/series/sr_10/sr10_244.pdf** 3. Akinbami LJ, Moorman JE, Liu X. Asthma Prevalence, Health Care Use, and Mortality: United States, 2005–2009. National Health Statistics Reports; No 32. Hyattsville, MD: National Center for Health Statistics, 2011. 4. Britton JR, Lewis SA, Epidemiology of childhood asthma. In Asthma: Epidemiology, Anti-Inflammatory Therapy and Future Trends; MA Giembycz and BJ O'Connor (Eds.),. Switzerland: Birkhäuser Verlag, 2000, pp. 25-56. 5. Gold DR, Wright R, Population disparities in asthma. Annu. Rev. Public Health 2005; 26: 89-113. 6. Lanphear BP, Aligne CA, Auinger P, et al., Residential exposures associated with asthma in US children. Pediatrics 2001; 107: 505-511. 7. Lanphear BP, Kahn RS, Berger O, et al., Contribution of residential exposures to asthma in US children and adolescents. Pediatrics 2001; 107: e98. 8. Redd SC. Asthma in the United States: Burden and current theories. Environ Health Perspect 2002; 110 (Suppl 4): 557-60. 9. Arif AA, Rohrer JE, Delclos GL. A population-based study of asthma, quality of life, and occupation among elderly Hispanic and non-Hispanic whites: a cross-sectional investigation. BMC Public Health 2005; 5: 97. 10. Pruss-Ustun A, Corvalan C. Preventing disease through health environments. Towards an estimate of the environmental burden of disease. World Health Organization. 2006. 11. Landrigan PJ, Schechter CB, et al. Environmental Pollutants and Disease in American Children: Estimates of Morbidity, Mortality, and Costs for Lead Poisoning, Asthma, Cancer, and Developmental Disabilities. Environ Health Perspect. 2002:110:721-728. 12. Bahadori K, Doyle-Waters MM, Marra C, Lynd L, Alasaly K, Swiston J, FitzGerald JM. Economic burden of asthma: a systematic review. BMC Pulm Med. 2009 May 19;9:24.

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CONTENT DOMAIN: BIRTH DEFECTS
INDICATOR: PREVALENCE OF BIRTH DEFECTS

Type of EPHT Indicator	Health Outcome
Measure	Five year prevalence rates of 12 birth defects per 10,000 live births. 1. Anencephaly 2. Spina bifida (without anencephaly) 3. Hypoplastic left heart syndrome 4. Tetralogy of Fallot 5. Transposition of the great arteries (vessels) 6. Cleft lip with or without cleft palate 7. Cleft palate without cleft lip 8. Hypospadias (male births only) 9. Gastroschisis 10. Upper limb deficiencies 11. Lower limb deficiencies 12. Trisomy 21 o Among mothers <35 years of age at delivery o Among mothers ≥35 years of age at delivery Five year prevalence rates at the state level are reported stratified by maternal age at delivery, maternal ethnicity/race, and infant sex. Five year prevalence rates at the county level are reported stratified by one demographic variable at a time: maternal age at delivery, maternal ethnicity/race, or infant sex.
Derivation of Measure(s)	Denominator is composed of all live-born infants in geographic region of interest during a calendar year. Numerator is composed of all live-born infants, fetal deaths (where available), and terminations (where available) with birth defect 'X' in the geographic region of interest during a calendar year. For states that ascertain fetal deaths and/or terminations, two sets of birth prevalence estimates are to be calculated for each birth defect—one including and one excluding fetal deaths and/or terminations. Diagnosis of cases may be made up to one year of age—ascertainment may be at any time.
Unit	Defect present at birth
Geographic Scope	State and National (tracking network states)
Geographic Scale	State, county
Time Period	1998-current
Time Scale	Five year

Rationale	Birth defects pose a significant public health problem. One in 33 babies is born with a structural birth defect in the United States. Birth defects are a leading cause of infant mortality; they are also responsible for considerable morbidity and disability with enormous economic and social costs. A lifetime of medical care and special education for a single child can cost more than $500,000. Approximately 60% of birth defects are of unknown etiology. The ambient environment remains a source of great public concern, but few environmental exposures have been well-studied. Most birth defects likely will be explained by a complex interaction between genetic predispositions and environmental factors. However, before the ability to conduct studies to explore these interactions is achieved, linking birth defects–outcome data with environmental hazard or exposure data is critical. The first step in effecting successful linkages of these data is the existence of high-quality birth defects prevalence data for which the geospatial and temporal patterns and distributions can be monitored. The environmental public health tracking (EPHT) initiative is well-positioned to bring together birth prevalence data from its state partners to begin analyses of these patterns, which will provide important clues to public health officials and researchers.
Use of the Measure	The basic procedure for calculating birth prevalence is the same for all the suggested birth defects. Once the input data are appropriately prepared, birth prevalence will be calculable for all defects at the same time. State Allow for consistent and rapid method for calculating and displaying (using GIS) prevalence at selected geographical areas (i.e., county level). Allow for a better understanding of spatial and temporal patterns of selected birth defects. National Allow for comparison of birth prevalence across states, which can be used to target interventions. Any comparison of birth prevalence, however, will need to account for the variability in data collection methods between state surveillance systems. (See "Limitations of Data Sources" below and introductory text in appended team recommendations). Local Concerned community members will be able to view the tracking network Web page to see the birth prevalence of selected birth defects (while protecting confidentiality) at specified geographical areas. A

	public health message will help interpret the results and provide more information on selected birth defects and prevention measures (i.e., folic acid for prevention of neural tube defects, smoking and clefts, alcohol and fetal alcohol syndrome, and known teratogenic medications). A link to a list of known teratogens can be provided to users.
Limitations of the Measure	Ideally, incidence rates would be used instead of birth prevalence to measure birth defects occurrence. The numerator of the incidence would be the number of new cases of birth defect A in an area and time period and the denominator would be the number of conceptions at risk for developing birth defect A in that area and time period. Because both the number of conceptions and the number of cases "lost" through spontaneous abortions (as well as terminations and later fetal losses depending on the source of ascertainment for the specific surveillance system) is unknown, incidence cannot be calculated. Birth prevalence is the only appropriate measure that can be reported for birth defects occurrence. It is not feasible, at this time, to recommend that individual-level birth defects surveillance data be made available on even a secure national portal. Most states have strict guidelines with respect to confidentiality, and even the publication of birth prevalence data based on <5 cases in a geographic region is generally not done.
Data Sources	State birth defects surveillance systems: The data sources that contribute to birth defects surveillance systems include the following (this varies by system type): • Vital records • Hospital records (discharge summaries or disease indices, nursery logs, NICU logs) • Administrative databases (Medicaid, state hospital discharge, HMO) • Specialty data sources (specialty clinics, programs for children with special health care needs) • Prenatal diagnostic centers or genetics clinics • Clinical examination • Local or national laboratories for cytogenetic testing Denominator data will come from state vital records—number of live births, by year, by maternal age, and by race/ethnicity. These data may be aggregated and provided to the birth defects surveillance system for calculating birth prevalence, or it may be made available on an individual level to the birth defects surveillance system. This varies by state.
Limitations of Data Sources	All states in the US do not have a birth defects surveillance program. Among those that do, there is significant variability between surveillance systems. These include:

	• Ascertainment method (active, passive, passive with follow-up/verification) ○ Primary differences are with data sources, coding, availability of verbatim description, and case verification • Ascertainment of spontaneous fetal deaths and variability in gestational age for inclusion. • Ascertainment of prenatally diagnosed cases and elective terminations • Case definitions • Classification as isolated, multiple, or syndromic Data for specific birth defects may not be collected by each state or may only have been collected recently, limiting historical data for that birth defect. Address data tend to be based on address at delivery, not conception (more relevant time period for birth defects-related exposure). Approximately 50% of birth defects surveillance systems do not geocode their address data.

CONTENT DOMAIN: CANCER
INDICATOR: INCIDENCE OF SELECTED CANCERS

Type of EPHT Indicator	Health Outcome
Measure	1. Annual number of cases for selected cancers, by state 2. Annual age-adjusted incidence rate for selected cancers per 100,000 population or per 1,000,000 for childhood cancers (<15 & <20 years of age), by state 3. Average annual number of cases for selected cancers over five year period, by county 4. Age-adjusted incidence rate for selected cancers per 100,000 population over a five year period, by county Measures for each of the selected cancer types are provided by sex and race/ethnicity groups. Some measures are also provided by age group as defined below.
Derivation of Measure(s)	Numerator is composed of counts of unique invasive primary incident cases of cancer "x" (bladder cancer also includes in situ) diagnosed during a specified calendar year or five year period within residents of a specified geographic region. Incident cancer data were originally collected by state and regional cancer registries. It is proposed that data for the National EPHT Network be obtained from the NCI and CDC joint venture, State Cancer Profiles. Denominator is composed of counts of the population residing in the geographic region of interest during a specified calendar year or five year period. Population data were originally collected by the U.S. Census. For these national cancer indicators, population data is obtained from the NCI and CDC's State Cancer Profiles, which use U.S. Census data as modified by SEER. Rates will be age-adjusted to year 2000 U.S. standard population. Cancer types: **Mesothelioma**: SEER Recode B 36010. ICD-O-3 codes: histologies 9050-9055. Malignant cases: ICD behavior code '3'. **Melanoma of the skin***: SEER Recode B 25010. ICD-O-3 codes: primary site C440-C449, histologies 8720-8790. Invasive melanoma (behavior code '3').

	Liver & Intrahepatic Bile Duct: SEER Recode B 21071, 21072. ICD-O-3 codes: primary sites C220, C221; excludes histologies: 9590-9989, 9050-9055, and 9140. Malignant cases: ICD behavior code '3'. **Kidney & Renal Pelvis**: SEER Recode B 29021, 29022. ICD-O-3 codes: C649, C659; excludes histologies: 9050-9055, 9140, 9590-9989. Malignant cases: ICD behavior code '3'. **Oral Cavity & Pharynx**: SEER Recode B Site Groups 20010-20100 (20010, 20020, 20030, 20040, 20050, 20060, 20070, 20080, 20090, 20100). ICD-O-3 site codes: C000-C009, C019-C069, C079-C119, C129-C140, C142-C148; excludes histologies 9050-9055, 9140, 9590-9989. **Esophageal**: SEER Recode B 21010. ICD-O-3 site codes: C150-C159; excluding histologies 9050-9055, 9140, 9590-9989. **Pancreas**: SEER Recode B 21100. ICD-O-3 codes: C250-C259; excluding histologies 9050:9055, 9140, 9590:9989. **Larynx**: SEER Recode B 22020. ICD-O-3 codes: C320-C329; excluding histologies 9050:9055, 9140, 9590:9989. **Lung & Bronchus**: SEER Recode B 22030. ICD-O-3 Site codes C340-C349; excludes histologies 9050-9055, 9140, 9590-9989. **Breast** (female)**: SEER Recode B 26001. ICD-O-3 Site codes C500-C509; excludes histologies 9050-9055, 9140, 9590-9989. **Bladder**: SEER Recode B 29010. ICD-O-3 Site codes C670-C679; excludes histologies 9050-9055, 9140, 9590-9989. [includes invasive and in-situ] **Brain & ONS***:** SEER Recode B 31010, 31040. ICD-O-3 Site codes C700-C709, C710-C719, C720-C729; excludes histologies 9050-9055, 9140, 9590-9989. **Thyroid**: SEER Recode B 32010. ICD-O-3 Site codes C739; excludes histologies 9050-9055, 9140, 9590-9989. **Non-Hodgkin Lymphoma**: SEER Recode B 33041, 33042. ICD-O-3 codes: histology 9590-9596, 9670-9671, 9673, 9675, 9678-9680, 9684, 9687, 9689-9691, 9695, 9698-9702,9705,9708-9709, 9714-9719, 9727-9729; histology 9823 or 9827 in all sites except C420, C421, C424.

	Leukemia: SEER Recode B 35011, 35012, 35013, 35021, 35022, 35023, 35031, 35041, 35043. ICD-O-3 codes: ALL – histology 9826,9835-9837; Other lymphocytic – histology 9820, 9832-9834, 9940; Acute monocytic – histology 9891; CML – histology 9863, 9875, 9876, 9945, 9946; Other – histology 9860, 9930, 9801, 9805, 9931, 9733, 9742, 9800, 9831, 9870, 9948, 9963, 9964. Site codes C420, C421, C424 – histology 9827. (Also include codes for CLL and AML.) **Chronic Lymphocytic Leukemia (CLL)**: SEER Recode B 35012. ICD-O-3 codes: C420, C421, C424 with histology 9823. **Acute Myeloid Leukemia** (**AML)**: SEER Recode B 35021. ICD-O-3 codes: histology 9840, 9861, 9866, 9867, 9871-9874, 9895-9897, 9910, 9920. **Child cancers:** SEER ICCC3 childhood cancer codes http://seer.cancer.gov/iccc/iccc3.html NOTE: SEER Recode B (Dec 2003) http://seer.cancer.gov/siterecode_b/icdo3_d12192003/ Tobacco-related cancers: consistent with SEER Recode B, CWG Cancer Team NCDM specifies Histology Exclusions 9050-9055 (Mesothelioma), 9140 (Kaposi Sarcoma), 9590-9989 (Lymphoma, Leukemia, Miscellaneous). * Grantee portals may choose to additionally display In-situ cases, both disaggregated and aggregated with invasive cases ("All combined"). ** Breast – Malignant/invasive only: The NEPHTN Metadata state "Counts and rates for in situ breast cancer cases among women are presented; these are reported separately and are not included in counts or rates for the "All Sites" category." (CDC-EHTB plans to delete this sentence from national portal Metadata.) The NCDM states "Numerator is composed of counts of unique invasive primary incident cases of cancer …" (in "Derivation of Measure"). Grantee portals may choose to additionally display In-situ cases, both disaggregated and aggregated with invasive cases ("All combined"). *** Brain/ONS – Malignant/invasive only: The NEPHTN Metadata state "Incidence data on nonmalignant primary brain and central nervous system (CNS) tumors are available on this Web site." (CDC-EHTB plans to delete this sentence from national portal Metadata.) The NCDM states "Numerator is composed of counts of unique invasive primary incident cases of cancer …" (in "Derivation of Measure").
Unit	Newly reported cancer case
Geographic Scope	State and national (tracking network states)
Geographic Scale	State and county.
Time Period	2000-current
Time Scale	Annual and 5 year period
Rationale	Approximately 1.4 million Americans are expected to be diagnosed with cancer during 2007. The National Cancer Institute (NCI) estimated that in January 2003, there were approximately 10.3 million living Americans with a history of cancer. The risk of being

	diagnosed with cancer increases as a person ages, and 77 % of all cancers are diagnosed in Americans age 55 years or older. Cancer, a diverse group of diseases characterized by the uncontrolled growth and spread of abnormal cells, is believed to be caused by both external and internal risk factors. Major risk factors for cancer include tobacco use, diet, exercise, and sun exposure (Clapp, Howe, Jacobs). For example, male smokers are about 23 times more likely to develop lung cancer than male non-smokers. Researchers have also identified genetic risks for cancer. Female first degree relatives (mother, sisters, and daughters) of women with breast cancer are about twice as likely to develop breast cancer as women who do not have a family history of breast cancer (*Cancer Facts and Figures, 2007*; ACS, 2007). However, the etiology of many cancer types is not well established. The physical environment (e.g., air quality, chemical pollution, and water quality) remains a source of great public concern but few community-level environmental exposures have been well-studied. Studies of occupational cohorts have identified numerous suggestive epidemiological associations between certain occupational exposures and elevated cancer rates. After reviewing the evidence regarding the causes of cancer in the United States, Doll and Peto published a seminal article in 1981 estimating that 35% of all U.S. cancer deaths were attributable to diet, 30% to smoking, 4% to occupation, and 2% to pollution. While some authors have agreed with Doll and Peto (Ames and Gold 1998), and others have cautioned against their approach: "there is substantial evidence that occupational and environmental exposures contribute to the burden of cancer" (Clapp, Howe, and Jacobs 2006). One way to assess cancer burden is to study geographic variation. In recent years, geographic information systems (GIS) have become an important tool for health and environmental research. GIS can extend the analysis of data beyond simple mapping by enabling the linkage, visualization, and analysis of multiple layers of health and environmental data from both spatial and temporal perspectives. One important use of geographic analysis of health data is in the analysis of regional variations in cancer mortality and incidence. The National Cancer Institute's *Atlas of Cancer Mortality for U.S. Counties: 1950–1969* (Mason et al. 1975), represented the first effort to map cancer mortality data at the county level throughout the United States. In 1999, the national level analysis of cancer mortality was updated by the NCI (*Atlas of Cancer Mortality in the United States, 1950–94*, Devesa et al. 1999). More recently, multiple Web-

	based data query systems have made U.S. cancer incidence and mortality datasets and or maps available at the county (NCI/CDC State Cancer Profiles: http://statecancerprofiles.cancer.gov/; NCI SEER data: http://seer.cancer.gov/data/; NJ DHSS cancer online: http://www.cancer-rates.info/nj/) and/or state level (NAACCR CINA+ Online: http://www.cancer-rates.info/naaccr/ ; CDC U.S. Cancer Statistics: http://apps.nccd.cdc.gov/uscs/).
Use of the Measure	At the local and state levels, the EPHT Network will: Allow interested persons to obtain information on environmental exposures (air pollution and drinking water quality) and cancer or other health outcomes (birth defects, asthma, and birth weight) for a selected geographic area and time interval. Standard suppression rules will be used to prevent the release of information that might reveal the identity of any person diagnosed with cancer. Public health messages will help interpret the results and provide linkages to additional information on cancer prevention, cancer etiology, and cancer treatment options. While many of these diverse health and environmental datasets are already available to the public, they are not currently available through "one-stop-shopping" via the Internet. Improve access to metadata regarding multiple health outcome datasets and environmental exposure datasets for public health practitioners and researchers. Enhanced access will provide better understanding of the strengths and limitations of the available datasets and may increase the use of the collected data. Allow for a better understanding of spatial and temporal patterns of selected cancers suggested to be linked to environmental exposures within states. At the national level, the EPHT Network will: Enhance the opportunity for multi-state epidemiological research by improving access to cancer incidence rates and environmental exposure information. This could be particularly helpful for uncommon cancer types or sub-types whereby incidence is too small for meaningful ecological studies in individual states.
Limitations of the Measure	Counts and rates will be calculated based upon residential address at time of diagnosis. No information is available on prior residences. Geocoding accuracy, level of geocoding, and geocoding completeness may vary by time and space. This could potentially create geographically non-random errors in calculated rates of cancer. No personal exposure information will be available, including smoking history, diet, lifestyle, or history of cancer.

	Data that will reveal the identity of any individual diagnosed with cancer can not be released. Suppression rules will govern the release of small case counts. No information will be available on the latency of cancer cases.
Data Sources	National Cancer Institute, Surveillance Epidemiology and End Results; CDC National Program of Cancer Registries
Strengths and Limitations of Data Sources	All of the 16 states and the 1 city participating in the EPHT Network are working with their state and/or regional cancer registry program(s). Registry training, data collection, data coding, data cleaning, and quality control programs are highly standardized and subject to annual evaluation. Documentation is available online from the North American Association of Centralized Cancer Registries (NAACCR). (http://www.naaccr.org/index.asp?Col_SectionKey=7&Col_ContentID=135). State cancer registry programs may vary, however, regarding the availability and quality of residential address information collected and completeness of geocoding efforts.

CONTENT DOMAIN: CARBON MONOXIDE
INDICATOR: HOSPITALIZATIONS FOR CARBON MONOXIDE POISONING

Type of EPHT Indicator	Health Outcome/Exposure
Measures	1. Number of hospitalizations for carbon monoxide (CO) poisoning 2. Crude rate of hospitalization for CO poisoning per 100,000 population 3. Age-adjusted rate of hospitalization for CO poisoning per 100,000 population
Derivation of measure	Numerator: Resident hospitalizations for CO poisoning that meet the 1998 CSTE case definition for public health surveillance for a "Confirmed" or "Probable" case of acute CO poisoning in administrative data sets. Frequencies for three unique groups: 1. Unintentional, non-fire related 2. Unintentional, fire-related 3. Unknown intent Denominator: Midyear resident population Adjustment: Age-adjustment by the direct method to year 2000 US Standard Population
Unit	Hospital admission (categorized by discharge diagnosis)
Geographic Scope	State and national (tracking network states)
Geographic Scale	State; county when feasible
Time Period	2000-current
Time Scale	Calendar year

Rationale	Carbon monoxide (CO) is an odorless, colorless gas that usually remains undetectable until exposure results in injury or death. Each year in the United States, an estimated 10,000 persons seek medical attention or lose at least one day of normal activity because of CO intoxication. There is limited information on CO hospitalization. In Florida, 1,494 were hospitalized with a diagnosis of CO poisoning from 1999–2007. Out of which 10% (n=143) were unintentional fire-related, 33% (n=493) were unintentional non-fire-related, and 17% (n=256) were from unknown cause of CO poisoning. During 2000–2009, a total of 68,316 CO exposures were reported to poison centers across United States. Persons hospitalized with CO poisoning are among the most severely poisoned cases. Unintentional CO poisoning is almost entirely preventable. These data are available in most states.
Use of the Measure	These data can be used to assess the burden of severe CO poisoning, monitor trends over time, identify high-risk groups, and enhance prevention, education, and evaluation efforts.
Limitations of the Measure	Hospitalization data, by definition, do not include: persons treated in outpatient settings (e.g., emergency departments, urgent care clinics, clinicians' offices or hyperbaric chambers but not hospitalized); persons who call poison control centers and are managed at the scene, and/or receive medical care but are not hospitalized; persons who do not seek any medical care; or persons who die immediately from CO exposure without medical care.
Data Sources	**Numerator:** State inpatient hospital discharge data **Denominator:** U.S. Census Bureau population data
Limitations of the Data Source	The use and quality of ICD9-CM coding varies across jurisdictions; this is especially true of the codes used to describe how an injury occurs, indicated as E-codes. Examples of this variation include: • The number of diagnostic fields available to specify cause of the injury; • Whether E-codes are mandated; • The completeness and quality of E-coding; for example, the reliability of ICD-9-CM coding to distinguish between cases of CO poisoning that are intentional or unintentional, and/or fire-or non-fire related The toxic effects of CO exposure are nonspecific and easily misdiagnosed when CO exposure is not suspected. These misdiagnosed cases will not be counted. These data usually do not include data from federal facilities such as Veteran's Administration hospitals.

	These data usually include only cases of state residents treated within the state. Health-care access is not restricted to these political boundaries so patients hospitalized for CO poisoning in another state may not be counted in their own state. Likewise, they may not be counted in the jurisdiction in which they were treated. Currently, few states have access to, or agreements to obtain, hospital discharge data from other states where their state residents may be hospitalized. To the extent that patients are treated out of state, there is undercounting of the rate of state residents poisoned by CO. Differences in rates between jurisdictions may reflect differences in hospital admissions practices for treating persons with severe CO poisoning. For example, some facilities may routinely admit all patients treated with hyperbaric oxygen; other facilities may release patients treated with hyperbaric oxygen after the treatment is completed if they are in stable condition. Race and ethnicity are important risk factors for CO poisoning, yet, many hospitalization data sets do not contain these data. Those that do may have data quality issues. *Census data:* • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the Census Bureau. These need to be extrapolated or purchased from a vendor.
References	**1.** Centers for Disease Control and Prevention, Perspectives in Disease Prevention and Health Promotion Carbon Monoxide Intoxication—A Preventable Environmental Health Hazard MMWR, 1982. 31(39): p. 529–31. **2.** Centers for Disease Control Prevention, Carbon monoxide exposures—United States, 2000–2009. MMWR, 2011. 60(30): p. 1014–7. **3.** Harduar-Morano, L. and S. Watkins, Review of unintentional non-fire-related carbon monoxide poisoning morbidity and mortality in Florida, 1999–2007. Public Health Rep, 2011. 126(2): p. 240–50. 4. King, M.E. and S.A. Damon, Attitudes about carbon monoxide safety in the United States: results from the 2005 and 2006 Health Styles Survey. Public Health Rep, 2011. 126 Suppl 1: p. 100–7.

CONTENT DOMAIN: CARBON MONOXIDE
INDICATOR: EMERGENCY DEPARTMENT VISITS FOR CARBON MONOXIDE POISONING

Type of EPHT Indicator	Health Outcome
Measures	1. Number of emergency department (ED) visits for CO poisoning 2. Crude rate of ED visits for CO poisoning per 100,000 population 3. Age-adjusted rate of ED visits for CO poisoning per 100,000 population
Derivation of measure	Numerator: Resident emergency department visits for CO poisoning that meet the 1998 CSTE case definition for public health surveillance for a "Confirmed" or "Probable" case of acute CO poisoning in administrative data sets. Frequencies for three unique groups: 1. Unintentional, non-fire related 2. Unintentional, fire-related 3. Unknown intent Denominator: Midyear resident population *Adjustment*: Age-adjustment by the direct method to year 2000 US Standard Population
Unit	Emergency department visit
Geographic Scope	State and national (tracking network states)
Geographic Scale	State
Time Period	2000-current
Time Scale	Calendar year

Rationale	Carbon Monoxide (CO) poisoning is preventable; nonetheless, unintentional, non-fire-related CO poisoning is responsible for approximately 15,000 emergency department visits and nearly 500 deaths annually in the United States. During 2004–2006, an estimated average of 20,636 ED visits for nonfatal, unintentional, non-fire-related CO exposures occurred each year. Approximately 73% of these exposures occurred in homes, and 41% occurred during winter months (December–February). Prevention efforts targeting residential and seasonal CO exposures can substantially reduce CO-related morbidity. During 2000–2009, a total of 68,316 CO exposures were reported to poison centers across United States. Persons admitted to emergency departments and diagnosed with CO poisoning range from suspected exposure to severe poisonings that may result in treatment and release, hospitalization, or death. Emergency department visits represent patients not counted in other clinical settings. Unintentional CO poisoning is usually preventable. Emergency department data are available in more than 50% of the states and that number is increasing.
Use of the Measure	These data can be used to assess the burden of CO poisoning and to monitor trends over time as well as to identify high risk groups, and enhance prevention, education, and evaluation efforts.
Limitations of the Measure	Measures based on emergency department data alone may underestimate its prevalence because these data may not include persons that are managed at the scene, persons who do not seek any medical care, persons admitted without first visiting an emergency department, or persons who die immediately from CO exposure without medical care.
Data sources	**Numerator:** State emergency department visit data **Denominator:** U.S. Census Bureau population data
Limitations of the Data Source	Emergency department data have limitations for comparisons across jurisdictions because the use and quality of ICD-9-CM coding may vary across jurisdictions; this is especially true of the codes used to describe how an injury occurs, indicated as E-codes. Examples of this variation include: • The number of diagnostic fields available to specify cause of the injury vary from nine to unlimited (in some states reaching more than 100); • E-codes are mandated in some jurisdiction but not in others;

	• The completeness and quality of E-coding vary by hospital as well as jurisdiction. In addition, the reliability of ICD-9-CM coding to distinguish between cases that are intentional or unintentional, fire-related, or of unknown intent is undocumented; • States are inconsistent in the use of intent codes. The toxic effects of CO exposure are nonspecific and easily misdiagnosed when CO exposure is not suspected. These misdiagnosed cases will not be counted. These data usually do not include data from federal facilities such as Veteran's Administration hospitals. These data usually include only cases of state residents who were treated within the state. Health care access is not restricted to these political boundaries so people discharged from the emergency department for CO poisoning in another state will neither be counted in their own state nor in the jurisdiction in which they were treated. Currently, few states have access to, or agreements to obtain, their emergency department data from other states in which their residents may have received treatment. To the extent that patients are treated out of state, there is undercounting of the rate of residents poisoned by CO. Regional variation between emergency departments in diagnosing CO poisoning may exist. Many emergency department visit data sets do not contain race or ethnicity information and those that do may have data quality issues. Yet, these characteristics are known risk factors for CO poisoning. *Census data:* • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the Census Bureau. These need to be extrapolated or purchased from a vendor.
References	**1.** Centers for Disease Control and Prevention. Perspectives in Disease Prevention and Health Promotion Carbon Monoxide Intoxication—A Preventable Environmental Health Hazard MMWR Morb Mortal Wkly Rep 1982;31(39):529–31. **2.** Centers for Disease Control Prevention. Nonfatal, unintentional,

	non-fire-related carbon monoxide exposures—United States, 2004-2006. MMWR Morb Mortal Wkly Rep 2008;57(33):896–9. **3.** Hampson NB. Emergency department visits for carbon monoxide poisoning in the Pacific Northwest. J Emerg Med 1998;16(5):695–8. 4. Kao LW, Nanagas KA. Carbon monoxide poisoning. Emerg Med Clin North Am 2004;22(4):985–1018. 5. Partrick M, Fiesseler F, Shih R, Riggs R, Hung O. Monthly variations in the diagnosis of carbon monoxide exposures in the emergency department. Undersea Hyperb Med 2009;36(3):161–7.

CONTENT DOMAIN: CARBON MONOXIDE
INDICATOR: CARBON MONOXIDE POISONING MORTALITY

Type of EPHT Indicator	**Health Outcome**
Measures	1. Number of deaths from CO poisoning 2. Crude rate of death from CO poisoning per 100,000 population 3. Age-adjusted rate of death from CO poisoning per 100,000 population
Derivation of measure	Numerator: Resident deaths from CO poisoning for three unique groups: 1. Unintentional, non-fire related 2. Unintentional, fire-related 3. Unknown intent Denominator: Midyear resident population Adjustment: Rates age-adjusted by the direct method to the Year 2000 U.S. Standard Population
Unit	Deaths due to CO poisoning
Geographic Scope	State and National
Geographic Scale	State
Time Period	2000-current
Time Scale	Calendar year

Rationale	CO is an odorless, colorless gas that usually remains undetectable until exposure results in injury or death. Carbon monoxide (CO) poisoning is a leading cause of unintentional poisoning deaths in the United States. CO poisoning is preventable; nonetheless, unintentional, non–fire-related CO poisoning is responsible for approximately 15,000 emergency department visits and nearly 500 deaths annually in the United States. During 1999–2004, CO poisoning was listed as a contributing cause of death on 16,447 death certificates in the United States and 2,631 (16%) were classified as both unintentional and non-fire-related deaths. The annual average age-adjusted death rate in the U.S. was 1.5 deaths per million persons. The US Consumer Product Safety Commission's historical data indicate that there is a statistically significant increasing trend in non-fire CO fatalities from 1999 through 2007. In 2007, 183 unintentional consumer product–related, non–fire-related CO deaths were reported. Out of which heating systems were associated with the largest percentage of non-fire CO poisoning fatalities at 38 percent (estimated 70 deaths); Engine-Driven Tools-related CO fatalities were also associated with 38 percent (69 deaths), and the remaining six product categories [Charcoal Grills or Charcoal (7 deaths); Ranges, Ovens (7 deaths); Water Heaters (3 deaths); Grills, Camp Stoves (3 deaths); Other Products (1 death); and Multiple Products (24 deaths)] combined were associated with a total of 25 percent. Death is the most severe outcome of CO poisoning. Unintentional CO poisoning deaths are almost entirely preventable. Most localities have access to data on their resident deaths.
Use of the Measure	These data can be used to assess the burden of severe CO poisoning, monitor trends over time, and enhance prevention, education, and evaluation efforts.
Limitations of the Measure	This measure understates the burden of CO poisoning because most cases do not result in death. Rates can be misleading (i.e., do not reflect risk of occurrence) if a relatively large proportion of deaths occur to non-residents poisoned within the jurisdiction (they are excluded from the rate calculation). Death investigation laws vary by locale.
Data Sources	**Numerator:** Death certificate records from vital statistics agency **Denominator:** Population counts or estimates from the U.S. Bureau of the Census

Limitations of the Data Source	Death investigation laws vary by locale. In addition, variations may occur between localities in how medical examiners/coroners/physicians assign intentionality. Thus an area where the ME/coroner/physician is disinclined to attribute a CO poisoning to suicide will have a higher unintentional CO poisoning death rate than a comparable locale. Finally, CO poisonings that are unrecognized by the ME/coroner/physician will be attributed to other causes.
References	**1.** Centers for Disease Control Prevention, Carbon monoxide--related deaths--United States, 1999-2004. MMWR Morb Mortal Wkly Rep, 2007. 56(50): p. 1309-12. **2.** Centers for Disease Control Prevention, Unintentional non-fire-related carbon monoxide exposures--United States, 2001-2003. MMWR Morb Mortal Wkly Rep, 2005. 54(2): p. 36-9. **3.** Mott, J.A., et al., National vehicle emissions policies and practices and declining US carbon monoxide-related mortality. JAMA, 2002. 288(8): p. 988-95. 4. Hnatov, MV. Non-Fire Carbon Monoxide Deaths Associated with the Use of Consumer Products 2007 Annual Estimates. Bethesda, MD: US Consumer Product Safety Commission. Available at: http://www.cpsc.gov/library/foia/foia11/os/co10.pdf. Accessed July 18, 2012

CONTENT DOMAIN: CARBON MONOXIDE
INDICATOR: REPORTED EXPOSURE TO CARBON MONOXIDE

Type of Indicator	**Exposure, Health Outcome**
Measures	1. Number of unintentional CO exposures reported to poison control centers by resulting health effect and treatment in a healthcare facility 2. Crude rate of unintentional CO exposures reported to poison control centers per 100,000 population by resulting health effect and treatment in a healthcare facility
Derivation of measures	Number of reported cases of unintentional carbon monoxide exposure stratified by presence of subsequent health effect and consequential treatment in a healthcare facility Denominator used is Midyear resident population
Unit	Reported exposure to CO
Geographic Scope	State and national (tracking network states)
Geographic Scale	County
Time Period	2000- current
Time scale	Annual
Rationale	PCCs serve the public and healthcare providers in the management of actual or potential exposure to hazardous substances, including CO. PCC calls are fielded by certified specialists in poisoning information (SPIs), and recorded in a standard electronic format. Regional PCC data are centralized nationally by AAPCC annually. PCC calls provide information about CO exposure that may not otherwise be captured in hospital discharge data or emergency department data. These include events where CO exposure was detected but did not result in symptoms, where symptoms were mild and did not require follow-up in a health care facility, and where the event resulted in symptoms but the patient refused to seek medical treatment. Two state-based evaluations (Connecticut [1] and Wisconsin [2]) found minimal overlap between persons using PCCs

	and persons treated in emergency departments. As such, tracking of PCC calls in addition to indicators of mortality, hospitalizations, and emergency room visits provides a more complete picture of the public health burden of CO exposure.
Use of the Measure	These data may be used to estimate the population's exposure to CO and to monitor trends over time. They may also be used to estimate symptomatic CO exposures among exposed persons who may not be treated in a health care facility and therefore would not be captured in other health outcome datasets.
Limitations of the Measure	Exposure status should not be considered confirmed. In some cases, ambient air sampling results or the patient's lab results may be reported in the case notes but only when this information is available or provided to the SPI. In addition, it should be noted that because they may contain identifiable and sensitive information, SPI notes are removed from case records by regional PCCs before submitting to the AAPCC and are therefore unavailable at the national level. Not all potentially hazardous CO exposures will be captured by PCC calls. For example, cases of moderately elevated exposure in the home are unlikely to be recognized if there are no acute symptoms and a CO alarm is not installed. Moreover, knowledge, attitudes, and practices around the use of PCCs likely vary both within and across jurisdictions. In the event of suspected exposure, callers may first notify their local fire department or call 911 or even their utility provider; in either case, the regional PCC may not be simultaneously notified. Practices by health care providers that use PCCs are also likely to vary from one jurisdiction to another. Generally speaking, healthcare providers use the PCC as a resource in the diagnosis and treatment of poisonings; in addition, in New York City, where CO poisoning was designated as an immediately reportable condition in 2004, the PCC plays an integral role in the management of reports from healthcare providers and in the rapid referral of the fire department for investigation at the site of exposure for the prevention of secondary cases (3). For these reasons, caution should be exercised in comparing rates of reported exposure across states.
Data Sources	**Numerator:** PCC calls (usually in standard Toxicall database) **Denominator:** U.S. Census Bureau population data
Limitations of the Data Sources	SPIs are not required to collect patient state/ZIP code unless the patient is the caller. Using caller state/ZIP code to determine

	residency may cause the number of calls pertaining to state residents to be overestimated—for example, when the caller is an out-of-state health care provider. The number of cases may differ slightly between datasets obtained directly from the state's PCC and the national AAPCC dataset for that state; this is typically due to calls that are re-routed to another state when the state's PCC is overloaded. The AAPCC national dataset is corrected for such instances. Age adjustment is not recommended since age is often estimated (such as "Adult > 19" or "50s").
References	1. Toal B. Comparison of Three CO Databases in Connecticut [PowerPoint presentation]. EPHT Web Seminar; 2006 June. 2. Bekkedal M, Sipsma K, Stremski ES, Malecki KC, Anderson HA. Evaluation of five data sources for inclusion in a statewide tracking system for accidental carbon monoxide poisonings. WMJ. 2006 Mar;105(2):36-40. 3. Wheeler K, Kass D, Hoffman R, Vecchi M, Allocca A. Preventing CO poisoning: tracking the impact of legislative and regulatory changes in New York City [PowerPoint Presentation]. Annual Meeting of the Council of State and Territorial Epidemiologists; 2006 June.

CONTENT DOMAIN: CARBON MONOXIDE
INDICATOR: HOME CARBON MONOXIDE DETECTOR COVERAGE

Type of Indicator	**Intervention**
Measure	Percent of Behavioral Risk Factor Surveillance System (BRFSS) respondents reporting at least one CO detector in their household
Derivation of Measure	**Numerator:** The number of respondents reporting CO detector in household **Denominator:** The number of respondents reporting CO detector in household plus respondents reporting no CO detector in household Proportion is adjusted using the survey's household weight
Unit	CO detector presence
Geographic Scope	State and national (tracking network states)
Geographic Scale	State
Time Period	2004; States' BRFSS surveys should include this question every 3–5 years and/or when implementing interventions, such as new legislation, to increase the use of CO alarms
Time Scale	Annual
Rationale	Correctly installed and maintained CO detectors can prevent injury and death from exposure to CO.
Use of the Measure	Collected data will determine the occurrence of CO detectors in homes. These data also can be combined with other data collected by the BRFSS survey, including respondent demographics (e.g., age, sex, and race of survey respondents and age and sex composition of household), socioeconomic characteristics (e.g., insurance status), and relevant health and prevention risk factors (e.g., smoking status, presence of fire alarms). The results of these analyses can be used to target and evaluate public health prevention strategies. **Notes about conducting the analysis:**

	BRFSS data should be analyzed by experts in analysis of sample survey data and the software available to conduct this type of analysis (e.g., SUDAAN and SAS survey procedures). The BRFSS survey is designed so that the primary sampling unit is the respondent. As such, BRFSS data are typically directly weighted to account for sampling error based on data collected at the individual level. However, the question about CO detectors is based on the household rather than the individual as the sampling unit. Using the weighting designed for individuals may bias the prevalence estimate of household risk factors. The indicator will therefore use a weight based on the potential error associated with sampling the household rather than the individual.
Limitations of the Measure	Carbon monoxide alarms must be properly installed and maintained to be effective; a single question does not capture information about either. Maine has developed two questions that can be asked to get supplemental information on maintenance: 1. Is your carbon monoxide detector battery powered or have a battery for back-up power? Response categories: Yes; No; Don't Know; Refused 2. When was the last time you checked the batteries? Response categories (Read only if needed): Within the past year; More than a year; Don't know/Not sure; Refused
Data Sources	BRFSS state-added question from the Indoor Air Pollution Module, question number 4: *A carbon monoxide or CO detector checks the level of carbon monoxide in your home. It is not a smoke detector. Do you have a carbon monoxide detector in your home?*
Limitations of the Data Resources	While the data collection methods are standardized to allow comparisons between states, there may still be bias introduced by "house-effects"—that is, the variation introduced by different organizations and individuals implementing the survey for different states. The BRFSS questionnaire is available in English or Spanish language versions; persons who are not conversationally fluent in English (or Spanish in the states that offer the Spanish-language option) are not eligible. This population of non-English speakers may differ systematically from English speakers in health and behavior

	characteristics, including the presence of a CO detector in their homes. The BRFSS is a telephone survey. While the effect of telephone non-coverage on estimates derived from BRFSS is small, the population without telephones is not likely representative of the general population. In particular, this population is less likely to have a CO detector in the household; therefore, these results should not be generalized to populations without telephone coverage. An increasing number of households use telephone technology that may result in changes in the population sampled and therefore may make the survey results less reliably generalized and introduce other bias. Two examples are: 1. Households with cellular telephones and no traditional telephone. These households are not in the sampling frame for the BRFSS 2. Households that use Caller ID to screen calls; their members may be less likely to pick up the call. Surveys based on self-reported information are likely less accurate than those based on physical measurements. However, when measuring change over time, this type of bias is likely to be constant and therefore not a factor in trend analysis.

CONTENT DOMAIN: CHILDHOOD LEAD POISONING
INDICATOR: TESTING COVERAGE AND HOUSING AGE

Type of EPHT Indicator	Hazard /Intervention
Measures	1. Number of children born in the same year and tested for lead before age 3 2. Percent of children born in the same year and tested before age 3 3. Number of homes built before 1950 (as measured in the 2000 Census) 4. Percent of homes built before 1950 (as measured in the 2000 Census)
Derivation of Measure(s)	Use birth year cohort to calculate the percentage of children with at least one test prior to age 36 months. Use 2000 Census, Summary file 3, to calculate the percentage of pre-1950 housing units
Unit	Proportion of houses by age-based hazard assessment
Geographic Scope	State and national
Geographic Scale	county and state
Time Period	2000-
Time Scale	annual; birth cohort
Rationale	Elevated BLLs in young children have been associated with adverse health effects ranging from learning impairment and behavioral problems to death. Because children may have elevated BLLs and not have any specific symptoms, CDC recommends a blood-lead test for young children at risk for lead poisoning. Risk factors identified in the National Health and Nutrition Examination Surveys (NHANES) include living in housing built before 1950, especially deteriorating condition, being African American and living in a family in poverty. Many states have adopted a targeted testing strategy (test children at high risk), and some states recommend universal testing (test all young children). Nevertheless, studies have documented low blood-lead testing rates among children at high risk. CDC recommends that state and local childhood lead poisoning prevention programs (CLPPPs) evaluate testing among high-risk populations. All CLPPPs have assessed testing in their states but many methods have been used and it is not possible to compare across states. CLPPPs also administer education campaigns for physicians and parents about childhood lead poisoning to enable them to identify

	children at risk. For both universal testing plans and targeted testing plans, children should be tested at least once before the age of 3 years. Some states require more than one test between the ages of 6 and 36 months. Using a birth cohort, the number of children born in a specific year tested before the age of 36 months can be determined.
Use of the Measure	State Identify populations that are not being tested adequately and improve testing Allow for a better understanding of what the blood-lead surveillance data represent National Allow for comparison across states; such comparison can be used to target interventions (especially CDC, EPA, HUD) Public/parents Determine if their community is at risk and the percentage of children being tested. There will be a public health message which will help interpret the results and provide more information on lead sources and prevention. Health care providers Identify children who should be tested for lead by identifying high-risk communities
Limitations of the Measure	This measure estimates testing rates in children living in communities which may be at greater risk of exposure due to older housing. It is a surrogate for a child's risk of lead poisoning due to lead paint in the home. A more direct measure would be based on individual children and the actual age of their housing. Some tested children's addresses are not in the CLPPP data system, while only the provider's address is provided for other children. This can result in some tests being attributed to the wrong county or not being counted at all. Counties are not homogenous with respect to the distribution of lead hazards or risk factors for lead exposure. Using number of pre-1950s housing from Census does not account for houses that have been renovated or have had lead removed. This measure does not account for other lead sources in the community.

	Children may be exposed to lead paint in neighboring counties (visiting family, day care) Many states require children be tested more than once. This indicator does not determine how many children are tested more than once to meet such state requirements.
Data Sources	• Childhood Blood Lead Surveillance Data • US Census (Summary file 3) for total number of housing units and number of pre-1950 units • Vital statistics birth data for number of births
Limitations of Data Sources	Childhood Blood Lead Surveillance Data • Surveillance data are not randomly sampled or representative of the population. • Addresses for all children tested are not included. • Address of the treating clinic is listed sometimes as the address of the child. • De-duplication by a standardized method will be required • Race and ethnicity are not always captured. Census data • Data are available only every 10 years. • Does not have information on renovation of pre 1950 housing is not available. • Does not have information on the condition of the housing is not available. • Address level information on the year the housing was built is not available. Vital Statistics Birth Data • Children may move to another county after birth

CONTENT DOMAIN: CHILDHOOD LEAD POISONING
INDICATOR: BLOOD LEAD LEVELS BY BIRTH COHORT

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Exposure
Measure(s)	1. Number of children born in the same year and tested , by county and state 2. Percent of children born in the same year and tested, by county and state 3. Number of children born in the same year and tested with confirmed blood lead levels ≥ 10 μg/dL [2], by county and state 4. Percent of children born in the same year and tested with confirmed blood lead levels ≥ 10 μg/dL [2], by county and state 5. Number of children born in the same year and tested with confirmed blood lead levels ≥ 10 μg/dL[2], by blood lead level category[3], by state 6. Percent of children born in the same year and tested with confirmed blood lead levels ≥ 10 μg/dL [2], by blood lead level category[3], by state [1] The current blood lead reference level is 5 μg/dL based on National Health and Nutrition Examination Survey (NHANES) 2007 – 2008 and 2009 – 2010 data published in the Fourth National Report on Human Exposure to Environmental Chemicals, and updated in 2012. Blood Lead Levels (BLLs) are confirmed if there is either: (1) one elevated venous test or (2) two elevated capillary and/or unknown tests at least 1 day but less than 12 weeks apart. [2]Details about selecting the appropriate test to classify a child are in the "How-To-Guide for Creating CLP-2 datasets." [3] BLL categories (in units of μg/dL) are <10, 10-<15, 15-<20, 20-<25, 25-<45, 45-<70, and ≥ 70. An additional category for unconfirmed single capillary or unknown specimen tests is used to calculate the total number of children tested. Data are presented by categories at the state level only.

Derivation of Measure(s)	Create CLP-2 (county level) dataset using the "How-To-Guide for Creating CLP-2 datasets." • Select children's records from childhood lead poisoning database. • Classify test results. • Aggregate by county of residence and birth cohort. • Merge with total number of county to obtain the denominator. From CLP-2 dataset, calculate the measures: 1. Number of children born in the same year and tested, by county and state • Sum all BLL categories including the unconfirmed 2. Percent of children born in the same year and tested, by county and state • Divide number of children tested by the total number of children in the birth cohort 3. Number of children born in the same year and tested with confirmed blood lead levels ≥ 10 μg/dL [2], by county and state • Sum number of children in BLL categories ≥ 10 μg/dL (BLLs10_14,…,BLLs70), excluding unconfirmed 4. Percent of children born in the same year and tested with confirmed blood lead levels ≥ 10 μg/dL [2], by county and state • Divide number of children tested with BLLs ≥ 10 μg/dL by the total number of children tested and multiply by 100 5. Number of children born in the same year and tested with confirmed blood lead levels ≥ 10 μg/dL[2], by blood lead level category[3], by state • Sum number of children by BLL categories ≥ 10 μg/dL (BLLs10_14,…,BLLs70), excluding unconfirmed 6. Percent of children born in the same year and tested with confirmed blood lead levels ≥ 10 μg/dL[2], by blood lead level category[3], by state • BLL Categories = Divide number of children for each BLL category by the total number of children tested and multiply by 100
Unit	Number and percent
Geographic Scope	State or National
Geographic Scale	County or State (measures 1-4 available by county and state; measures

	5 and 6 available by state)
Time Period	2000 (or first available) to current
Time Scale	Annual birth cohort
Rationale	Blood lead levels in young children have been associated with adverse health effects ranging from learning impairment and behavioral problems to death. No threshold for adverse effects has been identified. Because children may have elevated BLLs and not have any specific symptoms, CDC recommends blood lead testing for young children at risk for lead poisoning. The risk factors identified by the National Health and Nutrition Examination Surveys (NHANES) include living in housing built before 1950, especially housing in deteriorating condition, being African American, and living in poverty. Many states have adopted a targeted testing strategy (i.e., test children at high risk), whereas some states recommend universal testing (i.e., test all children), either statewide or within high-risk counties and cities. For both universal and targeted testing strategies, children should be tested at least once before the age of 3 years. Some states require more than one test between the ages of 6 and 36 months. In all states, a blood lead test is required for Medicaid-eligible children at 12 and 24 months of age. CDC updated its recommendations on children's blood lead levels in May 2012. The new recommendation is based on the U.S population of children aged 1-5 years who are in the top 2.5% of children tested for lead in their blood. This reference value is the 97.5th percentile, which is currently 5 μg/dL based on NHANES 2007 – 2008 and 2009 – 2010 data (CDC, 2012). The recommendation that chelation therapy should be considered for children with BLLs ≥45 μg/dL has not changed. BLL results ≥70 μg/dL represent a medical emergency. Many states initiate environmental investigations at either BLLs ≥20 μg/dL or persistent BLL results that are 15-19 μg/dL This indicator uses a birth cohort approach. Using these measures, it is possible to determine how many children born in a specific year were tested before the ages of 3 and how many of those tested had an elevated BLL. For children with more than one test before the age of 3, this indicator uses the highest venous specimen result or if there is no venous specimen the highest confirmatory capillary/unknown result. Using the highest results allows for examination of the peak BLLs for the birth cohort. Inclusion of multiple cohorts will allow for the evaluation of trends in testing and BLLs greater than the reference value.

Use of the Measure(s)	• To identify and monitor temporal and spatial changes in BLL testing and -BLLs by birth cohort. • To better understand BLL surveillance data when interpreting number of -BLLs. • To compare testing and BLLs within and across states for the purpose of targeting interventions. Comparisons should only be made between areas with similar testing and reporting rules. • To link data on risk factors and compare risk factors within and across states. • To guide interventions and allocation of resources related to BLL testing and prevention of lead exposure in young children.. • To develop and support public health policy and legislation related to BLL testing and prevention of childhood lead poisoning. • To monitor progress towards eliminating BLLs ≥5 μg/dL, the current reference value (NHANES 2007 – 2008 and 2009 – 2010 data).
Limitations of the Measure(s)	• The analysis uses the county of the child's residence at the time of the test, which may be different from the county where the child was exposed to lead. • Counties are not homogenous with respect to the distribution of lead hazards or risk factors for lead exposure. • Number and percent of BLLs cannot be interpreted as prevalence or incidence for the population. • State to state comparisons must be made cautiously and require additional information about the states' testing practices, confirmatory testing practices, and reporting laws. • Because the capillary test is subject to contamination it can result in a false positive BLL. The number and percent of BLLs may be overestimated when non-venous test results are used.
Data Sources	Childhood Blood Lead Surveillance Data Vital Statistics Birth Data
Limitations of Data Sources	Childhood Blood Lead Surveillance Data • Surveillance data are not randomly sampled or representative of the population. • Complete residential addresses are not available for all children tested. • Sometimes the address of the provider or another address is listed as the child's address when the data is not provided by the reporting authority. Vital Statistics Birth Data • The number of children born from Vital Statistics does not include children who have moved in or out of the area since birth. Therefore, as a denominator, it may under or over

	estimate the number of children in a birth cohort.
Presentation	Small numbers of children tested, births, or BLLs may exist when the measures are calculated at the county levels. These small numbers are not accurate estimates for childhood lead poisoning in these polygons. In addition, these small numbers will require additional data processing steps to preserve confidentiality. One or more of the following methods can be used: • Suppression of small numbers, • Aggregation of neighboring geographic units. • Aggregation to a lower resolved geographic level unit, • Aggregation of successive birth cohorts. Data on blood lead levels are presented by categories at the state level only. This indicator should be displayed with information about the lead testing program, including: • State and/or local testing policies or strategies (i.e., targeted or universal) • CDC-funded Childhood Lead Poisoning Prevention Program • Minimum BLL reported by laboratories to state or local lead program
Related Indicators	Blood Lead Testing and Housing Age Annual Blood Lead Levels
References	Centers for Disease Control and Prevention (CDC). 2012. CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention Recommendations in "Low Level Lead Exposure Harms Children: A Renewed Call of Primary Prevention".

CONTENT DOMAIN: CHILDHOOD LEAD POISONING
INDICATOR: ANNUAL BLOOD LEAD LEVELS

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Exposure
Measure(s)	1. Number of children tested, by age group[1], by county and state 2. Percent of children tested, by age group[1], by county and state 3. Number of children tested with confirmed blood lead levels ≥ 10 µg/dL[3,4], by age group[1], by county and state 4. Percent of children tested with confirmed blood lead levels ≥ 10 µg/dL [3,4], by age group[1], by county and state 5. Number of children tested with confirmed blood lead levels ≥ 10 µg/dL by blood lead level category[2,3,4], by age group[1], by state 6. Percent of children tested with confirmed blood lead levels ≥ 10 µg/dL, by blood lead level category[2,3,4], by age group[1], by state [1]Measures are available stratified by two age groups: <36 months and 36 to <72 months [2] The current blood lead reference level is 5 µg/dL based on National Health and Nutrition Examination Survey (NHANES) 2007 – 2008 and 2009 – 2010 data published in the Fourth National Report on Human Exposure to Environmental Chemicals, and updated in 2012. Blood Lead Levels (BLLs) ≥ 10 µg/dL are confirmed if there is either: (1) one elevated venous test or (2) two elevated capillary and/or unknown tests at least 1 day but less than 12 weeks apart. [3] Details about selecting the appropriate test to classify a child are in the "How-To-Guide for Creating CLP-4 datasets." [4] BLL categories (in units of µg/dL) are <10, 10-14, 15-19, 20-24, 25-44, 45-69, and ≥ 70. An additional category for unconfirmed elevated capillary or unknown specimen tests is used to calculate the total number of children tested. Confirmed BLLs ≥ 10µg/dL and BLLs 5-9µg/dL, reflecting the NHANES reference value, will be included by Spring 2013. Data on confirmed BLLs ≥ 10µg/dL will be presented by blood lead categories at the state level only.

Derivation of Measure(s)	Create CLP-4 (county level) dataset using the "How-To-Guide for Creating CLP-4 datasets." • Select children's records from childhood lead poisoning database. • Classify test results. • Aggregate by county of residence and year • Merge with total number of children by county to obtain the denominator. From CLP-4 dataset, calculate the measures: 1. Number of children tested • Sum all BLL categories including the unconfirmed 2. Percent of children tested • Divide number of children tested by the total number of children 3. Number of children tested with confirmed blood lead levels ≥ 10 μg/dL[4] • Sum number of children in BLL categories ≥ 10 μg/dL (BLLs 10-14,…,BLLs70), excluding unconfirmed 4. Percent of children tested with confirmed blood lead levels ≥ 10 μg/dL[4] • Divide number of children tested with blood lead levels ≥ 10 μg/dL by the total number of children tested and multiply by 100 5. Number of children tested with confirmed blood lead levels ≥ 10 μg/dL[4] • Sum number of children for each BLL category 6. Percent of children tested with confirmed blood lead levels ≥ 10 μg/dL[4] • Divide number of children for each BLL category by the total number of children tested and multiply by 100
Unit	Number and percent
Geographic Scope	State or National
Geographic Scale	County or State (measures 1-4 available at county and state; measures 5 and 6 available only at state)
Time Period	2000 to current
Time Scale	Annual
Rationale	Blood lead levels in children have been associated with adverse health effects ranging from learning impairment and behavioral problems to death. Lead can affect almost every organ and system in your body. The

	effects of lead are the same whether it enters the body through breathing or swallowing. Small children can be exposed by eating lead-based paint chips, chewing on objects painted with lead-based paint or swallowing house dust or soil that contains lead. Children are more vulnerable to lead poisoning than adults. The main target for lead toxicity is the nervous system in young children. A child who swallows large amounts of lead may develop blood anemia, severe stomachache, muscle weakness, and brain damage. If a child swallows smaller amounts of lead, much less severe effects on blood and brain function may occur. Even at much lower levels of exposure, lead can affect a child's mental and physical growth. Since children may have higher BLLs and not display any specific symptoms, CDC recommends blood lead testing for young children at risk for lead poisoning. The risk factors identified by the National Health and Nutrition Examination Surveys (NHANES) include living in housing built before 1950, especially housing in deteriorating condition, being African American, and living in poverty. States have developed and implemented assessment protocols for children to determine the need for a blood lead test. For both universal and targeted testing strategies, children should be tested at least once before the age of 3 years. Some states require more than one test between the ages of 6 and 36 months. Children not tested before the age of 3 should be tested at least once before the age of 6. In all states, a blood lead test is required for Medicaid-eligible children at 12 and 24 months. CDC updated its recommendations on children's blood lead levels in May 2012. The new recommendation is based on the U.S population of children aged 1-5 years who are in the top 2.5% of children tested for lead in their blood. This reference value is the 97.5^{th} percentile, which is currently 5 μg/dL based on NHANES 2007 – 2008 and 2009 – 2010 data (CDC, 2012). The recommendation that chelation therapy should be considered for children with BLLs ≥45 μg/dL has not changed. BLL results ≥70 μg/dL represent a medical emergency. Many states initiate environmental investigations at either BLLs ≥20 μg/dL or persistent BLL results that are 15-19 μg/dL This indicator provides information on the number of children tested each year and the number of those children tested with confirmed blood lead levels above 10 μg/dL. This information is used to direct resources for testing and management of elevated cases and be linked with environmental or the risk factor data to monitor trends over time.

Use of the Measure(s)	• To identify and monitor temporal and spatial changes in BLL testing and confirmed BLLs ≥ 10µg/dL[4] by year. • To better understand BLL surveillance data when interpreting number of confirmed BLLs ≥ 10µg/dL[4]. • To compare testing and BLLs within and across states for the purpose of targeting interventions. Comparisons should only be made between areas with similar testing and reporting rules. • To link data on risk factors and compare risk factors within and across states. • To guide interventions and allocation of resources related to BLL testing and prevention of EBLLs in children. • To develop and support public health policy and legislation related to BL testing and prevention of childhood lead exposure. • To monitor progress towards eliminating BLLs ≥5 µg/dL, the current reference value (NHANES 2007 – 2008 and 2009 – 2010 data).
Limitations of the Measure(s)	• The analysis uses the county of the child's residence at the time of the test, which may be different from the county where the child was exposed to lead. • Counties are not homogenous with respect to the distribution of lead hazards or risk factors for lead exposure. • Number and percent of EBLLs through surveillance data cannot be interpreted as prevalence or incidence for the population as a whole • State to state comparisons must be made cautiously and require additional information about the states' testing practices, confirmatory testing practices, and reporting laws. • Because the capillary test is subject to contamination it can result in a false positive EBLL. The number and percent of EBLLs would be overestimated if unconfirmed, non-venous test results are used.
Data Sources	Childhood Blood Lead Surveillance Data Census Population Data: Vintage bridged-race post-censal population estimates: **http://www.cdc.gov/nchs/nvss/bridged_race.htm**
Limitations of Data Sources	Childhood Blood Lead Surveillance Data • Surveillance data are not randomly sampled or representative of the population. • Complete residential addresses are not available for all children tested. • If the child's address is not provided the address of the provider may be used.
Related Indicators	Blood Lead Testing and Housing Age Blood Lead Levels by Birth Cohort
References	Centers for Disease Control and Prevention (CDC). 2012. CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention Recommendations in "Low Level Lead Exposure Harms Children: A Renewed Call of Primary Prevention".

INDICATOR TEMPLATE
CONTENT AREA: CLIMATE AND HEALTH
INDICATOR: HEAT STRESS HOSPITALIZATIONS

Type of EPHT Indicator	Health outcome
Measures	1. Age-adjusted rate of hospitalization for heat stress per 100,000 population 2. Crude rate of hospitalization for heat stress per 100,000 population 3. Number of hospitalizations for heat stress
Derivation of Measure(s)	*Numerator*: Hospital admissions having any ICD-9 code in the range of 992.0-992.9, or cause of injury code E900.0 or E900.9, EXCLUDING cases with a code of E900.1 (man-made source of heat) anywhere in the record. *Denominator:* Midyear resident population, by gender, for state and by county *Adjustment:* Age-adjustment by the direct method to year 2000 US standard population
Unit	1. Age-adjusted rate per 100,000 population 2. Rate per 100,000 population 3. Number
Geographic Scope	State and national
Geographic Scale	Residents of jurisdiction – State
Time Period	Hospital admissions between May 1 to September 30, inclusive, for each year, 2000–
Time Scale	May–September of each data year
Rationale	The Intergovernmental Panel on Climate Change (IPCC) projects with "virtual certainty" suggest that climate change will cause more frequent, more intense, and longer heat waves (1). Any individual, regardless of age, sex or health status can develop heat stress if engaged in intense physical activity and/or exposed to environmental heat (and humidity). Physiologic mechanisms maintain the core body temperature (i.e., the operating temperature of vital organs in the head or trunk) in a narrow optimum range around 37 °C (98.6 °F). When core body temperature rises, the physiologic response is to sweat and circulate blood closer to the skin's surface to increase cooling. If heat exposure exceeds the physiologic capacity to cool, and core body temperature rises, then a range of heat-related symptoms and conditions can develop. Heat stress or Heat-related illness ranges from mild heat edema and rash, heat syncope, heat cramps, to the most common type, heat exhaustion (2). Heat-related cramps, rash, and edema are relatively minor readily treatable conditions; however, they should be used as important warning signs to immediately remove the affected individual from the exposure situation.

	Heat cramps are brief, intermittent, and often severe muscular cramps occurring typically in muscles that are fatigued by heavy work (2). Individuals with heat cramp can also exhibit hyponatremia, hypochloremia (which are low serum sodium and chloride levels). *Heat syncope* is a temporary loss of consciousness as a result of prolonged heat exposure (2). Individuals adapt to hot, humid environment by dilation of cutaneous vessels in the skin to radiate heat. Peripheral vasodilation along with blood volume loss, results in lowering the blood pressure which can result in inadequate central venous return and cerebral perfusion, causing light-headedness and fainting. *Heat exhaustion* is a consequence of extreme depletion of blood plasma volume, which may be coincident with hyponatremia and/or peripheral blood pooling (2). Heat exhaustion often does not present with definitive symptoms and may be misdiagnosed, often as an acute viral illness. Symptoms include mild disorientation, generalized malaise, weakness, nausea, vomiting, headache, tachycardia (rapid beating of the heart), and hypotension. Because untreated heat exhaustion can progress to heat stroke, the most serious form of heat-related illness, treatment should begin at the first signs of heat exhaustion (3). *Heat stroke* is an extreme medical emergency that if untreated can result in death or permanent neurological impairment (2). Heat stroke occurs when a person's core body temperature rises above 40 °C (104 °F) as a result of impaired thermoregulation. High core body temperature and disseminated intravascular coagulation results in cell damage in vital organs, such as the brain, liver, and kidneys, which can lead to serious illness and death (3). Death may occur rapidly due to cardiac failure or hypoxia, or it can occur days later as a result of renal failure due to dehydration and/or rhabdomyolysis (i.e., the breakdown of muscle fibers with release into the circulation of muscle fiber contents, some of which are toxic to the kidney and can cause kidney damage) (4). Heat stroke is typically divided into two types. The two types are in general clinically the same, except that the individuals/population groups affected require medical interventions specific to their unique physiology and medical status (3). "Exertional Heat Stroke," as the name implies, involves strenuous physical activity under high temperature conditions to which the heat stroke victim was not acclimatized, and usually affects healthy young adults, such as athletes, outdoor laborers and soldiers. "Classic" heat stroke, by definition does not involve exertion, and usually affects susceptible individuals, such as infants and young children, the elderly, or people with chronic illness. Because heat stroke, even if treated, can have a death rate as high as 33%, and up to 17% of heat stroke survivors suffer permanent damage, measures should be taken to prevent heat-related illness, especially among vulnerable populations. The relationship between extreme heat and increased daily morbidity and mortality is well established. This indicator captures hospital admissions directly attributed to heat stress (e.g., heat illness, heat stroke, and hyperthermia). It is a measure that can

	be tracked easily and consistently across geography and time, and acts as a sentinel for the broader range of heat-related illness that is not recognized and/or coded as such.
Use of the Measure	Heat stress can manifest in a number of clinical outcomes, and people with chronic health problems (e.g., cardiovascular disease, diabetes, obesity) are more susceptible to the effects of heat than healthy individuals. For these reasons, heat stress may not be listed as the primary diagnosis. This indicator therefore includes all cases where heat stress is explicitly listed as the primary diagnosis or any other diagnosis. Increases in the rates of hospital admission for heat stress are one potential impact of rising global temperatures. Tracking these data can help document changes over place and time, monitor vulnerable areas, and evaluate the results of local climate-adaptation strategies.
Limitations of the Measure	Periods of extreme heat are frequently associated with increases in hospital visits and admissions for many causes. This measure does not capture the full spectrum of heat stress, especially where exposure to excess heat is not explicitly documented.
Data Sources	*Numerator:* State inpatient hospital discharge data (using admission date) *Denominator:* US Census Bureau population data
Limitations of Data Sources	*State hospital discharge data*: • Using a measure of all heat stress hospitalizations will include some transfers between hospitals for the same individual for the same heat stress event. Variations in the percentage of transfers or readmissions for the same heat stress event may vary by geographic area and impact rates. • Without reciprocal reporting agreements with abutting states, statewide measures and measures for geographic areas (e.g., counties) bordering other states may be underestimated because of health care utilization patterns. • Each state must individually obtain permission to access and, in some states, provide payment to obtain the data. • Veterans Affairs, Indian Health Services and institutionalized (e.g. Prison) populations are excluded. • Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers to hospitalize patients • Street address is currently not available in many states. • Sometimes mailing address of patient is listed as the residence address of the patient • Patients may be exposed to environmental triggers in multiple locations, but hospital discharge geographic information is limited to residence. • Since the data captures hospital discharges (rather than admissions), patients admitted toward the end of the year and discharged the following year will be omitted from the current year dataset

	• Data will need to be de-duplicated (i.e. remove duplicate records for the same event) • There is usually a two year lag period before data are available from the data owner. *Census data*: • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the Census
Related Indicators	• Heat vulnerability • Heat-related mortality • Temperature distribution • Emergency department visits for heat stress
References	1. Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, et al. 2007. Human health In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE. , editors. Climate Change 2007: Impacts, Adaptation and Vulnerability Contribution of Working Group II to: Fourth Assessment Report of the Intergovernmental Panel on Climate Change. New York: Cambridge University Press. pp. 391–431. 2. Rosen's Emergency Medicine: Concepts and Clinical Practice. 2010. Chapter 139: Heat illness. In JA Marx Editor-in-Chief; RS Hockberger & RM Walls Senior Editors; JG Adams … [et al] Editors (7th ed). Philadelphia: Mosby Elsevier. 3. American Medical Association. Heat-related Illness During Extreme Weather Emergencies (Report 10 of the Council on Scientific Affairs (A97), 1997; www.ama-assn.org/ama/pub/category/13637.html). 4. Centers for Disease Control and Prevention. Heat-related deaths--Los Angeles County, California, 1999-2000, and United States, 1979-1998. MMWR 2001;50(29):623-6.

INDICATOR TEMPLATE
CONTENT AREA: CLIMATE AND HEALTH
INDICATOR: EMERGENCY DEPARTMENT VISITS FOR HEAT STRESS

Type of EPHT Indicator	Health outcome
Measures	1. Annual age-adjusted rate of emergency department visits for heat stress per 100,000 population 2. Annual crude rate of emergency department visits for heat stress per 100,000 population 3. Annual number of emergency department visits for heat stress
Derivation of Measure(s)	*Numerator*: • Patients treated in an Emergency Department (ED) having any ICD-9 code in the range of 992.0-992.9, or cause of injury code E900.0 or E900.9. • Cases with a code of E900.1 (man-made source or heat) anywhere in the record are excluded. • *Denominator*: Midyear resident population, by gender, for state and by county *Adjustment*: • Age-adjustment by the direct method to the Year 2000 US Standard population • U.S. 2000 standard population by age categories from Surveillance Epidemiology and End Results (SEER), National Cancer Institute
Unit	5. Age-adjusted rate per 100,000 population 6. Rate per 100,000 population 7. Number
Geographic Scope	State and national
Geographic Scale	State (annual), County (aggregate years)
Time Period	Hospital admissions between May 1 to September 30, inclusive, for each year, 2000–
Time Scale	May–September of each data year
Rationale	The Intergovernmental Panel on Climate Change (IPCC) projects with "virtual certainty" suggest that climate change will cause more frequent, more intense, and longer heat waves (1). Any individual, regardless of age, sex or health status can develop heat stress if engaged in intense physical activity and/or exposed to environmental heat (and humidity). Physiologic mechanisms maintain the core body temperature (i.e., the operating temperature of vital organs in the head or trunk) in a narrow optimum range around 37 °C (98.6 °F). When core body temperature rises, the physiologic response is to sweat and circulate blood closer to the skin's surface to increase cooling. If heat exposure exceeds the physiologic capacity to cool, and core body temperature rises, then a range of heat-related symptoms and conditions can develop. Heat stress or Heat-related illness ranges from mild heat edema, rash, heat syncope, heat cramps, to the most common type,

	heat exhaustion (2). Heat-related cramps, rash, and edema are relatively minor readily treatable conditions; however, they should be used as important warning signs to immediately remove the affected individual from the exposure situation. *Heat cramps* are brief, intermittent, and often severe muscular cramps occurring typically in muscles that are fatigued by heavy work (2). Individuals with heat cramp can also exhibit hyponatremia, hypochloremia, and low serum sodium and chloride levels. *Heat syncope* is a temporary loss of consciousness as a result of prolonged heat exposure (2). Individuals adapt to hot, humid environment by dilation of cutaneous vessels in the skin to radiate heat. Peripheral vasodilation along with blood volume loss, results in lowering the blood pressure which can result in inadequate central venous return and cerebral perfusion, causing light-headedness and fainting. *Heat exhaustion* is a consequence of extreme depletion of blood plasma volume, which may be coincident with hyponatremia and/or peripheral blood pooling (2). Heat exhaustion often does not present with definitive symptoms and may be misdiagnosed, often as an acute viral illness. Symptoms include mild disorientation, generalized malaise, weakness, nausea, vomiting, headache, tachycardia (rapid beating of the heart), and hypotension. Because untreated heat exhaustion can progress to heat stroke, the most serious form of heat-related illness, treatment should begin at the first signs of heat exhaustion (3). *Heat stroke* is an extreme medical emergency that if untreated can result in death or permanent neurological impairment (2). Heat stroke occurs when a person's core body temperature rises above 40 °C (104 °F) as a result of impaired thermoregulation. High core body temperature and disseminated intravascular coagulation results in cell damage in vital organs, such as the brain, liver, and kidneys, which can lead to serious illness and death (3). Death may occur rapidly due to cardiac failure or hypoxia, or it can occur days later as a result of renal failure due to dehydration and/or rhabdomyolysis (i.e., the breakdown of muscle fibers with release into the circulation of muscle fiber contents, some of which are toxic to the kidney and can cause kidney damage) (4). Heat stroke is typically divided into two types. The two types are in general clinically the same, except that the individuals/population groups affected require medical interventions specific to their unique physiology and medical status (3). "Exertional Heat Stroke," as the name implies, involves strenuous physical activity under high temperature conditions to which the heat stroke victim was not acclimatized, and usually affects healthy young adults, such as athletes, outdoor laborers and soldiers. "Classic" heat stroke, by definition does not involve exertion, and usually affects susceptible individuals, such as infants and young children, the elderly, or people with chronic illness. Because heat stroke, even if treated, can have a death rate as high as 33%, and up to 17% of heat stroke survivors suffer permanent be taken to prevent heat-related illness, especially among vulnerable populations. The relationship between extreme heat and increased daily morbidity and mortality is well established. This indicator captures hospital admissions *directly* attributed to heat

	stress (e.g., heat illness, heat stroke, and hyperthermia). It is a measure that can be tracked easily and consistently across geography and time, and acts as a sentinel for the broader range of heat-related illness that is not recognized and/or coded as such.
Use of the Measure	Heat stress can manifest in a number of clinical outcomes, and people with chronic health problems (e.g., cardiovascular disease, diabetes, obesity) are more susceptible to the effects of heat than healthy individuals. For these reasons, heat stress may not be listed as the primary diagnosis. This indicator therefore includes all cases where heat stress is explicitly listed as the primary diagnosis or any other diagnosis. Increases in the rates of ED visits for heat stress are one potential impact of rising global temperatures. Tracking these data can help document changes over place and time, monitor vulnerable areas, and evaluate the results of local climate-adaptation strategies.
Limitations of the Measure	Periods of extreme heat are frequently associated with increases in hospital visits and admissions for many causes. This measure does not capture the full spectrum of heat-stress, where exposure to excess heat is not explicitly documented.
Data Sources	*Numerator:* State emergency department data *Denominator:* US Census Bureau population data
Limitations of Data Sources	*Emergency Department data*: • Data are not available for all states. • Number of diagnostic fields in hospital records varies from state to state. Utilization of EDs varies geographically. *Census data*: • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year.
Related Indicators	• Heat vulnerability • Heat-related mortality • Temperature distribution • Heat stress hopitalizations
References	1. Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, et al. 2007. Human health In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE. , editors. Climate Change 2007: Impacts, Adaptation and Vulnerability Contribution of Working Group II to: Fourth Assessment Report of the Intergovernmental Panel on Climate Change. New York: Cambridge University Press. pp. 391–431. 2. Rosen's Emergency Medicine: Concepts and Clinical Practice. 2010. Chapter 139: Heat illness. In JA Marx Editor-in-Chief; RS Hockberger & RM Walls Senior Editors; JG Adams … [et al] Editors (7th ed). Philadelphia: Mosby Elsevier. 3. American Medical Association. Heat-related Illness During Extreme Weather Emergencies (Report 10 of the Council on Scientific Affairs (A97), 1997; www.ama-assn.org/ama/pub/category/13637.html). 4. Centers for Disease Control and Prevention. Heat-related deaths--Los Angeles County, California, 1999-2000, and United States, 1979-1998. MMWR 2001; 50(29):623-6.

CONTENT DOMAIN: COMMUNITY WATER INDICATOR: ATRAZINE ENVIRONMENTAL PUBLIC HEALTH TRACKING	
Type of EPHT Indicator	Hazard, Exposure
Measures	**Level of Contaminant in Finished Water** 1. Quarterly distribution of number of Community Water Systems (CWS) by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 2. Yearly distribution of number of CWS by maximum atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 3. Yearly distribution of number of CWS by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 4. Mean concentration of atrazine at CWS-level, by year. **Potential Population Exposure to Contaminants in Finished Water** 1. Quarterly distribution of number of people served by CWS by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 2. Yearly distribution of number of people served by CWS by maximum atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 3. Yearly distribution of number of people served by CWS by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine).
Derivation of Measures	Atrazine measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	µg/L of Atrazine
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	**Atrazine and Public Health** Atrazine is a widely used herbicide active against broadleaf and grassy weeds. Atrazine was first registered as an herbicide in 1958. More than 70 million pounds have been applied

	annually in recent years, with about 75% of corn cropland receiving treatment. In addition to agricultural uses, atrazine is used in residential turf applications and on golf courses and sod farms to control weeds. Atrazine and its degradation products are the most commonly detected pesticides in ground and surface waters (Barr et al., 2007). The frequent detection of atrazine and its degradation products in streams, rivers, groundwater, and reservoirs is related directly to the volume of its use, its persistence in soils due to its resistance to photolysis and hydrolysis, and its ability to travel within water systems (Nelson et al., 2001). In water systems, atrazine is transformed over time by various chemical reactions into other compounds or its degradation products or metabolites, including dealkylated compounds such as desethylatrazine (DEA), desisopropylatrazine (DIA), and diaminochlorotriazine (DACT). In soil, atrazine degrades slowly to dealkylated compounds, which have half-lives of several months. Bacteria and plants can metabolize atrazine to hydroxylated products. In plants, atrazine is absorbed by the root system and tends to form hydroxylated metabolites that cannot be removed by washing contaminated vegetables (Nelson et al., 2001). Atrazine does not bioaccumulate. Studies suggest that in animals, the degradation products that retain the chlorine have biologic activity similar to that of atrazine, while the hydroxylated metabolites do not retain its biologic activity (Nelson et al., 2001). Use of atrazine in the presence of nitrogen fertilizers, has raised a possibility of N-nitrosation in soil (DeMarini and Zahm, 1999). There may also be endogenous formation of N-nitrosoatrazine from precursors ingested in the diet and drinking water. For the general population, drinking water is an infrequent source of atrazine exposure, but estimates of seasonal intakes from drinking water in a small number of communities have exceeded the recommended limits (U.S. EPA, 2003). As a result, atrazine use has progressively been restricted in an effort to reduce surface and ground water contamination. In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, atrazine was detected in 888 systems serving greater than 34 million people (EPA, 2009). Concentrations of atrazine were greater than the MCL in 98 systems serving 3.1 million people. Atrazine was the second highest occurring regulated synthetic organic chemical found based on the percent of detections found from the 6 Year Review data (EPA, 2009). While it is used on many crops, atrazine has not been found in many food samples, and then only at very low levels. Therefore, it is very unlikely that people would be exposed to atrazine by eating crops from atrazine-accumulated soil. Most people are not exposed regularly to atrazine. People living near areas where atrazine was applied to crops may be exposed through contaminated drinking water. Atrazine has been found at about 20 Superfund sites in the United States. People living near those sites may be exposed to higher levels of atrazine. Factory workers who work with atrazine may be exposed to higher amounts of atrazine than other workers. The government has estimated that approximately 1,000 people may be exposed to atrazine in this way (ATSDR, 2003). Applicators of atrazine may be exposed dermally and by inhalation. Atrazine is well absorbed orally, metabolized, and then eliminated in the urine over a few days (Bradway et al., 1982; Catenacci et al., 1993; Timchalk et al., 1990). Metabolism of atrazine and its degradation products is complex and results in many potential metabolites (Barr et al., 2007). As many as 8-12 metabolites of atrazine have been identified in animals and humans, with recent studies showing DACT as the primary

	metabolite (Barr et al., 2007); therefore, earlier biomonitoring studies measuring atrazine mercapturate alone misrepresent and underestimate total atrazine exposure. Panuwet et al., (2008) developed an analytical method that measures the seven primary urinary metabolites of atrazine, which are: hydroxyatrazine, DACT, DIA, DEA, desethylatrazine mercapturate, atrazine mercapturate, and atrazine itself. Human health effects of atrazine at environmental doses or at biomonitored levels from environmental exposure are unknown. In mammalian studies, atrazine is rated as having low acute toxicity. Atrazine product formulations can be mild skin sensitizers and irritants. Some human ecologic and epidemiologic studies of reproductive and cancer outcomes have shown either positive or no associations, but effects are difficult to attribute due to lack of exposure markers or due to mixed chemical or pesticide exposures (ATSDR, 2003; Gammon et al., 2005; Sathiakumar and Delzell, 1997). Studies of couples living on farms that use atrazine for weed control found an increase in the risk of pre-term delivery. These studies are difficult to interpret because most of the farmers were men who may have been exposed to several types of pesticides. A meta-analysis linked hypospadias to parental exposure to pesticides with possible endocrine-mediated effects (Rocheleau et al., 2009). Some epidemiological studies that looked at the potential impact of prenatal exposure to atrazine or its products of environmental degradation on pregnancy outcomes in the general population observed higher rates of babies born small-for gestational age (SGA) (Munger et al., 1997, Villanueva et al., 2005; Ochoa-Acuna et al., 2009). They also linked exposure of mothers who lived closer to sites with high atrazine concentrations with a higher risk of gastroschisis (Waller et al., 2010). Most of these studies were retrospective and relied on ecological assessment of exposure to atrazine. However, the most recent study that measured urinary biomarkers of prenatal atrazine exposure and was based on a prospective population-based cohort found associations between environmental exposure to atrazine and adverse effects on fetal growth, specifically birth weight, birth length, and small head circumference (Chevrier et al., 2011). Atrazine is not mutagenic and is not considered genotoxic. The International Agency for Research on Cancer (IARC) considers atrazine not classifiable with respect to human carcinogenicity, and the EPA considers atrazine unlikely to be a human carcinogen. However, IARC recommends future research to characterize the ability of atrazine to interfere with the hypothalamic-pituitary-ovarian axis in women. This research would help determine whether atrazine is a mammary carcinogen in women. Another area for future research is to explore atrazine's ability to alter immune and aromatase function in humans. Additional information is available from U.S. EPA at: http://www.epa.gov/pesticides/ ; from ATSDR at: http://www.atsdr.cdc.gov/toxpro2.html, and IARC at http://www.iarc.fr/ Children are likely to be exposed to atrazine in the same way as adults, primarily through contact with dirt that contains atrazine or by drinking water from wells that are contaminated with the herbicide. Little information is available about the effects of atrazine in children. Maternal exposure to atrazine in drinking water has been associated with low fetal weight and heart, urinary, or limb defects in humans. It is not known whether atrazine or its metabolites can be transferred from a pregnant mother to a developing fetus through the placenta or from a nursing mother to her offspring through breast milk. **Biomonitoring Information** Urinary levels of atrazine mercapturate reflect recent exposure. In the NHANES 2001–2002 subsample, levels of atrazine mercapturate were generally not detectable (CDC, 2005). In small studies of Maryland residents in 1995–1996 (MacIntosh et al., 1999) and 83

	Minnesota children with multiple urine collections during 1997 (Adgate et al., 2001), atrazine mercapturate was infrequently detected at the detection limit of 0.3 µg/L. In a study of 60 farm worker children, atrazine was detected in only four children (Arcury et al., 2007). Using immunoassay atrazine equivalents (detected mostly as atrazine mercapturate), the urinary geometric mean levels for herbicide applicators in Ohio and Wisconsin were about 6 µg/L (Hines et al., 2003; Perry et al., 2000). The geometric mean of urinary atrazine mercapturate was 1.2 µg/L in 15 farmers studied several days after spraying the pesticide (Curwin et al., 2005). In a small number of field workers, urinary concentrations ranged from 5-1756 µg/L (Lucas et al., 1993). However, biomonitoring studies that have evaluated only one urinary metabolite of atrazine (such as atrazine mercapturate) probably underestimated exposure (Barr et al, 2007). Finding measurable amounts of atrazine or its metabolites in urine does not mean that the levels of atrazine and its metabolites (e.g., atrazine mercapturate) cause an adverse health effect. Biomonitoring studies on levels of atrazine mercapturate provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of atrazine than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects. **Sources of Atrazine** Atrazine is the common name for an herbicide that is widely used to kill weeds. It is used mostly on farms. Pure atrazine—an odorless, white powder—is not very volatile, reactive, or flammable. It will dissolve in water. Atrazine is made in the laboratory; it does not occur naturally. Atrazine is used on crops such as sugarcane, corn, pineapples, sorghum, and macadamia nuts, and on evergreen tree farms and for evergreen forest re-growth. It has also been used to keep weeds from growing on both highway and railroad rights-of-way. Some of the trade names of atrazine are Aatrex®, Aatram®, Atratol®, and Gesaprim®. The scientific name for atrazine is 6-chloro-N-ethyl-N'-(1-methylethyl)-triazine-2,4-diamine. Atrazine is a Restricted Use Pesticide , which means that only certified herbicide users may purchase or use it. Certification for the use of atrazine is obtained through the appropriate state office where the herbicide user is licensed. Atrazine is usually used in the spring and summer months. For it to be active, atrazine needs to dissolve in water and enter the plants through their roots. It then acts in the shoots and leaves of the weed to stop photosynthesis. Atrazine is taken up by all plants, but in plants not affected by atrazine, it is broken down before it can affect photosynthesis. The application of atrazine to crops as an herbicide accounts for almost all of the atrazine that enters the environment, but some may be released from manufacture, formulation, transport, and disposal. Any atrazine that is washed from the soil into streams and other bodies of water will stay there for a long time, because chemical breakdown is slow in rivers and lakes. It also will persist for a long time in groundwater. This is one reason why atrazine is found commonly in the water collected from drinking water wells in some agricultural regions. If atrazine enters the air, it can be broken down by reactions with other reactive chemicals in the air. However, sometimes atrazine is on particles such as dust. When this happens, breakdown is not expected. Atrazine is removed from air mainly by rainfall. When atrazine is on dust particles, the wind can blow it long distances from the nearest application area.

	For example, atrazine has been found in rainwater more than 180 miles (300 kilometers) from the nearest application area. Atrazine does not tend to accumulate in living organisms such as algae, bacteria, clams, or fish, and, therefore, does not tend to build up in the food chain. **Atrazine Regulation and Monitoring** Congress established the Safe Drinking Water Act in 1974, which set enforceable Maximum Contaminant Levels (MCLs) and non-enforceable Maximum Contaminant Level Goals (MCLGs) for certain, specified contaminants. In the case of atrazine in drinking water, EPA has set an MCL of 3 µg/L. Atrazine is designated as a Restricted Use Pesticide, which means that only certified pesticide applicators can use atrazine. The Occupational Safety and Health Administration (OSHA) has set a limit of 5 milligrams of atrazine per cubic meter of workplace air (5 mg/m3) for an 8_hour workday and 40-hour work week. EPA has determined maximum levels allowed in foods of 0.02-15 parts atrazine per million parts of food (0.02-15 ppm).
Use of Measure	These measures assist by providing data that can be used for surveillance purposes. • Distribution measures provide information on the number of CWS and the number of people potentially exposed to atrazine at different concentrations. • Maximum concentrations provide information on the peak potential exposure to atrazine at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of the Measure	The current measures are derived for CWS only. Private wells are another important source of population exposure to atrazine in some agricultural regions. Transient non-community water systems, which are regulated by EPA, may also be an important source of atrazine exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be converted directly to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	Ground water systems may have many wells with different atrazine concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the atrazine concentration of people served by wells with higher atrazine concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different atrazine levels are averaged to estimate levels for the PWS.

Related Indicators	Public Water Use
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	18. Rocheleau C. M., P. A. Romitti, L. K. Dennis. Pesticides and hypospadias: a meta-analysis. J Pediatr Urol 2009; Vol. 5, 17-24. 19. Perry M, Christiani D, Dagenhart D, Tortorelli J, Singzoni B. Urinary biomarkers of atrazine exposure among farm pesticide applicators. Ann Epidemiol 2000;10(7):479. 20. Sathiakumar N, Delzell E. A review of epidemiologic studies of triazine herbicides and cancer. Crit Rev Toxicol 1997;27(6):599-612. 21. Timchalk C, Dryzga MD, Langvardt PW, Kastl PE, Osborne DW. Determination of the effect of tridiphane on the pharmacokinetics of [14C]-atrazine following oral administration to male Fischer 344 rats. Toxicology 1990;61(1):27-40. 22. U.S. Environmental Protection Agency (U.S. EPA). Interim Reregistration Eligibility Decision For Atrazine. Case No. 0062. 2003. Available at URL: http://www.epa.gov/oppsrrd1/REDs/atrazine_ired.pdf . 3/11/09 23. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009. 24. Villanueva C. M., G. Durand, M. B. Coutte, C. Chevrier, S. Cordier. Atrazine in municipal drinking water and risk of low birth weight, preterm delivery, and small-for-gestational-age status. Occupational Environ Med 2005; Vol. 62, 6, 400-405. 25. Waller S. A., K. Paul, S. E. Peterson, J. E. Hitti. Agricultural-related chemical exposures, season of conception, and risk of gastroschisis in Washington State. Am J Obstet Gynecol 2010, Vol. 202, 241.

CONTENT DOMAIN: COMMUNITY WATER INDICATOR: ARSENIC ENVIRONMENTAL PUBLIC HEALTH TRACKING	
Type of EPHT Indicator	Hazard, Exposure
Measures	**Level of Contaminant in Finished Water** 1. Yearly distribution of number of Community Water Systems (CWS) by maximum arsenic concentration (cut-points: 0-5, >5-10, >10-30, >30 µg/L arsenic). 2. Yearly distribution of number of CWS by mean arsenic concentration (cut-points: 0-5, >5-10, >10-20, >20-30, >30 µg/L arsenic). 3. Mean concentration of arsenic at CWS-level, by year. **Potential Population Exposure to Contaminants in Finished Water** 1. Yearly distribution of number of people served by CWS by maximum arsenic concentration (cut-points: 0-5, >5-10, >10-20, >20-30, >30 µg/L arsenic). 2. Yearly distribution of number of people served by CWS by mean arsenic concentration (cut-points: 0-5, >5-10, >10-20, >20-30, >30 µg/L arsenic).
Derivation of Measures	Arsenic measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	Concentration of arsenic, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	**Arsenic and Public Health** Exposures to higher than average levels of arsenic can come from elevated localized soil and ground water concentrations from application and runoff of

	arsenical pesticides and leachate from coal ash and landfills (ATSDR 2005). Exposure to hundreds of micrograms per liter of arsenic found in drinking water of Taiwan, Chile, Argentina, Mexico, Bangladesh, and India has been associated with many adverse health effects including lung, bladder, liver and skin cancers (NRC, 1999; Rahman et al. 2005; Salazar et al. 2004; Fazal et al., 2001). Arsenic has been identified as a human carcinogen by the International Agency for Research in Cancer (IARC) (IARC, 2004). Other adverse health effects include nausea, cardiovascular disease, (Chen et al., 2007; Chih-Hao et al., 2007; Bunderson et al., 2004), developmental and reproductive effects (Hopenhayn et al., 2003; Ahmad et al., 2001)), Diabetes Mellitus (Rahman et al., 1998), and skin keratosis and hyperpigmentation (Kapaj et al., 2006). Measured arsenic concentrations in finished drinking water can be used to understand the distribution of potential arsenic exposure levels for populations served by community water supplies. These measures allow for comparison of potential for arsenic exposures between the populations served by different water systems and water sources over time, and potentially across demographic groups. **Sources of Arsenic** Arsenic compounds (As (III) and As (V)) are found in both ground water and surface waters. The primary sources are geologic formations from which arsenic can be dissolved. Higher levels of arsenic tend to be found in ground water (e.g. aquifers) as compared to surface waters (e.g., lakes, rivers). **Arsenic Regulation and Monitoring** In 2001 EPA reduced the regulatory drinking water standard Maximum Contaminant Level (MCL) to 10 µg/L from 50 µg/L (effective January 23, 2006) on the basis of bladder and lung cancer risks (EPA 2001a). The cancer risks were extrapolated from the Taiwanese (Chen et al. 1985) study to U.S. risks. Lowering the MCL from 50 to 10 ppb statistically reduces bladder and lung cancer mortality and morbidity by 37-56 cancers a year in the U.S. (EPA 2001b). Based on the current understanding of the health impacts from arsenic exposure, the potential for adverse health effects from drinking water exposure to arsenic is very low for most municipal drinking water systems.
Use of Measure	These measures assist by providing data that can be used for surveillance purposes. • Distribution measures provide information on the number of CWS and the number of people potentially exposed to arsenic at different concentrations. • Maximum concentrations provide information on the peak potential exposure to arsenic at the state level. • Mean concentrations at the CWS level provide information on potential

	exposure at a smaller geographic scale.
Limitations of The Measure	Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	Samples are taken once a year (surface sources), once every three years (groundwater sources), or once every nine years (for sources with a waiver). Frequency of sampling is based on compliance with the MCL; the lower the measured concentration the fewer samples will be taken and some years there may be no sampling for arsenic. Ground water systems may have multiple wells with different arsenic concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the arsenic concentration of people served by wells with higher arsenic concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different arsenic levels are averaged to estimate levels for the PWS.
Related Indicators	Public Water Use
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CONTENT DOMAIN: COMMUNITY WATER INDICATOR: DI(2-ETHYLHEXYL)PHTHALATE (DEHP) ENVIRONMENTAL PUBLIC HEALTH TRACKING	
Type of EPHT Indicator	Hazard, Exposure
Measures	**Level of Contaminant in Finished Water** 1. Yearly distribution of number of Community Water Systems (CWS) by maximum DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP). 2. Yearly distribution of number of CWS by mean DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP). 3. Mean concentration of DEHP at CWS-level, by year. **Potential Population Exposure to Contaminants in Finished Water** 4. Yearly distribution of number of people served by CWS by maximum DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP). 5. Yearly distribution of number of people served by CWS by mean DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP).
Derivation of Measures	DEHP measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	DEHP, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	**Di (2-ethylhexyl)phthalate and Public Health** DEHP is the most commonly used of a group of related chemicals called phthalates or phthalic acid esters. Some people who drink water containing DEHP well in excess of the maximum contaminant level (MCL) for many years may have problems with their livers or could experience reproductive difficulties and may have an increased risk of getting cancer. (U.S.EPA, 2010)

	In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, DEHP was detected in 3,098 systems, which collectively serve more than 45 million people (EPA, 2009). Concentrations of DEHP were greater than the MCL in 460 systems serving 11.5 million people. DEHP was the highest occurring regulated synthetic organic chemical found based on the percent of detections found from the 6 Year Review data. This contamination could be due, in part, to sample contamination from older generation laboratory and field sampling equipment made of plastics that contained and released phthalates (EPA, 2009). Most of what we know about the health effects of DEHP comes from studies of rats and mice given high amounts of DEHP. Brief oral exposure to very high levels of DEHP damaged sperm in mice. Although the effect reversed when exposure ceased, sexual maturity was delayed in the animals. High amounts of DEHP damaged the liver of rats and mice. Whether or not DEHP contributes to human kidney damage is unclear. The Department of Health and Human Services has determined that DEHP may reasonably be anticipated to be a human carcinogen. The EPA has determined that DEHP is a probable human carcinogen. These determinations were based entirely on liver cancer in rats and mice. The International Agency for Research on Cancer has stated that DEHP cannot be classified as to its carcinogenicity to humans. People are exposed through ingestion, inhalation, and, to a lesser extent, dermal contact with products that contain phthalates. For the general population, dietary sources have been considered as the major exposure route, followed by inhaling indoor air. Infants may have relatively greater exposures from ingesting indoor dust containing some phthalates (Clark et al., 2003). Human milk can be a source of phthalate exposure for nursing infants (Calafat et al., 2004; Mortensen et al., 2005). The intravenous or parenteral exposure route can be important in patients undergoing medical procedures involving devices or materials containing phthalates. In settings where workers may be exposed to higher air phthalate concentrations than the general population, urinary metabolite and air phthalate concentrations are roughly correlated (Liss et al., 1985; Nielsen et al., 1985; Pan et al., 2006). Phthalates are metabolized and excreted quickly and do not accumulate in the body (Anderson et al., 2001). **Biomonitoring Information** Four metabolites of DEHP were measured for the Fourth National Report on Human Exposure to Environmental Chemicals: mono-(2-ethyl-5-hexyl) phthalate (MEHP), mono- (2-ethyl-5-oxohexyl) phthalate (MEOHP), mono-(2-ethyl- 5-hydroxyhexyl) phthalate (MEHHP) and mono-(2-ethyl- 5-carboxypentyl) phthalate (MECPP). MEHP is primarily formed by the hydrolysis of DEHP in the gastrointestinal tract and then absorbed. By contrast, DEHP present in medical devices and parenteral delivery systems results in the

	diester parent compound, rather than the monoester metabolite. being directly introduced into the blood. After parenteral administration hydrolysis of DEHP most likely also occurs in the blood, and subsequent metabolism is similar to that following ingestion (Koch et al., 2005*a*, 2005*b*, 2005*c*). MEOHP, MEHHP, and MECPP are produced by the oxidative metabolism of MEHP and are present at roughly three- to five-fold higher concentrations than MEHP in urine (Barr et al., 2003; Fromme et al., 2007; Koch et al., 2003). MEHP is the putative toxic metabolite of DEHP. Liver toxicity, decreased testicular weight, and testicular atrophy have been observed in rodents fed high doses over a short term or with chronic dosing (McKee et al., 2004; NTP-CERHR, 2000*c*, 2006). In contrast, marmoset monkeys fed high dose DEHP for longer than a year did not demonstrate testicular or liver toxicity (NTP-CERHR, 2006). Very high doses of DEHP have suppressed estradiol production in female rats (Lovecamp-Swan and Davis, 2003). The U.S. Food and Drug Administration determined that in adults, the amounts of DEHP or MEHP received from intravenous delivery systems or blood transfusions (DEHP is hydrolyzed to MEHP in stored blood) would result in short-term elevations similar to background levels (FDA, 2001). However, critically ill neonates and infants receiving selected or multiple intensive procedures, such as exchange transfusions, extracorporeal membrane oxygenation, and parenteral nutrition, could receive higher exposures than the general population (Calafat et al., 2004; FDA, 2001; Loff et al., 2000; Weuve et al., 2006). The levels of MEHP reported in NHANES 1999-2000, 2001-2002, and 2003-2004 appear roughly comparable to those reported previously in several small U.S. studies involving adults (Blount et al., 2000), pregnant women in New York City (Adibi et al., 2003), and low income African-American women in Washington, DC (Hoppin et al., 2002). In another sample of men attending an infertility clinic, the median and 95th percentile values of urinary MEHP were similar, but MEHHP and MEOHP were about three to five times higher than comparable values found in males in two NHANES survey periods (1999-2000, 2001-2002) (CDC, 2005; Hauser et al., 2007). In separate analyses of NHANES 1999-2000 and NHANES 2001-2002, the adjusted geometric mean levels of urinary MEHP were significantly higher in children compared with adolescents and adults, and in females compared with males (CDC, 2005; Silva et al., 2004). Studies of hospitalized neonates have reported urinary geometric mean levels of MEHP, MEOHP, and MEHHP that were two to five times higher, or more (depending on the intensity of DEHP-product exposure), than the geometric means of children in the NHANES subsamples for all three survey periods (Calafat et al., 2004; Weuve et al., 2006). Small studies of plasma and platelet donors have reported very high levels of MEHP, MEOHP, MEHHP and MECPP in urine collected shortly after these procedures (Koch et al., 2005*b*, 2005*c*). Finding a measurable amount of one or more DEHP metabolites in urine does not mean that the levels of the metabolites or the parent compound cause an adverse health effect. Biomonitoring studies on levels of urinary DEHP metabolites provide physicians and public health

	officials with reference values so that they can determine whether people have been exposed to higher levels of DEHP than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects. **Sources of DEHP** Phthalates are industrial chemicals, often called ***plasticizers***, that are added to plastics make them more flexible and resilient. Phthalates are also used in other applications as solubilizing and stabilizing agents. Numerous products contain phthalates: adhesives; automotive plastics; detergents; lubricating oils; some medical devices and pharmaceuticals; plastic raincoats; solvents; vinyl tiles and flooring; and personal-care products, such as soap, shampoo, deodorants, lotions, fragrances, hair spray, and nail polish. Phthalates are often used in polyvinyl chloride-type plastics, such as plastic bags, garden hoses, inflatable recreational toys, blood product storage bags, intravenous medical tubing, and toys (ATSDR, 2001, 2002). Because they are not chemically bound to the plastics to which they are added, phthalates can be released into the environment during use or disposal of the product. Various phthalate esters have been measured in specific foods, indoor and ambient air, indoor dust, water sources, and sediments (Clark et al., 2003). DEHP is primarily used to produce flexibility in plastics, mainly polyvinyl chloride, which is used for many consumer products, toys, packaging film, and blood product storage and intravenous delivery systems. Concentrations in plastic materials may reach 40% by weight. DEHP has been removed from or replaced in most toys and food packaging in the United States. Following ingestion, DEHP is metabolized to more than 30 metabolites which are rapidly eliminated in urine, and in humans, as glucuronide conjugates (Albro et al., 1982; Albro and Lavenhar, 1989; ATSDR, 2002; Peck and Albro, 1982). The major source of di(2-ethylhexyl) phthalate in drinking water is discharge from rubber and chemical factories (U.S. EPA, 2010). **DEHP Regulation and Monitoring** The EPA limits the amount of DEHP that may be present in drinking water to 6 parts of DEHP per billion parts of water (6 ppb), or 6 ug/L. The Occupational Safety and Health Administration (OSHA) sets a maximum average of 5 milligrams of DEHP per cubic meter of air (5 mg/m^3) in the workplace during an 8-hour shift. The short-term (15-minute) exposure limit is 10 mg/m^3.
Use of Measure	These measures assist by providing data that can be used for surveillance purposes. • Distribution measures provide information on the number of CWS and the

	number of people potentially exposed to DEHP at different concentrations. • Maximum concentrations provide information on the peak potential exposure to DEHP at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	The current measures are derived for CWS only. Private wells may be another source of population exposure to DEHP. Transient non-community water systems, which are regulated by EPA, may also be an important source of DEHP exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	Ground water systems may have many wells with different DEHP concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the DEHP concentration of people served by wells with higher DEHP concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different DEHP levels are averaged to estimate levels for the PWS.
Related Indicators	Public Water Use
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CONTENT DOMAIN: COMMUNITY WATER INDICATOR: DISINFECTION BYPRODUCTS ENVIRONMENTAL PUBLIC HEALTH TRACKING	
Type of EPHT Indicator	Hazard, Exposure
Measures	**Level of Contaminant in Finished Water** 1. Quarterly distribution of number of Community Water Systems (CWS) by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 2. Yearly distribution of number of CWS by maximum HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 3. Yearly distribution of number of CWS by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 4. Mean concentration of HAA5 at CWS-level, by year. 5. Quarterly distribution of number of CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) mg/L TTHM). 6. Yearly distribution of number of CWS by maximum TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) mg/L TTHM). 7. Yearly distribution of number of CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) mg/L TTHM). 8. Mean concentration of TTHM at CWS-level, by year. **Potential Population Exposure to Contaminants in Finished Water** 9. Quarterly distribution of number of people served by CWS by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 10. Yearly distribution of number of people served by CWS by maximum HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 11. Yearly distribution of number of people served by CWS by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 12. Quarterly distribution of number of people served by CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) mg/L TTHM). 13. Yearly distribution of number of people served by CWS by maximum TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) mg/L TTHM). 14. Yearly distribution of number of people served by CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80),

	(>80-100), (>100) mg/L TTHM).
Derivation of Measures	Disinfection byproducts measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Trihalomethanes comprise chloroform, bromodichloromethane, dibromochloromethane, bromoform and their sum, denoted total trihalomethanes (TTHM). Haloacetic acids comprise trichloroacetic acid, dichloroacetic acid, monochloroacetic acid, dibromoacetic acid, monobromoacetic acid, and their sum, denoted HAA5. Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	concentration of HAA5, µg/L concentration of TTHM, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	2002 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	**Disinfection By Products and Public Health** Disinfection byproducts (DBP) are formed when disinfectants used to inactivate microbial contaminants in water react with materials, primarily organic matter, in the water (Bellar et al. 1974, Rook 1974, Cedergren et al. 2002, Sadiq and Rodriguez 2004). Several hundred DBPs in over a dozen chemical classes have been identified (Woo et al. 2002, Krasner et al. 2006). Most commonly, DBPs form when chlorine reacts with naturally occurring organic matter in the source water. DBPs have been associated with both cancer and adverse pregnancy outcomes. High DBP levels, mainly for THMs, have been linked to bladder, colon and rectal cancer (King and Marrett 1996, Cantor et al. 1998, Amy et al. 2005, Villanueva et al. 2004, Villanueva et al. 2007), with bladder cancer reported most frequently. Although findings about adverse pregnancy outcomes have been less definitive, DBPs have been implicated in fetal loss (Swan et al. 1998, Waller et al. 1998, King et al. 2000, Dodds et al. 2004) and a variety of adverse birth outcomes involving growth (Bove et al. 1995, Gallagher et al. 1998, Wright et al. 2004, Infante-Rivard 2004, Toledano et al. 2005) and birth defects (Dodds et al. 1999, Klotz and Pyrch 1999, Dodds and King 2001, Cedergren et

	al. 2002, Shaw et al. 2003). In contrast, however, other research has found little effect on birth outcomes (Savitz et al., 2006). Animal, microbial, in vitro and modeling studies have also pointed to toxicity or carcinogenicity of a wide variety of DBPs (Boorman 1999, Komulainen 2004). Numerous studies have indicated that different DBPs among the THMs and HAAs have different health effects. A number of studies have suggested that iodinated and brominated DBPs are more toxic than their chlorinated counterparts (Plewa et al. 2002, 2004, Richardson 2005). It is therefore appropriate that the tracking network follow individual DBP species and not just class totals (*c.f.* Singer 2006). **Sources of DBPs** DPB levels tend to be highest in water derived from surface sources because ground water generally has little organic matter (Symons et al. 1975, Whitaker et al. 2003). Ground water can, however, produce relatively high levels of the more brominated DBPs when the water, due either to geological circumstances (Whitaker et al. 2003) or salt water intrusion in coastal areas (von Gunten 2003), has elevated levels of bromide. Bromate and chlorite are formed primarily after disinfection by ozone and chlorine dioxide, respectively. Sampling for these DBPs is required only for treatment plants that use the disinfectants that form them. Ozonation and chlorine dioxide are less common mechanisms of disinfection so these two DBPs will not be tracked initially. The disinfection processes that produce these two byproducts are likely to be used more often in the future so bromate and chlorite should be considered for eventual incorporation into the tracking network. **DBP Regulation and Monitoring** Safe Drinking Water Act (SDWA) regulation of DBPs began with the 1979 Total Trihalomethane Rule. This rule set an interim MCL for total trihalomethanes (TTHM), defined as the sum of four trihalomethanes, of 0.10 mg/L for community water systems (CWS) serving 10,000 or more people and using a disinfectant. The Stage 1 Disinfectants and Disinfection Byproducts Rule of 1998 (US EPA 1998) reduced the MCL for TTHM to 0.080 mg/L, added MCLs for the sum of five haloacetic acids (HAA5) of 0.060 mg/L, bromate of 0.010 mg/L and chlorite of 1.0 mg/L, and increased the scope of the rule to cover all CWS that disinfect. The rule had phased compliance with a date of 1 January 2002 for public water systems (PWS) with 10,000 or more people with a surface water or ground water under direct influence source and a date of 1 January 2004 for all other affected PWSs. The Stage 2 Disinfectants and Disinfection Byproducts Rule of 2006 (US EPA 2006) did not alter MCLs but did change how compliance with MCLs will be calculated and requires that PWSs evaluate their distribution systems for appropriate sampling locations. The results of this evaluation may affect the number and location of samples.

	The scope of the rule also increased to cover consecutive systems that receive finished water from other systems. The first reporting deadline for compliance with the Stage 2 rule was in 2006 but it will be a number of years before the rule requires the new compliance calculations based on routine DBP samples. Currently, therefore, Safe Drinking Water Act standards exist for two classes of halogenated organic DBPs, trihalomethanes (THM) and haloacetic acids (HAA), and for two inorganic compounds, bromate and chlorite (US EPA, 2007). Given the near ubiquity of chlorine disinfection, the THMs and HAAs are useful indicators of risk for other DBPs because they occur at high levels and are easily measured. In summary, evidence suggests that disinfection byproducts adversely affect human health. The THMs and HAAs are the most commonly formed DBPs that are routinely tracked in state Safe Drinking Water Act databases. Measures based on these contaminants thus provide a window into potential human exposure to DBPs in publicly provided drinking water. They show where people are potentially exposed to high levels of DBPs. These water supply systems are candidates for enhancement of source water quality, infrastructure improvements or other interventions to reduce DBP exposure.
Use of Measure	These measures assist by providing data that can be used for surveillance purposes. • Distribution measures provide information on the number of CWS and the number of people potentially exposed to nitrate at different concentrations. • Maximum concentrations provide information on the peak potential exposure to nitrate at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	The current measures are derived for CWS only. Transient non-community water systems, which are regulated by EPA, may also be an important source of DBPs exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	Safe Drinking Water Act compliance data include only a handful of the hundreds of known DBPs (Weinberg et al. 2002), most of which occur in chemical classes other than THMs and HAAs. While compliance sampling for THMs and HAAs is directed at the DBPs thought to be most commonly produced by chlorination, non-regulated DBPs exist even among the THMs and HAAs.

	Concern has also been expressed about iodinated THMs and HAAs which, while present in lower concentrations than the brominated and chlorinated THMs, are thought to be toxic at lower doses (e.g. Plewa et al. 2004). THMs and HAAs may not be the most satisfactory indicators of DBP levels in waters subject to alternative disinfection methods that produce different DBPs in different proportions than chlorination (Richardson 2002, Weinberg et al. 2002) and may result in high levels of unregulated DBPs. Little is known about the quantitative occurrence of these DBPs in the distribution system (Richardson et al. 2002, Krasner et al. 2006). While the health effects of different DBPs may vary, with some suspected to be hazardous, few have been characterized for their effects on human health (Woo et al. 2002). Correlations among different DBPs can be relatively low (King et al. 2004, Rodriguez et al. 2004a) so that the measured concentrations of THMs and HAAs may not be good predictors of exposure to other DBPs or overall DBP exposure. THM4 or HAA5, which are the only available data in some state databases, may therefore tell little about the relative concentrations of the THMs or HAAs. DBP levels vary seasonally (Singer et al. 1981, Whitaker et al. 2003, Rodriguez et al. 2004b). Quarterly samples may not capture maximum levels and may not even adequately reflect short term levels. They may therefore be inadequate for estimating exposure during critical periods of a pregnancy, which may be as short as tow to three weeks, especially if peak exposure matters more than average exposure. Furthermore, these fluctuations make it difficult to characterize levels with a single number such as an annual average and thus pose challenges to the development of meaningful synopses of patterns and trends. DBP levels are spatially and temporally labile within a distribution system (Rodriguez et al. 2004b). THM levels increase with time after disinfection and therefore with distance from the treatment plant (Chen and Weisel 1998, Rodriguez and Sérodes 2001). HAA levels may increase or decrease (Chen and Weisel 1998, Rodriguez et al. 2004b), depending upon distribution system conditions. Rechlorination further increases DBP levels. For all but small distribution systems it is therefore impossible to adequately characterize DBP levels with a single value. DBP sampling locations may change over time, making it more difficult to compare measurements from year to year. Better estimation of DBP levels will require spatial and hydraulic modeling of distribution systems. Water supply systems sample for DBPs on different schedules that range from quarterly to triennially. Different sampling frequencies complicate comparisons among different water supply systems. Long intervals between samples, although allowed only where THM and HAA levels have been found to be well under the MCL, create greater uncertainty about levels between sampling dates

	and require stronger assumptions when estimating exposure during short term events such as pregnancies. When allowed, annual or triennial monitoring takes place during the month of warmest weather and may therefore overestimate average DBP levels. Water supply systems that have disinfection waivers generally have no DBP sample results. While the default assumption that these water supply systems have DBP concentrations of zero is generally reasonable, low levels of DBPs can be found in raw ground water, e.g., from surface contamination or from movement of chlorinated water from onsite wastewater treatment systems into ground water. Human behavior greatly influences exposure, complicating efforts to estimate exposure from tap water measurements (Nieuwenhuijen et al. 2000, Kaur et al. 2004, Nuckols et al. 2005). Among the influences on exposure are showering and bathing time, consumption of tap water, use of bottled water, and exposure to water at workplaces or other locations outside the home. Moreover, ascertaining DBP levels in drinking water does not address other routes of exposure such as swimming (Villanueva et al. 2007, Zwiener et al. 2007). This consideration is not strictly a limitation of the measure but pertains to using the measure as an indicator of exposure. Some state SDWA databases may contain only totals for THMs and HAAs and may not record sample results for individual DBPs. Measures involving individual THMs and HAAs cannot be calculated for these states.
Related Indicators	Public Water Use
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CONTENT DOMAIN: COMMUNITY WATER INDICATOR: NITRATE ENVIRONMENTAL PUBLIC HEALTH TRACKING	
Type of EPHT Indicator	Hazard, Exposure
Measures	**Level of Contaminant in Finished Water** 15. Quarterly distribution of number of Community Water Systems (CWS) by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 16. Yearly distribution of number of CWS by maximum nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 17. Yearly distribution of number of CWS by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 18. Mean concentration of nitrate at CWS-level, by year. **Potential Population Exposure to Contaminants in Finished Water** 19. Quarterly distribution of number of people served by CWS by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 20. Yearly distribution of number of people served by CWS by maximum nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 21. Yearly distribution of number of people served by CWS by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate).
Derivation of Measures	Nitrate measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	Concentration of nitrate, mg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	**Nitrates and Public Health**

Nitrate was first identified as a public health threat in drinking water in 1945 when high nitrate levels from private wells were shown to cause methemoglobinemia or "blue baby syndrome" in infants who received formula made from well water. When an individual is exposed to nitrate it can be converted to nitrite ($NO2^-$) in the body and then oxidize the ferrous iron (Fe^{+2}) in deoxyhemoglobin in the blood to form methemoglobin containing ferric iron (Fe^{+3}). Methemoglobin cannot transfer oxygen to tissues; thus nitrate or nitrite can starve the body of oxygen and produce a clinical condition known as cyanosis, where the lips and extremities turn gray or blue. Infants younger than four months of age are more sensitive than adults, and can develop "blue baby" syndrome from intake of nitrate higher than 10 mg/L nitrate or 45 mg/L nitrate–nitrogen. Blue baby syndrome is fatal in about ten percent of the cases (ATSDR, 2007). Usually there are no outward signs of cyanosis at methemoglobin levels below 20 percent (Dabney et al, 1990).

In addition, there is some evidence to suggest that exposure to nitrate in drinking water is also associated with adverse reproductive outcomes such as spontaneous abortions, intrauterine growth retardation, and various birth defects such as anencephaly, related to fetal exposures to nitrate. However, the evidence is inconsistent (Manassaram et al, 2006).

Similarly, long term exposure to higher nitrate levels in drinking water has been suggested as a risk factor for cancer. Cancer at several sites (i.e. gastric, colorectal, bladder, urothelial, brain, esophagus, ovarian and non-Hodgkins lymphoma have been shown to be associated with nitrate in drinking water in some studies (Sandor et al, 2001; Weyer et al, 2001; Gulis et al, 2002; De Roos et al, 2003; Volkmer et al, 2005; Ward et al, 2005b; Chiu et al, 2007;). Other studies have not found any association (Ward et al, 2003; Ward et al, 2005, 2005c; Ward et al, 2006; Zeegers et al, 2006). Significant regional differences in cancer risk may occur (Mueller et al, 2001). Occupational exposures are also of concern as nitrate fertilizer workers have shown increased risk for stomach cancer (Zandjani et al. 1994).

Sources of Nitrate

Nitrate is the most commonly found contaminant in groundwater aquifers worldwide (Ward, 2005 from: Spalding and Exner 1993). Nitrate ($NO3^-$) originates in drinking water from nitrate-containing fertilizers, sewage and septic tanks, and decaying natural material such as animal waste. Nitrate is very soluble in water, can easily migrate, and does not evaporate (EPA Consumer Fact Sheet). Anthropogenic sources of nitrates are increasing resulting in increased nitrate levels in water resources. Surface water and shallow wells in both rural and urban areas can be affected. Consequently, private wells are especially vulnerable to excess levels of nitrates. Excess levels of nitrate and nitrite can occur in community water supplies. A U.S. Geological Survey (USGS) study found nitrate levels exceeded regulatory monitoring standards in 2% of a sample of 242 public drinking water wells between 1992 and 1999 (Squillace et al, 2002). Levels of nitrates in private wells are less well known; private wells are not regularly monitored and are often more vulnerable to higher levels of nitrates because they draw water from shallower groundwater aquifers. The

	USGS estimates approximately 22% of domestic wells in agricultural areas of the U.S. exceed the MCL (Ward, 2007). **Nitrate Regulation and Monitoring** Congress established the Safe Drinking Water Act in 1974, which set enforceable Maximum Contaminant Levels (MCLs) and non-enforceable Maximum Contaminant Level Goals (MCLGs) for certain specified contaminants. In the case of nitrate in drinking water, the MCLG of 10 mg/L (ppm) was established from human data from studies of methemoglobinemia in young children. (Johnson and Kross 1990; Walton, 1950). The MCL is also set at 10 ppm, and any exceedance of the MCL is potentially serious as there is no additional margin of safety between the MCLG and the MCL. 2002). The MCLG and MCL for nitrite are 1 mg/L. While evidence to suggest MCL exposures for chronic health endpoints remains inconclusive, there is some evidence to suggest that chronic exposure to nitrate levels below the MCL may be of concern (Ward, 2005).
Use of Measure	These measures assist by providing data that can be used for surveillance purposes. • Distribution measures provide information on the number of CWS and the number of people potentially exposed to nitrate at different concentrations. • Maximum concentrations provide information on the peak potential exposure to nitrate at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	The current measures are derived for CWS only. Private wells are another important source of population exposure to nitrate. Transient non-community water systems, which are regulated by EPA, may also be an important source of nitrate exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	Nitrate levels can vary substantially in groundwater; thus high levels may not be captured by even quarterly sampling. Estimates of the number of people potentially exposed may be unreliable as they are based on estimates made by the water system operator. Concentrations in drinking water cannot be directly converted to exposure because overall water consumption, and the proportion of water consumed that comes from the tap is quite variable (EPA 2004). In systems that have more than one Entry point to the Distribution system, the actual nitrate level at any given house is a mixture of the levels from all contributing sources. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to

	underestimate the nitrate concentration of people served by wells with higher nitrate concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different nitrate levels are averaged to estimate levels for the PWS.
Related Indicators	Public Water Use
References	51. ATSDR Case Studies in Environmental Medicine: Nitrate/Nitrite Toxicity. http://www.atsdr.cdc.gov/HEC/CSEM/nitrate/index.html Downloaded 08/07/07 52. Bosch, H. M., A. B. Rosenfield, R. Huston, H. R. Shipman, and F. L. Woodward. 1950. Methemoglobinemia and Minnesota well supplies. Am. Water Works Assoc J 42:161-170. 53. Chiu HF, Tsai SS, Yang CY. 2007. Nitrate in drinking water and risk of death from bladder cancer: an ecological case-control study in Taiwan. J Toxicol Environ Health A 70(12):1000-1004. 54. Coss A, Cantor KP, Reif JS, Lynch CF, Ward MH. 2004. Pancreatic cancer and drinking water and dietary sources of nitrate and nitrite. Am J Epidemiol 159(7):693-701. 55. Dabney BJ, Zelarney PT, Hall AH. 1990. Evaluation and treatment of patients exposed to systemic asphyxiants. Emerg Care Q 6(3):65-80 56. De Roos AJ, Ward MH, Lynch CF, Cantor KP. 2003. Nitrate in public water supplies and the risk of colon and rectum cancers. Epidemiology 14(6):640-649. 57. Gulis G, Czompolyova M, Cerhan JR. 2002. An ecologic study of nitrate in municipal drinking water and cancer incidence in Trnava District, Slovakia. Environ Res 88(3):182-187. 58. Johnson CJ and Kross BC. 1990. Continuing importance of nitrate contamination of groundwater and wells in rural areas. Am J Ind Med 18(4):449-456. 59. Mueller BA, Newton K, Holly EA, Preston-Martin S. 2001. Residential water source and the risk of childhood brain tumors. Environ Health Perspect 109(6):551-556. 60. Ruckart PZ, Henderson AK, Black ML, Flanders WD. 2007. Are nitrate levels in groundwater stable over time? J Expo Sci Environ Epidemiol Apr 11; [Epub ahead of print] 61. Sandor J, Kiss I, Farkas O, Ember I. 2001. Association between gastric cancer mortality and nitrate content of drinking water: ecological study on small area inequalities. Eur J Epidemiol 17(5):443-447. 62. U.S. Environmental Protection Agency Office of Water: Candidate Contaminants List. http://www.epa.gov/safewater/ccl/index.html Downloaded 08/02/07 63. U.S. Environmental Protection Agency. Office of Water (4606) Occurrence Estimation Methodology and Occurrence Findings Report for the Six-Year Review of Existing National Primary Drinking Water Regulations. EPA-815-R-03-006 www.epa.gov June 2003. http://www.epa.gov/safewater/standard/review/pdfs/support_6yr_occurancemethods_final.pdf Downloaded 08/02/07 64. U.S. Environmental Protection Agency (2007b): Technical Factsheet on: Nitrate/Nitrite. http://www.epa.gov/safewater/dwh/t-ioc/nitrates.html Downloaded 08/07/07 65. Volkmer BG, Ernst B, Simon J, Kuefer R, Bartsch G Jr, Bach D, Gschwend JE. 2005. Influence of nitrate levels in drinking water on urological malignancies: a community-based cohort study. BJU Int 95(7):972-976. 66. Walton, G. 1951. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. Am J Public Health 41:986-996. 67. Ward MH, Cantor KP, Riley D, Merkle S, Lynch CF. 2003. Nitrate in public water

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CONTENT DOMAIN: COMMUNITY WATER INDICATOR: PUBLIC WATER USE ENVIRONMENTAL PUBLIC HEALTH TRACKING	
Type of EPHT Indicator	Exposure
Measures	22. Number of people receiving water from community water systems.
Derivation of Measures	This measure will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format.
Units	1. Number of people
Geographic Scope	State
Geographic Scale	State
Time Period	2009 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	**Public Water Use and Public Health** The public water use index provides some data to explore the relative importance of community water supplies as sources of drinking water and to provide context for subsequent community drinking water system (CWS) indicators. SDWA collects data for a number of different types of public water systems of which community water systems (CWS) are a sub-set. The community water systems represent non-transient public water systems that serve year round community residents and are the focus of the initial indicators. The range of state populations served by CWS as their primary residential drinking water source varies from 95% to as low as 40% within the United States. Understanding the relative population coverage of these indicators by state helps to understand representativeness of these data for prioritization and evaluation across the United States and within individual states and communities.
Use of Measure	This measure can be useful in providing data for surveillance purposes. • Estimated population potentially exposed to contaminants in CWS.
Limitations of The Measure	The current measure is derived for CWS only. Private wells are another important source of population exposure to water contaminants. Transient non-community water systems, which are regulated by EPA, may also be an important source of potential exposure.
Data Sources	State grantee
Limitations of Data Sources	Population estimates are rough and may overestimate or underestimate the number of affected people.

Related Indicators	All other community water indicators.
Additional Information	1. U.S. Environmental Protection Agency, *Water On Tap*, Office of Water (4601) EPA 816-K-09-002, December 2009. **http://water.epa.gov/drink/guide/upload/book_waterontap_full.pdf** 2. U.S. Environmental Protection Agency, Public Drinking Water Systems: Facts and Figures **http://water.epa.gov/infrastructure/drinkingwater/pws/factoids.cfm** 3. U.S. Environmental Protection Agency, Public Drinking Water Systems Programs. **http://water.epa.gov/infrastructure/drinkingwater/pws/index.cfm**

CONTENT DOMAIN: COMMUNITY WATER INDICATOR: COMBINED RADIUM-226 AND -228 ENVIRONMENTAL PUBLIC HEALTH TRACKING	
Type of EPHT Indicator	Hazard, Exposure
Measures	**Level of Contaminant in Finished Water** 1. Yearly distribution of number of Community Water Systems (CWS) by maximum Radium concentration (cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium). 2. Yearly distribution of number of CWS by mean Radium concentration (cut-points: cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium). 3. Mean concentration of Radium at CWS-level, by year. **Potential Population Exposure to Contaminants in Finished Water** 4. Yearly distribution of number of people served by CWS by maximum Radium concentration (cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium). 5. Yearly distribution of number of people served by CWS by mean Radium concentration (cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium).
Derivation of Measures	Combined Radium-226 and -228 measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	**pCi/L combined Radium-226 & -228**
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	**Radium-226 and -228 and Public Health** Radium is a naturally occurring silvery-white radioactive metal that can exist in several forms called isotopes. Radium is produced constantly by the radioactive decay of uranium and thorium. Uranium and thorium are found in small amounts in most rocks and soil. Some of the radiation from radium is being released constantly into the environment. It is this radioactive decay that causes concern

	about the safety of radium and all other radioactive substances. Two of the main radium isotopes found in the environment are radium-226 and radium-228. The decay of radium-226 results in the formation of radon which exists as a gas and is mobile in environmental media. Radium has been used as a radiation source for treating cancer, in radiography of metals, and combined with other metals as a neutron source for research and radiation instrument calibration. Until the 1960s, radium was a component of the luminous paints used for watch and clock dials, instrument panels in airplanes, military instruments, and compasses (ATSDR, 2010). Everyone is exposed to low levels of radium in the air, water, and food. Higher levels may be found in the air near industries that burn coal or other fuels or near sites that mine or mill uranium. It also may be found at higher levels in drinking water from groundwater wells. Miners, particularly miners of uranium and hard rock, are exposed to higher levels of radium. It may also be found at radioactive waste disposal sites (ATSDR, 1990). It is not known whether long-term exposure to radium at the levels that are normally present in the environment (for example, 1 pCi of radium per gram of soil) is likely to result in harmful health effects. However, exposure to higher levels of radium over a long period of time may result in harmful effects including anemia, cataracts, fractured teeth, cancer (especially bone cancer), and death. Patients who were injected with radium in Germany, from 1946 to 1950, for the treatment of certain diseases including tuberculosis were significantly shorter as adults than people who were not treated. Some of these health effects may take years to develop and mostly are due to gamma radiation. Radium gives off gamma radiation, which can travel fairly long distances through air. Therefore, just being near radium at the high levels that may be found at some hazardous waste sites may be dangerous to your health. Exposure to high levels of radium results in an increased incidence of bone, liver, and breast cancer. The EPA and the National Academy of Sciences, Committee on Biological Effects of Ionizing Radiation, has stated that radium is a known human carcinogen. **Biomonitoring Information** Urine tests can determine if you have been exposed to radium. Another test measures the amount of radon (a breakdown product of radium) in exhaled air. Both types of tests require special equipment and cannot be done in a doctor's office. These tests cannot tell how much radium you were exposed to, nor can they be used to predict whether you will develop harmful health effects (ATSDR, 1990). Levels of radium in the U.S. population are unknown. **Sources of Radium** Radium forms from the decay of uranium or thorium in the environment.

	Radium -226 is formed from the decay of uranium-238; Radium-228 is formed from the decay of thorium. Radium is abundant in low levels everywhere because it originates from uranium which is commonly found in all rocks, soil and water. (EPA, 2010) **Radium Regulation and Monitoring** The EPA has set a drinking water limit of 5 picocuries per liter (5 pCi/L) for radium-226 and radium-228 (combined) (EPA, 2009). A gross alpha particle activity measurement may be substituted for the required radium-226 measurement provided that the measured gross alpha particle activity does not exceed 5 pCi/L. The EPA lifetime exposure cancer risk estimate for radium at the MCL, is approximately 1-2 cases per 10,000 people. **Monitoring frequency** Once a CWS has satisfied initial monitoring requirements (4 quarterly samples at every entry point to the distribution system within the first quarter after initiating the source); the required frequency for Combined Radium-226 and -228 monitoring is once every three years if the average of the initial monitoring results for the contaminant is greater than one-half the MCL but at or below the MCL. States may allow CWS to reduce the frequency of monitoring from once every three years to once every six or nine years at each sampling point, if the average of the initial monitoring results for each contaminant is below the detection limit. If a system has a monitoring result that exceeds the MCL while on reduced monitoring, the system must collect and analyze quarterly samples at that sampling point until the system has results from four consecutive quarters that are below the MCL, unless the system enters into another schedule as part of a formal compliance agreement with the State (CFR, 2002).
Use of Measure	These measures assist by providing data that can be used for surveillance purposes. • Distribution measures provide information on the number of CWS and the number of people potentially exposed to combined Radium-226 and -228 at different concentrations. • Maximum concentrations provide information on the peak potential exposure to combined Radium-226 and -228 at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	The current measures are derived for CWS only. Private wells may be another source of population exposure to combined Radium-226 and -228. Transient non-community water systems, which are regulated by EPA, may also be an important source of combined Radium-226 and -228 exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating

	populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	The required monitoring frequency for combined Radium-226 and -228 is infrequent and may be as intermittent as every nine years; therefore most states will have very little data on this contaminant. Ground water systems may have multiple wells with different combined Radium-226 and -228 concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the combined Radium-226 and -228 concentrations of people served by wells with higher combined Radium-226 and -228 concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different combined Radium-226 and -228 levels are averaged to estimate levels for the PWS.
Related Indicators	Public Water Use; Uranium
References	1. Agency for Toxic Substances and Disease Registry (ATSDR). Toxic Substances Portal. Radium. 2010. Available at: http://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=154 2. Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological Profile for Radium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service Available at: http://www.atsdr.cdc.gov/toxfaqs/TF.asp?id=790&tid=154 3. Code of Federal Regulations (CFR), 2002. Title 40 Protection of the Environment Chapter I--Environmental Protection Agency Part 141--National Primary Drinking Water Regulations 141.26 Monitoring frequency and compliance requirements for radionuclides in community water systems. Available at: URL: http://www.access.gpo.gov/nara/cfr/waisidx_02/40cfr141_02.html 4. U.S. Environmental Protection Agency (U.S. EPA). Radiation Protection, Radium, 2010. Available at: http://www.epa.gov/radiation/radionuclides/radium.html 5. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.

CONTENT DOMAIN: COMMUNITY WATER INDICATOR: TETRACHLOROETHENE (TETRACHLOROETHYLENE) (PCE) ENVIRONMENTAL PUBLIC HEALTH TRACKING	
Type of EPHT Indicator	Hazard, Exposure
Measures	**Level of Contaminant in Finished Water** 6. Yearly distribution of number of Community Water Systems (CWS) by maximum PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE). 7. Yearly distribution of number of CWS by mean PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE). 8. Mean concentration of PCE at CWS-level, by year. **Potential Population Exposure to Contaminants in Finished Water** 9. Yearly distribution of number of people served by CWS by maximum PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE). 10. Yearly distribution of number of people served by CWS by mean PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE).
Derivation of Measures	PCE measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	PCE, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be the approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	**Tetrachloroethene (PCE) and Public Health** Tetrachloroethene (PCE) is a volatile halogenated short-chain hydrocarbon. Tetrachloroethene is used in dry cleaning, metal cleaning, the synthesis of other chemicals, and household products such as water repellants, silicone lubricants, and spot removers. PCE is produced and used in high volumes in the U.S. and has been detected in urban and ambient air and occasionally in soils and drinking water most likely contaminated by industrial discharge (Moran et al., 2007; Rowe et al., 2007). Because of its volatility, this solvent does not persist in the soil or water following the discontinuation of contamination.

	Inhalation is the most common exposure route for the general population including indoor sources from paints, adhesives, and cleaning solutions. Volatilization from contaminated water (e.g., shower water) as well as the use of household products containing this solvent can result in higher indoor than outdoor air concentrations (ATSDR, 1997; Martin et al., 2005). Nearby dry cleaning establishments, industries producing PCE, and contaminated waste disposal sites can also contribute to human exposure (Armstrong and Green, 2004; ATSDR, 1997 and 2000; Schreiber et al., 1993; Wallace et al., 1991). Drinking water may contribute to exposure when underground drinking water supplies have been contaminated. Workers in industries such as dry cleaning, aircraft maintenance, electronics manufacturing, and chemical production may be exposed by inhalation or by dermal contact with PCE. The EPA has established drinking water standards and other environmental standards for PCE, and the FDA regulates PCE and trichloroethene as indirect food additives. Workplace standards have been established by OSHA, and ACGIH has recommended occupational guidelines and biological exposure indices for monitoring workers. Human health effects from PCE at low environmental doses or at biomonitored levels from low environmental exposures are unknown. PCE is well absorbed by ingestion and inhalation, and animal studies have demonstrated that liquid forms can be dermally absorbed. Following absorption, part of the solvent dose is excreted into expired air; for PCE, about 97-99% of the dose is eliminated unmetabolized into expired air, though it has an elimination half-life of several days (ATSDR 1997; Monster, 1986). The retained solvent can undergo hepatic metabolism. PCE is metabolized to trichloroacetic acid and trichloroethanol, which are eliminated in the urine. Accidental or intentional high dose acute exposure by ingestion or inhalation can result in loss of motor coordination, somnolence, and unconsciousness. Inhaling high doses of PCE may also produce cardiac arrhythmias attributed to enhanced sensitivity to catecholamines. High dose acute exposure to PCE has resulted in reversible kidney impairment, and prolonged, low level PCE exposure has been associated with altered renal enzyme excretion and liver enlargement (ATSDR, 1997). Chronic occupational exposure to PCE may be associated with mild degrees of neurological impairments, including reaction times, verbal skills, cognitive ability, and motor function (Armstrong and Green, 2004). Various epidemiologic studies of chronic PCE exposure in dry cleaning workers found increased incidences of esophageal and cervical cancers and non-Hodgkins lymphoma, but confounding exposures (e.g., other solvents and trichloroethene) were likely (IPCS, 2006). In animal studies, PCE-induced kidney and liver tumors and caused leukemia (IARC, 1995). IARC classifies PCE as a probable human carcinogen, and NTP classifies it as reasonably anticipated to be a human carcinogen (IARC, 1995; NTP, 2004). Additional information about these solvents is available from ATSDR at: **http://www.atsdr.cdc.gov/toxpro2.html**. In an analysis of occurrence data from the EPA 6 Year Review of National

	Primary Drinking Water Regulations, PCE was detected in 1,262 systems serving close to 32 million people (EPA, 2009). Concentrations of PCE were greater than the MCL in 241 systems serving close to 15 million people. PCE was the fifth highest occurring regulated volatile organic chemical found based on the percent of detections found from the 6 Year Review data (EPA, 2009). **Biomonitoring Information** Levels of halogenated solvents in blood reflect recent exposure. In the NHANES 2003-2004 subsample, the level of blood PCE for adults at the 75th percentile of the U.S. population appear similar to the levels at the 75th percentile reported for non-smoking adults in a subsample of NHANES 1999-2000 participants (CDC, 2009; Lin et al., 2008) and were similar or slightly less than levels reported in a nonrepresentative subsample of the earlier NHANES III (1988-1994) (Ashley et al., 1994; Churchill et al., 2001). A recent study of low income, urban children in the Midwest reported slightly lower median PCE levels (Sexton et al., 2005; Sexton et al., 2006) than the NHANES III levels (Ashley et al., 1994; Churchill et al., 2001). Comparatively higher blood levels of PCE and trichloroethene have been noted for urban and industrial residential settings than for rural settings (Barkley et al., 1980; Begerow et al., 1996; Brugnone et al., 1994). Residing near dry-cleaning facilities or storing recently dry-cleaned clothes at home can contribute to increased blood PCE levels (Begerow et al., 1996; Popp et al., 1992). In contrast, PCE blood levels in occupationally exposed workers have been reported to be many thousand times higher than the general population (Begerow et al., 1996; Furuki et al., 2000; Monster et al., 1983). The occupational biological exposure index associated with an 8-hour exposure of 25 ppm is 500 μg/L PCE in blood (ACGIH, 2007). Non-occupational exposures are usually well below this level. Finding a measurable amount of any of these solvents in blood does not mean that the level of the solvent causes an adverse health effect. Biomonitoring studies of blood halogenated solvents can provide physicians and public health officials with reference values so that they can determine whether or not people have been exposed to higher levels of halogenated solvents than levels found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects. **Sources of PCE** The major source of PCE in drinking water is discharge from factories and dry cleaners. A federal law called the Emergency Planning and Community Right to Know Act requires facilities in certain industries, which manufacture, process, or use significant amounts of toxic chemicals, to report annually on their releases of these chemicals. For more information on the uses and releases of chemicals in your state, contact the Community Right-to-Know Hotline: (800) 424-9346 (EPA, 2010).

	PCE Regulation and Monitoring The EPA limits the amount of PCE that may be present in drinking water to 5 parts of PCE per billion parts of water (5 ppb), or 5 ug/L.
Use of Measure	These measures assist by providing data that can be used for surveillance purposes. • Distribution measures provide information on the number of CWS and the number of people potentially exposed to PCE at different concentrations. • Maximum concentrations provide information on the peak potential exposure to PCE at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	The current measures are derived for CWS only. Private wells may be another source of population exposure to PCE. Transient non-community water systems, which are regulated by EPA, also may be an important source of PCE exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	Ground water systems may have multiple wells with different PCE concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the PCE concentration of people served by wells with higher PCE concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different PCE levels are averaged to estimate levels for the PWS.
Related Indicators	Public Water Use
References	1. ACGIH. TLVs and BEIs Based on the documentation of the threshold limit values for chemical substances and physical agents and biological exposure indices. 2007. Signature Publications. Cincinnati OH. p.104. 2. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for tetrachloroethylene update. 1997 [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/ tp18.html. 4/22/09 3. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for Tetrachloroethylene update. 2000 [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/ tp14.html. 4/22/09 4. Armstrong SR, Green LC. Chlorinated hydrocarbon solvents. Clin Occup Environ Med 2004;4(3):481-496. 5. Ashley DL, Bonin MA, Cardinali FL, McCraw JM, Wooten JV. Blood concentrations of volatile organic compounds in a nonoccupationally exposed US

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CONTENT DOMAIN: COMMUNITY WATER INDICATOR: TRICHLOROETHENE (TRICHLOROETHYLENE) (TCE) ENVIRONMENTAL PUBLIC HEALTH TRACKING	
Type of EPHT Indicator	Hazard, Exposure
Measures	**Level of Contaminant in Finished Water** 3. Yearly distribution of number of CWS by maximum TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE). 4. Yearly distribution of number of CWS by mean TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE). 5. Mean concentration of TCE at CWS-level, by year. **Potential Population Exposure to Contaminants in Finished Water** 6. Yearly distribution of number of people served by CWS by maximum TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE). 7. Yearly distribution of number of people served by CWS by mean TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE).
Derivation of Measures	TCE measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	TCE, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be the approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	**Trichloroethene (TCE) and Public Health** Trichloroethene (TCE) is a volatile halogenated short-chain hydrocarbon. TCE is used primarily as an industrial degreaser, solvent, and in the synthesis of other chemicals. In the past, it was used in dry cleaning, food processing, household cleaners, and as a general anesthetic. TCE is produced and used in high volumes in the U.S. and has been detected in urban and ambient air and occasionally soils and drinking water most likely contaminated by industrial discharge (Moran et al., 2007; Rowe et al., 2007). Because of its volatility, this solvent does not persist in the soil or water following the discontinuation of contamination.

	Drinking or breathing high levels of TCE may cause nervous system effects, liver and lung damage, abnormal heartbeat, coma, and possibly death (ATSDR, 2003). Inhalation is the most common exposure route for the general population including indoor sources from paints, adhesives, and cleaning solutions. Volatilization from contaminated water (e.g., shower water) as well as the use of household products containing this solvent can result in higher indoor than outdoor air concentrations (ATSDR, 1997b; Martin et al., 2005). Nearby dry cleaning establishments, industries producing this solvent, and contaminated waste disposal sites can also contribute to human exposure (Armstrong and Green, 2004; ATSDR, 1997a, 1997b, and 2000; Schreiber et al., 1993; Wallace et al., 1991). Drinking water may contribute to exposure when underground drinking water supplies have been contaminated. Workers in industries such as dry cleaning, aircraft maintenance, electronics manufacturing, and chemical production may be exposed by inhalation or dermal contact. The EPA has established drinking water standards and other environmental standards for TCE, and the FDA regulates TCE as an indirect food additive. OSHA has established workplace standards , and ACGIH has recommended occupational guidelines and biological exposure indices for monitoring workers (ACGIH, 2007). Human health effects from TCE at low environmental doses or at biomonitored levels from low environmental exposures are unknown. TCE is well absorbed by ingestion and inhalation, and animal studies have demonstrated that liquid forms can be dermally absorbed. Following absorption, part of the solvent dose is excreted into expired air (ATSDR1997a; Monster, 1986). The retained solvent can undergo hepatic metabolism. TCE is metabolized to trichloroacetic acid and tricholoroethanol, which are eliminated in the urine. Accidental or intentional high dose acute exposure by ingestion or inhalation can result in loss of motor coordination, somnolence, and unconsciousness. Inhaling high doses of TCE may also produce cardiac arrhythmias attributed to enhanced sensitivity to catecholamines. Prolonged, low level exposure to TCE has been associated with altered renal enzyme excretion and liver enlargement (ATSDR, 1997a, b). Chronic occupational exposure to TCE may be associated with mild degrees of neurological impairments, including reaction times, verbal skills, cognitive ability and motor function (Armstrong and Green, 2004). In animal studies, TCE induced kidney and liver tumors; and caused lung and testicular tumors (IARC, 1995). A recent EPA toxicological review (EPA/635/R-09/011F) characterized TCE as carcinogenic in humans by all routes of exposure (EPA, 2011). For cancer, the inhalation unit risk is 2×10^{-2} per ppm [4×10^{-6} per µg/m3], based on human kidney cancer risks (Charbotel et al.; 2006) and adjusted, using human epidemiologic data, for potential risk for non-Hodgkin lymphoma (NHL) and liver cancer. The oral unit risk for cancer is 5×10^{-2} per mg/kg/day, resulting from physiologically based pharmacokinetic model-based route-to-route extrapolation of the inhalation unit risk based on the human kidney cancer risks (Charbotel et al. 2006) and adjusted, using human epidemiologic data, for potential risk for NHL and liver cancer. There is high confidence in these unit

	risks for cancer, as they are based on good quality human data, as well as being similar to unit risk estimates based on multiple rodent bioassays. Evidence is sufficient to conclude that TCE operates through a mutagenic mode of action for kidney tumors. Evidence is insufficient and TCE-specific quantitative data are lacking on early-life susceptibility. Additional information about TCE is available from ATSDR at: http://www.atsdr.cdc.gov/toxpro2.html. In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, TCE was detected in 1,013 systems serving 29.5 million people (EPA, 2009). Concentrations of TCE were greater than the MCL in 195 systems serving close to 12 million people. TCE was the fifth highest occurring regulated volatile organic chemical found based on the percent of population served by systems with at least one sample detection found from the 6 Year Review data (EPA, 2009). Biomonitoring Information Levels of halogenated solvents in blood reflect recent exposure. Blood levels of TCE were generally not detected in the NHANES 2003-2004 subsample and were detected infrequently in previous U.S. surveys (CDC, 2009). Comparatively higher blood levels of tetrachloroethene and TCE have been noted for urban and industrial residential settings than for rural settings (Barkley et al., 1980; Begerow et al., 1996; Brugnone et al., 1994). Finding a measurable amount of any of these solvents in blood does not mean that the level of the solvent causes an adverse health effect. Biomonitoring studies of blood halogenated solvents can provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of halogenated solvents than levels found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects. Sources of TCE TCE does not occur naturally in the environment. However, it has been found in underground water sources and many surface waters as a result of the manufacture, use, and disposal of the chemical (ATSDR, 2003). TCE Regulation and Monitoring The EPA has set a maximum contaminant level for TCE in drinking water of 0.005 milligrams per liter (0.005 mg/L) or 5 parts of TCE per billion parts water. The EPA has also developed regulations for the handling and disposal of trichloroethylene. OSHA has set an exposure limit of 100 parts of TCE per million parts of air (100 ppm) for an 8-hour workday, 40-hour work week (ATSDR, 2003).
Use of Measure	These measures assist by providing data that can be used for surveillance purposes.

	• Distribution measures provide information on the number of CWS and the number of people potentially exposed to TCE at different concentrations. • Maximum concentrations provide information on the peak potential exposure to TCE at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	The current measures are derived for CWS only. Private wells may be another source of population exposure to TCE. Transient non-community water systems, which are regulated by EPA, also may be an important source of TCE exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	Ground water systems may have multiple wells with different TCE concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the TCE concentration of people served by wells with higher TCE concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different TCE levels are averaged to estimate levels for the PWS.
Related Indicators	Public Water Use
References	1. ACGIH. TLVs and BEIs Based on the documentation of the threshold limit values for chemical substances and physical agents and biological exposure indices. 2007. Signature Publications. Cincinnati OH. p.104. 2. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for tetrachloroethylene update. 1997*a* [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/ tp18.html. 4/22/09 3. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for trichloroethylene update. 1997*b* [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/ tp19.html. 4/22/09 4. Agency for Toxic Substances and Disease Registry (ATSDR). ToxFAQs™ for Trichloroethylene (TCE), July 2003. Available at: **http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=172&tid=30** 5. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for Methylene chloride update. 2000 [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/ tp14.html. 4/22/09 6. Armstrong SR, Green LC. Chlorinated hydrocarbon solvents. Clin Occup Environ Med 2004;4(3):481-496. 7. Barkley J, Bunch J, Bursey JT, Castillo N, Cooper SD, Davis JM, et al. Gas chromatography mass spectrometry computer analysis of volatilie halogenated hydrocarbons in man and his environment—a multimedia environmental study.

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14. Monster AC. Biological monitoring of chlorinated hydrocarbon solvents. J Occup Med 1986;28:583-588.
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16. Moran MJ, Zogorski JS, Squillace PJ. Chlorinated solvents in groundwater of the United States. Environ Sci Technol 2007;41:74-81.
17. National Toxicology Program (NTP). Report on Carcinogens, 11th ed. 2004. [online]. Available at URL: http://ntp.niehs.nih. gov/ntp/roc/eleventh/profiles/s066dich.pdf. 4/22/09
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CONTENT DOMAIN: COMMUNITY WATER INDICATOR: URANIUM (U) ENVIRONMENTAL PUBLIC HEALTH TRACKING	
Type of EPHT Indicator	Hazard, Exposure
Measures	**Level of Contaminant in Finished Water** 1. Yearly distribution of number of Community Water Systems (CWS) by maximum Uranium concentration (cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium). 2. Yearly distribution of number of CWS by mean Uranium concentration (cut-points: cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium). 3. Mean concentration of Uranium at CWS-level, by year. **Potential Population Exposure to Contaminants in Finished Water** 4. Yearly distribution of number of people served by CWS by maximum Uranium concentration (cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium). 5. Yearly distribution of number of people served by CWS by mean Uranium concentration (cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium).
Derivation of Measures	Uranium measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	Uranium, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	**Uranium (U) and Public Health** Uranium is a silver-white metal that is extremely dense and weakly radioactive. It usually occurs as an oxide and is extracted from ores containing less than 1% natural uranium. Natural uranium is a mixture of three isotopes: 238U (greater than 99%), 235U (about 0.72%), and 234U (about 0.01%). Uranium has many commercial uses, including nuclear weapons, nuclear fuel, in some ceramics,

	and as an aid in electron microscopy and photography. Depleted uranium (DU) refers to uranium in which the proportions of 235U and 234U isotopes have been reduced compared with the proportion in natural uranium. Since the 1990's, DU has been used by the military in armor-piercing ammunition and as a component of protective armor for tanks. Natural and depleted uranium are primarily chemical toxicants, with radiation playing a minor role or no role at all (ATSDR, 2009). Everyone is exposed to uranium in food, air, and water as part of the natural environment. (ATSDR, 2009). Variable concentrations of uranium occur naturally in drinking water sources. In some locations the natural concentrations may have increased due to mining and milling of uranium. Thus, the primary exposure sources for non-occupationally exposed persons are likely dietary and drinking water. Populations most heavily exposed to uranium are those employed in mining and milling operations, or in uranium enrichment and processing activities (ATSDR, 2009). In workplaces that involve uranium mining, milling, or processing, human exposure occurs primarily by inhaling dust and other small particles. Exposure to DU may occur in military personnel from retention of internal shrapnel that contains DU or exposure to dust generated from ammunition impact. Absorption of uranium compounds is low by all routes of exposure (i.e., ingestion, inhalation, and skin contact). Depending upon the specific compound and solubility, 0.1%-6% of an ingested dose may be absorbed. Inhaled uranium-containing particles are retained in the lungs, where limited absorption occurs (less than 5%). After long term or repeated exposure, kidneys, liver, and bones can accumulate uranium with the largest amounts being stored in bones (Li et al., 2005). Uranium is eliminated in feces and urine; about 50% of the absorbed dose is eliminated in the urine within the first 24 hours. After exposure to soluble uranium salts, the initial half-life of uranium is about 15 days (Bhattacharyya et al., 1992), which represents distribution and excretion, with much slower elimination from bone. After inhalation, the half-life of insoluble uranium in the lungs is several years (Durakovic et al., 2003). Human health effects from uranium at low environmental doses or at biomonitored levels from low environmental exposures are unknown. Health outcomes that may occur with uranium overexposure, based on both observed human effects and animal studies, include non-malignant respiratory disease (fibrosis, emphysema) and nephrotoxicity. Studies of persons with chronic exposure to elevated uranium salts in drinking water have shown changes in urinary biomarkers potentially associated with impaired kidney function (Kurttio et al., 2006). IARC and NTP have no ratings for uranium human carcinogenicity. Radiation risks from exposure to natural uranium are very low. Alpha radiation (such as that from uranium) is classified as a human carcinogen. However, human studies have not found elevated rates of cancer from uranium exposure, and high-dose animal studies have not found cancer

following inhalation, oral, or dermal exposure to uranium.

Workplace air standards and guidelines for external exposure to soluble and insoluble uranium compounds have been established by OSHA and ACGIH, respectively. Drinking water and other environmental standards have been established by U.S. EPA. Information about external exposure (i.e., environmental levels) and health effects is available from ATSDR at: http://www.atsdr.cdc.gov/toxpro2.html.

In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, uranium was detected in 4,101 systems serving close to 55 million people (EPA, 2009). Concentrations of uranium were greater than the MCL in 448 systems serving close to 8.4 million people (EPA, 2009).

Biomonitoring Information

Levels of urinary uranium reflect recent and ongoing or accumulated exposure. A previous nonrandom subsample from NHANES III (n = 499) (Ting et al., 1999) and other small populations have shown urinary concentrations that are similar to those in NHANES 1999-2000, 2001-2002, and 2003-2004 (Dang et al.,1992; Galletti, 2003; Karpas et al.,1996; Tolmachev et al., 2006). Older studies have demonstrated urinary uranium concentrations that are consistent with levels in the U.S. population, in that the levels were below their respective detection limits (Byrne et al., 1991; Hamilton et al., 1994; Komaromy-Hiller et al., 2000). In a study of 105 persons exposed to natural uranium in well water, urinary levels of uranium were as high as 9.55 μg/L (median 0.162 μg/L) (Orloff et al., 2004). Eighty-five percent of those levels were above the 95th percentile of the NHANES 1999-2000 population. The U.S. Nuclear Regulatory Commission (NRC) has set an action level of 15 μg/L urinary uranium to protect people who are occupationally exposed (NRC, 1978). Finding a measurable amount of uranium in urine does not mean that the level of uranium causes an adverse health effect. Biomonitoring studies on levels of uranium provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of uranium than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

Sources of Uranium

Uranium is a naturally-occurring element found in the earth's crust. It is naturally abundant in rocks, soil and water. Significant concentrations of uranium can occur in phosphate rock deposits, and in minerals such as pitchblende and uraninite. The total amount of Uranium on earth stays virtually the same because it has such a long half-life (4.47x109 years for U-238) (EPA, 2010).

	Uranium Regulation and Monitoring The EPA limits the amount of uranium that may be present in drinking water to 30 ug/L (EPA, 2009). A gross alpha particle activity measurement may be substituted for the required uranium measurement provided that the measured gross alpha particle activity does not exceed 15 pCi/l. **Monitoring frequency** Once a CWS has satisfied initial monitoring requirements (4 quarterly samples at every entry point to the distribution system within the first quarter after initiating the source); the required frequency for Uranium monitoring is once every three years if the average of the initial monitoring results for the contaminant is greater than one-half the MCL but at or below the MCL. States may allow CWS to reduce the frequency of monitoring from once every three years to once every six or nine years at each sampling point, if the average of the initial monitoring results for each contaminant is below the detection limit. If a system has a monitoring result that exceeds the MCL while on reduced monitoring, the system must collect and analyze quarterly samples at that sampling point until the system has results from four consecutive quarters that are below the MCL, unless the system enters into another schedule as part of a formal compliance agreement with the State (CFR, 2002).
Use of Measure	These measures assist by providing data that can be used for surveillance purposes. • Distribution measures provide information on the number of CWS and the number of people potentially exposed to Uranium at different concentrations. • Maximum concentrations provide information on the peak potential exposure to Uranium at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	The current measures are derived for CWS only. Private wells may be another source of population exposure to Uranium. Transient non-community water systems, which are regulated by EPA, may also be an important source of Uranium exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	The required monitoring frequency for Uranium is infrequent (every 3 to 6 years) and may be as intermittent as every nine years; therefore most states will have very little data on this contaminant. Ground water systems may have multiple wells with different Uranium concentrations that serve different parts of the population. Compliance samples

	are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the Uranium concentrations of people served by wells with higher Uranium concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different Uranium levels are averaged to estimate levels for the PWS.
Related Indicators	Public Water Use; combined Radium-226 and -228
References	1. Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological Profile for uranium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. 2. Bhattacharyya MH, Breitenstein BD, Metivier H, Muggenburg BA, Stradling GN, Volf V. Guidebook for the treatment of accidental internal radionuclide contamination of workers. In: Gerber GB, Thomas RG, eds. Radiation protection dosimetry. Vol. 41 (1). Kent (England): Nuclear Technology Publishing; 1992. pp. 1-49. 3. Byrne AR, Benedik L. Uranium content of blood, urine and hair of exposed and non-exposed persons determined by radiochemical neutron activation analysis, with emphasis on quality control. Sci Total Environ 1991;107:143-157. 4. Centers for Disease Control and Prevention (CDC). Third National Report on Human Exposure to Environmental Chemicals. Atlanta (GA). 2005. 4/20/09 5. Code of Federal Regulations (CFR), 2002. Title 40 Protection of the Environment Chapter I--Environmental Protection Agency Part 141--National Primary Drinking Water Regulations 141.26 Monitoring frequency and compliance requirements for radionuclides in community water systems. Available at: URL: http://www.access.gpo.gov/nara/cfr/waisidx_02/40cfr141_02.html 6. Dang HS, Pullat VR, Pillai KC. Determining the normal concentration of uranium in urine and application of the data to its biokinetics. Health Phys 1992;62:562-566. 7. Durakovic A, Horan P, Dietz LA, Zimmerman I. Estimate of the time zero lung burden of depleted uranium in Persian Gulf War veterans by the 24-hour urinary excretion and exponential decay analysis. Mil Med 2003;168(8):600-605. 8. Ejnik JW, Carmichael AJ, Hamilton MM, McDiarmid M, Squibb K, Boyd P, et al. Determination of the isotopic composition of uranium in urine by inductively coupled plasma mass spectrometry. Health Phys 2000;78:143-146. 9. Galletti M, D'Annibale L, Pinto V, Cremisini C. Uranium daily intake and urinary excretion: a preliminary study in Italy. Health Phys 2003;85:228-235. 10. Gwiazda RH, Squibb K, McDiarmid M, Smith D. Detection of depleted uranium in urine of veterans from the 1991 Gulf War. Health Phys 2004;86:12-18. 11. Hamilton EI, Sabbioni E, Van der Venne MT. Element reference values in tissues from inhabitants of the European community. VI. Review of elements in blood, plasma and urine and a critical evaluation of reference values for the United Kingdom population. Sci Total Environ 1994;158:165-190. 12. Karpas Z, Halicz L, Roiz J, Marko R, Katorza E, Lorber A, et al. Inductively coupled plasma mass spectrometry as a simple, rapid, and inexpensive method for determination of uranium in urine and fresh water: comparison with LIF. Health Phys 1996;71(6):879-885. 13. Komaromy-Hiller G, Ash KO, Costa R, Howerton K. Comparison of representative ranges based on U.S. patient population and literature reference intervals for urinary trace elements. Clin Chim Acta 2000;296(1-2):71-90. 14. Kurttio P, Auvinen A, Salonen L, Saha H, Pekkanen J, Makelainen I, et al. Renal effects of uranium in drinking water. Environ Health Perspect 2002;110(4):337-342.

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CONTENT DOMAIN: REPRODUCTIVE HEALTH OUTCOMES
INDICATOR: PREMATURITY

Type Of EPHT Indicator	**Health Outcome**
Measure	1. Percent of preterm (less than 37 weeks gestation) live singleton births 2. Percent of very preterm (less than 32 weeks gestation) live singleton births
Derivation of Measure	1. Number of live singleton births before 37 weeks of gestation to resident mothers, divided by total number of live singleton births to resident mothers 2. Number of live singleton births before 32 weeks of gestation to resident mothers, divided by total number of live singleton births to resident mothers
Unit	1. Preterm live singleton births 2. Very preterm live singleton births
Geographic Scope	State and national
Geographic Scale	State and County
Time Period	2000-current
Time Scale	Preterm: Annual Very Preterm: 5 yr annual average

Rationale	Preterm birth (at less than 37 completed weeks of gestation and among all births regardless of plurality) affects more than 500,000, or 12.5%, of live births in the United States and is a leading cause of infant mortality and morbidity (8, 9, 13). Of those births, the majority (about 84%) of premature babies are born *moderately preterm* (between 32 and 36 completed weeks of gestation). The remaining 16% of those are born *very preterm* (at less than 32 weeks of gestation), representing more than 80,000, or 2%, of live births in the United States. Of those infants born very preterm, about 63% are born between 28–31 weeks of gestation, and about 37% are born at less than 28 weeks of gestation. The preterm birth rate rose 18% between 1990 and 2004 (from 10.6% in 1990 to 12.5% in 2004) and more than 30% since 1981 (from 9.4%) (9). For 2003–2004, increases were seen among both moderately preterm and very preterm births. The percentage of infants born very preterm increased from 1.92% to 2.01% between 1990 and 2004 (9); it also increased between 2003 and 2004 from 1.97% to 2.01%, respectively. Preterm birth rates are higher among black mothers compared to Hispanic and white mothers. Between 2002 and 2003, the rates increased for the three largest race and ethnic groups: non-Hispanic white (11.0 to 11.3%), non-Hispanic black (17.7 to 17.8%), and Hispanic (11.6 to 11.9 %) (9). Since 1990, preterm birth rates have risen by one-third (about 33%) for non-Hispanic white births (from 8.5%) and by 8% for Hispanic births (11.0%). In contrast, preterm rates among non-Hispanic black infants have declined slightly over this period (from 11.9%). However, the preterm birth risk of non-Hispanic blacks continues to be substantially higher that the risk of other race and ethnic groups. Of particular concern is the very preterm rate, about twice as high among non-Hispanic black infants compared to non-Hispanic white and Hispanic births (3.99% compared to 1.6% and 1.73%, respectively). Preterm birth is a leading cause of infant mortality, morbidity, and long-term disability (8, 9, 13, 14). All infants born preterm are at risk for serious health problems; however, those born earliest are at greater risk of medical complications, long-term disabilities, and death. Studies have shown that infants born prematurely, especially those with VLBW, have an increased risk for neurological problems ranging from attention deficit hyperactivity disorder to cerebral palsy or mental retardation compared with infants born at term gestation (1, 6, 8, 14). Preterm birth is associated with nearly half of all congenital neurological defects such as cerebral palsy (9); it is also associated with congenital gastrointestinal defects such as gastroschisis. Preterm infants are at greater risk for serious health problems for several reasons: the earlier an infant is born, the less it will weigh, the less developed its organs will be, and the more medical complications it will likely face later in life. Very preterm infants have the greatest risk of death and lasting disabilities, including mental retardation, cerebral palsy, respiratory (premature lung) and gastrointestinal problems (including birth defects such as gastroschisis), and vision and hearing loss. Preterm births account for health care expenditure of more than $3 billion per year (14).

	Studies have shown that major risk factors associated with preterm birth include (2, 4, 7, 8, 10, 14): 1. Plural births 2. Previous preterm birth 3. Certain uterine or cervical abnormalities of the mother 4. Mother's age, race, poverty (for example, black women, women younger than 17 and older than 35 years, and poor women are at greater risk than other women) 5. Male fetal gender (associated with singleton preterm birth) 6. Certain lifestyles and environmental factors, including: o Late or no prenatal care, o Maternal smoking, alcohol consumption (especially in early pregnancy), illegal drug use, exposure to the medication diethylstilbestrol (DES), domestic violence, lack of social support, stress, long working hours with long periods of standing, being underweight before pregnancy, obesity, marital status, and spacing (less than 6–9 months between giving birth and the beginning of the next pregnancy), o Neighborhood-level characteristics, o Environmental contaminants (e.g., exposure to air pollution and drinking water contaminated with chemical DBP or lead). Certain medical conditions during pregnancy (e.g., infections, diabetes, hypertension, blood clotting disorders/thrombophilia, vaginal bleeding, certain birth defects of the fetus) may also increase the risk of preterm birth. The strength of the association of each of these risk factors with preterm birth varies, and remains a subject of significant debate in the literature (14). The rise in the occurrence of multiple/plural births, which are much more likely than singleton births to be preterm, influenced the overall preterm birth rate over the past two decades. However, preterm rates for singleton births have also increased, up to 11% since 1990 (9). This increase in singleton preterm births was only in infants born moderately preterm; the singleton very preterm birth rate declined slightly, from 1.69% in 1990 to 1.61% in 2004. Preterm births are associated with many modifiable risk factors, and prevention of preterm births may greatly contribute to the overall reduction in infant illness, disability, and death. Several studies are being conducted to improve our understanding of the precise causes of preterm births, especially those with VLBW, and to learn how to prevent them. These studies look at how genes, maternal stress, race, occupational and environmental factors, and infections may contribute to preterm birth (8). Better understanding of the specific causes of preterm births is needed before tailored interventions can be developed. Neighborhood-level characteristics have proven to be useful predictors of preterm birth risks (10). Neighborhoods are the geographic units where interventions can be targeted, and those interventions can be an effective way to reduce preterm birth rates and other adverse birth outcomes. Neighborhood-level characteristics contributing to prematurity include the social, economic, and environmental risk factors such as certain aspects of the built

	environment. Preterm births data are readily available in all state health departments and can be used to examine trends. These trends may reflect the contributions of environmental exposures and other modifiable risks to preterm births. These trends can also be used to evaluate the effectiveness of existing and new prevention programs. "*Live birth* means the complete expulsion or extraction from its mother of a product of human conception, irrespective of the duration of pregnancy, which, after such expulsion or extraction, breathes, or shows any other evidence of life, such as beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles, whether or not the umbilical cord has been cut or the placenta is attached. Heartbeats are to be distinguished from transient cardiac contractions; respirations are to be distinguished from fleeting respiratory efforts or gasps." All states require the reporting of live births regardless of length of gestation or birth weight (3).
Use Of The Measure	These measures can be utilized to enhance public health prevention actions and interventions, and inform policy makers and the public regarding risk factors management and mitigation.
Limitations Of The Measure	Uncertainties associated with gestational age estimates: The interval between the first day of the mother's last normal menstrual period (LMP) and the day of birth is one method used to determine the gestational age of the newborn. However, this measurement is subject to error for many reasons, including imperfect maternal recall or misidentification of the LMP due to postconception bleeding, delayed ovulation, or intervening early miscarriage (9). Thus, for the purpose of calculating national statistics of preterm births, these data are being edited for gestational ages that are clearly inconsistent with the infant's plurality and birth weight, but substantial inconsistencies in the data still persist (9). The National Center for Health Statistics (NCHS) and most state vital records offices report gestational age based on an algorithm that uses both the mother's reported last normal menses and the clinician's estimate of gestational age. The LMP indicator is used unless its value appears to be inconsistent with birthweight, falls outside likely parameters, or was not reported. If any of these circumstances exist, the clinical estimate is used. Nationwide in 2004, approximately 5.9% of gestational age values were based on the clinical estimate (9). Changes in reporting of the gestational age over time may affect trends in preterm birth rates, especially by race (9). These reporting problems may occur more frequently among some subpopulations and among births with shorter gestations.

	Difficulties of interpreting preterm and very preterm birth rates: The preterm birth rates might be an indicator of pregnancy outcome that does not necessarily predict the true health risk associated with early birth. Preterm rates based on live singleton births may be affected by maternal characteristics; a low preterm birth rate might indicate a low-risk population, and a high preterm birth rate might indicate maternal characteristics that predispose to preterm birth.
Data Sources	Birth certificate data from Vital Statistics state systems (both numerator and denominator); National Vital Statistics System (NVSS), CDC, NCHS http://www.cdc.gov/nchs/VitalStats.htm; CDC Wonder: Natality Data Request, CDC http://wonder.cdc.gov/natality.html CDC GIS Reproductive Health Atlas: http://cdc.gov/reproductivehealth/gisatlas/index.htm
Limitations Of Data Sources	Vital statistics data are readily available, of high quality, and useful for various purposes, including public health surveillance; however, they cannot be correctly interpreted unless various qualifying factors and classification methods are considered (see "Limitations of the Measure"). The factors to be considered will vary depending on the intended use of the data; however, most of the limiting factors result from imperfections in the original records, and they should not be ignored. Yet, their existence does not lessen the value of the data for calculating/estimating this measure. One important limitation of the national data is the timeliness of when the data are available. The national file cannot be compiled until all states have submitted their data. Often times there is delay of 2-3 years before national statistics are available. There are also some differences between national data and state data handling of unknowns, imputation rules, and close out dates. There may be differences or delays in processing resident births that occur out of state. These process issues, along with the need to close off national statistics at specified intervals following a reporting period, may lead to small discrepancies between national data compiled by NCHS and data maintained by state vital statistics registries.
Related Indicators	Low birthweight
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CONTENT DOMAIN: REPRODUCTIVE HEALTH OUTCOMES
INDICATOR: LOW BIRTHWEIGHT

Type Of EPHT Indicator	**Health Outcome**
Measure	1. Percent of low birthweight (less than 2500 grams) live term singleton births 2. Percent of very low birthweight (less than 1500 grams) live singleton births
Derivation of Measure	Number of singleton infants live born at term (at or above 37 completed weeks of gestation) with a birthweight of less than 2,500 grams, divided by the total number of singleton infants live born at term to resident mothers Number of live singleton births with a birthweight of less than 1,500 grams, divided by total number of live singleton births to resident mothers
Unit	LBW: live singleton term births VLBW: live singleton births
Geographic Scope	State and national
Geographic Scale	State and County
Time Period	2000-current
Time Scale	Low birthweight: Annual Very low birthweight: 5 yr annual average
Rationale	LBW, a weight of less than 2,500 grams, or 5 pounds, 8 ounces, at birth (regardless of gestational age and plurality), affects about 1 of every 13 babies born each year in the United States (7). Studies have shown that LBW is an important predictor of future morbidity and mortality. Note however, that the percent of LWB babies among all births (a percentage that is confounded by gestational age and plurality) is not recommended as a population-level measure of perinatal morbidity and mortality (1, 11). It is not recommended as a measure because preterm delivery, decreased fetal growth, and genetically determined small body size commonly occur in LBW infants (1). Compared to infants of normal weight, LBW infants may be at increased risk of perinatal morbidity, infections, and the longer-term consequences of impaired development such as delayed motor and social development or learning disabilities. Mortality risk is lowest for infants born weighing 3,500–4,500 grams (8). Nationally, the percentage of LBW infants (regardless of gestational age and plurality) has been increasing steadily; it reached 8.2% of all births in 2005, the highest level reported since 1968 (4). The 2005 rate was 17% higher than the 1970 (7%) rate, which was 22% higher than the 1984 low (6.7%). In addition, this rate is 64% higher than the Healthy People 2010 goal of 5% (5). The percentage of LBW births also increased among singleton births, from 5.9% in 1990 to 6.31% in 2004 (7% increase). Increases in the multiple birth rate, obstetric interventions (e.g., induction of labor and

	cesarean delivery), older maternal age at childbearing, and increased use of infertility therapies likely have affected the trends toward lower birthweights (8). Environmental exposures have also been implicated as possible risk factors for LBW, but the magnitude of the contribution to these increased rates remains relatively uncertain. The percentage of LBW increased among each of the largest racial and ethnic groups: non-Hispanic whites (from 7.0% in 2003 to 7.2% in 2004), non-Hispanic blacks (from 13.6% in 2003 to 13.7% in 2004), and Hispanics (from 6.7% in 2003 to 6.8% in 2004) (8). LBW in singleton births rose between 2003 and 2004 among non-Hispanic white and Hispanic infants; the increase for non-Hispanic black infants was not statistically significant (8). Since 1990, singleton LBW rates have risen 8% and 14% for Hispanic and non-Hispanic white infants, respectively; the rates have declined 2% among non-Hispanic black infants. The youngest and oldest mothers are the most likely to deliver LBW infants. In 2004, the lowest LBW levels were reported for women aged 25–34 years (7.3% for women aged 25–29 years and 7.5% for women 30–34 year old); the highest LBW levels were for teenagers younger than 15 years (13.6%) and women aged 45–54 years (21.2%) (8). However, much of the elevated LBW risk among older mothers can be attributed to their higher multiple birth rates; in fact, the LBW rate declined from 21% to 10% for the oldest mothers of singleton births. LBW rates also vary widely between states or reporting areas (8). In 2004, more than 10% of all infants born in Alabama, Louisiana, Mississippi, South Carolina, and the District of Columbia were LBW., This compares with less than 6.5% of newborns in Alaska, Maine, Oregon, Vermont, and Washington that were LBW. Different demographic characteristics of these populations, including maternal age, race, or ethnicity, may explain some of these differences. Infants weighing less than 1,500 grams, or 3 pounds, 4 ounces, at birth are considered VLBW (3); most of them are also premature (born before 37 weeks gestation). (Note that the percent of VLBW babies among all births is also confounded by plurality; therefore, the percent of VLBW births among singleton births is recommended as a population-level measure of prematurity.) Studies have shown that the infant's birthweight is a predictor of future morbidity and mortality (8), especially for VLBW infants. VLBW infants have about a 25% chance of dying in the first year of life; this risk is estimated to be about 100 times higher for VLBW infants than for normal-weight infants (≥2,500grams) (8). VLBW infants have an increased risk for developing neurological and intellectual problems (including attention deficit hyperactivity disorder, cerebral palsy, developmental delay and mental retardation), visual problems (including blindness), hearing loss, infections, and chronic lung diseases compared with infants of normal weight or infants born at term gestation (2, 5, 6, 7). Nationally, the percentage of VLBW infants (regardless of plurality) increased slightly from 1.45% in 2003 to 1.49% in 2005, and has increased from 1.27% in 1990 (5). The 2005 rate is 66% higher than the Healthy People 2010 goal of 0.9% (5). The VLBW has

	increased since 1990 among whites, blacks, Puerto Ricans, American Indians, and other population groups (5). For 2004–2005, increases in VLBW rates were statistically significant for non-Hispanic black infants but not for non-Hispanic white infants (8). The increase in the rate of multiple births, in which the infants tend to be much smaller than in singleton births, has likely affected the upward trend in the VLBW rate (8). However, the VLBW rate among singleton births also increased slightly from 1.12% in 2004 to 1.14% in 2005 (8). Increases in obstetric interventions (e.g., induction of labor and cesarean delivery), teenage pregnancy, and older maternal age at childbearing likely contributed to the increased VLBW rates. Teen mothers, especially those younger than aged 15 years, have a higher chance of giving birth to a VLBW infant. Environmental exposures, including exposure to air pollution, drinking water contaminated with chemical DBP, and exposure to pesticides, have also been implicated as possible risk factors for VLBW, but the exact magnitude of the contribution to the increased VLBW rates remains relatively uncertain Birthweight is a multifactorial and heterogeneous birth outcome. Birthweight of an infant is directly related to its gestational age. As noted above, multiple births are usually LBW, even those delivered at term. Therefore, the focus of the measure is restricted to singleton term births. As such, the measure distinguishes between preterm and multiple birth categories and decreased fetal growth that may be affected by other risk factors, including environmental factors. LBW rate is associated with many modifiable risk factors, and preventing LBW may contribute to the overall reduction in infant illness, disability, and death. Several studies are being conducted that may help understand the biological, social, and environmental factors that contribute to LBW births and learn how to prevent them. These studies look at how genes, hormonal changes, maternal stress, race, occupational and environmental factors, and infections may contribute to prematurity and LBW (7). Specific causes of LBW births must be better understood before tailored interventions can be developed. Neighborhood-level characteristics have proven to be useful predictors of LBW risks (9). Neighborhoods are the geographic units where interventions can be targeted, and those interventions can be an effective ways to reduce LBW rates, infant mortality, and other adverse birth outcomes. Neighborhood-level characteristics contributing to LBW include social, economic, and environmental risk factors, such as certain aspects of the built environment. The percentage of LBW among term singleton births is a useful and feasible measure of perinatal health. LBW, gestational age, and plurality data are readily available in all state health departments, and can be used to examine trends that occur over time and space. These trends may reflect the contributions of environmental exposures and other modifiable risk factors for LBW. Exposure to air pollution (both indoor and outdoor) and drinking water contaminated with

	chemical DBPs or lead may serve as examples of environmental risk factors. Maternal smoking, alcohol consumption, or inadequate weight gain are associated with an increased risk of intrauterine growth retardation and LBW. Socioeconomic factors, including low income and lack of education, are reported as risk factors for LBW (10). Women younger than 15 years or older than 35 years, unmarried mothers, and women who have had previous preterm birth are at increased risk of having LBW babies. Women who experience excessive stress, domestic violence, or other abuse also may be at increased risk of having a LBW baby (7). "*Live birth* means the complete expulsion or extraction from its mother of a product of human conception, irrespective of the duration of pregnancy, which, after such expulsion or extraction, breathes, or shows any other evidence of life, such as beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles, whether or not the umbilical cord has been cut or the placenta is attached. Heartbeats are to be distinguished from transient cardiac contractions; respirations are to be distinguished from fleeting respiratory efforts or gasps." All states require the reporting of live births, regardless of length of gestation or birth weight (3). *Birthweight* is the first weight of the newborn obtained after birth (3). *Low birthweight* is defined as less than 2,500 grams or 5 pounds, 8 ounces (3). Before 1979, low birthweight was defined as 2,500 grams or less. *Very low birthweight* is defined as less than 1,500 grams or 3 pounds, 4 ounces (3). Before 1979, very low birthweight was defined as 1,500 grams or less. *Term birth* is defined here as the birth at or above 37 completed weeks of gestation.
Use Of The Measure	This indicator can be used to influence public health prevention actions and interventions and policy makers and inform the public regarding risk factors management and mitigation. The LBW measure can be used to track the perinatal health in states, regions, counties, and smaller geographic areas or communities, as needed. Baseline data can be used to monitor changes or trends. This measure can also be used to evaluate the effectiveness of existing and new prevention programs.
Limitations Of The Measure	**Difficulties of interpreting LBW birth rates among term singleton births:** Using LBW rates alone as a pregnancy outcome measure might not inform the user about the true health risk associated with LBW. **Difficulties of interpreting VLBW birth rates:** Although the percentage of VLBW births has increased during the past 20 years, in large part this could be due to improvements in fetal health. Conditions that may have resulted in a fetal death decades ago might today result in fetal survival and a live VLBW birth (6).

	Recommendations: LBW rates should be interpreted with caution. The LBW rate should be only one of the reproductive outcome measures being tracked, and it should be accompanied by the infant mortality rate (neonatal and postneonatal), fetal death rate if reliable, and morbidity measures. If feasible, an infant's anthropometric parameters should also be monitored; this could include a reduced head circumference measure because smaller head size may predict lower IQ and cognitive abilities and may be associated with ADD/ADHD.
Data Sources	Birth certificate data from Vital Statistics state systems (both numerator and denominator) National Vital Statistics System (NVSS), CDC, NCHS; CDC Wonder: Natality Data Request, CDC http://wonder.cdc.gov/natality.html CDC GIS Reproductive Health Atlas: http://cdc.gov/reproductivehealth/gisatlas/index.htm
Limitations Of Data Sources	Although vital statistics data are readily available, of high quality, and otherwise useful for various purposes, including public health surveillance, they cannot be correctly interpreted unless various qualifying factors and classification methods are considered (see also "Limitations of the Measure"). The factors to be considered will vary, depending of the intended use of the data; however, most of the limiting factors result from imperfections in the original records, and they should not be ignored. Yet, their existence does not lessen the value of the data for the purpose of calculating this measure. At the minimum, the following data quality attributes should be evaluated: completeness of registration, reporting and quality control procedures, and records geocoding procedures and quality. One important limitation of the national data is the timeliness of when the data are available. The national file cannot be compiled until all states have submitted their data. Often times there is delay of 2-3 years before national statistics are available. There are also some differences between national data and state data handling of unknowns, imputation rules, and close out dates. There may be differences or delays in processing resident births that occur out of state. These process issues, along with the need to close off national statistics at specified intervals following a reporting period, may lead to small discrepancies between national data compiled by NCHS and data maintained by state vital statistics registries.
Related Indicators	Prematurity
References	1. Adams M., Andersen A-M. N., Andersen P. K., Haig D., Henriksen T. B., Hertz-Picciotto I., Lie R. T., Olsen J., Skjerven R., and Wilcox A. Sostrup Statement on Low Birthweight. Int J Epidemiol 2003, 32: 884-885 2. Ananth C. W., Joseph K. S., Oyelese Y., Demissie K., Vintzileos A. M. Trends in Preterm Birth and Perinatal Mortality Among Singletons: United States, 1989 through 2000. Obstet Gynecol,2005, Vo. 105, No. 5, 1084-1091 3. Centers for Disease Control and Prevention, National Center for Health Statistics (NCHS), NCHS Definitions. Available from:

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CONTENT DOMAIN: REPRODUCTIVE HEALTH OUTCOMES
INDICATOR: MORTALITY (USING PERIOD LINKED BIRTH/INFANT DEATH APPROACH)

Type of EPHT Indicator	Health Outcome
Measures	1. Average Infant (less than 1 year of age) Mortality Rate per 1000 live births 2. Average Neonatal (less than 28 days of age) Mortality Rate per 1000 live births 3. Average Perinatal (equal to or greater than 28 weeks gestation to less than 7 days of age) Mortality Rate per 1000 live births (plus fetal deaths equal to or greater than 28 weeks gestation) 4. Average Postneonatal (equal to or greater than 28 days to less than 1 year of age) Mortality Rate per 1000 live births
Derivation of Measures	1. Infants: Number of deaths occurring in infant residents under 1 year of age (under 366 days during a leap year) in a given year divided by the number of live births in the same year. 2. Neonates: Number of deaths occurring in infant residents less than 28 days of age in a given year divided by the number of live births in the same year 3. Perinates: Number of fetal deaths in infant residents greater than or equal to 28 weeks gestation plus infant deaths less than 7 days old in a given year divided by the number of live births plus fetal deaths at greater than or equal to 28 weeks gestation in the same year 4. Postneonates: Number of deaths occurring in infant residents at 28 days to less than1 year of age (under 366 days during a leap year) in a given year divided by the number of live births in the same year Both birth and death counts are geographically classified based on maternal residence at the time of birth.
Units	1. Deaths per 1,000 live births 2. Deaths per 1,000 live births 3. Deaths per 1,000 live births plus fetal deaths at 28 or greater weeks gestation 4. Deaths per 1,000 live births
Geographic Scope	State and national
Geographic Scale	State and County
Time Period	2000-current
Time Scale	Five year

Rationale	Fetuses and young children may be particularly susceptible to harmful effects of environmental contaminants. Many environmental contaminants have been proposed to be particularly toxic in utero; many cross the placenta and make their way into the circulatory system of the developing fetus. However, specific health effects are often not well understood for years Therefore, gross indicators of childhood health—such as mortality—should be tracked as part of an EPHT system. Furthermore, data on births and deaths in a region may be far more complete than data on other health-related events. Overall, congenital malformations, deformations, and chromosomal abnormalities are the leading cause of infant deaths (20.1% of deaths) (1). Disorders related to short gestation and LBW are second, making up 16.6% of deaths. However, importantly, cause of death varies over the first year of life, and combining all causes obscures the fact that sudden infant death syndrome is the leading cause of death in the postneonatal period. Disorders related to short gestation and LBW are the leading cause of neonatal death (24.3% of deaths) (1). This is in contrast to the leading cause of postneonatal death, which is sudden infant death syndrome (21.8%). Congenital malformations, deformations, and chromosomal abnormalities are the second-leading cause of neonatal deaths (21.4%) and postneonatal deaths (17.5%) (1). Restricting infant mortality to deaths during the perinatal, neonatal, or postneonatal period may limit the etiologic heterogeneity inherent in a gross measure such as overall infant mortality. Also, it may be more likely that infants who died within 7 or 28 days, respectively, were living in reasonable proximity to where they were born, making ecological associations with environmental exposures potentially more meaningful. Specifically, exclusion of infants who died within 28 days might reduce etiologic heterogeneity due to differences in early prenatal care and other non-environmental factors likely to influence neonatal survival. When a fetus or an infant dies around the time of labor and delivery, it is not always clear whether to classify this event as a live birth and infant death, or a fetal death. Diagnostic ability for detecting signs of life, such as breathing or beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles after expulsion or extraction from the mother may vary across obstetric clinics. Unexplained fetal death and death related to growth restriction are the leading causes of fetal loss (2). Fetal death is an important contribution

	to reproductive loss, with the rate being many times higher than the rate of sudden infant death syndrome among infants (1). Although the rate of late fetal loss (greater than or equal to28 weeks gestation) has been decreasing in past decades, the rate of intermediate fetal loss (20–27 weeks gestation) has remained relatively constant (3). Markers of increased risk for fetal loss include pre-pregnancy obesity, lower socioeconomic status, non-Hispanic black race, and advanced maternal age.
Use of the Measure	Identifying populations with higher infant, neonatal, perinatal, and postneonatal mortality rates may indicate where potential environmental problems are. It will assist in targeting outreach intervention activities and improve our understanding of geographic variation, time trends, and demographic patterns of infant death.
Limitations of the Measure	An important limitation of this health outcome measure is the heterogeneity in its etiology. Environmental exposure-related causes of infant death are only one piece of a puzzle that includes many other factors, such as access to and quality of health care, competency in childcare, and understanding of injury prevention. The maternal residence during pregnancy and the infant's residence during the first year of life are critical data for linking deaths to environmental hazards/exposures; these residences may differ from maternal residence at birth or infant residence at death. The mother may have lived far from the place at which she gave birth during part or all of the pregnancy. The infant who died may have been born and lived for a major portion of its life far from the place of death; it may be less likely that neonates and perinates who died were born and lived far from the place of death. NCHS currently uses a period linkage approach that links death certificates to birth certificates. This approach would allow stratification of deaths according to place of birth. However, it does not address the possibility that migration across states or other geographies occurred *during* pregnancy or infancy.
Data Sources	Local, state, or national vital statistics systems (birth, death, and fetal death records)
Limitations of Data Sources	It may be reasonable to assume universal reporting of live births and infant deaths in the United States; however, some births/deaths may be excluded because of the difficulty in distinguishing a death shortly after birth as a live birth; a death soon after birth might be reported as a fetal death rather than as a live birth and infant death. In addition, some fetal deaths may be missed in some regions, although those occurring at greater than or equal to28 weeks are less likely to be missing. Data on fetal death certificates may not provide all the information that

	can be collected from birth certificates linked to infant deaths within 7 days; however, many variables used for environmental health tracking (maternal race/ethnicity and age, place of residence) have relatively complete reporting on the fetal death certificate. Births and deaths will be tabulated according to maternal race/ethnicity, using linked data from birth certificates.
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CONTENT DOMAIN: REPRODUCTIVE HEALTH OUTCOMES
INDICATOR: FERTILITY

Type of EPHT Indicator	**Health outcome**
Measure	Total Fertility Rate per 1000 women of reproductive age
Derivation of Measure(s)	TFR = sum of age-specific fertility rates * 5
Unit	Rate per 1,000 women of reproductive age
Geographic Scope	State and national
Geographic Scale	State and County
Time Period	2000-current
Time Scale	Year
Rationale	The cause of approximately 10% of fertility problems is unknown, and environmental contaminants, including endocrine disruptors, have been considered major contributors. The case of diethylstilbestrol revealed that environmental contamination can have multi-generational effects on reproduction that should be studied and tracked long-term. Several indicators have been used to track fertility on a global, national, state, and local level. Indicators most commonly used are the general fertility rate (GFR), which is defined as the number of live births divided by the total number of women of reproductive age (aged 15–44 years), and the total fertility rate (TFR). The TFR differs from the GFR in that it adjusts for age-specific differences in fertility. It also shows the potential impact of current fertility patterns on reproduction, allowing for more valid comparisons of rates across time and space. *Fecundity*: The physical ability of a woman or couple to conceive and carry a child to term birth. *Fertility:* The ability to conceive a child.
Use of the Measure	The TFR indicates the average number of births to a hypothetical cohort of 1,000 women if they experienced the age-specific birth rates observed in a given year. Understanding the geographic distribution and trends in fertility will provide basic descriptive clues to changes that may be influenced by environmental risk factors. As more is learned regarding the link between adverse exposures and fertility, these rates will provide important background information about how fertility varies geographically in relation to changes in potentially related environmental risk factors and how it has varied over time within the United States. Similar to the GFR, the TFR may not be

	specific enough to permit tracking of specific changes related to environmental risk factors. However, if the estimate of 10% is correct, this measure can be used with other measures, including ambient concentrations of pollutants, to examine potential associations with population-level changes in fertility and generate some well- informed hypotheses or areas for future investigations.
Limitations of the Measure	The fertility measure is influenced by social/demographic choices for reproduction, maternal age, parity, and social class measures, as well as the use of contraception and infertility treatments leading to multiple births. These factors all may determine variations in overall fertility across populations and geographic locations; therefore social and demographic factors would need to be controlled for to examine any environmental effects on total fertility.
Data Sources	**Numerator:** U.S. National Center for Health Statistics—Vital Statistics Reports and/or state-specific vital statistics (for more recent years of data) **Denominator:** U.S. Census Bureau
Limitations of Data Sources	National-level data sources may differ slightly from state-level vital statistics data sources

CONTENT DOMAIN: REPRODUCTIVE HEALTH OUTCOMES
INDICATOR: SEX RATIO AT BIRTH AMONG SINGLETON BIRTHS

Type of EPHT Indicator	**Health outcome**
Measure	Male to Female sex ratio at birth (term singletons only)
Derivation of Measure(s)	Sex ratio=total males/total females at birth among term singleton births only
Unit	Ratio
Geographic Scope	State and national
Geographic Scale	State and county
Time Period	2000-current
Time Scale	Year
Rationale	Population growth is, in part, related to the number of live male children (1). Numerous studies have reported changes in the ratio of males to females at birth; many of the studies have found a reduction in male relative to female births in different countries throughout the world (2-5). Although the mechanism that determines the sex of the infant is not completely understood, some (6-12), but not all (3-4), have suggested that environmental hazards can affect the number of males. Biological parent(s) and/or the fetus can come in contact with and become exposed to different hazards referred to as endocrine disruptors (7-8, 10, 12). Fewer males are conceived when exposure to endocrine disruptors results in a decrease in testosterone. Because states have accurate Vital Statistics (VS) records on the sex of live births, changes over time in the sex ratio of infants can be measured as the ratio of males to females. This ratio of total males/total females born in a pre-defined polygon (e.g., state, county, ZIP code, census tract, block group) at a certain time (one birth year or multiple years) is referred to as the Sex Ratio (SR).
Use of the Measure	The SR can be used to monitor the proportion of males to females in states, counties, or smaller-resolution polygons, when data are available and such analyses are justified. Baseline data can be used to determine if the proportion of males is changing over time. When the number of male births is the same as the number of female births, the SR is equal to 1.000. Many studies have observed baseline SR values that are usually higher than 1.000, and closer to 1.050(1, 3, 13). In 2002, the U.S. SR was 1.048 (1). If the SR is decreasing over time, the implication is that fewer males than females are born for that period of time. If consistent decreases in the SR occur, this outcome could be used to determine if such changes are the result of environmental hazards that can disrupt the endocrine system or some other

	physiological system related directly or indirectly to the expression of the neonates' sex at birth.
Limitations of the Measure	Unfortunately, other factors besides endocrine disruptors can affect the expression of sex (6, 13-15). Decreases in male births inversely related to parental smoking, gestation length, parental age, and birth order. Reproductive practices and social morays regarding sex preferences—males over females, for example, can affect the observed SR (3, 4, 7). Case-control studies have to be carried out to determine if decreases in the SR over time are due to contact with and exposure to endocrine disruptors; but effect modifiers have to be controlled in order to understand this relationship, factors that modify it need to be better accounted for. (8).
Data Sources	State's VS data, CDC Wonder, CDC VS data, and U.S. Census 2000 data in Summary File (SF) 1.
Limitations of Data Sources	There may be discrepancies between national and state data as noted in the templates for measures of prematurity and growth retardation above.
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