Rashes Among Schoolchildren—14 States, October 4, 2001–February 27, 2002

FOURTEEN STATES (ARIZONA, CONNECTICUT, NEW YORK, VIRGINIA, WASHINGTON, AND WEST VIRGINIA) have reported investigations of multiple schoolchildren who have developed rashes. This report summarizes the investigations by state and local health departments of these rashes, which have occurred during October 2001 through February 2002, and provides examples for four states. Preliminary findings indicate that further investigation is needed to determine whether a common etiology for these rashes exists.

United States

The first reported incident occurred October 4, 2001, in Indiana, followed by cases in Virginia that began November 20. Subsequent cases of rashes began in late January and occurred as recently as February 21. Rashes have been reported primarily from elementary schools but also among students in a few middle and high schools. The number of affected students in each state ranges from <10 to approximately 600. A few teachers and school staff have been affected, but rarely parents or siblings.

Characteristics of the rashes vary, but onset has generally been acute, typically with maculopapular erythematous lesions—possibly in a reticulated pattern—on the face, neck, hands, or arms; duration of the rash varied but in most reports it was highly pruritic. The rashes were not attributed to a defined environmental exposure or infectious agent. Children with rashes were afebrile and usually had no other associated signs or symptoms. The rashes lasted from a few hours to 2 weeks and appeared to be self-limiting. Secondary transmission has not been reported, but in-school “sympathy” cases have reportedly occurred. Diagnoses by clinicians who have examined children have included viral exanthem, contact or atopic dermatitis, eczema, chemical exposure, impetigo, and poison ivy. Approximately 40 serum samples collected in four states have been PCR or IgM negative for parvovirus B19; 22 nasal swab samples have been negative for enterovirus. Environmental assessments have not identified environmental causes.

Case Reports

Indiana. During October 4–November 2, 2001, rashes appeared among 18 third-grade students in an elementary school of 390 students; one substitute teacher also developed rash. No rashes among family members were reported. The rash most often began on the face, then spread to the upper extremities; most rashes occurred on exposed skin. Clinical signs—including reddish welt-type itchy rash on face and upper extremities, swollen eyes, and smooth pink cheeks—degrees of coloration, and prominence of rash varied among the children. Diagnoses in the few children examined by family physicians varied and included contact dermatitis, chemical exposure, impetigo, and poison ivy. Because parvovirus B19 infection was diagnosed in one third-grade student on August 30, 2001, the Indiana State Department of Health collected serum specimens from four students with rashes to assess whether they had parvovirus B19 infection. All specimens tested negative for the presence of IgM antibodies. Laboratory data analysis, interviews, a building survey, and examination of the children did not identify a cause for the rashes.

Pennsylvania. The initial report of rash occurred on January 31, 2002, among 54 elementary school students who had contact dermatitis diagnosed by a local health-care provider. To date, approximately 375 cases of rashes have been reported to the Pennsylvania Department of Health; 58 schools and child-care centers have reported cases (range: one-168 cases per facility). Most cases are in elementary and middle school students, with female cases outnumbering males. The rash has been characterized as bright-red, itchy or burning, and macular, occasionally with an urticarial or papular component. The rash may be evanescent, or remain for as long as 2 weeks; recurrent cases have been reported. There have been no other associated symptoms. Among the 54 students reported initially, serologies for parvovirus B19 were drawn on 13 cases; all were negative for IgM. PCR for parvovirus B19 was negative for 10 cases; results are pending for the remainder. Another health-care provider reported that results of nonserological (biopsy) specimens from his patients were consistent with viral exanthem. Environmental investigations at five schools have not yet identified an environmental source of the rashes. These investigations have included sampling for dust mite and cockroach allergens, solvents and cleaners, and fungal or bacterial culture growth. Air and surface cultures are still pending.

Oregon. During February 2002, outbreaks of rashes of acute onset and short duration occurred among students in two Oregon schools. Starting February 4, rashes were reported in 53 children and 11 adults in an elementary school of 589 students in southwestern Oregon; 54 (84%) were female. The rash, which appeared on cheeks and arms, was itchy and had a sunburned appearance but no systemic symptoms. A panel of dermatologists who examined 28 of the affected children reported that the rash resembled fifth disease but that several
characteristics were not compatible with that diagnosis. Testing for parvovirus in two children was negative. Extensive questioning and environmental inspection did not uncover a source of the rash. Beginning February 21, rashes were reported by 84 children and seven adults in a middle school of 314 students in northwestern Oregon; 67 (74%) affected persons were female. No known links existed between the two schools. Rashes were characterized in a variety of ways, including eczema, and as a sunburned, itchy rash on face, arms, neck, and back; no other symptoms were reported. Tests for parvovirus in six persons were negative. An environmental evaluation of the school found no explanation for the rash. In both schools, rash improved in several children when they left school but recurred when they returned to school.

Connecticut. On February 20, the Connecticut Department of Public Health was notified of nine elementary schoolchildren with rashes. On February 21, an additional 16 children were identified with a similar rash. The children, all fourth-graders, represented four classrooms in a school of 253 students and 12 classrooms. The acute rash appeared on the trunk and extremities and was characterized by erythema and pruritis. The children were alebrile and had no other symptoms. The illness lasted 24–72 hours. A dermatologist who examined three children attributed the rash to an allergic reaction to an environmental exposure. Rashes were not reported among parents or siblings of affected children. The local health director and the state Environmental Epidemiology Program are collaborating to identify potential environmental causes. The school was closed for 1 day to clean the classrooms, check air-handling units, and replace air filters.

Public Health Response

CDC is working with state and local health and education agencies in these investigations to determine if affected children within and between schools have developed rash as a result of a common etiology. CDC is systematically compiling information about (1) date of onset and duration of rash; (2) settings of and circumstances surrounding the rash’s appearance; (3) the number, age, and sex of affected persons; (4) the appearance and characteristics of the rash; (5) additional signs or symptoms, diagnoses, and treatments; and (6) investigational methods used (e.g., interviews or questionnaires, biologic sampling, and environmental sampling). To facilitate the collection of standardized information, CDC has developed and distributed to health departments a document with suggested approaches for investigating reports of rashes among groups of schoolchildren. In addition, CDC requests that dermatologists and other health-care providers who have examined affected children share their clinical observations, diagnoses, and photographs with a CDC dermatologist (bdtt1@cdc.gov). This information will help CDC assess whether affected children within and between schools developed rash caused by a common etiology. Local health and school officials with information about rashes among groups of schoolchildren in their jurisdiction are asked to report this information to their state health department.


CDC Editorial Note: With 53 million young people attending 117,000 schools each school day in the United States, it is expected that rashes from a wide range of causes will be observed. Environmental factors or infectious agents can cause rashes among groups of school-aged children. Rashes caused by infectious agents usually are preceded or accompanied by symptoms such as headache or fever. However, in these reports, none of the children showed signs of systemic illness, and the rash appeared to be self-limiting.

Potential environmental causes of rashes include biologic contaminants (e.g., bacteria and fungi), chemical agents (e.g., cleaning products and pesticide residues), physical agents (e.g., fiberglass), insects (e.g., biting flies and moths), and allergens (e.g., dust mites). If one of these environmental causes is suspected, appropriate environmental experts should be consulted.

The most commonly identified viral agent associated with rashes in school-aged children is parvovirus B19, which causes erythema infectiosum (i.e., fifth disease). Fifth disease is a mild rash illness characterized by a “slapped-cheek” rash on the face and a lacy red rash on the trunk and limbs, which may itch; it usually resolves within 7–10 days. Low-grade fever, malaise, or upper respiratory symptoms usually precede the rash. Other manifestations of parvovirus B19 infection include arthritis and arthralgia (especially in adults), transient crisis of aplastic anemia (in persons with certain hematologic disorders such as sickle-cell anemia), neutropenia, and thrombocytopenia. In pregnant women, parvovirus B19 infection may be associated with miscarriage or nonimmune hydrops fetalis.

Public health response to rashes of unknown etiology involves an epidemiologic investigation that includes consultation with facilities and maintenance staff familiar with the physical plant, examination of the rash by a dermatologist, and, when appropriate, collection and analysis of biologic specimens. To date, reports from states do not document a common cause or demonstrate that all children are experiencing the same rash. State and local health departments, in collaboration with CDC, continue to investigate these and other reports of rashes among groups of schoolchildren.

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2 figures, 1 table omitted

In 1998, 53% of all cancer-related deaths in the United States were associated with four sites: lung/bronchus, colon/rectum, prostate, and female breast.1 Cancer-related death does not affect racial/ethnic populations similarly. In 1996, the National Cancer Institute (NCI) published cancer incidence and death rates during 1988-1992 in 10 categories of race/ethnicity.2 To examine trends during 1990-1998 in annual death rates for the four major cancers by sex and race/ethnicity (i.e., blacks, whites, Hispanics, American Indians/Alaska Natives [AI/ANs], and Asians/Pacific Islanders [APIs]), CDC analyzed data from the National Center for Health Statistics’ National Vital Statistics System.3 This report summarizes the results of that analysis, which indicated that, except for lung cancer in women and lung, colorectal, and breast cancer in AI/ANs, trends in death rates from these cancers have generally declined. But the rates remained high for blacks, have not decreased equally among all populations, and have increased in certain instances. Continuing research and prevention efforts are needed to reach high-risk and underserved populations and to understand the reasons for differences in cancer mortality among racial/ethnic populations.

In each state, attending physicians report and file causes of death on death certificates, which then are consolidated into the National Vital Statistics System. For this analysis, the International Classification of Diseases, Ninth Revision, (ICD-9) codes4 for cause of death from these cancers and NCI software were used to compute death rates as the number of deaths per 100,000 population, age-adjusted to the 1970 U.S. population by using 5-year age intervals. Deaths and death rates were presented for 1990-1998 for whites, blacks, Hispanics, AI/ANs, and APIs. To test for significant trends in death rates during 1990-1998, linear regression was used to estimate the annual percentage change for this period.

Data from 1998 indicated that death rates for lung and bronchus cancer were higher for blacks and whites than for other races/ethnicities. Death rates for black men were higher than for white men. Among men, death rates from lung and bronchus cancer decreased 1% to 2% per year for each race/ethnicity except AI/ANs. Among AI/ANs, death rates increased 1.7% per year among men and 2.9% per year among women. Death rates also increased for white and black women.

For 1998, death rates for colorectal cancer for each race/ethnicity were approximately 40% higher among men than women. Blacks had the highest death rate for colorectal cancer, followed by whites. Death rates for colorectal cancer decreased 2.2% per year for white men and 1.8% per year for white women. Declines in death rates among black men and women were approximately 50% less than that for whites. Death rates for colorectal cancer increased 4.5% per year for AI/AN men, although the increase was not significant because of year-to-year variations in rates.

Death rates for prostate cancer were more than twice as high for blacks than
for whites (Table 1). Rates were lowest among APIs. Death rates decreased for men of each race/ethnicity except AI/ANs. The declines in death rates for whites (2.8% per year) and APIs (3.4% per year) were approximately twice the decreases for blacks, Hispanics, and AI/ANs.

Female breast cancer death rates were highest for blacks, followed by whites, Hispanics, AI/ANs, and APIs. During 1990-1998, breast cancer-related death rates decreased for white (2.5% per year) and Hispanic (1.2% per year) women and were unchanged for black, AI/AN, and API women.

**CDC Editorial Note:** The findings in this report indicate that death rates have declined for lung and bronchus, colorectal, prostate, and female breast cancers among most racial/ethnic populations; however, death rates remained high for certain cancers among blacks and are generally increasing among AI/ANs. Trends in cancer death rates might reflect changes in cancer risk behaviors, new screening modalities, and the development and use of new and more effective treatments.

Lung and bronchus cancer was the most important cause of cancer mortality in the 1990s, accounting for approximately 28% of all cancer-related deaths. Approximately 90% of these deaths have been attributed to smoking.

Changes in death rates reflect substantial decreases in smoking during 1965-1985 among men (51.9% to 32.6%) and approximately 28% of all cancer-related deaths.5

The reporting of race/ethnicity to the U.S. Bureau of the Census and on death certificates usually is reliable for blacks and whites6; however, underreporting for other races/ethnicities can underestimate death rates from 2% among Hispanics to 21% among AI/ANs. Second, because this study was a description of national trends and not an evaluation of cancer intervention studies, the findings should be interpreted with caution.

One of the goals of the national health objectives for 2010 is to eliminate health disparities among racial/ethnic populations. CDC supports several initiatives that address the four major cancers: national tobacco-control efforts; *Screen for Life*, a multimedia campaign promoting prevention and early detection of colorectal cancer; and Racial and Ethnic Approaches to Community Health (REACH 2010). CDC also funds research on the high death rates from prostate cancer among blacks and the National Breast and Cervical Cancer Early Detection Program, which provides screening to underserved women. Additional information about CDC’s cancer prevention and control programs is available at http://www.cdc.gov/tobacco; http://www.cdc.gov/cancer; and http://www.cdc.gov/cancer/minorityawareness.htm.

Differences in cancer death rates result from a combination of factors such as behaviors (e.g., smoking and nutrition); access to preventive, diagnostic, therapeutic, and screening services; and aggressiveness of treatment. If these factors were modified, more than half of the cancer deaths could be prevented andmost racial/ethnic disparities in cancer death rates could be eliminated.

**REFERENCES**


*Codes: lung and bronchus 162.2-162.5, 162.8-162.9; colon and rectum 153, 154.0-154.1, 159.0; prostate 185; breast 174.