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

Version 2.0
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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 to this document 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

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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:

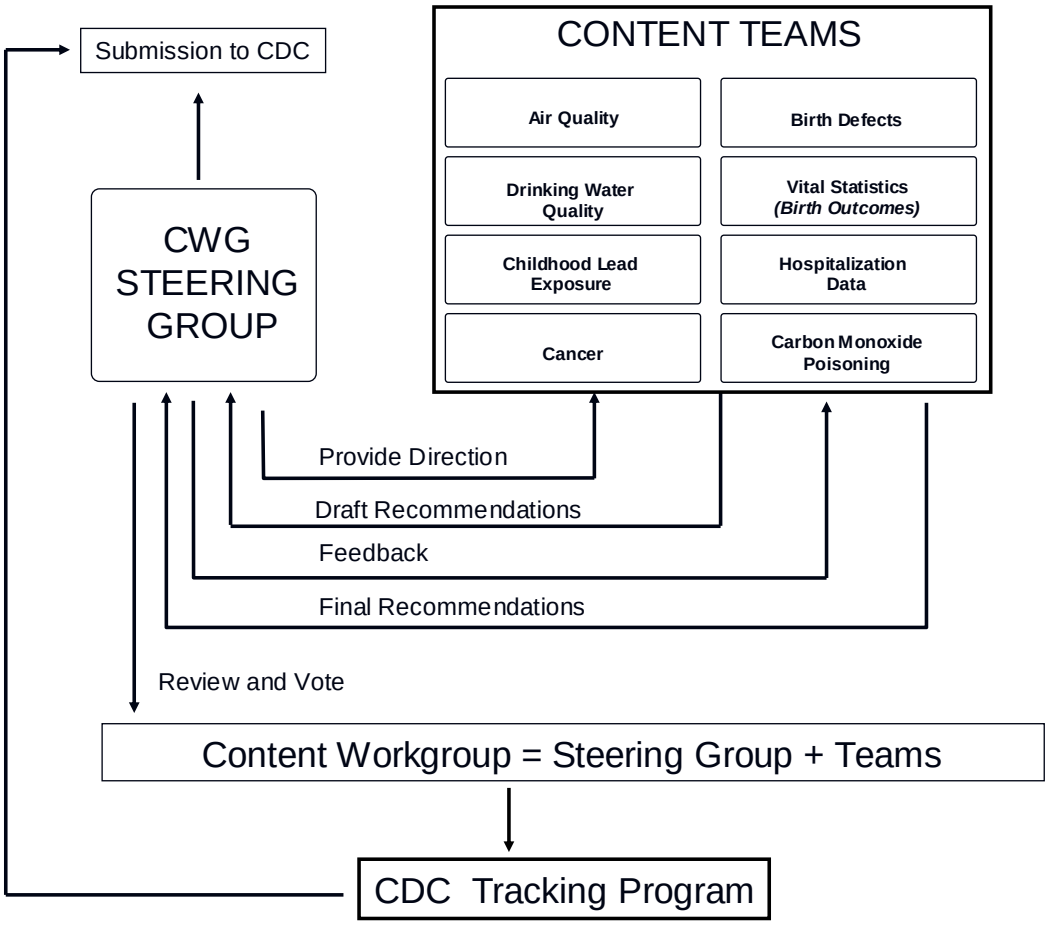
1. Identify and recommend core measures for the Tracking Network;
2. Examine the availability and applicability of existing data and identify approaches for deriving or collecting needed data;
3. Identify and adapt standards and guidelines to facilitate nationally consistent data collection and ensure 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: Acute Myocardial Infarction (AMI)

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
AMI	Number of hospitalizations for AMI	Annual	State and county	Grantee Provided	Required
	Average daily number of hospitalizations for AMI, by month	Annual	State and county	Grantee Provided	Optional
	Maximum daily number of hospitalizations for AMI by month	Annual	State and county		
	Minimum daily number of hospitalizations for AMI by month	Annual	State and county		
	Rate of hospitalization for AMI 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 AMI 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		

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	Annual	State		

	by Age group (<50, ≥50, total)	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 population	Annual	State		
		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	Annual	State		

	unknown intent)				
	Age-adjusted 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		
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	Annual	State		

	resulting health effect and treatment in a healthcare facility				
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 Coverage and Age of Housing	Number of children born in the same year and tested for lead before age 3	3 year testing period by annual birth cohort	State and county	Nationally Derived	Required
	Percent of children born in the same year and tested before age 3	3 year testing period by annual birth cohort	State and county		
	Number of children younger than 5 years living in poverty (as measured in 2000 census)	Annual	State and county		
	Percent of children younger than 5 years living in poverty (as measured in 2000 census)	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		

* The Childhood Lead Poisoning measures can be displayed as the one indicator described above or as two indicators splitting the age of housing measures from the testing and poverty measures. The two indicators would be (1) Testing Coverage and (2) Age of Housing. At the time of this publication, revised and new Childhood Lead Poisoning indicators are under review by the CWG.

Content Domain: Drinking Water

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Arsenic Level and Potential Population Exposures	Distribution of number of community water systems by mean arsenic concentrations (micrograms per liter) by year	Annual	State	Grantee Provided	Required
	Distribution of number of people served by community water systems by mean arsenic concentrations (micrograms per liter)by year	Annual	State		
	Distribution of number of community water systems by maximum arsenic concentrations (micrograms per liter)by year	Annual	State		
	Distribution of number of people served by community water systems by maximum arsenic concentrations (micrograms per liter)by year	Annual	State		
	Distribution of number of community water systems by mean arsenic concentrations (micrograms per liter)by quarter	Quarterly	State		
	Distribution of number of people served by community water	Quarterly	State		

	systems by mean arsenic concentrations (micrograms per liter)by quarter				
Nitrate Level and Potential Population Exposures	Distribution of number of community water systems by mean nitrate concentrations (milligrams per liter)by year	Annual	State	Grantee Provided	Required
	Distribution of number of people served by community water systems by mean nitrate concentrations (milligrams per liter) by year	Annual	State		
	Distribution of number of community water systems by maximum nitrate concentrations (milligrams per liter)by year	Annual	State		
	Distribution of number of people served by community water systems by maximum nitrate concentrations (milligrams per liter)by year	Annual	State		
	Distribution of number of community water systems by mean nitrate concentrations (milligrams per liter)by quarter	Quarterly	State		
	Distribution of number of people served by community water systems by mean nitrate concentrations (milligrams per	Quarterly	State		

	liter) by quarter				
Disinfection Byproducts (DBP) Level and Potential Population Exposure (TTHM)	Distribution of number of community water systems by mean trihalomethane (THM) concentrations (micrograms per liter)by year	Annual	State	Grantee Provided	Required
	Distribution of number of people served by community water systems by mean trihalomethane (THM) concentrations (micrograms per liter) by year	Annual	State		
	Distribution of number of community water systems by maximum trihalomethane (THM) concentrations (micrograms per liter) by year	Annual	State		
	Distribution of number of people served by community water systems by maximum trihalomethane (THM) concentrations (micrograms per liter) by year	Annual	State		
	Distribution of number of community water systems by mean trihalomethane concentrations (micrograms per liter) by quarter	Quarterly	State		

	Distribution of number of people served by community water systems by mean trihalomethane (THM) concentrations (micrograms per liter) by quarter	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) by year	Annual	State	Grantee Provided	Required
	Distribution of number of people served by community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter) by year	Annual	State		
	Distribution of number of community water systems by maximum haloacetic acids (HAA5) concentrations (micrograms per liter) by year	Annual	State		
	Distribution of number of people served by community water systems by maximum haloacetic acids (HAA5) concentrations (micrograms per liter) by year	Annual	State		
	Distribution of number of people served by community water systems by mean haloacetic acids concentrations (micrograms per	Quarterly	State		

	liter) by quarter				
	Distribution of number of people served by community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter) by quarter	Quarterly	State		
Public Water Use	Number of people receiving water from community water systems	Annual	State	Grantee Provided	Required

*At the time of publication of this document, these water measures and additional water measures were under review by the CWG.

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	Required

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: ACUTE MYOCARDIAL INFARCTION
INDICATOR: HOSPITALIZATIONS FOR ACUTE MYOCARDIAL INFARCTION

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 **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	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.

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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.

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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 $\mu g/m^3$ 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 $\mu g/m^3$. 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
References	Dockery DW and Pope CA. Acute respiratory effects of particulate air pollution. Annu Rev Public Health 1994;15:107–132. Cannon J. The Health Costs of Air Pollution: A Survey of Studies Published 1984– 1989. New York: American Lung Association; 1990. 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: 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 affect 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 IOM 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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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" 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. Cancer sites/types to be included in the EPHT Network are: • Breast cancer (females) o <50 years o ≥50 years • Cancer of the lung and bronchus • Bladder cancer (including in situ) • Cancers of the brain and other nervous system (ONS) • Cancers of the brain and central nervous system (children <15 and children <20) • Thyroid cancer • Liver and bile duct cancers

	• Kidney and renal pelvis cancers
	• Mesothelioma
	• Melanoma
	• Cancers of the pancreas
	• Oral/Pharynx cancers
	• Esophageal cancers
	• Larynx cancers
	• Non-Hodgkin's lymphoma
	• Leukemias
	  o Chronic lymphocytic leukemia
	  o Acute myeloid leukemia
	• Leukemias (children <15 and children <20)
	• Acute lymphocytic leukemia
	  o Acute myeloid leukemia
	
	Rates will be age-adjusted to year 2000 U.S. standard population.

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	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 used is Midyear resident population Rates age-adjusted by the direct method to the Year 2000 U.S. 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	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.

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	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 used is Midyear resident population Rates age-adjusted by the direct method to the Year 2000 U.S. 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	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.

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	Resident deaths from CO poisoning for three unique groups: 1. Unintentional, non-fire related 2. Unintentional, fire-related 3. Unknown intent Denominator used is midyear resident population 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	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).

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.

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").
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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) 5. Number of children younger than 5 years living in poverty (as measured in 2000 census) 6. Percent of children younger than 5 years living in poverty (as measured in 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 and percentage of children under 5 living in poverty. Merge testing and housing data files by geography.
Unit	Tested child Proportion of houses by age-based hazard assessment
Geographic Scope	State and national (tracking network states)
Geographic Scale	county and state
Time Period	Begin with year 2000 birth cohort and repeat for each succeeding birth cohort once they reach age 3 years.
Time Scale	3 year testing period by 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: DRINKING WATER
INDICATOR: ARSENIC LEVEL OF CONTAMINANT IN FINISHED WATER
POTENTIAL POPULATION EXPOSURE TO CONTAMINANTS IN FINISHED WATER

Type of EPHT Indicator	Hazard, Exposure
Measures	1. Distribution of number of community water systems by mean arsenic concentrations (micrograms per liter) by year 2. Distribution of number of people served by community water systems by mean arsenic concentrations (micrograms per liter) by year 3. Distribution of number of community water systems by maximum arsenic concentrations (micrograms per liter) by year 4. Distribution of number of people served by community water systems by maximum arsenic concentrations (micrograms per liter) by year 5. Distribution of number of community water systems by mean arsenic concentrations (micrograms per liter) by quarter 6. Distribution of number of people served by community water systems by mean arsenic concentrations (micrograms per liter) by quarter
Data Sources and Derivation of Measures	Arsenic measures will be developed from water system attribute and water quality data stored in state 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 water source type) to generate the measures and a simple public use dataset from which statistics similar to the measures can be generated.
Purpose and 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.
Limitations	***Limitations:*** Measures do not account for the variability in sampling, numbers of sampling repeats, etc Furthermore, 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/underestimate the number of affected people. Levels of arsenic are likely to be higher in private drinking water wells (Karagas et al. 2002), and these indicators would not capture that data.

	Surface water CWSs started meeting the 10 ppb standard in 2006 (1/23/06), and ground water systems must take their compliance samples by the end of 2007. Therefore, running annual averages demonstrating violations in groundwater systems (78% of CWSs) will not all be in the database until late 2008. 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.
Measurement Unit	Arsenic concentration in µg/L
Geographic Scope	National
Geographic Scale	Community water systems (CWSs)
Time Period	1999 to most recent Analytic methods changed over time. (In 1994 (59 FR 62456; US EPA, 1994, as cited in EPA 2000a), the Agency approved the use of the updated Methods for the Determination of Metals in Environmental Samples, that lowered the detection levels for some methods .
Time Scale	Annual and quarterly mean and annual maximum for each community water system as this should be the best time period for normalizing values across time--- the mean for each CWS will represent the mean of entry point means within a particular time period.
General Limitations of the Measures and Data Source	Consistent measurements of potential arsenic exposure to populations are only available for populations on community water supplies. Specific measurements of arsenic data for unregulated small water systems and private domestic wells are generally not available and must be estimated from aggregated data sets. Estimates of the population served by each CWS as reported yearly by the water purveyor may not be accurate as they are usually based on the number of residential connections and an assumed average number of residents per connection. In some cases, a connection to a multi-family unit may be counted as a single connection. Estimates of the potential for exposure to populations are only applicable to populations on community water supplies. Arsenic data are generally not available

	for unregulated small water systems and private domestic wells. Demographic information about specific populations, needed to generate distributions for demographic sub-populations, is not collected or reported by purveyors. Using demographic data from the US Census or other population-based surveys requires a method for linking a specific water system to a population unit of analysis (e.g. block group). This requires knowledge of the extent of the distribution system for each water system, or some geographic aggregation of water systems. The spatial extent of distribution systems is generally not available. The estimate of the number of community water system (CWS) expected to exceed different arsenic levels are based on the distribution of average arsenic concentrations in water systems. Measurements are taken at every entry-point-to-distribution-system (EPTDS). The means of these values may not represent actual concentrations at residential service connections due to system hydraulic characteristics and differences in flow through each EPTDS. Samples are taken once a year (surface sources), once every three years (groundwater sources), or once every nine years (after 2006 for sources with a waiver). This may not adequately capture temporal variation, particularly for low levels. However health risks are linked to long-term exposure. The change in sampling frequency may affect the ability to make meaningful comparisons over time using this measure. Sampling points that exceed the 10 μg/L standard are sampled each quarter while the running annual average exceeds 10 μg/L. The change in sampling frequency may affect our ability to make meaningful comparisons over time using this measure. The actual levels of exposure depend on the quantity of tap water consumed (and any filtration systems installed by each resident). Daily intake of inorganic arsenic from food is about 10 micrograms (NRC, 1999; MacIntosh et al., 1997, as cited in EPA, 2000), and the lifetime tap water consumption value in the U.S. is 1 liter per day (EPA, 2001a), which at 10 micrograms per liter, is equivalent to what people eat in food. Most public systems have much lower than 10 ppb arsenic in their drinking water. Also the per-capita water ingestion varies with demographic sub-population (EPA 2004). This may result in variations in actual exposures within a water system population. While this limits the indicator as a measure of exposure, it is still a valuable measure of potential exposure, and helpful in identifying regions or water systems which may need to be targeted for improvement. Although acute or short-term exposures to high doses of inorganic arsenic can cause adverse effects, such exposures do not occur from public water supplies in the U.S. at the former MCL of 50 □g/L. "From human acute poisoning incidents, the LD_{50} of arsenic has been estimated to range from 1 to 4 mg/kg (Vallee et all, 1960; Winship, 1984). This dose would correspond to a lethal dose range of 70 to 280 mg for 50% of adults weighing 70 kg (EPA 2000a)." The background rate

	for bladder cancer in the U.S. is roughly 60,000 incident cases and 12,500 mortalities per year (NCI, 2003), approximately 4,000 systems exceeded 10 ppb (EPA 2001c) out of the 72,213 CWSs and NTNCWSs (EPA 2005). EPA's drinking water regulation addresses the long-term, chronic effects of exposure to low concentrations of inorganic arsenic in drinking water.
Related Indicators	FOR DISTRIBUTIONS BY DEMOGRAPHIC FACTORS: geographic boundaries of PWS, spatially linked to US Census data 1990 including demographic characteristics for each block/block group. FOR ESTIMATES OF EXPOSURE: Tap water consumption patterns for demographic sub-populations.
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CONTENT DOMAIN: DRINKING WATER
INDICATOR: DISINFECTION BYPRODUCTS
LEVEL OF CONTAMINANT IN FINISHED WATER
POTENTIAL POPULATION EXPOSURE TO CONTAMINANTS IN FINISHED WATER

Type of EPHT Indicator	Hazard, Exposure
Measures	1. Distribution of number of community water systems by mean trihalomethane (THM) concentrations (micrograms per liter) by year 2. Distribution of number of people served by community water systems by mean trihalomethane (THM) concentrations (micrograms per liter) by year 3. Distribution of number of community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter) by year 4. Distribution of number of people served by community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter) by year 5. Distribution of number of community water systems by maximum trihalomethane (THM) concentrations (micrograms per liter) by year 6. Distribution of number of people served by community water systems by maximum trihalomethane (THM) concentrations (micrograms per liter) by year 7. Distribution of number of community water systems by maximum haloacetic acids (HAA5) concentrations (micrograms per liter) by year 8. Distribution of number of people served by community water systems by maximum haloacetic acids (HAA5) concentrations (micrograms per liter) by year 9. Distribution of number of community water systems by mean trihalomethane concentrations (micrograms per liter) by quarter 10. Distribution of number of people served by community water systems by mean trihalomethane (THM) concentrations (micrograms per liter) by quarter 11. Distribution of number of people served by community water systems by mean haloacetic acids concentrations (micrograms per liter) by quarter 12. Distribution of number of people served by community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter) by quarter

Data Sources and Derivation of Measures	DBP measures will be developed from data stored in state SDWA databases such as the Safe Drinking Water Information System (SDWIS/State). Distribution system sample result concentrations of trihalomethanes and haloacetic acids will be extracted from these databases. Trihalomethanes comprise chloroform, bromodichloromethane, dibromochloromethane, bromoform and their sum, denoted total trihalomethanes (TTHM) or THM4. 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. Inventory information about each CWS such as service population and water source type will also be extracted from the state SDWA database. All samples taken on the same day will be averaged for each individual THM, THM4, each individual HAA, and HAA5 to determine the values for the CWS. Quarterly and annual values by CWS will be derived from these daily averages using algorithms appropriate for the sampling frequency.
Purpose and Rationale	*Disinfection Byproducts 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. 2005, 2006). Animal, microbial, in vitro and modeling studies have also pointed to toxicity or carcinogenicity of a wide vari7ety 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 CWSs 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 1998). 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.
Limitations	*Description and Development:* The number of people receiving water from a CWS in which the quarterly average is greater than a set of fixed reference values for any DBP during a calendar year enables annual comparisons and progress tracking relative to a baseline year. Reference values will be obtained from the distribution of population exposure with respect to DBPs in a small number of consecutive baseline years. The reference levels for each DBP or sum will be the 50^{th}, 75^{th}, 90^{th} and 95^{th} percentiles of population exposure in 2005. The reference levels, while arbitrary, provide fixed points of comparison to which changes in DBP levels from year to year can be compared. This measure is obtained from state SDWA databases for each DBP by obtaining the quarterly average of DBP sample results. The system population is determined for any CWS that has an average for any DBP that exceeds each reference level for a DBP at any time during a calendar year. The populations of these CWSs are added over the desired spatial unit, such as a state or the nation, to provide the measure. *Advantages:* This measure provides a simple to calculate and readily comprehended indicator of the number of people potentially exposed to water with DBP concentrations that exceed reference levels. It is easily replicated, allowing interannual comparisons and progress tracking, and can be calculated for different spatial aggregations. *Limitations:* The reference levels and the criteria by which they are obtained are arbitrary. Useable health based benchmarks are not available for individual DBPs. The measure is based on a binary rather than quantitative criterion. As it is based only on whether a reference level was exceeded, it does not provide information on the number of DBPs for which a reference level was exceeded, the duration of the exceedance, or the magnitude of the exceedance. The DBP value used to determine exceedance of the reference level is an average of a spatially varying measurement. The average may not exceed the reference level in a water supply system in which a DBP level in part of the distribution system exceeds the reference level, leading to an underestimate of the number of affected people. Conversely, levels may be below the reference level in part of a distribution system in which the average exceeds the reference level, thereby overestimating the number of affected people. This limitation can be overcome when sufficient spatial data are available to enable the assignment of each DBP sample to the part of the distribution system for which it is most representative.
Measurement Unit	Concentrations are routinely reported in either mg/L or µg/L. EPHTN reporting should be standardized to µg/L.
Geographic Scope	National.
Geographic Scale	Community water system.
Time Period	1999 – present: THMs for water systems serving 10,000 or more people. 2002 – present: THMs and HAAs for surface water systems and ground water systems

	under direct influence of surface water serving ≥ 10,000 people. 2004 – present: THMs and HAAs for all other community water systems. Note: requirements apply only to water supply systems using disinfectant.
Time Scale	Quarterly and annual means and annual maximum values. Most CWSs are required to sample quarterly but those with lower DBP levels may sample annually or triennially. Some CWSs choose to sample monthly. CWSs with disinfection waivers do not sample.
General Limitations of the Measures and Data Source	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. HAA9, defined as HAA5 plus bromochloroacetic acid, bromodichloroacetic acid, chlorodibromoacetic acid, and tribromoacetic acid, can exceed HAA5 by a considerable amount (Roberts et al. 2002). 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	Bromide concentrations indicate potential for formation of more brominated DBPs. Evidence suggests that brominated THMs are more toxic than chlorinated forms. Organic matter concentration has a substantial effect on the potential formation of DBPs, both by providing a reaction substrate and being associated with the need for greater disinfection amounts.
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CONTENT DOMAIN: DRINKING WATER
INDICATOR: NITRATES
LEVEL OF CONTAMINANT IN FINISHED WATER
POTENTIAL POPULATION EXPOSURE TO CONTAMINANTS IN FINISHED WATER

Type of EPHT Indicator	Hazard, Exposure
Measures	1. Distribution of number of community water systems by mean nitrate concentrations (milligrams per liter) by year 2. Distribution of number of people served by community water systems by mean nitrate concentrations (milligrams per liter) by year 3. Distribution of number of community water systems by maximum nitrate concentrations (milligrams per liter) by year 4. Distribution of number of people served by community water systems by maximum nitrate concentrations (milligrams per liter) by year 5. Distribution of number of community water systems by mean nitrate concentrations (milligrams per liter) by quarter 6. Distribution of number of people served by community water systems by mean nitrate concentrations (milligrams per liter) by quarter
Data Sources and Derivation of Measures	Nitrate measures will be derived using data each state (primacy agency) is required to collect and report under the SDWA (e.g. SDWIS/State). Only community water systems (CWS) regulated under the SDWA will be used due to the lack of consistent data for smaller systems and private wells. Specific elements will be extracted and used to create three standardized tables: CWS attributes, nitrate sampling results, and nitrate MCL violations. Nitrate sampling results will be summarized by sampling point, and then by CWS, to generate mean and maximum nitrate values for each CWS and year. These results will be merged with the CWS attribute data and the MCL violation data to create a public use dataset, from which the measures can be generated.
Purpose and 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 methemoglobimia or "blue baby syndrome' in infants who received formula from well water. When an individual is exposed to nitrate it can be converted to nitrite (NO_2^-) 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 in-consistent (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 (NO_3^-) 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 and has 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 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 often more vulnerable to higher levels of nitrates because they draw water from shallower groundwater aquifers. The U.S. geological survey 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).
Measurement Unit	Nitrate – nitrogen in mg/l
Geographic Scope	Depends on who does this.
Geographic Scale	State may be lowest level of feasible aggregation as CWS service areas may span county jurisdictions.
Time Period	1999 – present
Time Scale	Annual and quarterly means and annual maximum values.
General Limitations of the Measures and Data Source	The current measures are derived for CWS only. A large number of studies have show that private wells are another important source of population exposure to nitrates. Nitrate levels in private wells were stable over time in one study (Ruckart et al, 2007). While the MCLs and MCLGs may protect most people, they were based on a small sample size of only 144 cases (Bosch et al, 1950; Walton, 1951). Depending on the dose of nitrate or nitrite from other possible sources such as the diet, some individuals may have higher "baseline" levels of methemoglobin than others, and may thus develop cyanosis from lower levels of nitrate/nitrite in drinking water. A similar argument may hold for cancer risk (De Roos et al, 2003). Therefore it is important to know how close CWS may be to the MCLs. There are many gaps in information on chronic exposures and possible health risks of nitrate and/or nitrosation products in drinking water (Ward et al, 2005b). Hence the need for more complete and accessible data on levels of nitrates and nitrites in drinking water.
Related Indicators	
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CONTENT DOMAIN: DRINKING WATER
INDICATORS: PUBLIC WATER USE

Type of EPHT Indicator	*Exposure*
Measures	Number of people receiving water from community water systems
Data Sources and Derivation of Measures	Measures will be developed from data stored in state SDWA databases such as the Safe Drinking Water Information System (SDWIS/State) that is used to report monitoring compliance to EPA.
Purpose and 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.
Measurement Unit	count
Geographic Scope	National.
Geographic Scale	CWS, state
Time Period	2000 and later
Time Scale	Annual.

General Limitations of the Measures and Data Source	Population estimates are updated at irregular intervals (every 1 to 5 years) by community water supplies. Once population estimates are updated, historical information are no longer retained within the system.
Related Indicators	Estimated proportion of population on private wells.

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): • Plural births • Previous preterm birth • Certain uterine or cervical abnormalities of the mother • 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) • Male fetal gender (associated with singleton preterm birth) • 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
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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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