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Community-Associated MRSA: Disparities and Implications for AI/AN Communities

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Summary

Infection caused by community-associated methicillin-resistant Staphylococcus aureus is a recently-emerged epidemic disproportionately borne by AI/AN communities in the US. This review traces the roots of this epidemic, and highlights relevant clinical features so that IHS providers may be better equipped to respond to this evolving threat.

An Old Foe Develops New Fangs¹: Resistance and Virulence

With the selective pressures introduced by the increasing use of penicillin in the 1940s, resistance to β -lactam antibiotics began to emerge in Staphylococcus aureus strains. Methicillin resistant Staphylococcus aureus (MRSA) was first reported in the early 1960s,² and by the 1980s had become a common nosocomial pathogen.³ Since the 1999 publication of a report of four deaths of pediatric patients that resulted from MRSA infection,⁴ including an American Indian girl in North Dakota, growing attention became focused on the role of MRSA as a community-associated pathogen. Increasingly, otherwise healthy individuals living in the community with no identifiable risk factors for nosocomial pathogens were being reported as having MRSA infections. These infections became categorized as community-associated MRSA (CA-MRSA) infections, and the Centers for Disease Control and prevention (CDC) developed criteria to distinguish CA-MRSA infections from the health care-associated type (HA-MRSA).⁵

A pathogen that represents a unique convergence of resistance and virulence,⁶ CA-MRSA emerged de novo in the community, exhibiting genetic origins and a phenotype distinct from those of HA-MRSA⁷ (Table 1). CA-MRSA primarily causes skin and soft tissue infections (SSTI; prevalence, 77%); it can also cause traumatic wound infection (prevalence, 10%), urinary tract infection, sinusitis, bacteremia, and pneumonia (prevalence, <5% each).³ The gene encoding the Panton-Valentine Leukocidin (PVL) toxin has been implicated in its virulence, although controversy exists over whether PVL is necessary for pathogenesis

or simply a marker for other virulence factors.⁸ The enhanced virulence of CA-MRSA compared with HA-MRSA is demonstrated by more severe skin infections⁹ and association with poor patient outcomes and serious complications.^{10,11}

Table 1. Characteristics of healthcare-associated and community-associated MRSA Strains^{6,35,41}

	Healthcare-associated (HA-MRSA)	Community-associated (CA-MRSA)
Genotype (SCC ^{mec} type)	I, II, or III	IV
Common strains	USA 100 USA 200	USA 300 USA 400
Usual Antibiotic susceptibility		
Clindamycin	R	S ^c
TMP-SMZ ^b	S	S
Fluoroquinolones	R	Geographic variability
Erythromycin	R	R
Tetracyclines	R	S
Glycopeptides	S	S
Panton-Valentine Leukocidin (PVL) exotoxin	Uncommon	Common

^a Staphylococcal cassette chromosome mec

^b Trimethoprim/sulfamethoxazole

^c Inducible clindamycin resistance has been documented

PVL-producing CA-MRSA strains have a high transmission and clinical attack rate,¹² not only within families, but also on a larger scale in community settings (e.g., prisons, schools, sport teams) and among certain high risk groups (Table 2). Primary modes of transmission include skin-to-skin contact (including

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unabraded skin) and indirect contact with contaminated shared objects (e.g., towels, sheets, sports equipment). Where there are shared contaminated items, poor hygiene, and crowded living conditions, transmission appears more likely.¹³ Of concern is the observation that these strains are now showing the propensity to not only spread through the community, but also into hospitals, undermining efforts at infection control in these settings.^{13,14,17}

Table 2. Risk Factors for CA-MRSA infection^{21,23,34}

- High local prevalence
- Personal history of MRSA infection or colonization
- Report of a "spider bite"
- Close contact with infected individual
- Young age
- Crowded and/or unsanitary conditions
- Immunocompromised
- Participation in contact sports/ sharing athletic equipment or towels
- Intravenous drug abuse
- Men who have sex with men

Further, while most CA-MRSA infections involve the skin and soft tissue, severe and sometimes fatal infections have been observed even in healthy patients, including sepsis, necrotizing pneumonia, purpura fulminans, pyomyositis and necrotizing fasciitis.^{15,16} A recent study of invasive MRSA infections in 2005 found that almost 95,000 patients in the US developed an invasive infection (31.8 per 100,000), and nearly one in five died (standard mortality, 6.3 per 100,000).¹⁷

The etiologic role of CA-MRSA in post-influenza community-acquired pneumonia is of growing concern in light of preparations for the next influenza pandemic.³ During the 2003 - 2004 US influenza season, 15 of 17 cases of community-acquired staphylococcal pneumonia that were reported to the CDC were caused by MRSA, and death occurred in 5 of the patients (4 with MRSA).¹⁸

CA-MRSA Goes Global

Outbreaks of CA-MRSA infections have been described in numerous communities throughout North America^{19,20} and the world.²¹ Multiple individual hospitals have reported increased occurrence of CA-MRSA infections. One hospital emergency department reported that among patients presenting with SSTI, the prevalence of MRSA isolates increased from 29% in 2001 - 2002 to 64% in 2003 - 2004.²² A prospective population-based surveillance found that 8 to 20 percent of all MRSA isolates were CA-MRSA, with incidence varying geographically and between ethnicities.²³

An Epidemic Among AI/AN Communities

While the prevalence of CA-MRSA varies according to geography and ethnicity,²³ high rates of CA-MRSA have been observed among American Indian/Alaska Native (AI/AN) communities since its first description as a unique pathogen.

Beginning in the 1980s and 1990s, reports surfaced of methicillin-resistant strains of *S. aureus* present in indigenous

communities in Canada,²⁴ Australia,²⁵ and Alaska²⁶; and a 1996 national survey of IHS facilities found that already 40% (600/1490) of *S. aureus* isolates tested from the Midwest and Northern Plains were MRSA.²⁷ As early as 2000, it was reported that CA-MRSA was an issue disparately represented among AI/AN, and that at some rural clinics serving AI/AN, over 60% of *S. aureus* isolates were methicillin-resistant.²⁸

Subsequently, numerous studies have documented the disproportionate burden of CA-MRSA among AI/AN communities.²⁹⁻³³ A seminal paper published in 2001 documenting work in a rural midwestern IHS clinic reported the spread of MRSA beyond the nosocomial setting, first suggesting that CA-MRSA was replacing community-acquired methicillin-sensitive *S. aureus* as the dominant strain in the community.²⁷ A study of Minnesota health facilities examining MRSA infections during 1996 - 1998 found disproportionately high rates of CA-MRSA among AI/AN patients, with Native Americans comprising 40% of the cases, whites 21%, and blacks 18%.³⁰ A study of a large outbreak of community-onset MRSA infections among AN in southwestern Alaska determined that most (77%) of the patients with MRSA skin infections had community-acquired MRSA.³¹ A study of the prevalence of and risk factors for CA-MRSA nasal carriage in AI patients of a rural IHS clinic in Washington found prevalence of CA-MRSA colonization to be approximately 1%; colonization was associated with recent antimicrobial use and larger household.³²

Increased prevalence of CA-MRSA among AI/AN communities likely reflects the ontogeny of the pathogen, as socioeconomic and demographic factors present in AI/AN communities may have provided necessary selective pressure to foster the initial emergence of CA-MRSA. Studies in Wisconsin demonstrated that CA-MRSA was found to have emerged largely in Native American communities. During 1989 - 1999, 581 MRSA isolates were collected, 17.2% of which came from patients who were treated at five Native American clinics.³³ Subsequent molecular characterization of the CA-MRSA strains suggested that CA-MRSA in Wisconsin likely first originated in Native American communities in the early 1990s and subsequently became widespread throughout the state.³⁴

As they have chronicled this important health disparity among AI/AN communities, multiple IHS-affiliated researchers have significantly advanced scientific understanding of the evolving CA-MRSA epidemic. Among others, particularly noteworthy contributions have been made by Drs. Jim Cheek, Tim Naimi, Amy Groom, and Richard Leman.

Clinical Implications for Providers

The emergence of MRSA in the community has several significant therapeutic implications for clinical providers, particularly for those working in AI/AN communities (Table 3). First, CA-MRSA should figure prominently in the differential diagnosis of all SSTIs. A presenting chief complaint of "spider bite" should heighten one's suspicion of *S. aureus* infection.³⁵ Given the high documented prevalence rates of CA-MRSA among

AI/AN communities, a reasonable approach to complicated community-acquired infections may be to assume the presence of CA-MRSA, unless evidence suggests the local prevalence to be particularly low.³⁶ If antibiotics are indicated (see below), appropriate empiric choices for SSTI treatment should therefore include trimethoprim-sulfamethoxazole and clindamycin.

Table 3. Clinical implications for providers

<ol style="list-style-type: none"> 1. Be aware of local prevalence of CA-MRSA <ul style="list-style-type: none"> • Include CA-MRSA in your in your differential diagnosis, especially with "spider bites"! 2. Always obtain material for culture whenever possible <ul style="list-style-type: none"> • Tailor antibiotic choices accordingly 3. Surgical incision and drainage is the priority intervention <ul style="list-style-type: none"> • "never let the sun set on an undrained abscess..." 4. With severe infections, include vancomycin for CA-MRSA coverage <ul style="list-style-type: none"> • Sepsis syndrome, osteomyelitis, septic arthritis, severe pneumonia, necrotizing fasciitis, purpura fulminans 5. Community awareness and broad-based infection control measures are key! <ul style="list-style-type: none"> • Reduce unnecessary antibiotics, improve community surveillance

Second, in light of evolving antibiotic resistance patterns among CA-MRSA strains,³⁷ specimens should be collected and cultured whenever possible so that results of culture and sensitivity testing can appropriately guide treatment regimens. Importantly, if the isolate is resistant to erythromycin but susceptible to clindamycin, the clindamycin D-zone test should be performed if clindamycin therapy is being considered.³⁸

Third, surgical drainage and debulking of SSTIs should be considered the priority intervention.³⁸ If unconvinced by the surgical aphorism, "never let the sun set on an undrained abscess," consider some recent evidence. In a randomized trial of cephalexin for treatment of uncomplicated skin abscesses in a population at risk for CA-MRSA, the 90.5% cure rate observed in the placebo arm (84.1% cure rate, cephalexin arm) suggests that antibiotics may be unnecessary after surgical drainage of uncomplicated SSTIs caused by CA-MRSA.³⁹ The observation that after adequate surgical drainage, SSTIs severe enough to warrant hospitalization resolved regardless of whether the antimicrobial agent given to the patient had in vitro activity⁴⁰ is further reminder of the primacy of this intervention.

Fourth, CA-MRSA should be included in the differential diagnosis for patients with clinical signs of serious infection (including sepsis syndrome, osteomyelitis, septic arthritis, pneumonia that is severe or follows an influenza-like illness, necrotizing fasciitis, and purpura fulminans), and these patients should receive aggressive therapy including empiric coverage for CA-MRSA with vancomycin. Unfortunately reports of treatment failure associated with vancomycin have become increasingly common;⁴¹ for now, alternate agents do exist, if necessary, including linezolid, daptomycin, and quinupristin-dalfopristin,⁴² each of which has shown clinical efficacy in CA-MRSA therapy.

Finally, containing the CA-MRSA epidemic in our communities will require much more than appropriate antibiotics. Efforts to raise awareness among community members and health care personnel, to reduce unnecessary antibiotic use, improve community surveillance, and to bolster infection control measures will be required to help mitigate the effect of this evolving

pathogen in our midst.

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Low Prevalence of Asthma Among American Indian Youth in Southeastern Montana

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Abstract

Objectives: To highlight the variability in asthma prevalence among American Indian children, the authors determined the period prevalence of current asthma in youth accessing care at the Crow Service Unit (CSU) in southeastern Montana from 1987-2006.

Methods: The authors performed retrospective electronic and manual chart review to identify patients aged 0 - 20 years, stratified by age and gender, with a diagnosis of asthma who had at least one clinic visit during the preceding three calendar years.

Results: Prevalence of diagnosed asthma among youth aged 0 - 20 years at the CSU was low, and increased during 1987 - 2006 from an age-adjusted 2.8 per 100 to 5.1 per 100, by an average of 3.5% per year. Age adjusted prevalence of diagnosed asthma was higher in males than females, and decreased with age for both sexes.

Conclusion: Contrary to published data, the asthma prevalence rate in our population is lower than previously cited rates for all races and for AI/AN populations, likely due to local environmental factors. Future research should include focused analysis of elements of the social and environmental microclimate to determine which factors predispose and protect against the development of asthma in our population.

Introduction

Asthma is the most common chronic disease of childhood,¹⁻⁷ afflicting approximately 6.2 million American children in 2004,^{1,2} and posing significant burdens of morbidity, mortality and economic costs worldwide.^{1-3,7} Myriad studies describing disparities in disease prevalence among different populations highlight variable contributions of environmental, social, and innate (i.e., genetic and biologic) factors to the distribution of this disease.⁸⁻¹³

Recent years have witnessed marked progress in describing the prevalence and severity of asthma among American Indian and Alaska Native (AI/AN) children,^{14,16-21} and while early reports noted asthma among AI/AN groups to be rare,^{22,23} more recent studies suggest much higher prevalences.^{14-21,39} However, there is broad social and biological

heterogeneity among the ethnic category of AI/AN.^{24,25} Given the marked regional differences in local factors that may play a significant etiological role in asthma prevalence within a given AI/AN subgroup (i.e., tribe, band, village, urban), there is a need for ongoing epidemiological investigation within these subgroups. A recent study of asthma hospitalization rates for the AI/AN population revealed that there were wide regional variations in the hospitalization rates in the AI/AN population.²¹ Understanding population and regional variances in the prevalence of asthma is important for optimal design of local interventional strategies as well as for elucidating important insights into the social and biologic determinants of asthma.

Located in rural southeastern Montana, the Crow Service Unit is a subset of the Indian Health Service (IHS) comprising one hospital and two satellite clinics whose estimated user population in 2006 was approximately 13,936.²⁶ Children receiving care at these facilities are predominantly enrolled members of the Crow and Northern Cheyenne tribes, although 20% of pediatric patients seen in 2006 were from a variety of other tribes. The Apsáalooke (Crow) reservation is located in southeastern Montana and had a 2000 census AI population of 6890, 44% of whom were under 21 years of age. The Northern Cheyenne reservation (home of the Tsitsistas and So'taa'eo'o People), which is immediately adjacent to the east of the Crow reservation, had a 2000 census AI population of 4,470 persons, of whom 48% were under 21 years of age. The two reservations span a collective area of 4300 square miles.

Multiple factors that may play a significant role in asthma epidemiology are common among the two reservations, including low income levels,^{26,28,31} obesity,^{27,33} poor housing condition,^{29,30} and high rates of tobacco smoking.³² Conversely, several protective factors are also locally prevalent: point sources of concentrated industrial air pollution are relatively few in number over a wide geographic distribution^{34,35}; children have ready access to health care through the IHS system³⁶; and most children live in families with multiple siblings.²⁸

This study seeks to define the prevalence of asthma among the children receiving healthcare at the Crow Service Unit over the past 20 years.

Methods

We sought to establish the prevalence rate of current asthma among children age 20 or younger receiving care at the Crow Service Unit during the years 1987 - 2006. Permission

for the study was obtained from the Crow Tribal Chairman, the Crow Tribal Health Department; the Institutional Review Board, Billings Area Indian Health Service; and the Institutional Review Board, University of Washington, Seattle.

We defined current asthma as a visit diagnosis of asthma (International Classification of Diseases, Ninth Revision, Clinical Modification, code 493) during the past 12 months. We analyzed data from an electronic health record system (Resource and Patient Management System, RPMS), a database that includes unduplicated case reports from patients who visited the service unit one or more times during each of the years studied. Using an approach employed in previous studies using RPMS,³⁷ we calculated prevalence using the AI/AN population age <21 years that received health care services at the Crow Service Unit at least once during the preceding three years. We used these overall population data and the number of persons age <21 years identified in RPMS as persons with diagnosed asthma to estimate the age-specific prevalence of diagnosed diabetes among children in four age groups: 0 - 4, 5 - 10, 11 - 15, and 16 - 20 years. We age adjusted prevalence by the direct method, on the basis of the 2000 US standard population, and modeled average annual percentage changes (APCs) using regression analysis³⁸ (Joinpoint Regression Program, National Cancer Institute).

Results

Among all youth age <21 years receiving care at the Crow Service Unit, prevalence of diagnosed asthma during 1987 - 2006 increased from an age-adjusted 2.8 per 100 to 5.1 per 100, increasing by an average of 3.5% per year (Table). Prevalence of diagnosed asthma decreased with age, and in 2006, ranged from 6.0 per 100 population among youth aged 0 - 4 years to 2.8 per 100 population among those aged 16 - 20 years. In 2006, the age-adjusted prevalence of diagnosed asthma was 5.6 per 100 among males and 4.7 per 100 among females (Table).

During 1987 - 2006, prevalence of diagnosed asthma was greater among males than females in age groups 0 - 4 years and 5 - 10 years (Figure). Gender differences in prevalence rates were not present among age groups 11 - 15 and 16 - 20 years.

During 1987 - 2006, prevalence of diagnosed asthma increased steadily for both sexes and in all age groups, with the exception of males and females age 0 - 4 years (Figure). Among males and females in this age group, there was a non-significant trend towards decreased prevalence (Table). Among all age groups, females aged 11 - 15 years had the highest APC (9.4%).

Discussion

Our study demonstrates that the prevalence of diagnosed asthma at the Crow Service Unit among youth age <21 years has been increasing over the past two decades, paralleling a similar nationwide trend.^{1,3} Our results corroborate national trends in gender distribution of asthma as well.^{1,3,21,40} In the

Table. Prevalence* and annual percentage change (APC) of diagnosed asthma during the previous 12 months among children aged <21 years, by sex and age group--Crow Service Unit, 1987-2006

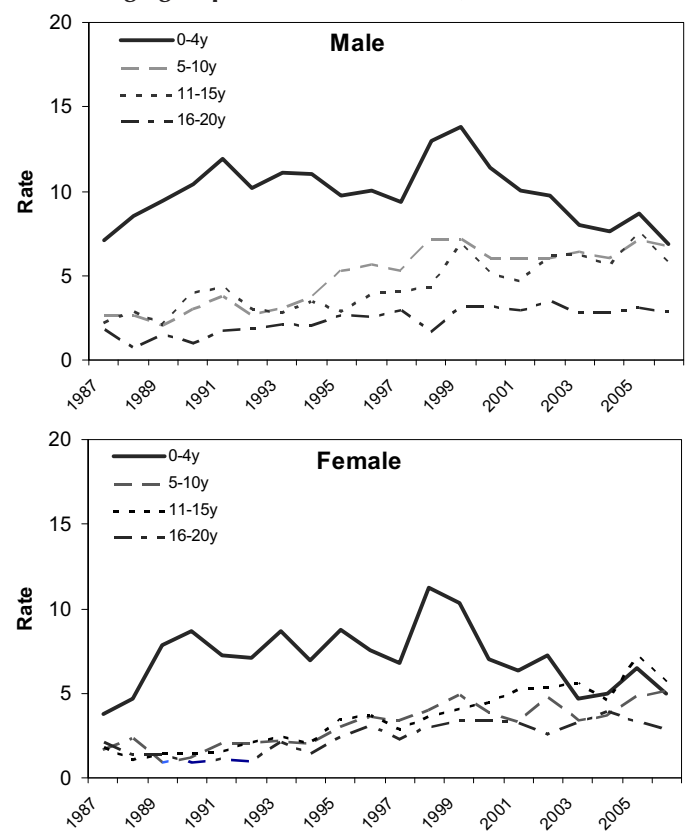
Age Group (yrs)	Rate		Trend	
	1987	2006	APC	(95% CI [†])
Male				
0-4y	7.1	6.8	-0.5	(-2.1, 1.0)
5-10y	2.6	6.7	6.4	(4.7, 8.2)
11-15y	2.2	5.6	5.6	(3.9, 7.3)
16-20y	1.8	2.9	5.7	(3.3, 8.1)
<21	3.8	5.6	2.2	(1.1, 3.3)
<21 [‡]	3.4	5.6	3.1	(1.9, 4.2)
Female				
0-4y	3.8	5.0	-0.3	(-2.6, 2.0)
5-10y	1.7	5.1	7.1	(4.8, 9.4)
11-15y	1.8	5.6	9.4	(7.8, 11.1)
16-20y	2.1	2.8	6.4	(3.8, 9.0)
<21	2.4	4.6	3.4	(2.2, 4.6)
<21 [‡]	2.3	4.7	4.1	(2.9, 5.4)
Both sexes				
0-4y	5.5	6.0	-0.5	(-2.2, 1.3)
5-10y	2.2	5.9	6.6	(4.8, 8.3)
11-15y	2.0	5.6	7.0	(5.9, 8.1)
16-20y	2.0	2.8	5.9	(3.9, 7.9)
<21	3.1	5.1	2.7	(1.6, 3.8)
<21 [‡]	2.8	5.1	3.5	(2.4, 4.6)

* Per 100 population in age group

[†] Confidence interval

[‡] Age adjusted to the 2000 US standard population

Figure. Prevalence* of diagnosed asthma during the previous 12 months among children aged < 21 years, by sex and age group--Crow Service Unit, 1987-2006.



* per 100 population in age group

younger age groups, males had higher rates of asthma than their female counterparts, though this effect decreased with age. However, contrary to previously-reported studies, the overall prevalence rate in our population is lower than most previously cited rates,^{1,3,14-17,19,20} both for all races and for AI/AN populations. Further, also distinct from published data,^{1,3} we found a decreasing prevalence of asthma with age for both males and females.

Our data indicate an average annual asthma prevalence during the previous 12 months among children aged <21 years of 5.1% for 2006. Comparable investigations, although involving different geographic areas and different tribes, report significantly higher prevalence rates for AI/AN children. Examining data from the National Health Interview Survey, Akinbami¹ reported nationwide current asthma prevalence (2004 - 2005) for all races of 8.7%, with a rate of 9.9% for AI/AN children. A survey of children in three non-IHS community health centers in Montana³⁹ noted an asthma prevalence (2000 - 2002) of 5.0% for all races, with a rate of 12.1% for AI/AN children. A review of patient encounters for children age 18 and under at the Fort Peck IHS Service Unit in northeastern Montana¹⁹ demonstrated a prevalence rate of 15.5% during the period 1996 - 1999. Conversely, a study of pediatric AI/AN patients age less than 21 years at an IHS site in North Dakota¹⁸ found a prevalence rate of 3.6% during 2001 - 2002.

There are several possible explanations for our apparently discordant results. First, we note that the marked biological, social, and environmental heterogeneity among and between various AI/AN groups would be expected to result in a broad distribution of asthma prevalence, and that studies of individual subgroups may yield divergent results. Recognizing that race/ethnicity is a social, not a biological, category,⁴³⁻⁴⁵ we submit that the prevailing social and ecological microclimate would be most relevant in determining the local asthma prevalence. In our population, where there is a high degree of homogeneity in socioeconomic position,^{26,28,31} history and culture, the social determinants of health, while not completely uniform, are relatively constant.

Likewise, the outdoor ambient air quality, which is low in particulate matter and other air pollutants, is similar across the geographic area of our study.^{34,35} Anecdotal reports suggest that indoor air quality is low, and that exposure rates to indoor allergens linked to development of asthma are high (CSU, unpublished data, 1996). Previous studies suggest that exposure to indoor air pollutants may have a more important effect on the development of childhood asthma and on promoting asthma exacerbations than may exposure to outdoor air pollutants.⁴⁶ One possibility, then, for the inverse relationship between prevalence and age is that excessive indoor allergen exposure promotes high rates of asthma exacerbations in the youngest children. Older children, who are more mobile and spend more time in higher quality

ambient outdoor air, may have less frequent asthma exacerbations; it is possible that those with quiescent asthma would present less often to the clinic between asthma flares, giving the appearance of diminishing prevalence with age. A similar decreasing rate of asthma hospitalizations with age was also demonstrated²¹ among AI/AN children under 19 years old, with the highest hospitalization rates found among children ages 1 to 4 years.

The relatively low overall rate of pediatric asthma in our study may reflect the protective effect of more communal living styles observed in our community. Housing shortages in our study area result in conditions of widespread overcrowding in households. Previous studies indicate that higher levels of household crowding mitigate against the development of asthma, likely via timing and mode of endotoxin exposure.⁴² Similarly, larger family size has also been identified as correlating with lower rates of asthma.⁴⁷

Potential confounders in our study include reliance on physician diagnosis for case ascertainment of asthma. The high prevalence observed in young children may represent overdiagnosis, including diagnostic substitution of asthma in place of bronchiolitis or recurring wheezing after bronchiolitis within the first year of life. Conversely, low rates in older children may be due to underdiagnosis. Evidence exists to suggest that AI/AN populations are at risk for underdiagnosis of asthma.⁴¹ However, given the consistency of our data over a 20-year period of analysis, during which time multiple physicians comprised the diagnosing provider, these biases are less likely. The possible confounding role of diagnostic transfer cannot be elucidated by our data.

Areas for future research include focused analysis of elements of the social and environmental microclimate to determine which factors predispose and protect against the development of asthma in our population. In particular, elucidating the contribution of type and timing of exposures to specific indoor allergens, in addition to evaluating factors such as low birth weight/gestational age, BMI, breast-feeding, and antecedent viral infections would be useful to guide strategies of asthma prevention and health promotion.

Acknowledgments

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This is a page for sharing “what works” as seen in the published literature, as well as what is being done at sites that care for American Indian/Alaskan Native children. If you have any suggestions, comments, or questions, please contact Steve Holve, MD, Chief Clinical Consultant in Pediatrics at sholve@tcimc.ihs.gov.

IHS Child Health Notes

Quote of the month

“Logic is an organized procedure for going wrong with confidence and certainty.”

C. F. Kettering

Articles of Interest

Stephen S. Hall. Small and Thin: The controversy over the fetal origins of adult health. *The New Yorker*, November 19, 2007.

http://www.newyorker.com/reporting/2007/11/19/071119fa_fact_hall.

Weight in infancy and death from ischemic heart disease. *Lancet*. 1989 Sep 9;2(8663):577-80.

Mother’s weight in pregnancy and coronary heart disease in a cohort of Finnish men: follow up study. *BMJ*. 1997 Oct 4;315(7112):837-40.

Trajectories of growth among children who have coronary events as adults. *N Engl J Med*. 2005 Oct 27;353(17):1802-9.

The belief that maternal health in pregnancy can have life-long effect on chronic illnesses in their offspring has gone from heresy to orthodoxy in the past 20 years. The driving force behind this theory is David Barker, who was featured recently in an article in *The New Yorker*. Dr. Barker’s original hypothesis was based on population studies in England. It was confirmed by similar work in Finland. This epidemiologic work has found correlates in the new field of epigenetics, which postulates that environmental factors can produce permanent changes in the activity of genes. Birth effects are not predestination but they do have measurable effects over a population. Dr. Barker argues we need to put more emphasis on maternal nutrition as a cost effective intervention for future health.

Editorial Note

What do skinny children born to skinny, poorly nourished mothers in England in the early 20th century have to do with AI/AN children today? Studies have shown that the children at greatest risk for coronary heart disease as adults are not fat babies: it is thin babies who gain unusual amounts of weight after birth. This perfectly describes infants born 20 to 40 years ago as many AI/AN populations went in one generation from

under nutrition to over nutrition. We now have an epidemic of diabetes and coronary heart disease in young adults. It is unclear what will be seen 20 years from now in the current group of large for gestational infants being born to overweight mothers. We can only be sure that there will be epigenetic effects.

Infectious Disease Updates.

Rosalyn Singleton, MD, MPH

2008 Childhood Immunization Schedule: Few Changes Planned.

There were numerous changes to the Childhood (0 - 6 year) and Adolescent Immunization Schedules in 2007. Few additional footnote changes are proposed for the 2008 schedule:

1. Hep B. “If mother is HBsAg-negative, the birth dose can be delayed in rare cases with providers order and copy of mother’s negative HBsAg lab report in infant’s medical record.”
2. Pneumococcal vaccine. “At ages 24 - 59 months, administer 1 dose of PCV to incompletely vaccinated healthy children and 2 doses of PCV at least 8 weeks apart to incompletely vaccinated children with certain high risk conditions. Administer Pneumovax to children 2 years and older with certain high-risk conditions.”
3. Meningococcal vaccine. “Administer Menactra to children ages 2 - 10 years with terminal complement deficiencies or anatomic or functional asplenia or HIV infection.”
4. Influenza vaccine.
 - a. Yearly for children 6 - 59 months, close contacts of children 0 - 59 months
 - b. Yearly for children 5 years + with certain risk factors
 - c. FluMist can be used in healthy children 2 years and older (with out asthma or recurrent wheezing)
 - d. Give 2 doses (separated by at least 4 weeks) to children <9 years receiving Flu vaccine for the first time; or who were vaccinated for the first time last season but only received 1 dose.

Summary: The biggest new change is that Menactra, currently recommended for 11 - 18 year olds, will be recommended for children down to 2 years of age with certain high risk conditions. Depending on the supply and finances, ACIP may eventually expand Menactra to one dose for any child 2 - 18 years. You can download the new schedules in January at: <http://www.cdc.gov/vaccines/recs/schedules/>.

Recent literature on American Indian/Alaskan Native Health

Doug Esposito, MD

Singleton RJ, Holman RC, Yorita KL, et al. Diarrhea-associated hospitalizations and outpatient visits among American Indian and Alaska Native children younger than five years of age, 2000-2004. *Pediatr Infect Dis J*. 2007;26(11):1006-1013.

http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=ShowDetailView&TermToSearch=17984807&ordinalpos=14&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_RVDocSum

Editorial Comment

This study scrutinizes IHS hospital discharge and outpatient visit data for diarrhea in children <5 years of age for calendar years 2000 - 2004, inclusive. Hospitalization and outpatient visit rates (events/10,000 population) were constructed using the 2004 IHS service-population as the denominator, with statistical adjustments applied to earlier years. The data were analyzed on a regional basis; for the AI/AN population, Northern Plains, Southern Plains, Southwest, East, Alaska, and West designations were used, while the general US population was regionalized using Northeast, Midwest, South, and West. Additional analysis was made based on age (<12 months and 1 - 4 years), sex, and diarrhea etiology.

Childhood diarrhea hospitalization rates for the general US population were examined using the 2003 Kids' Inpatient Database (KID). Comparison outpatient visit data for the general US population derived from the 2000 - 2004 National Ambulatory Medical Care Survey (NAMCS) and the National Hospital Ambulatory Medical Care Survey (NHAMCS). Age-specific rates were constructed using 2003 US census and US natality data.

It appears that AI/AN children <5 years of age had similar or slightly lower rates of hospitalization for diarrhea than the general US population [65.9 (95% CI: 63.8 - 68) vs. 79.3 (95% CI: 74.9 - 83.6)/10,000], while rates of diarrhea hospitalization for AI/AN infants were almost two-fold higher [(262.6 (95% CI: 252.3 - 273.3) vs. 154.7 (95% CI: 145.6 - 163.8)/10,000]. Also of note is that the hospitalization rate for AI/AN infants was 10 times the rate for 1 - 4 year-old AI/AN children! This difference was not as striking among the general US population. Higher rates of outpatient visits for diarrhea were found for AI/AN children <5 years of age [2255.4 (95% CI:

2245.1-2265.7) vs. 1647.9 (95% CI: 1398.4 - 1897.4)/10,000], with infants being seen at about twice the rate of the general US population.

The pattern of diarrhea-associated hospitalization and outpatient visits for AI/AN children varied by region, but the regions of highest and lowest burden did not completely match. Overall, hospitalization rates were highest in the Southwest and in Alaska and lowest in the Northern and Southern Plains while outpatient visit rates were highest for the East and the Southwest and lowest for the Southern Plains and the West. Please refer to the article itself for details of these differences and the regional variability based on age group. Seasonal variation occurred for both in- and out-patient events and mimicked what might be expected based on the known epidemiology of viral etiologies (especially rotavirus) of childhood diarrhea.

Given that these data reflect IHS hospital discharge and outpatient visit data, some inherent peculiarities exist that might reasonably be expected to have underestimated hospitalization rates overall and to have possibly impacted the variability observed by IHS region. For example, in regions where hospitalizations occur mainly outside the IHS or contract facilities (e.g., the Northern and Southern Plains), hospitalization rates would be expected to be biased lower (i.e., the disparity between AI/AN children and the general US populations is actually worse than it appears). Additionally, differences in hospital admission criteria, health care seeking patterns, diagnosis and coding issues, and issues with the denominator (user population) all could serve to skew the rate estimates. These issues are discussed by the authors both in the current article and in another article employing an identical methodology to investigate rates of hospitalization for a different condition.¹ Finally, given that AI/AN groups are known to be very diverse and not uniform with regard to traditions, lifestyle, health behaviors, socioeconomic factors, and others, variability in health status and health outcomes is to be expected. American Indian/Alaska Native is a highly diverse racial designation, which is reflected in the health statistics of individual band and tribal designations.

Although disparities in diarrhea still exist and are possibly even greater than demonstrated by this study, one cannot argue the impressive progress that has been made over the years with regards to the burden of diarrhea among AI/AN children. Substantial credit for this progress belongs to the IHS itself and to the dedicated individuals who have worked with and within the system to accomplish so very much with so very little.² These improvements are certainly the result in large part of the targeted deployment of the same basic public health interventions that have so positively impacted health status throughout the world, namely safe water systems, improved sanitation, and better hygiene practices.

According to the authors, further improvements and reductions in diarrhea-associated hospitalization and outpatient visit rates, especially for infants, might be expected as a result

of the newly available rotavirus vaccine. That, however, remains to be seen. At a minimum, continued deployment of technologies known to be effective should be a focus of our efforts. Significant proportions of AI/AN populations still lack basic systems of safe and available water and sanitation that most of us take for granted. Additionally, components of poverty that are widely known to adversely affect susceptibility and transmission of infectious disease (e.g., overcrowding, unhygienic living environments, nutritional integrity), must continue to be targeted. To me, disparities in diarrhea reflect basic and unacceptable societal inequities. Until we as a society address these fundamental facts, health equity and the complete elimination of health disparities will be a long time in coming.

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Additional Reading

Holman RC, Parashar UD, Clarke MJ, et al. Trends in diarrhea-associated hospitalizations among American Indian and Alaska native children, 1980-1995. *Pediatrics*. 1999;103(1):E11.

http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=ShowDetailView&TermToSearch=9917491&ordinalpos=2&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_RVDocSum.

The 13th Annual Elders Issue

The May 2008 issue of THE IHS PROVIDER, to be published on the occasion of National Older Americans Month, will be the twelfth annual issue dedicated to our elders. Indian Health Service, tribal, and Urban Program professionals are encouraged to submit articles for this issue on elders and their

health and health care. We are also interested in articles written by Indian elders themselves giving their perspective on health and health care issues. Inquiries or submissions can be addressed to the attention of the editor at the address on the back page of this issue.

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