

## Heat Illness and Deaths — New York City, 2000–2011

Heat waves kill more persons, on average, than any other extreme weather event in the United States (1), and additional heat-related deaths are caused by hot weather not classified as heat waves (2). Summer temperatures in New York City (NYC) are increasing, with longer and hotter heat waves projected into the next century and beyond (3). To assess current risk factors and vulnerable populations among NYC residents, hospital data, death certificate data, and medical examiner records involving cases of heat illness, including hyperthermia (also known as heat stroke), were analyzed by the NYC Department of Health and Mental Hygiene for the period 2000–2011. On average, 447 patients each year were treated for heat illness and released from emergency departments, 152 were hospitalized, and 13 persons died from heat stroke. Chronic diseases, mental health disorders, and obesity were common comorbidities. Among fatality investigation records with information available about cooling, none found a working air conditioner in use. Outreach to city residents at high risk and their caregivers should emphasize the dangers of heat and importance of protective cooling measures during hot weather. Improving awareness of chronic health conditions that increase vulnerability to heat is also important.

The New York Statewide Planning and Research Cooperative System (SPARCS) provided de-identified electronic patient records from NYC emergency departments (EDs) and hospitals from 2000 through 2010.\* Records were limited to NYC residents and homeless persons with any diagnosis of heat illness from *International Classification of Diseases, Ninth Revision, Clinical Modification* (ICD-9-CM) codes 992.0–992.9, “effects of heat and light,”† or External Cause of Injury (E-code) of E900.0, “excessive heat due to weather conditions.” Records with an E-code of E900.1 (i.e., “due to man-made conditions”), and records of patients whose disposition was “death” (n = 75) were excluded. De-identified electronic death certificate data

were obtained from the NYC Department of Health and Mental Hygiene Office of Vital Statistics for the years 2000–2011 for NYC residents and homeless persons. Hyperthermia deaths were defined as cases having *International Classification of Diseases, 10th Revision*, (ICD-10) codes X30, “exposure to excessive natural heat” or T67, “heatstroke and sunstroke” listed as causes of death anywhere in the record, for deaths occurring in the months of May–September. Records having a man-made cause of heat exposure (W92) were excluded.

Counts and rates of heat illness ED visits, admissions, and deaths were summarized by age, sex, neighborhood poverty, and place of illness onset (Table 1). Average annual rates were estimated using 2005 population estimates produced by the NYC Department of Health and Mental Hygiene based on the U.S. Census Bureau Estimate Program and housing data from the NYC Department of City Planning. Neighborhood poverty was classified as the percentage of individuals, by postal code, below 100% of the federal poverty level, according to the U.S. Census American Community Survey 2007–2011.

A protocol was established after 2006 whereby the Department of Health hyperthermia investigation team is notified by the Office of the Chief Medical Examiner and the Office of Vital Statistics of deaths involving hyperthermia

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\* Complete emergency department records became available in 2005.

† Includes heat stroke, sunstroke, heat syncope, heat cramps, heat exhaustion, heat fatigue, and heat edema.



during or as a result of an extreme heat event, defined in NYC as  $\geq 2$  days when the heat index, which incorporates both air temperature and relative humidity, reaches at least 95°F (35°C), or at least 1 day when the heat index reaches 100°F (37.8°C). Electronic records were reviewed at the Office of the Chief Medical Examiner after extreme heat events during 2008–2011 that involved five or more deaths to obtain details on medical history and postmortem height and weight measurements for 48 decedents and information about the home environment for 41 decedents overcome by heat in their own residence.

Diabetes and obesity prevalences were compared with city-wide data from the 2010 NYC Community Health Survey.<sup>§</sup> Air conditioning prevalence was compared with the 2007 NYC Community Health Survey, and other housing characteristics were compared with the Census Bureau's Housing and Vacancy Survey for NYC. Confidence intervals were calculated using exact binomial methods for deaths. Statistical software was used for survey data to account for weighting, and normal approximation methods were used for 2010 decennial U.S. Census data.

## Death Certificates and Hospital Data

During 2000–2011, an annual average of 447 NYC residents were treated and released from an ED for heat illness, another 152 were hospitalized, and another 13 persons died. Of the

154 total deaths that occurred over the 12-year period, 70 (45%) died as a result of two severe heat waves in 2006 and 2011. The 2006 heat wave lasted for 10 days, with maximum heat indices above 90°F (32.2°C), three of which exceeded 100°F (37.8°C), and the 2011 heat wave lasted 4 days, with a peak heat index above 110°F (43.3°C). Rates of heat illness and death increased with age, were typically higher among males than females for those aged <65 years, and increased with neighborhood poverty (Table 1).<sup>¶</sup> Approximately 3% of hospitalized patients and 5% of decedents were homeless.

Comorbid conditions recorded for hospitalized patients and contributing causes of death from death certificates included cardiovascular disease (64% and 55%, respectively), diabetes (23% and 13%), substance abuse (14% and 11%), and other mental health conditions (24% and 11%). Respiratory conditions were common among hospitalized patients (22%) but not often indicated as contributing causes of death (4%) (Table 1).

## Medical Examiner Case Review

After heat waves during the summers of 2008–2011, the Department of Health reviewed medical examiner records for 48 hyperthermia deaths. The records noted evidence of cardiovascular disease for 36 decedents (75%), acute or chronic substance abuse for 14 (29%), and a history of schizophrenia or schizoaffective disorder for five (10%). Three (6%) of the

<sup>§</sup> Available at <http://www.nyc.gov/html/doh/html/data/survey.shtml>.

<sup>¶</sup> Additional information available at <http://www.nyc.gov/health/trackingportal>.

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TABLE 1. Number, percentage, and rate of residents treated for heat-related illness, by selected characteristics, type of treatment, and outcome — New York City, 2000–2011\*

Characteristic	Emergency department visits, excluding hospital admissions or deaths (2005–2010)			Hospital admissions, excluding deaths (2000–2010)			Deaths (2000–2011)		
	No.	(%)	Average annual rate per million	No.	(%)	Average annual rate per million	No.	(%)	Average annual rate per million
<b>Total</b>	<b>2,680</b>	<b>(100)</b>	<b>54.9</b>	<b>1,675</b>	<b>(100)</b>	<b>18.7</b>	<b>154</b>	<b>(100)</b>	<b>1.6</b>
<b>Female age group (yrs)</b>									
0–4	21	(1)	12.1	8	(<1)	2.5	0	—	—
5–14	146	(5)	48.2	11	(1)	2.0	1	(1)	0.2
15–34	435	(16)	61.3	35	(2)	2.7	5	(3)	0.4
35–64	414	(15)	41.0	164	(10)	8.9	21	(14)	1.0
65–84	208	(8)	69.3	361	(22)	65.6	26	(17)	4.3
≥85	63	(2)	99.2	206	(12)	177.0	14	(9)	11.0
<b>Male age group (yrs)</b>									
0–4	40	(1)	22.0	13	(1)	3.9	2	(1)	0.5
5–14	133	(5)	42.1	14	(1)	2.4	0	(0)	0.0
15–34	519	(19)	75.3	114	(7)	9.0	7	(5)	0.5
35–64	563	(21)	61.9	389	(23)	23.3	53	(34)	2.9
65–84	120	(4)	59.2	272	(16)	73.2	21	(14)	5.2
≥85	18	(1)	65.9	88	(5)	175.8	4	(3)	7.3
<b>Neighborhood poverty level†</b>									
Low (<10%)	368	(14)	36.7	233	(14)	12.7	24	(16)	1.2
Medium (10% to <20%)	947	(35)	52.4	614	(38)	18.5	51	(35)	1.4
High (20% to <30%)	647	(24)	55.2	408	(25)	19.0	36	(24)	1.5
Very high (≥30%)	707	(26)	76.5	357	(22)	21.1	36	(24)	1.9
Other/Unknown‡	11	—	—	63	—	—	7	—	—
<b>Homeless¶</b>	<6	NA	NA	58	(3)	NA	7	(5)	NA
<b>Comorbidities</b>									
Cardiovascular	233	(9)	NA	1,069	(64)	NA	84	(55)	NA
Diabetes	106	(4)	NA	387	(23)	NA	20	(13)	NA
Respiratory	102	(4)	NA	373	(22)	NA	6	(4)	NA
Substance use/dependency	44	(2)	NA	240	(14)	NA	17	(11)	NA
Mental illness	63	(2)	NA	402	(24)	NA	17	(11)	NA
<b>Place of heat illness</b>									
Residence	270	(15)	NA	636	(48)	NA	37	(80)	NA
Street	237	(14)	NA	229	(17)	NA	4	(9)	NA
Industrial place or worksite	126	(7)	NA	34	(3)	NA	1	(2)	NA
Other	978	(56)	NA	363	(27)	NA	4	(9)	NA
Unspecified/Unknown‡	930	—	—	353	—	—	108	—	—

**Abbreviation:** NA = not available.

\* Hospital outpatient data available only for 2005–2010, inpatient through 2010. Data restricted to events in months of May–September.

† Neighborhood poverty level was based on postal code and defined as the percentage of residents with incomes below 100% of the federal poverty level, according to the American Community Survey 2007–2011. Rates were based on 2010 census data.

‡ Excluded from the denominator used to calculate percentages.

¶ Based on homeless indicator in hospital data and residence unknown in death certificates.

decedents had cerebral palsy, including one child. Based on postmortem height and weight, 10 (48%) decedents aged 18–64 years with known body mass index were classified as obese, compared with 16% of NYC adults of the same ages in 2010 ( $p < 0.05$ ). The prevalence of diabetes (15%) was not significantly different from the citywide estimate of 9% (Table 2).

Of the 48 decedents, 41 (85%) were overcome by heat in their own home. Of 26 deaths with information available on the presence or absence of air conditioning, 23 (88%) did not have any air conditioner, and the remaining three (12%) had an

air conditioner that was broken or not in use. By comparison, 13% of NYC adults reported living in residences without air conditioning in 2007 (Table 3). The proportion of decedents who lived alone (18%) was not significantly different from the percentage of NYC adults who lived alone (14%). Compared with all city residents, a lower proportion of decedents lived in buildings with elevators (23% versus 35%) or in public housing (2% versus 6%), but these differences were not statistically significant (Table 3).

**TABLE 2. Number and percentage of heat stroke decedents (n = 48), by selected medical characteristics — New York City, 2008–2011**

Medical characteristic	Heat stroke decedents		
	No.	(%)	(95% CI)
Evidence of cardiovascular disease	36	(75)	(60–86)
Evidence of alcohol or substance abuse	14	(29)	(17–44)
History of diabetes	7	(15)	(6–28)
History of schizophrenia/ schizo-affective disorder	5	(10)	(3–23)
Cerebral palsy	3	(6)	(1–17)
<b>Known body mass index among those aged 18–64 yrs (n = 21)</b>			
Normal/Underweight	5	(24)	(8–47)
Overweight	6	(29)	(11–52)
Obese	10	(48)	(26–70)

Abbreviation: CI = confidence interval.

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### Editorial Note

During 2000–2011, approximately 600 cases of serious illness and 13 deaths occurred annually in NYC as a result of heat illness. Although these cases do not capture the full spectrum of the health effects of extreme heat, such as exacerbations of chronic conditions leading to hospital admissions or deaths that are not recognized or coded as heat-related (9), cases of hyperthermia and other forms of heat illness can be directly identified, counted, and investigated to better understand risk factors and potential gaps in public health communications and interventions.

Older adults continue to have the highest rates of heat illness and death. However, persons of all ages are at risk, especially those with underlying physical or mental health conditions and those taking medications that can impair thermoregulation (4). The association of obesity with heat-related death in NYC is consistent with biologic evidence that adiposity increases vulnerability to heat exhaustion (4,5).

In NYC, the majority of hospitalized and fatal cases of heat illness occurred in the home. Among the 26 deaths reviewed that had information about the presence of home air conditioning, none of the decedents had a working air conditioner. Aspects of the urban environment can cause city apartments without air conditioning, in some cases, to reach temperatures more than 18°F (10°C) higher than outdoor temperatures on hot days (6).

Unlike findings from a study of a 1995 Chicago heat wave (7), NYC hyperthermia decedents were not statistically more likely to live in multifamily apartment buildings than the

### What is already known on this topic?

Heat waves cause more deaths in the United States, on average, than any other type of extreme weather event. Older adults, those with underlying physical or mental health conditions, and those without access to working home air conditioning are most at risk for hyperthermia death.

### What is added by this report?

During 2000–2011, approximately 447 heat-related emergency department visits, 152 hospital admissions, and 13 heat-related deaths occurred each year in New York City. Higher rates of heat illness and death were associated with older age and neighborhood poverty; chronic physical and mental health conditions were prevalent comorbidities in decedents. Based on medical examiner records for 48 decedents, 85% were exposed at home and, among records with information regarding the presence of air conditioning, none of the decedents had a working air conditioner. Among decedents aged 18–64 years, 48% were obese and another 29% were overweight. Unlike in some previous studies, decedents in this analysis were not more likely to live in multifamily apartment buildings or to live alone.

### What are the implications for public health practice?

Rising summer temperatures from climate change, a growing older adult population, and the increasing prevalence of obesity and chronic disease might increase the number of serious heat illnesses and deaths in New York City. Adaptation efforts in urban settings should focus on neighborhoods with high poverty, promoting greater access to air conditioning, and encouraging members of the public to check on vulnerable family members and contacts.

general city population. Also, unlike other previous studies (4), NYC hyperthermia decedents were not more likely to live alone. Hyperthermia can progress rapidly, and many persons might not be aware of the warning signs, including lack of sweating in late-stage illness.

The findings in this report are subject to at least three limitations. First, cases of heat illness identified in this report might not include heat-associated increases in rates of hospital care for other conditions such as cardiovascular disease, renal disease, and diabetes, and do not reflect heat-associated increases in overall mortality rates from natural causes (4,8). In 2006, 100 excess deaths from natural causes in NYC were attributed to a severe heat wave, based on typical summer mortality rates from a statistical model (9). Second, place of injury is not determined by a standard protocol and often is recorded as unspecified or unknown. Similarly, data on air conditioning was available only for a small number of decedents. Finally, the prevalence of obesity in the general NYC population was based on self-reported height and weight, which tends to underestimate obesity.

Although heat waves typically cause less mortality today, compared with years past (10), reductions in summer heat

**TABLE 3. Number and percentage of heat stroke decedents with onset at home (n = 41), by selected housing characteristics,\* compared with percentage of city residents overall — New York City, 2008–2011**

Housing characteristic	Heat stroke decedents			Residents overall		p-value
	No.	(%)	(95% CI)	(%)	(95% CI)	
<b>Air conditioner in the home</b>						
Present, working, and in use <sup>†</sup>	0	—	—	(87)	(87–88)	
Not working or not in use	3	(12)	(3–30)			
No	23	(88)	(70–98)	(13)	(12–13)	<0.001
Unknown <sup>§</sup>	15	—	—			
<b>Lived alone</b>						
Yes	7	(18)	(6–31)	(14)	(14–14)	
No	31	(82)	(66–92)	(86)	(86–86)	0.432
Unknown <sup>§</sup>	3	—	—			
<b>Building type</b>						
1 or 2 units	14	(36)	(21–53)	(33)	(33–34)	
≥3 units, walk-up	16	(41)	(26–58)	(32)	(31–33)	
≥3 units, elevator	9	(23)	(11–39)	(35)	(34–36)	0.262
Unknown <sup>§</sup>	2	—	—			
<b>No. of floors</b>						
≤2	16	(39)	(24–56)	(30)	(29–31)	
3–5	12	(29)	(16–46)	(35)	(34–36)	
≥6	13	(32)	(18–48)	(36)	(35–36)	0.395
<b>Public housing</b>						
Yes	1	(2)	(1–13)	(6)	(6–6)	
No	40	(98)	(87–99)	(94)	(94–95)	0.337

**Abbreviation:** CI = confidence interval.

\* Comparison with percentage of New York City residents overall is from the 2010 U.S. Census for householders living alone; household occupancy type and structure type is from the 2008 New York City Housing and Vacancy Survey. Comparison with citywide air conditioning prevalence is from the 2007 New York City Community Health Survey for residents living in households with or without air conditioning. Additional information available at <http://www.nyc.gov/html/goh/html/data/survey.shtml>.

<sup>†</sup> Percentages for residents overall include all homes with air conditioners, whether working or not.

<sup>§</sup> Excluded from the denominator.

illness and mortality over the past century in NYC might be threatened by rising temperatures, a growing older adult population, and the increasing prevalence of obesity and chronic disease. This investigation highlights risks to vulnerable persons living in NYC homes without air conditioning. Before and during heat waves, outreach to seniors and those with chronic physical and mental health conditions, as well as their caregivers, should emphasize protective measures to avoid heat illness. Susceptible persons should be encouraged to stay hydrated and use air conditioning, if available, during periods of extreme heat. For those without air conditioning who are able to leave their homes, cooling centers and other air-conditioned public places can provide respite during heat waves. Pools, cool showers, or baths also can provide some relief. Surveillance for hyperthermia illness and mortality can help identify local patterns of vulnerability to best target heat emergency response activities and prevention efforts.

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## Fatal West Nile Virus Infection After Probable Transfusion-Associated Transmission — Colorado, 2012

West Nile virus (WNV) is transmitted to humans primarily by infected mosquitoes. However, WNV also can be transmitted through infected blood products or solid organs (1). Since 2003, the U.S. blood supply has been routinely screened for WNV RNA. The Food and Drug Administration (FDA) recommends that blood collection agencies perform WNV nucleic acid testing (NAT) year-round on all blood donations, either in minipools of six or 16 donations (MP-NAT) or as individual donations (ID-NAT) (2). Since implementation of screening, 12 transfusion-associated transmissions of WNV have been documented (3–5). This report describes a case of fatal WNV encephalitis in an immunosuppressed patient after probable transfusion-associated transmission. The implicated donation was reactive by MP-NAT but nonreactive by ID-NAT on routine screening and was released for transfusion. During the subsequent investigation, retrospective testing of the donated unit showed discrepant ID-NAT results and evidence of WNV-specific immunoglobulin M (IgM) and neutralizing antibodies. Although WNV is rarely transmitted through screened blood products, clinicians should consider WNV disease in patients with compatible symptoms who were recently transfused. Further evaluation is required to determine the relative risks and benefits of different strategies to manage MP-NAT-reactive minipools when all constituent donations are nonreactive by subsequent ID-NAT

### Case Report

In August 2012, a man with non-Hodgkin's lymphoma was admitted to hospital for chemotherapy and autologous stem cell transplant. His cancer was in partial remission after six cycles of chemotherapy. He had been screened for subclinical infections 14 days before stem cell collection, and all testing, including WNV NAT, was negative. Stem cells were collected 8 days before admission and transplanted on hospital days 8 and 9. The patient developed gastrointestinal symptoms on hospital day 18, followed by fever and hypotension on hospital day 28. On hospital day 29, he developed altered mental status, somnolence, and respiratory failure; sedation and mechanical ventilation precluded a full neurologic assessment. Cerebrospinal fluid (CSF) collected on hospital day 30 showed an elevated glucose (103 mg/dL [normal: 40–80 mg/dL]) and normal protein (44 mg/dL [normal: 5–50 mg/dL]) with two white blood cells/mm<sup>3</sup> (normal: 0–5/mm<sup>3</sup>) and 46 red blood cells/mm<sup>3</sup> (normal: 0–5/mm<sup>3</sup>). This CSF sample was negative for bacteria by Gram and acid-fast stains, for cryptococcal

antigen by latex agglutination, and for cytomegalovirus, BK virus, herpes simplex virus, JC virus, human herpes virus 6, and varicella-zoster virus by polymerase chain reaction (PCR); however, the sample was not tested for WNV. Magnetic resonance imaging showed meningeal and cortical changes consistent with inflammation, and electroencephalogram findings were consistent with diffuse encephalopathy. The patient's mental status did not improve after discontinuation of sedation; support was withdrawn, and he died on hospital day 47. Postmortem evaluation showed diffuse encephalitis. WNV IgM antibodies were identified in serum collected on hospital day 43. WNV RNA was detected by TaqMan reverse transcription-PCR (RT-PCR) on brain and spinal cord tissues collected at autopsy.

### Public Health Investigation

The patient was hospitalized continuously for 4 weeks before illness onset without known outdoor exposure; therefore, mosquito-borne WNV transmission was unlikely. A public health investigation was conducted to determine the source of his WNV infection and prevent further transmission by removal of any contaminated blood products. The patient had received allogeneic, leukoreduced, irradiated blood products during hospital days 13–30, including 6 units of red blood cells, 2 units of platelets, and 2 units of fresh frozen plasma. He received 2 additional units each of red blood cells and platelets on day 44.

The hospital blood bank investigated the blood products received before hospital day 43, when the patient's first WNV-positive serum was collected. WNV serologic testing was conducted on all 10 associated donors. Nine donors had no evidence of WNV infection by WNV IgM enzyme-linked immunosorbent assay. One apheresis donor was positive for WNV IgM antibodies and for WNV neutralizing antibodies by plaque reduction neutralization testing performed on serum collected 56 days after the implicated donation. This donor reported no history of illness before or after donation and no travel, but did report outdoor exposure with mosquito bites. The apheresis platelet unit was donated on the recipient's hospital day 9 and transfused on day 14. The implicated donation was initially screened as part of a minipool with five other donations; the MP-NAT was reactive using the Cobas TaqScreen West Nile Virus Test (Roche Molecular Systems). However, all of the units comprising the minipool were nonreactive when tested by ID-NAT using the same assay and were

released into the blood supply per FDA guidance at the time of the investigation (2).

In addition to the implicated unit of platelets, fresh frozen plasma was also derived from the original donation but was not transfused. The quarantined unit of fresh frozen plasma was retrieved and tested for WNV. Freshly thawed aliquots from this unit were reactive for WNV on four of five replicate Cobas TaqScreen West Nile Virus Tests, but WNV RNA could not be detected by conventional Taqman RT-PCR. An aliquot from the same plasma unit, tested 58 days after being thawed and stored at 39.2°F (4.0°C) was nonreactive by the Procleix WNV NAT assay (Gen-Probe Incorporated). The unit was positive for WNV IgM antibodies by microsphere-based immunosorbent assay, WNV immunoglobulin G antibodies by enzyme-linked immunosorbent assay, and WNV neutralizing antibodies by plaque reduction neutralization testing.

Per FDA guidelines, the donor was deferred from making further donations for 120 days after the implicated donation. The donor had made seven donations of platelets and plasma in the 120 days before the implicated donation, and one donation of platelets and plasma 35 days after the implicated donation. The blood products from those donations had been transfused to nine recipients. Four of these recipients had since died; however, their deaths were thought to be related to causes other than WNV infection because none had discernible signs or symptoms of WNV disease. However, no clinical samples were available for testing. The five surviving recipients were monitored clinically and remained asymptomatic but were not tested for WNV infection.

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#### Editorial Note

This report describes a fatal case of WNV disease after probable transfusion-associated transmission via platelets collected from an asymptomatic infected blood donor. Although transfusion-transmitted WNV has been reported after initial screening was nonreactive by MP-NAT, it has not been documented after screening was nonreactive by ID-NAT. This case demonstrates the potential for transmission of WNV through

#### What is already known on this topic?

Blood collection agencies in the United States routinely screen donors for West Nile virus (WNV) RNA using minipool nucleic acid testing (MP-NAT) augmented by individual donation testing (ID-NAT). Since 2003, approximately 3,500 reactive units have been removed from the blood supply, preventing transfusion transmission of WNV. This system is effective; however, 12 transfusion-associated cases have been identified since implementation of WNV NAT screening.

#### What is added by this report?

This report describes the first probable case of transfusion-associated WNV infection in which the donation was reactive by MP-NAT but nonreactive by ID-NAT on initial screening. The case occurred in an immunosuppressed patient. Virus-specific immunoglobulin M and neutralizing antibodies could be detected in the implicated donation. These findings are suggestive that transmission occurred from a blood donation with a low concentration of WNV even in the presence of presumptively protective WNV-specific antibodies.

#### What are the implications for public health practice?

Clinicians should maintain a high index of suspicion for cases of transfusion-associated WNV disease, especially in immunosuppressed patients with compatible symptoms who have recently received blood products. Further evaluation is required to determine the relative risks and benefits of different strategies to manage MP-NAT-reactive minipools when all constituent donations are nonreactive by subsequent ID-NAT.

transfusion of blood products that screen nonreactive by ID-NAT, despite having detectable WNV-specific IgM and neutralizing antibodies.

Persons infected with WNV typically develop a transient viremia that occurs 1–2 weeks after infection and lasts approximately 7 days. Viremia, detectable by NAT, is present before symptom onset and early in the course of illness, but usually resolves as the patient develops WNV antibodies. FDA recommends that blood collection agencies screening for WNV by MP-NAT establish criteria to switch to ID-NAT, which is more sensitive than MP-NAT, during periods of high WNV activity in their collection areas (2). At the time of the implicated donation, the blood testing agency had not reached its threshold for conversion to ID-NAT. During initial screening, the implicated donation was reactive by MP-NAT. ID-NAT was performed to identify the infected donation(s) in the minipool; however, none of the constituent donations were reactive, so the products were released into the blood supply. The blood collection and testing agencies involved have now decided to discard all constituent units of reactive minipools when an ID-NAT reactive donation cannot be identified.

It is likely that this transfusion-associated transmission occurred because the donor had a waning viremia that was sufficiently low to be inconsistently identified by either MP-NAT

or ID-NAT. NATs performed during initial screening and during retrospective testing of the plasma co-component from the implicated donation yielded discrepant results. The Cobas TaqScreen West Nile Virus Test and Procleix WNV NAT assays typically detect lower levels of WNV RNA than conventional RT-PCR. However, at very low levels of viremia, NAT assays performed on minipools or individual units can exhibit test-to-test variability and might require replicate testing to detect viral RNA (6–8). In addition, the nonreactive Procleix NAT might reflect lower WNV RNA concentration in the sample because of viral degradation during prolonged storage at 39.2°F (4.0°C).

In published reports of transfusion-associated transmissions of WNV, IgM could not be detected in implicated donations when stored donor segments or co-components were available for testing (1,3). However, researchers at a conference in 2003 reported detection of WNV IgM in one of 13 WNV RNA-positive plasma co-components identified during multiple transfusion-transmission investigations during that year; neutralizing antibody results were not reported (9). An *in vitro* study using serum from blood donors containing both WNV RNA and IgM antibodies showed that 10 (36%) of 28 of these specimens could infect Vero cells (10). In the case described in this report, WNV transmission appears to have occurred despite the presence of detectable WNV-specific IgM and neutralizing antibodies in the implicated donation. The protective benefit of these antibodies might have been insufficient to prevent infection in this severely immunosuppressed patient.

Since the implementation of routine screening of blood products for WNV, transfusion-associated WNV infections have been rare. However, clinicians should consider WNV disease in any patient with compatible symptoms who has received a blood transfusion during the 28 days before illness onset. This is particularly important in immunosuppressed patients, such as stem cell recipients, who might be more susceptible to WNV infection at very low viral concentrations in the transfused blood product and, once infected, are at greater risk for developing WNV neurologic disease. Suspected cases

should be reported immediately to hospital blood banks and public health authorities to facilitate prompt investigation and quarantine of potentially infectious blood products. Further evaluation is required to determine the relative risks and benefits of different strategies to manage MP-NAT-reactive minipools when all constituent donations are nonreactive by subsequent ID-NAT.

### Acknowledgments

Carol Gullickson, Infection Prevention, Don Lenderink, Blood Bank, Presbyterian/St. Luke's Medical Center, Denver, Colorado.

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## CDC Grand Rounds: Public Health Approaches to Reducing U.S. Infant Mortality

Infant mortality is defined as the death of an infant before his or her first birthday. The infant mortality rate (IMR) measures this occurrence per 1,000 live births. In addition to being a key marker of maternal and child health, the IMR has been called the most sensitive indicator of overall societal health. In the United States, substantial progress has been made over the last 50 years in reducing the IMR; however, further reduction of preventable infant deaths remains a challenge. Based on preliminary data, the IMR in 2011 had declined to 6.05 overall, but that number obscures persistent racial and geographic disparities (Figure 1) (1). Non-Hispanic black infants continue to die at nearly twice the rate of non-Hispanic white infants. Additionally, preterm-related causes of death among black infants occur at a rate three times greater than that of white infants (2,3). Geographically, the majority of states in the top quartile for infant mortality are in the southern United States (Figure 2) (2).

Infant mortality is divided into two age periods: neonatal (birth–27 days) and postneonatal (28–364 days). Approximately two thirds of all infant deaths occur in the neonatal period, and one third occur in the postneonatal period. Infant deaths in the neonatal period are caused by complications arising from preterm births, birth defects, maternal health conditions, complications of labor and delivery, and lack of access to appropriate care at the time of delivery. Infant deaths in the postneonatal period are driven by sudden unexpected infant death (SUID) (including sudden infant death syndrome [SIDS]), injury, and infection. An increasing proportion of postneonatal infant deaths occur among infants who were born preterm but survived the neonatal period (4). Prevention of infant deaths should begin in the preconception period; opportunities are available to improve the health of mothers, and thus avoid preventable infant deaths.

### Infant Mortality in the United States

A major contributor to the decline in U.S. infant mortality is the decline in neonatal deaths associated with low birthweights (Figure 3). An infant born in 1950 with a birthweight <1,000 grams had only a 10%–15% chance of survival, whereas an

infant born in 2008 with a birthweight <1,000 grams had a >60% chance of surviving the neonatal period.

Since 1950, medical technology has helped to reduce infant mortality, but the United States still has a relatively poor global standing. In 2010, the United States ranked 32nd among the 34 nations of the Organization for Economic Cooperation and Development in infant mortality, and the overall IMR was three times that of the countries with the lowest IMRs: Iceland (2.2 per 1,000), Finland (2.3), and Japan (2.3) (5). The main reason that the U.S. IMR remains higher than that of European nations is because the United States has a high percentage of preterm births (6). The United States ranks 130th out of 184 countries for preterm births; approximately 12% of U.S. births are preterm.\*

Opportunities exist to reduce the mortality rate among infants born preterm by addressing key risk factors. Prenatal smoking contributes to low birthweight, preterm delivery, preterm-related death, and SIDS. Many very low birthweight infants in the United States are not born in hospitals that have level III neonatal intensive-care units, which have been shown to significantly reduce mortality (7). To reduce the IMR, the rates of preterm birth, including rates of late preterm birth (births between 34 and 36 weeks of gestation), need to be reduced (8).

### State and Local Efforts to Reduce Deaths in Infancy

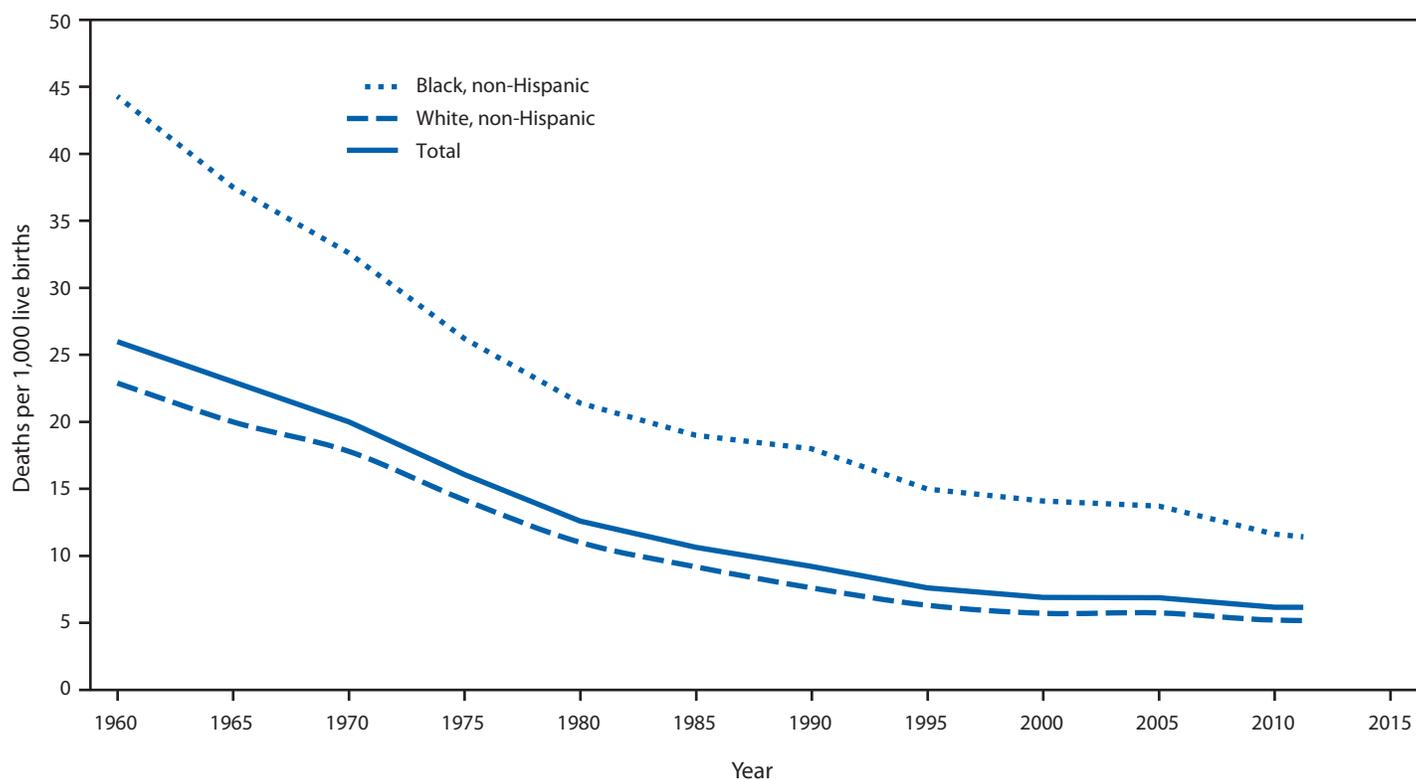
The Pregnancy Risk Assessment Monitoring System (PRAMS) has been operating for the past 25 years.† In 1987, Congress appropriated funds for CDC to administer state-based programs of surveillance to collect data that would be helpful for reducing maternal and infant morbidity and mortality. Data collected would be used to direct the efforts of health programs. Forty states and New York City currently participate in PRAMS, and three additional states (California, Nevada, and Idaho) conduct PRAMS-like surveys. Information is reported to PRAMS by new mothers in response to a mailed questionnaire; if no response to the mailed questionnaire is received, the mother is contacted by telephone. Approximately 77,000 responses are received annually. In 2010, response rates ranged from 54% (Virginia) to 83% (Vermont). Key survey questions focus on topics such as breastfeeding, prenatal care, cigarette smoking during pregnancy, and infant sleep position.

\* Additional information available at <http://www.everywomaneverychild.org/borntoosoon>.

† Additional information available at <http://www.cdc.gov/prams>.

*This is another in a series of occasional MMWR reports titled CDC Grand Rounds. These reports are based on grand rounds presentations at CDC on high-profile issues in public health science, practice, and policy. Information about CDC Grand Rounds is available at <http://www.cdc.gov/about/grand-rounds>.*

FIGURE 1. Infant mortality rates, by race/ethnicity and year — United States, 1960–2011

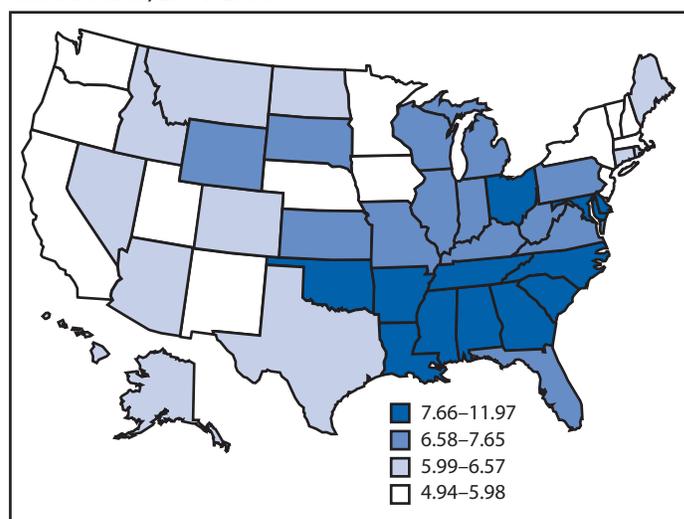


Through the years, local and state efforts have been successful in identifying infant mortality risks through the use of PRAMS data. For example, white mothers and mothers aged <19 years have had the highest prevalence of smoking during pregnancy. Among PRAMS participants, West Virginia has had the largest percentage (>30%) of respondents who smoked cigarettes during the last 3 months of pregnancy, compared with New York City, which had the smallest percentage (2.3%). To help reduce smoking among pregnant women, West Virginia launched the “Tobacco Free Pregnancy Initiative” in 2009, with resulting increases in calls to tobacco quitlines by pregnant women and their families.<sup>§</sup> In Michigan, PRAMS data revealed that black non-Hispanic mothers were 20% less likely than mothers of other races/ethnicities to place infants on their backs to sleep. In 2004, the Michigan governor’s office launched the “Infant Safe Sleep Campaign,” which included educational and policy components. Safe sleep messages were integrated into state services and programs. As one example, Michigan required child care centers to adhere to safe sleep recommendations as a condition for licensure.<sup>¶</sup> Each of these state programs used PRAMS data to discover prevention opportunities and target interventions.

<sup>§</sup> Additional information available at [http://c.ygcdn.com/sites/www.naquitline.org/resource/resmgr/2009\\_conference\\_materials/wvpostpartum.pdf](http://c.ygcdn.com/sites/www.naquitline.org/resource/resmgr/2009_conference_materials/wvpostpartum.pdf).

<sup>¶</sup> Additional information available at [http://www.michigan.gov/dhs/0,4562,7-124-5453\\_7124\\_57836---,00.html](http://www.michigan.gov/dhs/0,4562,7-124-5453_7124_57836---,00.html).

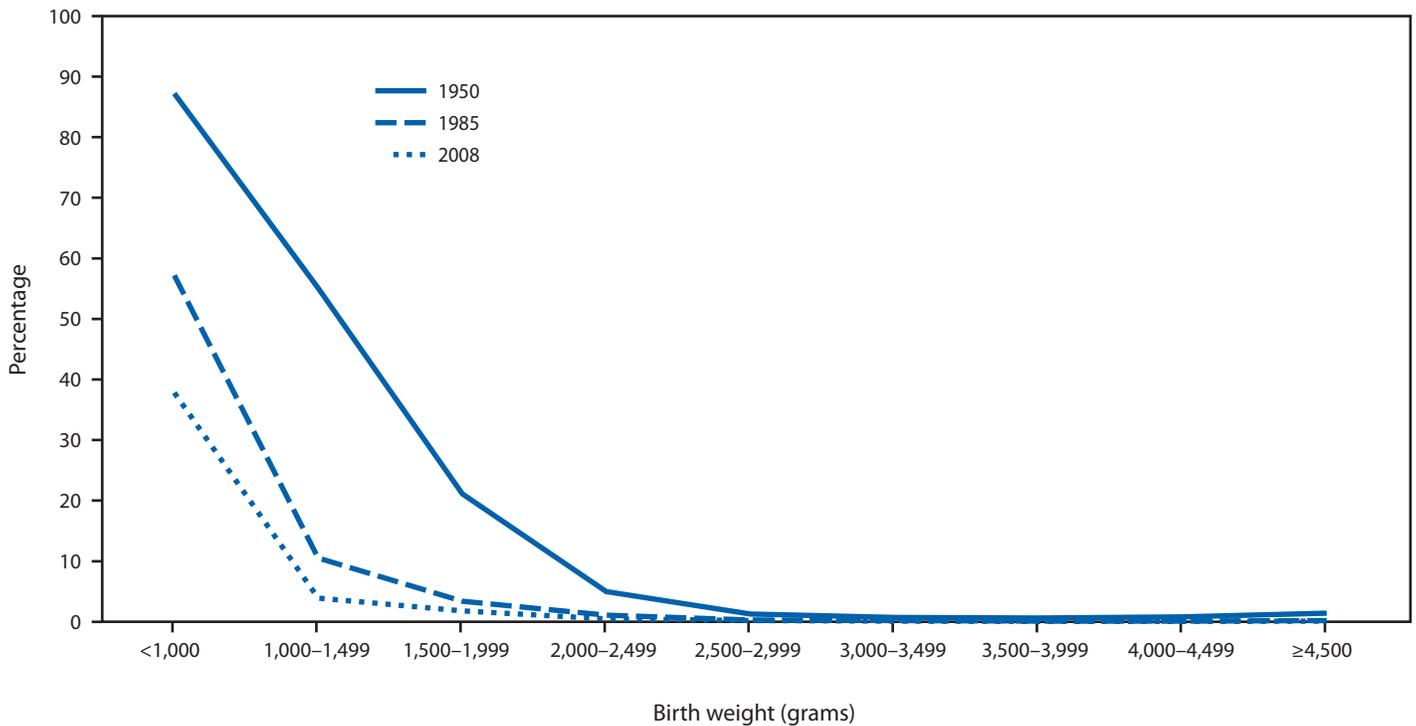
FIGURE 2. Infant mortality rates per 1,000 live births, by state — United States, 2004–2008



## Reducing SUID and SIDS

SUID comprises three main categories of death: accidental suffocation and strangulation in bed (ASSB), ill-defined deaths, and SIDS. Approximately 4,500 deaths per year are attributable to SUID. Of these deaths, approximately half result from SIDS, which is the leading cause of infant mortality in the

FIGURE 3. Birthweight-specific neonatal mortality — United States, 1950, 1985, and 2008



postneonatal period. During 1995–2005, the proportion of SUID deaths attributed to SIDS declined, and the proportion of deaths attributed to ill-defined causes and ASSB increased.

Rates of SIDS and ASSB are highest among American Indians/Alaska Natives, followed by non-Hispanic blacks (9). Risk factors for SIDS include side or stomach sleeping position, bed sharing, soft bedding, and exposure to smoke, as well as prenatal drug or alcohol use by the mother. Protective actions against SIDS include room sharing without bed sharing, breastfeeding, pacifier use, and immunization. Based on epidemiologic studies, the American Academy of Pediatrics (AAP) published revised recommendations in October 2011, with level A recommendations considered the strongest recommendations (10). AAP recommends putting infants on their backs to sleep every time, using a firm sleeping surface, room sharing without bed sharing, keeping soft objects away from infants, regular prenatal care, breastfeeding, avoiding smoke exposure, and breastfeeding as much and for as long as a mother can. Additional level A recommendations include offering a pacifier at nap time and bedtime, avoiding overheating, and refraining from the use of home cardiorespiratory monitors as a strategy for reducing the risk for SIDS. AAP expanded the national campaign to include a major focus on the safe sleep environment and ways to reduce the risks for all sleep-related infant deaths.

### National Efforts

From 2007 through 2011, the U.S. IMR declined approximately 3% per year, from 6.75 to 6.05 per 1,000 live births, as a result of targeted efforts. If the trend continues, the U.S. IMR would be reduced to 4.5 per 1,000 live births by the year 2020. The *Healthy People 2020* target is to reduce the rate of infant death to 6.0 per 1,000 live births.\*\* On June 14, 2012, the U.S. Department of Health and Human Services announced that it would work with state agencies to develop a national strategy for addressing infant mortality. The Secretary's Advisory Committee on Infant Mortality was created as an independent body, administered by the Health Resources and Services Administration, to advise the Secretary on the programs created to reduce infant mortality. The committee provides the Secretary with advice on policies and resources required as well as advice on coordinating efforts with state agencies and programs. Strategies include 1) improving women's health before pregnancy, 2) promoting quality and safety in prenatal care, 3) investing in prevention and health promotion, 4) promoting coordination among health services, 5) strengthening surveillance and research, and 6) promoting public/private and community collaboration.

\*\* Additional information available at <http://www.healthypeople.gov/2020/topicsobjectives2020/objectiveslist.aspx?topicid=26>.

The Collaborative Improvement and Innovation Network (CoIIN) to reduce infant mortality is a collaborative effort of states, the Health Resources and Services Administration, CDC, the Centers for Medicare & Medicaid Services, the Association of Maternal and Child Health Programs, the Association of State and Territorial Health Officials, the March of Dimes, and other agencies.<sup>††</sup> CoIIN was initiated in 13 mostly southern states<sup>§§</sup> in January 2012. Each state designs and shares their state's plan to reduce infant mortality. The focus of CoIIN's strategy teams include reducing elective deliveries at <39 weeks' gestation, expanding interconception care in Medicaid, reducing SIDS/SUID, increasing smoking cessation among pregnant women, and expanding the regionalization of perinatal services to provide more appropriate levels of neonatal medical care for high-risk infants. High-risk infants need to be born in facilities where they can receive the best medical care, and decades of data demonstrate that high-risk infants born and cared for in level III neonatal intensive-care units have better outcomes. The goal of these programs and related collaborative efforts is to improve access to quality preconceptional, periconceptional, and prenatal health care across racial/ethnic and geographic divides, and to provide the best available care to mothers and infants.

#### Reported by

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<sup>††</sup> Additional information available at <http://mchb.hrsa.gov/infantmortality/coiin/index.html>.

<sup>§§</sup> Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississippi, New Mexico, North Carolina, Oklahoma, South Carolina, Tennessee, and Texas.

## Vital Signs: Obesity Among Low-Income, Preschool-Aged Children — United States, 2008–2011

On August 6, 2013, this report was posted as an MMWR Early Release on the MMWR website (<http://www.cdc.gov/mmwr>).

### Abstract

**Background:** The prevalence of obesity among U.S. preschoolers has doubled in recent decades. Childhood obesity increases the risk for adult obesity and is associated with negative health consequences. Trends in the state-specific prevalence of obesity among low-income U.S. preschool children have not been examined since 2008. State-specific obesity prevalence surveillance helps determine the need for and impact of state and local obesity prevention strategies.

**Methods:** Measured weight and height data from approximately 11.6 million low-income children aged 2–4 years from 40 states, the District of Columbia, and two U.S. territories who participated in the Pediatric Nutrition Surveillance System during 2008–2011 were used to estimate state obesity prevalence. Obesity was defined as having an age- and sex-specific body mass index  $\geq 95$ th percentile, according to the 2000 CDC growth charts. Logistic regression models adjusted for age, sex, and race/ethnicity were used to examine trends in the state-specific obesity prevalence.

**Results:** During 2008–2011, statistically significant downward trends in obesity prevalence were observed in 18 states and the U.S. Virgin Islands. Florida, Georgia, Missouri, New Jersey, South Dakota, and the U.S. Virgin Islands had the largest absolute decreases in obesity prevalence, each with a decrease of  $\geq 1$  percentage point. Twenty states and Puerto Rico experienced no significant change, and obesity prevalence increased significantly in three states.

**Conclusions and Implications for Public Health Practice:** Small but significant declines in obesity among low-income preschoolers were observed in 19 of 43 states/territories examined. Continued prevention efforts are needed to sustain and expand the implementation and evaluation of population-level interventions to prevent childhood obesity.

### Introduction

Data from the National Health and Nutrition Examination Survey indicate that nationally the prevalence of obesity remains high among all youths, including preschool-aged children, however there was no statistically significant change in obesity prevalence between 2007–2008 and 2009–2010. During 2009–2010, the prevalence of obesity was 12.1% among U.S. children aged 2–5 years with higher rates among some subgroups including non-Hispanic black (18.9%) and Hispanic (16.2%) children (1). Childhood obesity is associated with negative physical and mental health consequences (2). Overweight or obese preschoolers are five times as likely as to become overweight or obese adults as their non-obese peers (3). Preventing obesity early in life is a public health priority to improve health across the lifespan.

National estimates of the prevalence of obesity describe the extent of the problem for the U.S. population. However, state-specific obesity prevalence estimates provide opportunities for state and local health departments to monitor progress in controlling and preventing obesity. In addition, state-specific estimates are important because there is variability among states in demographic and other factors that might influence obesity and appropriate prevention strategies and decision-making related to programmatic efforts, environmental supports, and policies might differ.

Limited studies are available on state-specific trends in obesity prevalence among young children. The few published reports focus on older school-aged children and adolescents (4–6) and use parent/child reports of height and weight to calculate body mass index (BMI) (4,5). Other reports on young children (7,8)\* have used data from CDC's Pediatric Nutrition Surveillance System (PedNSS); one report showed that among 44 states/territories during 2003–2008, nine showed a significant decline in obesity, 24 showed significant increases, and 11 showed no statistically significant change (7). The prevalence of obesity among low-income preschool children remains high. In 2011, the prevalence of obesity for all contributors to the PedNSS was 14.4%, was approximately 4% points higher than in the early 1990s<sup>†</sup> and varied considerably among states/territories. This report extends earlier PedNSS analyses (7,8) by examining the state-specific prevalence and trends of obesity during 2008–2011.

\* Dalenius K BE, Smith B, Polhamus B, Grummer-Strawn L. Pediatric nutrition surveillance 2010 report. Atlanta, GA: US Department of Health and Human Services, CDC; 2012.

<sup>†</sup> CDC. Pediatric Nutrition Surveillance System. Atlanta, GA: US Department of Health and Human Services, 2013. Available at [http://www.cdc.gov/pednss/pednss\\_tables/pdf/national\\_table12.pdf](http://www.cdc.gov/pednss/pednss_tables/pdf/national_table12.pdf).

## Methods

PedNSS is a state-based public health surveillance system that monitors the nutritional status of low-income children from birth through age 4 years. Data are primarily collected from participants in the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC). About 50% of low-income children eligible for WIC are enrolled (9), but data from other programs such as the Early and Periodic Screening, Diagnosis and Treatment program, and the Maternal and Child Health Bureau Title V program are also included. Not all WIC agencies submitted data to the PedNSS during 2008–2011 (9). Data were collected from children and their caregivers during routine visits to local public health clinics approximately twice per year. Children's heights and weights were directly measured by trained clinical staff using standardized protocols. All data were submitted electronically to CDC. One randomly selected clinical record per child, per year, was used in this study. Obesity was defined as having an age- and sex-specific BMI  $\geq 95$ th percentile according to the 2000 CDC growth charts.<sup>§</sup>

This study included approximately 12.1 million children aged 2–4 years from 43 PedNSS contributors, including 40 states, the District of Columbia, and two U.S. territories (U.S. Virgin Islands and Puerto Rico) that consistently reported data annually to PedNSS and used consistent methodology during 2008–2011. Height, weight, and BMI data that were missing ( $n = 258,310$ ; 2.1%), miscoded ( $n = 8,124$ ; 0.1%), or biologically implausible<sup>¶</sup> ( $n = 285,572$ ; 2.4%; e.g., height-for-age z-score  $< -5.0$  or  $> 3.0$ , weight-for-age z-score  $< -4.0$  or  $> 5.0$ , or BMI-for-age and sex z-score  $< -4.0$  or  $> 5.0$ ) were excluded from all analyses, leaving an analytic sample of 11,590,087 low-income preschool children. The average sample size among states/territories across all years of study ranged from 2,516 to 294,209.

The state-specific prevalence of obesity among low-income preschoolers during 2008–2011 was calculated. To examine trends in the state-specific obesity prevalence during the study period, logistic regression models were used, adjusted for age, sex, and race/ethnicity. Adjusted odds ratios (AORs) were calculated to determine the estimated annual change in odds of obesity. To determine whether changes in obesity prevalence could be explained by socioeconomic status of participants, a secondary analysis was conducted that included household income as part of the adjusted model. AORs were considered statistically significant at  $p < 0.05$ . Compared with the 2008 study population, the 2011 study population was older and included a slightly

smaller proportion of non-Hispanic whites and a slightly larger proportion of non-Hispanic blacks (Table 1).

## Results

During 2008–2011, a total of 19 states/territories reported significant downward trends in obesity prevalence among low-income preschoolers (Figure 1). Among them, the largest decline in obesity prevalence was in the U.S. Virgin Islands (AOR = 0.92; CI = 0.87–0.97), where there was a decrease in the prevalence of obesity from 13.6% in 2008 to 11.0% in 2011, an absolute decrease of 2.6 percentage points (Table 2). In five additional states (Florida, Georgia, Missouri, New Jersey, and South Dakota) the absolute decrease in obesity prevalence from 2008 to 2011 was  $\geq 1$  percentage point. Across the 19 states/territories with significant downward trends, the absolute decrease in obesity prevalence from 2008 to 2011 ranged from 0.3 to 2.6 percentage points. The relative decreases in obesity prevalence among the 19 states/territories ranged from 1.8% to 19.1%.

An additional 21 states/territories experienced no significant trend in obesity prevalence. Three states experienced a significant upward trend in obesity prevalence. The absolute increase in obesity prevalence from 2008 to 2011 for the three states ranged from 0.6 to 0.7 percentage points. The relative increase in obesity prevalence among the three states ranged from 5.2% to 6.4%.

In a secondary analysis of the 34 states/territories that had complete data on household income, household income was added to the logistic regression model. Among these states/territories, significance changed for only one state. Montana went from no significant trend to a significant decrease in the prevalence of obesity (AOR = 0.97, CI = 0.94–1.00).

In 2011, the prevalence of obesity among states/territories in the study ranged from 9.2% to 17.9% (Table 2). Ten states/territories had an obesity prevalence  $\geq 15\%$  (Figure 2), with the highest prevalence in Puerto Rico (17.9%). Six states/territories had an obesity prevalence  $< 12\%$ . The lowest obesity prevalence was in Hawaii (9.2%).

## Discussion

In recent years there have been small but significant decreases in childhood obesity. The finding that 19 states/territories experienced a decrease in obesity among low-income preschoolers, a vulnerable population, adds to recent findings from local data of low-income preschoolers (10,11) and studies observing decreases among children with higher socioeconomic status (SES) (12). The specific factors that might have contributed to the differential changes in obesity prevalence by state could not be readily identified. States likely differ by cultural and other factors that affect diet, activity, and weight as well as the

<sup>§</sup> Kuczumarski RJ, Ogden CL, Guo SS, et al. 2000 CDC growth charts for the United States: methods and development. *Vital Health Stat* 11 2002;(246). Available at [http://www.cdc.gov/nchs/data/series/sr\\_11/sr11\\_246.pdf](http://www.cdc.gov/nchs/data/series/sr_11/sr11_246.pdf).

<sup>¶</sup> Additional information available at <http://www.cdc.gov/nccdphp/dnpa/growthcharts/resources/biv-cutoffs.pdf>.



**TABLE 2. Estimated annual change in odds of obesity among low-income children aged 2–4 years, by state/territory — Pediatric Nutrition Surveillance System, United States, 2008–2011\***

State/Territory	2008		2009		2010		2011		Crude OR	(95% CI)	AOR <sup>†</sup>	(95% CI)
	No.	%	No.	%	No.	%	No.	%				
Alabama	56,813	13.8	60,572	14.4	65,914	14.1	67,246	14.1	1.01	(1.00–1.02)	0.99	(0.98–1.00)
Arkansas	38,591	13.9	42,270	14.2	32,615	14.1	42,455	14.2	1.01	(1.00–1.02)	1.01	(0.99–1.02)
Arizona	75,338	14.6	85,020	14.3	87,897	14.2	86,516	14.5	1.00	(0.99–1.00)	1.00	(0.99–1.01)
California	301,643	17.3	332,663	17.0	282,216	17.3	260,314	16.8	0.99	(0.99–1.00)	0.99 <sup>§</sup>	(0.99–1.00) <sup>§</sup>
Colorado	43,476	9.4	51,659	9.0	52,292	9.1	27,467	10.0	1.02	(1.00–1.03)	1.02 <sup>§</sup>	(1.00–1.04) <sup>§</sup>
Connecticut	25,623	15.5	28,432	16.0	28,401	15.8	27,561	15.8	1.00	(0.99–1.02)	1.00	(0.98–1.01)
District of Columbia	6,195	13.3	6,742	13.6	6,954	13.7	6,940	13.1	1.00	(0.96–1.03)	0.97	(0.94–1.00)
Florida	209,671	14.1	238,542	13.7	242,399	13.4	240,022	13.1	0.97	(0.97–0.98)	0.97 <sup>§</sup>	(0.96–0.97) <sup>§</sup>
Georgia	124,533	14.8	134,173	14.2	136,379	13.5	138,622	13.2	0.96	(0.95–0.96)	0.95 <sup>§</sup>	(0.95–0.96) <sup>§</sup>
Hawaii	16,106	9.3	17,252	9.3	17,827	9.1	17,819	9.2	0.99	(0.97–1.02)	0.99	(0.97–1.01)
Iowa	33,548	15.1	36,225	15.0	35,783	14.7	34,327	14.4	0.98	(0.97–0.99)	0.98 <sup>§</sup>	(0.97–0.99) <sup>§</sup>
Idaho	20,081	12.3	22,620	11.9	22,973	11.4	22,238	11.5	0.97	(0.95–0.99)	0.97 <sup>§</sup>	(0.95–0.99) <sup>§</sup>
Illinois	121,608	14.7	133,023	14.6	135,408	14.6	132,671	14.7	1.00	(0.99–1.01)	1.00	(0.99–1.01)
Indiana	66,499	14.5	75,671	14.3	78,634	14.2	73,247	14.3	1.00	(0.99–1.00)	0.99	(0.98–1.00)
Kansas	34,352	13.3	36,956	13.2	37,838	13.0	37,419	12.7	0.98	(0.97–1.00)	0.98 <sup>§</sup>	(0.96–0.99) <sup>§</sup>
Kentucky	62,832	15.7	68,450	15.8	75,189	15.6	33,008	15.5	0.99	(0.98–1.01)	0.99	(0.98–1.00)
Massachusetts	59,297	16.7	63,567	16.8	60,433	16.1	61,094	16.4	0.99	(0.98–1.00)	0.98 <sup>§</sup>	(0.97–0.99) <sup>§</sup>
Maryland	54,866	15.7	62,194	15.8	63,951	15.7	64,773	15.3	0.99	(0.98–1.00)	0.98 <sup>§</sup>	(0.97–0.99) <sup>§</sup>
Michigan	103,523	13.9	114,489	13.7	106,019	13.3	115,608	13.2	0.98	(0.97–0.99)	0.97 <sup>§</sup>	(0.97–0.98) <sup>§</sup>
Minnesota	65,607	13.4	68,997	13.1	68,594	12.7	70,353	12.6	0.98	(0.97–0.99)	0.97 <sup>§</sup>	(0.96–0.98) <sup>§</sup>
Missouri	60,908	13.9	60,150	13.9	67,547	13.6	67,650	12.9	0.97	(0.96–0.98)	0.97 <sup>§</sup>	(0.96–0.98) <sup>§</sup>
Mississippi	44,807	14.6	51,741	13.9	52,112	13.7	47,494	13.9	0.98	(0.97–0.99)	0.98 <sup>§</sup>	(0.96–0.99) <sup>§</sup>
Montana	10,428	12.4	10,105	12.5	8,958	12.2	10,681	11.7	0.98	(0.95–1.00)	0.98	(0.95–1.00)
North Carolina	96,381	15.7	104,323	15.2	105,392	15.5	103,565	15.4	1.00	(0.99–1.00)	0.99	(0.99–1.00)
North Dakota	6,551	13.8	6,968	14.1	6,836	14.1	6,665	13.1	0.98	(0.95–1.01)	0.99	(0.95–1.02)
Nebraska	20,658	13.9	20,811	14.2	22,194	13.8	22,136	14.3	1.01	(0.99–1.02)	1.00	(0.99–1.02)
New Hampshire	8,082	15.5	8,963	14.4	8,621	14.2	8,219	14.6	0.98	(0.95–1.00)	0.97 <sup>§</sup>	(0.94–0.99) <sup>§</sup>
New Jersey	68,163	17.9	75,191	18.4	78,181	17.3	77,476	16.6	0.97	(0.96–0.97)	0.96 <sup>§</sup>	(0.95–0.97) <sup>§</sup>
New Mexico	22,295	12.0	31,433	12.0	31,043	11.7	30,269	11.3	0.98	(0.96–0.99)	0.97 <sup>§</sup>	(0.96–0.99) <sup>§</sup>
Nevada	23,348	12.9	28,159	13.9	32,080	13.6	33,427	12.7	0.99	(0.97–1.00)	0.99	(0.98–1.01)
New York	209,713	14.6	224,130	14.4	224,243	14.5	229,291	14.3	1.00	(0.99–1.00)	0.99 <sup>§</sup>	(0.99–1.00) <sup>§</sup>
Ohio	125,011	12.2	130,792	12.3	128,754	12.4	121,624	12.4	1.01	(1.00–1.01)	1.00	(0.99–1.01)
Oregon	49,193	14.7	52,713	15.0	54,150	15.1	54,212	14.9	1.00	(0.99–1.02)	1.01	(1.00–1.02)
Pennsylvania	111,767	11.5	119,134	12.0	117,337	12.0	119,812	12.2	1.02	(1.01–1.03)	1.02 <sup>§</sup>	(1.01–1.03) <sup>§</sup>
Puerto Rico	99,828	17.9	100,313	18.1	89,438	18.3	89,463	17.9	1.00	(0.99–1.01)	1.00	(0.99–1.01)
Rhode Island	11,466	16.2	12,456	16.2	12,922	15.5	12,644	16.6	1.00	(0.98–1.02)	1.00	(0.98–1.02)
South Dakota	9,125	16.2	9,705	16.4	10,106	16.1	10,312	15.2	0.97	(0.95–1.00)	0.97 <sup>§</sup>	(0.95–1.00) <sup>§</sup>
Tennessee	69,015	13.5	71,914	14.0	71,349	14.5	69,276	14.2	1.02	(1.01–1.03)	1.02 <sup>§</sup>	(1.01–1.03) <sup>§</sup>
U.S. Virgin Islands	2,339	13.6	2,587	11.9	2,587	11.2	2,552	11.0	0.92	(0.88–0.98)	0.92 <sup>§</sup>	(0.87–0.97) <sup>§</sup>
Vermont	7,009	13.3	7,051	13.2	6,921	12.2	6,168	12.9	0.98	(0.95–1.01)	0.98	(0.95–1.01)
Washington	92,980	14.4	104,389	14.4	105,886	14.4	106,346	14.0	0.99	(0.98–1.00)	0.99 <sup>§</sup>	(0.98–1.00) <sup>§</sup>
Wisconsin	55,875	13.6	60,280	13.7	59,975	14.1	58,745	14.0	1.01	(1.00–1.02)	1.01	(1.00–1.02)
West Virginia	22,689	13.5	23,739	13.4	23,205	13.7	22,581	14.0	1.02	(1.00–1.03)	1.02	(1.00–1.03)

**Abbreviations:** OR = odds ratio; AOR = adjusted odds ratio; CI = confidence interval.

\* Includes 43 states and territories with consistent data during 2008–2011.

<sup>†</sup> Estimated annual change in odds of obesity, calculated from logistic regression models controlling for age, sex, and race/ethnicity.

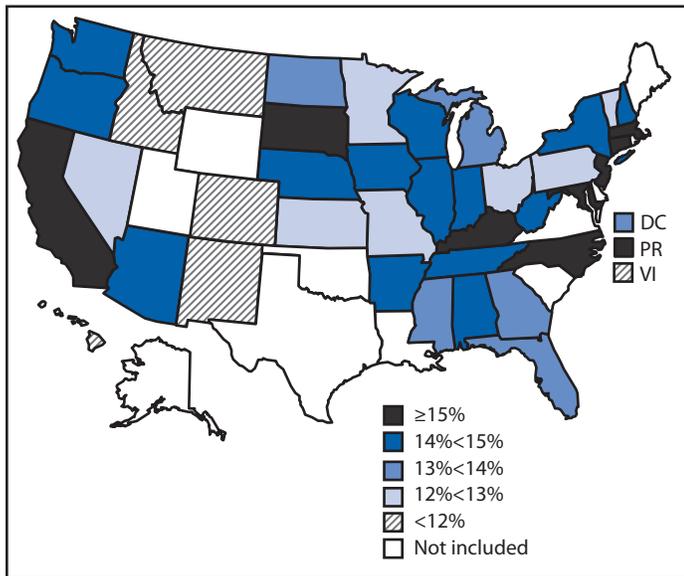
<sup>§</sup> Statistically significant at  $p < 0.05$ ; some 95% CIs include 1.00 because of rounding.

that officials can help communities prevent childhood obesity. State and local officials also can engage communities in obesity prevention by leading or participating in coalitions (e.g., an obesity task force or food advisory council), and partnerships with groups in other sectors (18) like agriculture, retail, transportation, and economic development.

The findings in this report are subject to at least three limitations. First, PedNSS is limited to low-income children who participated in federal nutrition programs. These findings might not reflect the obesity prevalence and trends among all low-income

U.S. preschool-aged children. In addition, the results might not be reflective of preschool-aged children of higher socioeconomic status who might have experienced more substantial declines in obesity prevalence (12). Second, this study included 43 states/territories that consistently collected PedNSS data during 2008–2011. Trends in other states/territories might differ. Finally, compared with 2008, the number of children in PedNSS was higher in subsequent years (approximately 2.7 million in 2008, 3.0 million in 2009, 3.0 million in 2010, and 2.9 million in 2011). This might have been caused, in part, by the economic downturn, which

FIGURE 2. Prevalence of obesity among low-income, preschool-aged children — Pediatric Nutrition Surveillance System, United States, 2011



\* Obesity defined as having an age- and sex-specific body mass index  $\geq$ 95th percentile, according to the 2000 CDC growth charts.

might have led to previously ineligible families becoming eligible for these nutrition programs. Whether or the degree to which these changes might have contributed to declines in obesity is unknown. As compared to the 2008 cohort, the 2011 cohort was slightly older and included a slightly smaller proportion of non-Hispanics whites and a slightly larger proportion of non-Hispanic blacks. Adjustments for these characteristics in logistical regression models might not have eliminated all confounding. Further adjustment for household income (for 34 states/territories with complete income data) however had little effect; one state went from no change to a significant decline.

Small decreases in the prevalence of obesity among low-income preschool children have been observed in certain states/territories. Continued prevention efforts remain necessary to ensure that this downward trend continues. State and local officials have an opportunity to lead these efforts through the continued development, implementation, and evaluation of obesity prevention initiatives and by leading partnerships with public and private sectors.

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#### Key Points

- The prevalence of obesity declined slightly among low-income preschoolers in 19 of 43 states and territories during 2008–2011.
- Obesity rates are high among preschool children in the United States. Approximately one child in eight aged 2–5 years is obese.
- Overweight or obese preschoolers are five times as likely to become overweight or obese adults, compared with their normal weight peers. In older children and adolescents, obesity is associated with high cholesterol, high blood sugar, asthma, and mental health problems.
- To continue the downward trend in obesity, continued communitywide action is needed. State and local officials can help prevent obesity in young children by 1) creating partnerships with community members such as civic leaders and child-care providers to make changes that promote healthy eating and active living; 2) making it easier for families with children to buy healthy, affordable foods and beverages in their neighborhood; and 3) providing opportunities for children to play safely through access to community parks and other recreation areas.
- Additional information is available at <http://www.cdc.gov/vitalsigns>.

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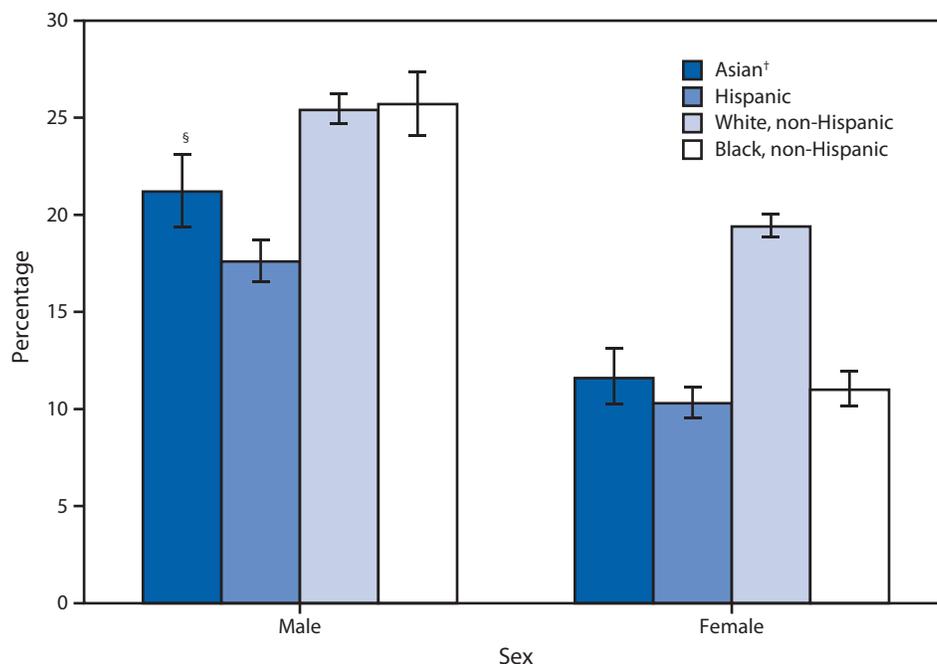
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## QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

## Percentage of Adults Aged $\geq 18$ Years Who Met the Aerobic Activity and Muscle Strengthening Guidelines,\* by Sex and Selected Race/Ethnicity — National Health Interview Survey, United States, 2009–2011



\* Respondents defined as meeting both aerobic and strengthening guidelines reported moderate physical activity for  $\geq 150$  minutes per week, vigorous physical activity for  $\geq 75$  minutes per week, or an equivalent combination of moderate and vigorous-intensity activity, and engaging in physical activities specifically designed to strengthen muscles at least twice per week.

† The Asian race group includes persons who identify themselves as members of one or more Asian race subgroups.

§ 95% confidence interval.

During 2009–2011, males were more likely than females to have met both the aerobic activity and muscle strengthening guidelines, regardless of race/ethnicity. Non-Hispanic white and non-Hispanic black males were more likely to have met the aerobic activity and muscle strengthening guidelines compared with Hispanic and Asian males. Non-Hispanic white females were more likely to have met both guidelines compared with non-Hispanic black, Hispanic, and Asian females.

Sources: CDC. National Health Interview Survey, 2009–2011.

CDC. Health Data Interactive. Available at <http://www.cdc.gov/nchs/hdi.htm>.

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