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disease prevention news

Meningococcal Disease

Approximately 2,000 persons are infected by meningococcal disease each year in the United States. Despite treatment, 10% to 15% percent of these persons die of their infections while others suffer complications such as brain damage, loss of hearing, kidney failure, or amputations. Children less than 2 years old are at highest risk;[1] however, a rising number of teenagers and young adults are becoming infected.[2] Many of these young adults are first-year college students living in dorms or residence halls. See DPN *1999;59(21):3. This article describes the epidemiology of meningococcal disease, its signs and symptoms, a community-wide outbreak that occurred in Texas in the mid-1990s, as well as recommendations for prevention and control.*

Meningococcal disease is caused by a gram-negative diplococcus, *Neisseria meningitidis*. Nasopharyngeal carriage of *N. meningitidis* by otherwise healthy individuals may occur; as high as 10% to 15% of the population are carriers in countries where the disease is endemic.[3] *N. meningitidis* is transmitted from person to person by the respiratory route.[4] A small minority of carriers will progress to invasive disease involving one or more of the following clinical syndromes: bacteremia, sepsis, meningitis, or pneumonia.[3] A case of meningococcal disease is defined as follows:

- A disease which manifests itself most commonly as meningitis and/or meningococcemia that may progress rapidly to purpura fulminans, shock, and death. However, other manifestations may be observed.
- Isolation of *Neisseria meningitidis* from a normally sterile site (eg, blood or cerebrospinal fluid (CSF) or, less commonly, joint, pleural, or pericardial fluid).

Classification of meningococcal disease is as follows:

- **Probable:** a case with a positive antigen test in CSF or purpura fulminans in the absence of a positive blood culture.
- **Confirmed:** a clinically compatible case that is laboratory confirmed[5]

Several factors determine an individual's transition from nasopharyngeal carriage to invasive disease, including crowded living conditions, poor economic status,[6] active and passive smoking,[7] and preceding or coincident respiratory infections from viruses or mycoplasma.[4,8] Not surprisingly, as the number of carriers in a population rises, outbreaks of disease become more likely.[4] Meningococcal disease shows a seasonal pattern with the highest number of cases occurring in the late winter and early spring (December-March) and the fewest in the late summer and early fall (July-October) (Table 1).

The individuals at highest risk for meningococcal disease are children < 2 years of age, probably because of frequent exposure to respiratory infections.[6] Other risk groups include close personal contacts of cases, individuals with terminal complement component deficiencies, persons with anatomic or functional asplenia, laboratorians who handle *N. meningitidis* isolates, travelers to sub-Saharan Africa or the Middle East, and first-year college students living in dormitories.[1] Community-wide outbreaks also occur occasionally and are defined as "3 or more confirmed or probable cases in the same community during a period of 3 months and resulting in a primary attack rate of 10 cases per 100,000 population."[1,9]

From 1989 to 1998 incidence rates of meningococcal disease in the United States have remained at or near 1 case per 100,000 population (Figure 1). The average US case-fatality rate for meningococcal disease from 1990 to 1997 was 9%.[10] Over

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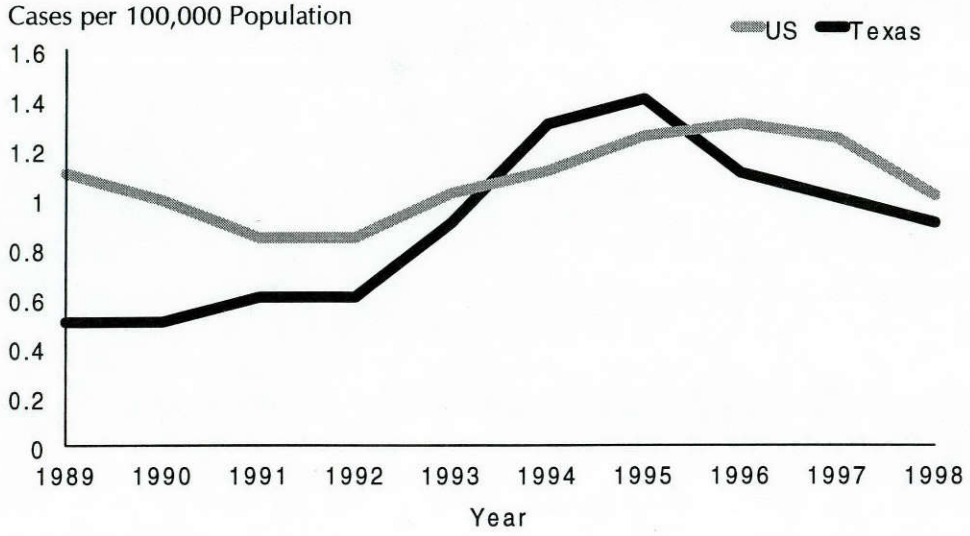
Also in this issue:
Preventing Birth Defects in Texas

Figure 1. Meningococcal Disease Incidence Rates, US and Texas, 1989-1998



the past 10 years, the number of Texas cases has ranged from 93 to 253 per year, for a yearly average of 166 cases. The yearly incidence rate in Texas has ranged from 0.5 to 1.4 cases per 100,000 population. The rise in average annual occurrence from 1994 to 1996 is largely attributable to an outbreak in the northeastern part of the state.

While there are 13 *N. meningitidis* serogroups, most serious disease is caused by groups A, B, C, Y, and W-135. According to one study, children less than a year old are more likely to become infected by serogroup B, while older children and young adults (age 5 - 22) are more likely to develop serogroup C or Y meningococcal disease. This study also found that while nearly all the cases of meningitis and more than half the cases of meningococcemia resulted from serogroup B infections, half of the fulminant meningococcemia cases resulted from serogroup C.[11] Outbreaks of meningococcal disease in developed countries may be due to serogroup B or C. In less developed countries, outbreaks are usually due to serogroup A or B.[4]

Gregg County Outbreak, 1994 - 1996

From 1994 to 1996, an outbreak of 95 cases of serogroup C meningococcal disease took place in Public Health Region 4 (Figures 2&3), population 950,000, for an average annual incidence rate of 3.4 cases per 100,000 population. The majority of the patients were male (53%) and White (78%). Eighty-one percent

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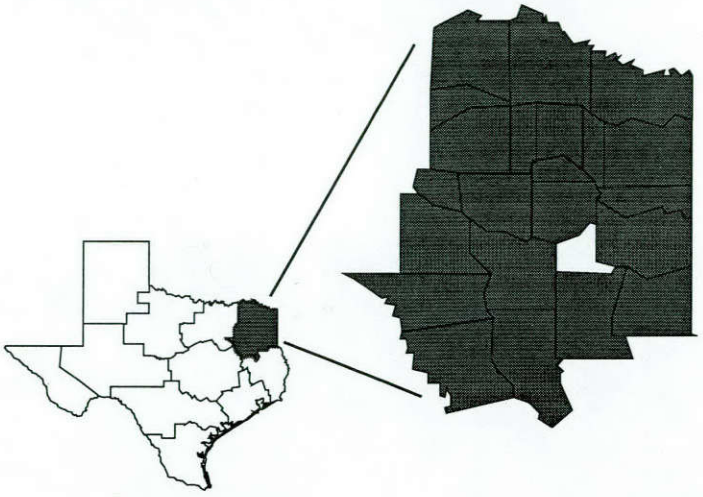
Table 1. Meningococcal Disease by Month, Texas, 1990 to 1999

Month	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	Total	Average
January	16	13	14	15	36	27	49	30	30	16	244	24
February	12	12	12	19	29	29	22	28	25	9	192	19
March	6	7	10	22	26	37	21	25	27	14	191	19
April	6	6	15	14	17	28	22	23	19	11	160	16
May	6	8	8	6	12	28	23	13	7	9	116	12
June	7	10	8	10	15	16	10	7	18	12	105	11
July	3	6	10	3	14	13	8	8	12	6	81	8
August	6	2	5	11	10	9	7	5	13	5	70	7
September	5	8	5	12	20	8	14	13	7	4	93	9
October	9	3	1	8	16	15	10	9	16	3	89	9
November	8	7	7	11	22	11	15	14	8	5	105	11
December	9	18	16	26	20	32	17	20	12	12	177	18
Total	93	100	111	157	237	253	218	195	194	106	1623	162

Figure 2. Public Health Region 4: Gregg County



were under age 29. During the same outbreak, 16 cases of other serogroups occurred, and 59 cases occurred for which the serogroup was unknown. The 1994 outbreak in a cluster of six Region 4 counties continued to spread: by 1996, 20 (56%) of the counties in Regions 4 and 5 had at least 1 case.

The focal point of the outbreak was Gregg County (population 110,000) where 39 people became ill from December 1993 through September 1995; 7 (18%) died. From December 1993 through February 1994, four Gregg County cases of serogroup C meningococcal disease occurred in the 2- to 9-year age group (population 13,600). By the end of February, the 3-month incidence rate in this age group (24.9 cases per 100,000) had met the case definition for an outbreak. In 1998 CDC recommended controlling an outbreak of group C meningococcal disease in Gregg County by immunizing all susceptibles in the population: all 2- to 9-year-olds. Because the incidence rate among 2- to 9-year-olds in adjacent Rusk County had also exceeded the CDC threshold, vaccine was recommended for this age group in Rusk County as well.

By 1995 the outbreak in Gregg County included older children and adults. In February 1995 the incidence rate for persons aged 2 to 29 (population 45,000) exceeded the recommended CDC threshold and vaccination clinics were resumed. In all, nearly 75,000 doses of vaccine were administered in Public Health Region 4—50,000 in Gregg County alone. The vaccine initiative was successful in controlling the outbreak.[12]

Chemoprophylaxis and Vaccination

In the United States, the primary means for prevention of sporadic meningococcal disease is antimicrobial chemoprophylaxis of close contacts of infected persons (Table 2). Close contacts include a) household members, b) day-care center contacts, and c) anyone directly exposed to the patient's oral secretions (eg, through kissing, mouth-to-mouth resuscitation, endotracheal intubation, or endotracheal tube management). The attack rate for household contacts exposed to patients who have sporadic meningococcal disease is an estimated 4 cases per 1,000 persons exposed, which is 500 to 800 times greater than the rate for the general population. Because the rate of secondary disease for close contacts is highest during the first few days after onset of disease in the index patient, antimicrobial chemoprophylaxis should be administered as soon as possible (ideally within 24 hours after identification of the index patient). Conversely, chemoprophylaxis administered > 14 days after onset of illness in the index patient is probably of limited or no value. Oropharyngeal or nasopharyngeal cultures are not helpful in determining the need for chemoprophylaxis and may unnecessarily delay institution of this antibiotic administration.[1]

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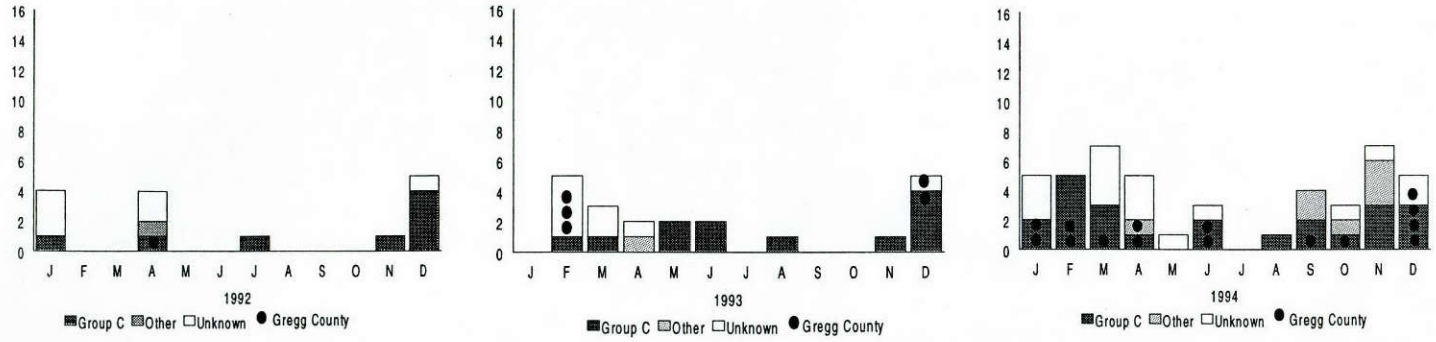
Figure 3. Meningococcal Infections in PHR 4/5, 1992-1996



Rifampin, ciprofloxacin, and ceftriaxone are all 90% to 95% effective in reducing nasopharyngeal carriage of *N. meningitidis* and are equally acceptable for chemoprophylaxis. Systemic antimicrobial therapy of meningococcal disease with agents other than ceftriaxone or other third-generation cephalosporins may not reliably eradicate nasopharyngeal carriage of *N. meningitidis*. If other agents have been used for treatment, the index patient should receive chemoprophylactic antibiotics for eradication of nasopharyngeal carriage before being discharged from the hospital.[1]

One meningococcal vaccine is available in the United States. Menomune®, a quadrivalent polysaccharide vaccine, provides protection against *N. meningitidis* serogroups A, C, Y and W-135 in persons ≥2 years of age. (There is no vaccine to prevent serogroup B disease.) Efficacy against serogroups A and C is 85% to 95%, and immunity develops within 7 to 10 days after vaccination and remains at protective levels for 3 to 5 years. Menomune® also provides some protection against serogroups Y and W-135. This biological is distributed by Aventis-Pasteur (formerly Connaught), 800/822-2463, and is available in single dose (0.5 mL) vials, 5 X 0.5 mL vial packs, and 10 dose (5.0 mL) vials. Vaccine must be used within 5 days of being reconstituted for subcutaneous injection. Side effects are generally mild and infrequent, consisting primarily of erythema and swelling at the injection site lasting up to two days. Any physician can prescribe, and any pharmacy can order Menomune®.

CDC recommends routine meningococcal vaccination for individuals with terminal complement component deficiencies or anatomic or functional asplenia and for clinical laboratory personnel who are routinely exposed to *N. meningitidis* in solutions that may be aerosolized.[1]

Epidemics of meningococcal disease, usually due to serogroups A or C, occur regularly in sub-Saharan Africa (the "meningitis belt") during the dry season (typically December through June). Although the risk of infection is low, the Centers for Disease Control and Prevention (CDC), United State Public Health Service, recommends meningococcal vaccination for travelers to the region during the dry season, especially those (ie, missionaries or health care providers) who anticipate close contact with local people.[13]

As a result of an outbreak of serogroup A meningococcal disease during the 1987 Hajj, Saudi Arabia requires proof of recent immunization for entering religious pilgrims. Following the most recent Hajj (concluded March 17, 2000), 3 cases of serogroup W-135 disease were recognized in the US, including one in a returned pilgrim, another in a household contact of a returned pilgrim, and one in a person who may have had close contact with returned pilgrims or their family

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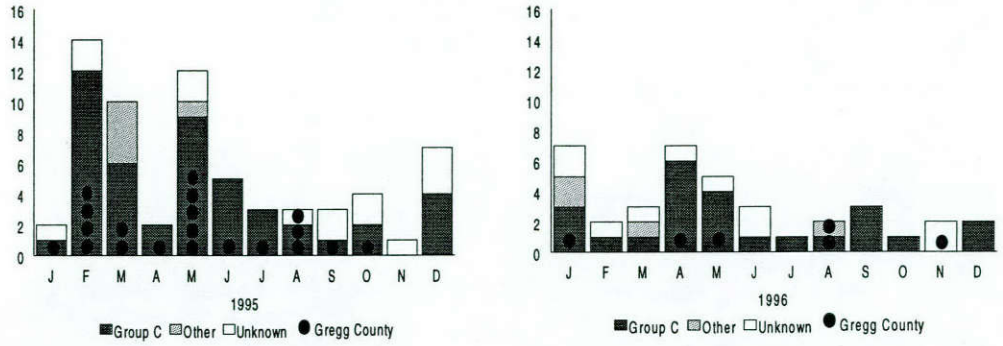

members; 40 cases of serogroup W-135 disease were reported among Hajj pilgrims and their close contacts in Europe and Oman. Additionally, 199 cases of meningococcal disease were reported in Saudi Arabia, including 30 of serogroup W-135 and 55 of serogroup A. This is the largest recorded outbreak of serogroup W-135 meningococcal disease.[14]

Occasionally, the CDC recommends immunization for travelers to areas with recognized outbreaks caused by vaccine-preventable serogroups. No such recommendations are currently in effect, but in recent years CDC has recommended vaccine for some travelers to India, Nepal, Mongolia, Saudi Arabia, Kenya, Tanzania, and Burundi.[11] For further information about foreign travel immunizations, see the CDC website: www.cdc.gov/travel.

From 100 to 125 cases of meningococcal disease occur on college campuses each year; 5 to 15 die.[2] In Texas, 18- to 24-year-olds (the typical age-range of most college students) are twice as likely to die of their meningococcal infections as are members of the general public (OR = 2.06; 95% CI 1.35, 3.76). See *DPN* 1999;59(21):3. Concerned about the increase in the risk of meningococcal disease among college students, the CDC began surveillance of the disease among college students during the 1998-1999 school year. Results of this surveillance found that freshmen living in dormitories had an incidence rate (4.6 cases per

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Table 2 . Schedule for Administering Chemoprophylaxis for Meningococcal Disease

Drug	Age	Dosage	Duration and route of administration
Rifampin*	≤1 month	5 mg/kg every 12 hours	2 days, orally
	>1 month	10 mg/kg (maximum 600 mg) every 12 hours	2 days, orally
	≥18 years	600 mg every 12 hours	2 days, orally
Ciprofloxacin†	≥18 years	500 mg	Single dose, orally
Ceftriaxone	≤12 years	125 mg	Single dose, IM§
Ceftriaxone	>12 years	250 mg	Single dose, IM§

*Rifampin is not recommended for pregnant women because the drug is teratogenic in laboratory animals. Because the reliability of oral contraceptives may be affected by rifampin therapy, alternative contraceptive measures should be considered while rifampin is being administered.

†Ciprofloxacin is not generally recommended for pregnant and lactating women because the drug causes cartilage damage in immature laboratory animals. However, ciprofloxacin can be used for chemoprophylaxis of children when no acceptable alternative therapy is available.

§Intramuscular.

Source: American Academy of Pediatrics. Red Book 2000: Report of the Committee on Infectious Diseases (25th Edition). Elk Grove Village, IL, American Academy of Pediatrics, 2000.

100,000 population) second only to that of children less than two years old. However, because the incidence rate was less than 10 cases per 100,000 population, routine vaccination of college freshmen was not recommended. Health care professionals are urged to inform first-year college students, particularly those who will be living in dorms, and their parents of the risk of meningococcal disease and the benefits of vaccination. Students who want to reduce their risk should receive vaccine.[1]

Because of its relative ineffectiveness in children under 2 years of age and its relatively short duration of protection, routine vaccination of the general population against meningococcal disease is not recommended. However, as previously discussed the vaccine is recommended for use in the control of serogroup C meningococcal outbreaks including community-wide outbreaks.[1]

Confirmed and suspected cases of invasive meningococcal infections **must** be reported immediately by telephone to the local health department (800/705-8868 routes callers to their closest local health authority) or TDH Austin (800/252-8239). Required case/patient information includes name, age, sex, race/ethnicity, DOB, address, telephone number, date of onset, method of diagnosis. The name, address, and telephone number of the physician also must be included.

Isolates from all cases **must** be submitted to either the City of Houston Health Department or TDH Austin laboratories for serogrouping. Because the meningococcal case rate is currently increased somewhat in PHR 6, **isolates from Houston should be PROMPTLY submitted to the Houston Health & Human Services Department laboratory** (1115 S. Braeswood, Houston 77030, 713/558-3400). **All other isolates from PHR 6 MUST be sent by overnight mail** (with a completed TDH G-1A laboratory specimen submission form, call 512-458-7582 for additional information) to the TDH Bureau of Laboratories at 1100 West 49th Street, Austin, Texas 78756.

It may be possible to determine serogroup on probable (CSF culture-negative) cases by PCR testing (available only at CDC). CSF from probable (culture-negative) cases should be held for 3 weeks in the event PCR testing is indicated.

Prepared by
Mardi VanEgdom; Neil Pascoe, RN, BSN; and James Perdue, Infectious Disease Epidemiology and Surveillance

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Monitoring Birth Defects in Texas

Nationally, 1 in 33 babies is born with at least one birth defect. Approximately 9,000 to 10,000 babies are born in Texas each year with at least one birth defect. Birth defects are the leading cause of infant mortality in Texas and the third most common cause of death among children 1 to 14 years of age. In addition, birth defects account for about one-third of pediatric hospital admissions in Texas.

Birth defects are abnormal conditions that are present at birth. They can result in physical and/or mental disability and can be fatal. The number and type vary, depending on family history and the parents' age, race, ethnicity, diet, medical care, and exposure to harmful substances. Some common birth defects are heart defects, spina bifida, cleft lip, and Down syndrome.

The causes of two-thirds of all birth defects remain unknown. Therefore, it is essential that accurate information about the occurrence of congenital anomalites be available to communities and researchers. A recent report describes the primary efforts of the Texas Birth Defects Monitoring Division (TBDMD) to meet this challenge.

The TBDMD mission is to protect and promote the health of the people of Texas through

- Identifying and describing the patterns of birth defects in Texas
- Collaborating with others in finding causes of birth defects
- Working toward prevention and linking families with services

TBDMD publishes annual reports of activities using data contained in the Texas Birth Defects Registry. The latest report, which is the third official report of Registry data, provides statistical information on birth defects among deliveries to residents of areas covered by the Registry in 1996 and 1997. The following executive summary provides statistical highlights of that report.

Overall Prevalence. A total of 9,636 cases were detected with one or more of the birth defects monitored in 1996 and 1997. Of these, 9,300 were live born, corresponding to 3.1% of all live births in the registry coverage area. The 3 most common birth defects were heart defects: patent ductus arteriosus, atrial septal defect, and ventricular septal defect. Rounding out the 10 leading birth defects were hypospadias or epispadias, obstructive genitourinary defect, pyloric stenosis, Down syndrome, cleft lip with or without cleft palate, hydrocephaly, and cleft palate without cleft lip.

Age. There were 14 birth defects with statistically significant variation among mothers of different age groups. Younger mothers had the highest rates for reduction defects of the upper limbs, reduction defects of the lower limbs, and gastroschisis. Both younger mothers and older mothers had higher rates for microcephaly and stenosis or atresia of the large intestine, rectum, or anal canal. The highest rates were found among older mothers for the following defects: hydrocephaly, tetralogy of Fallot, ventricular septal defect, atrial septal defect, endocardial cushion defect, pulmonary valve atrisia or stenosis, patent ductus arteriosus, Down syndrome, and Edwards syndrome.

Race/Ethnicity. Fifteen birth defects showed statistically significant differences among mothers of different racial/ethnic groups. The rates of cleft palate without cleft lip and pyloric stenosis were highest among births to non-Hispanic White mothers. The rate of microcephaly was highest among births to African American mothers, while rates of hypoplastic left heart syndrome and craniosynostosis were significantly lower among African American mothers. Rates were highest among

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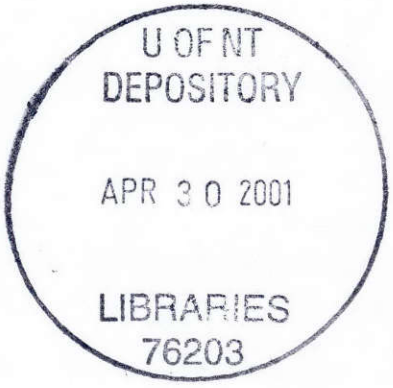

births to Hispanic mothers for the following conditions: spina bifida without anencephaly; hydrocephaly; anotia or microtia; ventricular septal defect; atrial septal defect; patent ductus arteriosus; cleft lip with or without cleft palate; stenosis or atresia of the large intestine, rectum, or anal canal; and reduction defects of the upper limbs. The rate of hypospadias was significantly lower among births to Hispanic mothers.

Sex. Fourteen birth defects showed statitstically significant differences between males and females. Birth defects that were more common among females than males were microcephaly, ventricular septal defect, cleft palate without cleft lip, and congenital hip dislocation. Conditions that occurred more frequently among males were transposition of the great vessels, aortic valve stenosis, cleft lip with or without cleft palate, pyloric stenosis, Hirschsprung disease, hypospadias or epispadias, renal agenesis or dysgenesis, obstructive genitourinary defect, reduction defects of the lower limbs, and craniosynostosis.

Public Health Regions. Nine birth defects showed statistically significant differences among regions. Region 11 had the highest rates for ventricular septal defect, atrial septic defect, pulmonary valve atresia or stenosis, and patent ductus arteriosus. The higher rates observed in Region 11 for these heart defects of lesser severity may result from differences between regions in the use of diagnostic tests and procedures, differences in reporting in medical records, or true higher rates in Region 11. Further analyses are underway to assess what may be contributing to these observations. Other birth defects with statistically significant differences among the regions were microphthalmia (highest rate in Region 8), pyloric stenosis (highest in Region 2), hypospadias or epspadias Region 3), obstructive genitourinary defect (Region 3), and congenital hip dislocation (Region 9).

To obtain the full report, as well as other information from the TDH Birth Defects Monitoring Division, call 512/458-7232 or visit the following website: www.tdh.state.tx.us/tbdmd/index.htm

January is Birth Defects Prevention Montn.