Chapter 14

SEXUALLY TRANSMITTED INFECTIONS AMONG MILITARY RECRUITS

DAVID W. NIEBUHR, MD, MPH, MSC[*]; STEVEN K. TOBLER, MD, MPH[†]; NIKKI N. JORDAN, MPH[‡]; AND DARRELL E. SINGER, MD, MPH[§]

[*]*Lieutenant Colonel, Medical Corps, US Army, Preventive Medicine Physician, Division of Preventive Medicine, Walter Reed Army Institute of Research, 503 Robert Grant Ave., Silver Spring, Maryland 20910-5000*
[†]*Major, Medical Corps, US Army, United States Army Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, Maryland 21010-5403*
[‡]*Epidemiologist, United States Army Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, Maryland 21010-5403*
[§]*Major, Medical Corps, US Army Walter Reed Army Institute of Research, 503 Robert Grant Ave., Silver Spring, Maryland 20910-5000*

INTRODUCTION

There is a wide range of morbidity associated with sexually transmitted infection (STI), including pelvic inflammatory disease (PID), chronic pain, low birth weight, ectopic pregnancy, infertility, neurologic disorders, joint disorders, cervical cancer, and immune suppression, to name a few. If left untreated and allowed to progress, STI can be debilitating and fatal. The World Health Organization estimates that approximately 340 million new cases of curable STI (eg, gonorrhea, chlamydia, trichomoniasis, and syphilis) occur annually worldwide. A substantial number of incurable STIs are also acquired each year; approximately 5 million newly acquired human immunodeficiency virus (HIV) infections worldwide were reported by the World Health Organization in 2004, and over 6 million cases of infection with human papilloma virus (HPV) are reported annually by the Centers for Disease Control (CDC) in the United States alone. Additionally, a substantial number of STIs are asymptomatic and go unreported. When the full spectrum of STI—which includes multiple infections and clinical syndromes caused by more than 30 bacteria, viruses, protozoa, and other infecting organisms—is taken into account, the significance of the global burden of STI is overwhelmingly evident.

Although the effects are often most pronounced in developing countries, where medical care and preventive services are lacking, STIs continue to pose a major health problem in industrialized nations. Approximately 18.9 million STIs occur annually in the United States, causing a financial burden conservatively estimated at $17 billion each year.[1,2]

Within the United States, the military is arguably a high-risk group for STI. There are recognized demographic, geographic, behavioral, and situational factors common to military members that facilitate acquisition, maintenance, and transmission of these infections. Prostitution and STIs have historically flourished around military settlements, particularly when troops are deployed during conflicts. During World War I, STIs were second only to influenza as a cause of lost productivity in the US forces. From 1965 to the end of the Vietnam War, US Army monthly morbidity reports listed venereal disease as the most common diagnosis among diseases reported.[3] Although many examples showcase the high rate of STI transmission between the military and surrounding civilian communities, the military continues to have considerably more infections than are reported in the civilian sector.[4]

The increased STI prevalence observed in the military is largely because its population is skewed towards younger individuals, who tend to be more susceptible to peer pressure and prone to riskier behavior than older people. They are also vulnerable because their experience with STI preventive and clinical services is often limited, resulting from a lack of routine health care before recruitment and concerns about stigma and confidentiality in seeking care. A higher prevalence of STI among adolescents and adults under the age of 25 has been well documented. Almost half of new STI cases occurring each year in the United States are among persons 15 to 24 years of age.[1]

The youthful nature of military populations is clear when comparing military and civilian workforce demographics: approximately 49% of the 2003 active duty enlisted force was 17 to 24 years of age, compared to roughly 14% of the civilian labor force. This population dynamic is most notable in the recruit setting, where recruits are 20 years old on average, and about 83% and 92% of active duty and reserve enlisted accessioned personnel, respectively, are under the age of 25.[5] This contributes to high baseline STI rates upon entry into the military.

Despite the many challenges of preventing STI among young, risk-prone recruits, the regimented recruit training environment provides a number of advantages to implementing prevention strategies, which makes controlling transmission among the force an obtainable goal. Military entry processing stations (MEPS) include some STI and associated sequelae screening in the applicant accession process. Additionally, reception stations or points of entry for recruits entering basic or advanced training routinely perform physical exams, laboratory screening, and vaccinations. Studies performed to date indicate that screening for common STIs among high-risk groups (eg, chlamydia screening for female recruits under the age of 25) at reception stations that conduct multiple screenings and vaccinations is a cost-effective strategy for the military, despite the fact that 50% of recruits typically return to civilian life within 2 years of beginning service.[6-8] Linkage between screening programs and STI educational programs for recruits has likewise been demonstrated to be both feasible and effective.[9,10] Additional savings might be gained through sharing resources used for screening and education among training sites, which would facilitate negotiation of reduced rates for laboratory testing and allow for a consistent preventive medicine approach. Implementation of such proven and cost-effective STI countermeasures upon entrance into the military should enhance the maintenance of a healthy, deployable force. Such measures would also increase retention by curbing transmission rates and effectively identifying and treating infections before complications develop.

TABLE 14-1

DEMOGRAPHIC CHARACTERISTICS OF ACTIVE DUTY AND RESERVE/NATIONAL GUARD ACCESSIONED PERSONNEL, FISCAL YEAR 2003

	Enlisted Accessions				Officer Accessions			
	Active Duty		Reserve/Guard		Active Duty		Reserve/Guard	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Total	176,408	(100.0)	64,390	(100.0)	18,808	(100.0)	16,132	(100.0)
Service								
Army	67,940	(38.5)	45,957	(71.4)	6,334	(33.7)	8,657	(53.7)
Air Force	36,186	(20.5)	7,557	(11.7)	7,028	(37.4)	3,059	(19.0)
Navy	40,204	(22.8)	6,208	(9.6)	4,123	(21.9)	3,552	(22.0)
Marines	32,078	(18.2)	4,668	(7.2)	1,323	(7.0)	864	(5.4)
Gender								
Men	145,732	(82.6)	48,368	(75.1)	14,891	(79.2)	13,180	(81.7)
Women	30,676	(17.4)	16,022	(24.9)	3,917	(20.8)	2,952	(18.3)
Age								
17-24	161,806	(91.7)	53,282	(82.7)	9,477	(50.4)	1,246	(7.8)
25+	14,602	(8.3)	11,108	(17.3)	9,331	(49.6)	14,717	(92.2)
Unknown	0		15		15		169	
Race/Ethnicity*								
White	105,283	(78.2)	37,195	(78.9)	14,695	(85.5)	9,389	(85.0)
Black	20,821	(15.5)	7,788	(16.5)	1,658	(9.6)	1,276	(11.6)
Other	8,534	(6.3)	2,132	(4.5)	836	(4.9)	379	(3.4)
Unknown	4,276		3,126		1,619		1,269	
Marital Status								
Married	15,377	(8.7)	5,379	(8.4)	NA	(31.4)	NA	(55.5)
Not married/Unknown	161,031	(91.3)	59,011	(91.6)	NA	(68.6)	NA	(44.5)

*Racial/ethnicity data were not available for the first quarter, therefore categories do not add up to the total.
NA: not available
Data source: Population Representation in the Military Services, Fiscal Year 2003. Office of the Undersecretary of Defense, Personnel and Readiness Web site. Available at: http://www.dod.mil/prhome/poprep2003/. Accessed November 21, 2005.

RECRUIT POPULATION

Recruits are typically a young and healthy population, composed primarily of single white males under the age of 25, with an average age of 20 years. There are minor demographic variations between active duty and reserve/guard component accessions: there are more female accessioned reservists, and reservists are slightly older on average. Similarly, accessioned officers tend to be older than their enlisted counterparts, especially reserve officers, and accessioned officers are also more likely to be married. Although recruits are composed of both enlistees and officers, enlisted accessions outnumber officer accessions by roughly 7 to 1. Thus enlistees make up the bulk of the recruit population and, not surprisingly, enlisted personnel bear the burden of most STI cases. A summary of fiscal year (FY) 2003 demographics for the enlisted recruit population by active and reserve components is provided in Table 14-1.

RISK FACTORS

There are a number of inherent risk factors that place military recruit populations at high risk for acquiring STI, the most influential of which is age. As previously noted, the vast majority of military recruits are under the age of 25, the demographic group that makes up roughly half of STI cases within the United States. Additionally, it has been suggested that adolescents who represent a significant proportion of the military recruit pool are more prone to acquiring infections because of greater biological susceptibility and barriers to accessing care.[11] This is of particular concern because younger age groups may be more prone to

severe complications such as PID. Women are disproportionately affected by STIs and their consequences because of anatomical differences; they are more likely to acquire an STI from a single sexual experience and to have more asymptomatic infections, which are more difficult to diagnose.[11,12]

In addition to these biological differences, underlying social determinants of STI epidemics, such as poverty, inequality, racial/ethnic discrimination, unemployment, sex ratio, volume of migration, and health care coverage and quality, influence sexual behavior and fuel the establishment of high-risk sexual networks.[13] Other risk factors include having new or multiple sex partners, a history of STI, the presence of another STI, oral contraceptive use, and lack of barrier contraception.[12]

Adolescents as a group tend to exhibit riskier sexual behavior; they have shorter-term relationships than other sexually active individuals. The CDC's National Youth Risk Behavior Survey data from 1997 indicate that 61% of 12th grade students have had sexual intercourse, with 18% of 12th graders reporting four or more partners, and only 57% of currently sexually active 9th through 12th grade students report having used a condom during their last sexual encounter.[14] Racial disparities were also observed among this group, with black and Hispanic students reporting riskier behavior; these findings are consistent with other studies showing similar ethnic differences in infection rates.[14]

The military has reportedly faired worse than these national statistics indicate, with 1998 rates of condom use during the last sexual encounter of unmarried sailors reported as low as 39%.[15] In one study conducted among male marines, approximately half of the participants reported having had more than 10 sexual partners in their lifetime, 36% admitted to having paid for sex in the past, and 80% reported that they did not consistently use condoms.[16]

Substance abuse is also a notorious predictor of risky sexual behavior, including failure to use condoms.[16] In a 2002 Department of Defense survey of health-related behavior, reported illicit drug use within the past 30 days was considerably lower among military members than civilians (roughly 3% compared to 12%).[15] Mandatory screening of military applicants for marijuana and cocaine use and periodic screening after accession is attributed to dissuading high-risk groups from enlistment. On the other hand, alcohol use, which includes heavy and binge drinking, is largely unregulated within the military community and reported use was higher among military personnel than civilians. Compared to their civilian counterparts, military personnel aged 18 to 25 were significantly more likely to engage in heavy drinking (27% compared to 15%) and in binge drinking (42% compared to 17%), although rates of reported binge drinking were roughly equivalent to those reported by college students in 2001.[16]

Lastly, the posting of military personnel around the world and their frequent and rapid travel to distant lands from their military installations and homes in the United States provide the opportunity for acquisition and spread of STI agents. These occasional deployments separate members from their normal social supports and constraints and expose them to diverse populations where endemic STI rates may be substantially higher than at home. This worldwide mobility may also increase the risk of unwittingly introducing infections and antibiotic resistance into the United States.[4,17]

SURVEILLANCE IN THE MILITARY

The first opportunity for STI surveillance within the military occurs at MEPS processing sites. Located throughout the country, MEPS perform administrative and medical screenings of potential recruits. Department of Defense Instruction 6130.4, *Criteria and Procedure Requirements for Physical Standards for Appointment, Enlistment, or Induction in the Armed Forces*, identifies diseases and conditions that are disqualifying for military service. For STI it states: "Current or history of genital infection or ulceration, including, but not limited to herpes genitalis or condyloma acuminatum, if of sufficient severity to require frequent intervention or to interfere with normal function, is disqualifying." Also disqualifying is current untreated syphilis.[18] The accession medical examination is limited to questions regarding history of STI, physical examination of external genitalia, and HIV antibody testing. No other laboratory screening is performed unless indicated by the history or physical examination. In the case of infection due to the syphilis agent *Treponema pallidum*, diagnosis used to be made at the MEPS by serology, and documentation of treatment or cure was sufficient to warrant reconsideration of enlistment. However, an evaluation of the need for syphilis screening revealed that the prevalence of positive tests was extremely low among applicants and that there was ample opportunity for detecting syphilis during the course of a person's military enlistment. A cost-effectiveness analysis supported elimination of the test.[19] As a result, the MEPS abandoned syphilis screening in June 1998.

More robust STI surveillance measures are simply beyond the scope of the MEPS, which are responsible

for multiple in-processing procedures in an extremely fast-paced environment. Therefore, surveillance currently performed is cursory, consisting predominantly of detecting visibly symptomatic patients. The information is recorded primarily on paper, although some of the information captured for disqualified individuals is made available in electronic format to the Accessions Medical Standards Analysis and Research Activity (AMSARA) at the Walter Reed Army Institute of Research. This data only recently included codes for STI; AMSARA reports that STIs are not within the top 20 reasons for disqualification. This is not surprising, given both the limitations in screening practices and the fact that STI, if appropriately managed, should not preclude an individual from performing his or her duties.

The majority of STI diagnoses are derived from patient-initiated medical encounters; additional infections may be identified through contact tracing, periodic physical exams, or screening programs. Cases of STI confirmed at a laboratory or clinic that are considered to be notifiable conditions (eg, chlamydia, gonorrhea, nongonococcal urethritis [NGU], syphilis, hepatitis B, and hepatitis C) are then tracked through the reportable medical events system (RMES) managed by the Defense Medical Surveillance System (DMSS). Over the past 10 years, STIs (specifically chlamydia, NGU, and gonorrhea) have been consistently identified as the leading reportable medical event (RME) among active duty personnel. Service-specific STI rates tabulated from RMES data are presented in Table 14-2. Although data specific to recruit populations are not available from DMSS, rates among a comparably aged active duty population have been substantially higher, so rates among recruits are likely to be elevated as well. Additionally, there is considerable variation in RME rates between services, due in part to variable service reporting and screening practices.

The RME rates provided should be interpreted with caution. Because this surveillance system is passive and depends on multiple reporting chains and data systems (both service-specific and inter-service), under-reporting is common.[20-22] The actual number of infections that occur is likely to be considerably higher than reported for a number of reasons, most notably the large number of asymptomatic individuals who

TABLE 14-2

REPORTABLE MEDICAL EVENTS AMONG ACTIVE COMPONENTS OF THE US ARMED FORCES, RATES PER 1,000 BY CALENDAR YEAR

STI	Service	2000	2001	2002	2003	2004
Chlamydia	Army	12.32	13.40	15.28	11.14	11.74
	Navy	1.15	1.81	3.79	2.71	2.20
	Air Force	8.48	10.32	12.93	12.35	10.76
	Marines	0.95	3.61	4.41	2.96	2.95
	Total	**6.90**	**8.24**	**10.21**	**8.18**	**7.88**
	Total, < 25 year-old subgroup*	*12.88*	*15.18*	*19.02*	*14.94*	*14.17*
Gonorrhea	Army	3.66	3.90	4.33	2.62	2.68
	Navy	0.33	0.51	0.94	0.49	0.42
	Air Force	1.13	1.12	1.11	1.21	0.90
	Marines	0.27	0.55	0.90	0.62	0.65
	Total	**1.69**	**1.85**	**2.15**	**1.43**	**1.36**
	Total, < 25 year-old subgroup*	*2.96*	*3.19*	*3.75*	*2.39*	*2.22*
NGU	Army	2.59	2.09	1.65	0.93	1.05
	Navy	0.25	0.04	0.19	0.05	0.06
	Air Force	0.13	0.07	0.06	0.03	0.02
	Marines	0.20	0.05	1.83	0.09	0.54
	Total	**1.03**	**0.76**	**0.86**	**0.35**	**0.45**
	Total, < 25 year-old subgroup*	*1.57*	*1.18*	*1.26*	*0.54*	*0.71*

*Negligible rates of hepatitis B, hepatitis C, and syphilis were also reported.
STI: sexually transmitted infection
NGU: nongonococcal urethritis
Data source: Army Medical Surveillance Activity, Defense Medical Epidemiology Database (DMED) query of Reportable Medical Events, Active Components, US Armed Forces.

do not seek care. Additionally, healthcare providers may opt to treat infections presumptively without laboratory testing or notification of community health workers through RME channels.

As proof of the underreporting phenomenon, STI rates generated from these passive surveillance systems are low in comparison to those observed through active screening programs, which have been conducted periodically at military installations. For example, prevalence studies of female recruits have repeatedly demonstrated chlamydia infection rates of approximately10%.[10,23-26]

PREVENTION

Despite the absence of accurate, representative data to quantify the burden of STI in military populations, the high prevalence observed, both anecdotally and through targeted screening studies, provides sufficient evidence that preventive action is warranted. The first official approach taken by the US Army to discourage soldiers from putting themselves at risk for STI was a 1778 regulation establishing fines of $10 and $4 for officers and enlisted soldiers, respectively, diagnosed with an STI. By the end of World War I, the US military was using shame as a means of punishment for contracting an STI; posters and pamphlets portrayed venereal disease as an accomplice of the Axis, implying that soldiers who failed to use prophylactic measures were neglecting their duty. By World War II, the military realized that stigmatizing and penalizing STI patients did not work. Prophylactics are now readily available, and regulations reflect a policy designed to provide appropriate support to those newly diagnosed while recognizing the importance of confidentiality.[27]

A number of methods are now advocated for reducing the burden of STI, and many have been shown to be effective in military settings. The first step in preventing STI is typically education and counseling on safe sex. However, education-based programs have not been shown to be highly effective in changing risk behaviors or attitudes over the long term. To maximize effectiveness, STI education programs should be interactive, targeted to the risk behaviors of the individual, and performed by people educated on the topic. Counseling that stresses safer sex instead of abstinence may be more effective in changing behaviors in the long term.[12] One study found that using a health risk appraisal and an interactive video disc worked better than either a standard briefing or targeted situational behavior interventions.[28] Evaluations have found that the military basic training environment is conducive to implementation of STI educational campaigns, that basic trainees have accepted these campaigns, and that the campaigns have been effective at least in the short term.[9]

Education efforts should endorse consistent, correct condom use (male or female condoms), which may be the best method for preventing most STIs. Randomized, controlled trials provide evidence that the newer female condoms confer as much protection against STI as male condoms.[29] Failures due to condom breakage have been shown to be minimal. Valappil and colleagues[30] found that condom breakage occurred in only 0.1% (95% confidence interval [CI] = 0.05–0.21) and 3.1% (95% CI = 2.8–3.4) of sexual encounters using female and male condoms, respectively; slippage errors were likewise shown to be minimal: 5.6% (95% CI = 5.1–6.1) and 1.1% (95% CI = 0.9–1.3) with the female and male condoms, respectively. Unfortunately, many people do not realize the need to have a condom in place anytime body fluids are exchanged, and when condoms are used, they are often employed incorrectly. Also, some people are unaware that improper storage and the use of non–water-based lotions can weaken the condom. Spermicides are not effective in preventing many STIs. In fact, they may increase risk for HIV and other STI transmission.[31]

Education is most effective in curbing STI transmission when used in tandem with countermeasures such as screening. STI screening campaigns can decrease the prevalence of infection in the population and the likelihood of transmission through detection and treatment of asymptomatic individuals. Many experts recommend STI testing before starting sexual contact with a new partner. Although this is helpful, false-negative tests performed during the latent period (time between acquisition of the infection and accumulation of enough organisms to allow detection) remain a problem.

Screening for comorbid STIs at the time of an initial STI diagnosis is also useful because many STIs exist concurrently. For this reason most authorities advocate screening for HIV, hepatitis B, and syphilis during most STI visits and for other infections in selected populations. Unfortunately, many opportunities for screening high-risk populations such as STI clinic patients are missed. In one study in a military setting, one third of people diagnosed with chlamydia had no follow-up testing for HIV, syphilis, or hepatitis B.[32]

With the introduction of molecular-based diagnostic assays, rapid and noninvasive testing for chlamydia, gonorrhea, and potentially other genitourinary pathogens can be readily accomplished. These emerging technologies offer promising novel diagnostics to

support requirements peculiar to military populations. For example, a diagnostic system for STI in females (vaginal swab) that can be self-administered and mailed to a central processing laboratory was recently shown to be at least as sensitive and specific for diagnosis of *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, and *Trichomonas vaginalis* as routine clinic-based tests. This assay system, which also detects HPV, offers potential advantages to military women who may be deployed in remote settings for prolonged periods of time and for whom adequate STI diagnostics or routine Papanicolaou (Pap) screening may be unavailable or impractical.

In May 1999, the Armed Forces Epidemiology Board advised that all female military service members younger than 25 be annually screened for chlamydia at the time of each recommended Pap smear. The recommendation advocated that the initial screening take place at recruit reception stations, which can easily provide the recommended screening in conjunction with STI education, additional STI testing, and treatment. To date, the US Navy and Marine Corps have fully enacted the recommendation. All women and men at the Great Lakes Naval Training Center, Illinois, and the Marine Corps Recruit Depot, Parris Island, South Carolina, receive comprehensive STI training, screening, and treatment when indicated. The Air Force began providing screening, specifically for chlamydia among female recruits, through a pilot program in 2005, and testing will likely become policy. The Coast Guard also screens female personnel for chlamydia and provides STI education and treatment as indicated. Diagnosis and treatment of STI in new Army recruits is limited to clinical encounters when recruits seek health care or undergo physical examinations; STI classes that encourage recruits to seek care if they think they may have a problem are common during basic training (even mandatory at some sites), but procedures vary by location.

Partner management, including screening and treatment, is an important component of the screening process. Routine contact tracing by public health officials (civilian or military) is recommended to prevent reinfection and further transmission, although it has yet to be proven effective in reducing rates of STI.[11] In general, the CDC recommends that partners who have had sexual contact with the infected individual within 60 days prior to diagnosis or the appearance of symptoms, or were the infected person's most recent partner regardless of the time since their last sexual encounter, should be treated prior to resuming sexual relations. Ideally, the treatment should be efficacious and easy to complete. For example, one-dose therapy under observation has become the standard for gonorrhea and chlamydia treatment of partners in emergency department settings. The CDC has also recently supported expedited partner therapy, the practice of allowing patients to take medicine to their partner(s) in cases where partner evaluation is difficult.[12]

Lastly, immunization is an important countermeasure for many infectious diseases. Unfortunately, vaccines against most STIs do not exist or are in development. The only STI vaccine currently approved for use is the hepatitis B vaccine, which has been demonstrated to be very effective in preventing transmission. Two others, the HPV virus-like particle (VLP) and herpes simplex virus (HSV) 2 glycoprotein D subunit vaccines, are showing encouraging results in clinical trials.[33,34]

SELECTED INFECTIONS

Diagnosis of STI begins with the recognition of common STI syndromes and knowledge of the relative prevalence of the infections that cause these symptoms. This knowledge guides examination and testing. In some areas laboratory support is limited or costly, or follow-up is unlikely. Under these circumstances, empiric treatment based on symptomatology is often prescribed. Although presumptive diagnosis and treatment in a single visit sounds attractive, antibiotic overuse, the lack of testing for drug resistance, and the tendency for syndromic management to work poorly in women are significant disadvantages that argue against this practice.

This section deals with selected and relatively common STIs, specifically their epidemiology, clinical presentations, and diagnosis and management. The specific bacterial infections considered are chlamydia, gonorrhea, syphilis, and NGU and cervicitis. The protozoal infection trichomoniasis is also discussed. The specific viruses considered are HSV, HPV, and HIV. Less common STIs, such as lymphogranuloma venereum and chancroid, are not discussed. Because of changing drug regimens, development of new antibiotics, and emerging antibiotic resistance patterns, treatment of STIs is not covered. Clinical practice guidelines are available in regularly updated references.

Bacterial Infections

Chlamydia

Epidemiology. *C trachomatis* is the most commonly reported and most commonly transmitted STI bacteria in the United States. Worldwide, chlamydia is probably

the third most common STI in prevalence and incidence. Unlike some of the other STIs, the rates of infection in the US population are increasing in both males and females. It is estimated that over 4 million cases occur annually (2.6 million in women), and that up to 1 million women develop PID each year because of the large number of asymptomatic chlamydial infections.[2] In 2003, passive surveillance detected 877,478 cases of chlamydia (304 cases per 100,000 persons) within the United States, the highest rates since reporting began in the mid-1980s.[35] Although some of this increase may be due to increased screening and the availability of better tests, chlamydia appears to be increasing in frequency at the same time that many other STIs are decreasing.[35,36]

Annual rates reported by the CDC continue to be significantly higher for females than males.[35] Chlamydia is more concentrated in adolescents than any other STI; risk correlates inversely with age, and in direct relation to the number of sex partners. In general, infections have been most prevalent in sexually active individuals from younger age groups, African-Americans, individuals who have a previous history of STI, and individuals who have a home of record in a southern state.[36,37]

Similar demographic differences have been observed among the military; however, rates within the military community have been higher than national averages.[4,38,39] The 2003 rate of chlamydia reported through the CDC (304 cases per 100,000) was considerably less than that reported through the military RMES (818 cases per 100,000). Within both civilian and military communities the true prevalence is believed to be substantially higher because of the previously noted limitations of passive surveillance. Prevalence studies among large female recruit populations have repeatedly found chlamydia infection rates as high as 10%.[10,23-26] Even higher rates were detected in a prevalence study among nearly 3,000 active duty personnel tested at Fort Bragg, California, from July 1998 to June 1999 in which 19.1% of females and 14.1% of males tested positive.[38] Additional studies among male military members have demonstrated rates of 2.5%, 4%, and 5.3% among Reserve Officer Training Corps (ROTC) cadets, marines, and Army recruits, respectively; the ROTC cadets and Army recruits who screened positive were overwhelmingly asymptomatic (93.6% and 86%, respectively).[40-42]

Clinical Manifestations. The high rate of asymptomatic cases is characteristic of chlamydial infection; the CDC estimates that 70% to 90% of infected females and 50% to 90% of infected males are asymptomatic.[36] Among infected individuals who become symptomatic, the incubation period ranges from several weeks to several months. The most common presentation among men is urethritis or urethral discharge and testicular pain; epididymitis, proctitis, prostatitis, bartholinitis, salpingitis, and pharyngitis also occur. Among females, the most common complaints are mucopurulent cervicitis and pelvic pain; sequelae of particular concern include PID, ectopic pregnancies, and tubal infertility. It is estimated that up to 40% of women with untreated infection will develop PID and one fifth of the women who develop PID will become infertile.[36] Chlamydia infection also can facilitate the transmission of HIV and HPV.[43-45]

Diagnosis and Management. Based on the availability of laboratory support and prevalence of infection, a number of strategies are used to diagnose and treat chlamydia. In many areas of the world syndromic diagnosis and treatment are used. However, in the United States it is recommended that laboratory testing be used to confirm the diagnosis. A comparison of diagnostic methods, summarized by Gaydos and colleagues, is provided in Table 14-3.[46] Although culture is highly specific, it is of only moderate sensitivity, requires epithelial scraping, and takes up to 48 to 96 hours to obtain a result. It is therefore not an ideal screening method for most populations. Alternatively, direct fluorescent antibody testing, enzyme-linked immunosorbent assay (ELISA), and hybridization probes are quick and provide a result in the minutes. However, the sensitivity of these methods is too low for many uses. Testing has recently been simplified by the development of nucleic acid amplification tests that are highly sensitive and specific, can be rapidly turned around, and allow the use of urine or swabs; the drawback of these tests is the high cost.[46] Also, 6% to 30% of screens may be falsely negative if the infection is limited to the cervix. In general, urine tests are appropriate for screening in most populations.

Testing for chlamydia infection can be performed in response to symptoms or for routine screening of high-risk groups. Young females have been shown to have the highest rates of infection and to be the most prone to severe sequelae such as PID. Although young males have high infection rates, severe sequelae in this group are much less common. Use of diagnostic tests for screening in populations with a high prevalence of infection (both military and civilian) has been shown in multiple studies to be cost-effective because it prevents significant sequelae. Much of the cost benefit comes from preventing PID, which is most common within the first year of infection, and the chronic pain that often occurs during the second year of infection.[47] These sequelae are likely to occur during the period of first military enlistment. It is currently recommended by a variety of organizations, including the Armed Forces Epidemiology Board and the CDC, that all sexually active females younger than 25 be screened for chla-

TABLE 14-3

DIAGNOSTIC TESTS FOR *CHLAMYDIA TRACHOMATIS*

Diagnostic Method	Sensitivity	Specificity
Tissue culture	70–85%	100%
Direct fluorescent antibody	80–85%	> 99%
Enzyme immunoassay	53–76%	95%
Hybridization (Pace2; GenProbe, San Diego, Calif)	65–83%	99%
Polymerase chain reaction (COBAS; Roche Molecular Diagnostics, Indianapolis, Ind)		
Cervical	89.7%	99.4%
Female urine	89.2%	99.0%
Male urine	90.3%	98.4%
Strand displacement amplification		
Cervical	92.8%	98.1%
Female urine	80.5%	98.4%
Male urine	93.1%	93.8%
Transcription-mediated amplification		
Cervical	94.2%	97.6%
Female urine	94.7%	98.9%
Male urine	97.0%	99.1%
Male urethral	95.2%	98.2%

Reproduced from: Gaydos CA, Quinn TC. Urine nucleic acid amplification tests for the diagnosis of sexually transmitted infections in clinical practice. *Curr Opin Infect Dis*. 2005;18:56.

mydia at least yearly, even if they are asymptomatic. It is also recommended that older women with a risk factor for chlamydia (eg, new or multiple sexual partners) be screened, and that screening be performed during prenatal examinations. For people suspected of having chlamydia, testing to rule out gonorrhea, syphilis, hepatitis, and HIV infection should also be performed. Because at least 50% of males are asymptomatic, the CDC and other organizations are also considering routine screening of sexually active men. Additionally, because reinfection or recrudescence is common, it is now recommended that females be retested 3 to 4 months after treatment is completed.[11,48,49]

Gonorrhea

Epidemiology. During 2003, a total of 335,104 cases of gonorrhea (116.2 cases per 100,000) were reported in the United States, representing a decrease of 10.1% since 1999. However, it is believed that gonorrhea is underreported; infections are sometimes estimated at over 600,000 each year.[36] Unlike chlamydia, the rates of gonorrhea are decreasing. In fact, the 2003 gonorrhea rates are the lowest the United States has ever documented, but the rates still considerably exceed the Healthy People 2010 (HP 2010) target of 19 cases per 100,000.[35]

Although there were no significant gender differences noted, in 2003 the reported national gonorrhea rate among women (118.8 cases per 100,000) was greater than that reported for men (113.0 cases per 100,000) for the first time.[35] For both genders, sexually active persons younger than 25 were most likely to be infected. Racial differences in gonorrhea transmission were also observed: 2003 rates among black women aged 15 to 19 years (2,947.8 cases per 100,000) and black men aged 20 to 24 years (2,649.8 cases per 100,000) remained higher than those for any other racial/ethnic or age group. Rates are considerably higher among African Americans, although they are decreasing, while rates among whites, Latinos, and Asian-Pacific Islanders are increasing.[35]

Studies within military populations have shown similar group differences in terms of gonorrhea prevalence; younger age, lower enlisted rank, and being African American have been shown to be significant risk factors.[38,50] Overall rates among the military have been substantially higher than those reported at the national level. Gonorrhea is the second most frequently reported communicable disease in members of all four armed services, surpassed only by chlamydia. The 2003 rate reported among active duty personnel was 143

cases per 100,000, as compared to 116 cases per 100,000 reported nationwide. Yet these rates are much lower than those derived from active screening programs. For example, the prevalence rate among nearly 3,000 active duty soldiers screened at Fort Bragg from July 1998 to June 1999 was as high as 8.8% in males and 3.3% among females.[38]

Despite these rates, most authorities, including the US Preventive Services Task Force, do not recommend routine screening for gonorrhea, but rather selective screening of high-risk women, particularly pregnant women, since infection increases their risk for complications such as PID, infertility, and congenital transmission. As a result, the majority of gonorrhea cases are detected after the development of symptoms. There is a considerable difference in rate of symptomatic disease between genders; males with genital gonococcal infections are estimated to be asymptomatic only 10% of the time, whereas roughly half of females with genital infections are asymptomatic.[12]

Clinical Manifestations. The incubation period of gonorrhea is usually 2 to 14 days. Men commonly present with urethritis or epididymitis; approximately 80% of infected men have urethral discharge and 50% have dysuria. In women, gonorrhea typically presents as mucopurulent cervicitis or urethritis. Symptoms can include abnormal vaginal discharge, intermenstrual bleeding, lower abdominal pain, salpingitis, endometritis, and dysuria. Complications among women include glandular infection (Skene's and Bartholin's glands), PID, and infertility. Both sexes can present with anorectal infection and proctitis following anoreceptive sex acts or pharyngeal infection from oral sex; these infections are more likely to be asymptomatic. If untreated, gonorrhea may lead to perihepatitis (Fitz-Hugh Curtis syndrome), sepsis, infected joints, endocarditis, rash, meningitis, and blindness. However, disseminated gonorrhea is estimated to occur less than 1% of the time. Rates of HIV infection are also increased in patients with active gonorrhea.[12]

Diagnosis and Management. Although gonorrhea is often treated based on symptoms without laboratory confirmation in some areas of the world, laboratory confirmation is recommended within the United States. Microscopic examination of Gram-stained exudates is valuable in diagnosing gonorrhea infection. Sensitivity and specificity estimates vary based on whether individuals are symptomatic or asymptomatic and whether the exudate is from the urethra, cervix, pharynx, or anorectum. A summary of diagnostic tests for gonorrhea from Gaydos and colleagues is presented in Table 14-4.[46] In symptomatic males, the presence

TABLE 14-4

DIAGNOSTIC TESTS FOR *NEISSERIA GONORRHOEAE*

Diagnostic Method	Sensitivity	Specificity
Culture	80–95%	100%
Gram stain		
Males, symptomatic	90–95%	95–100%
Males, asymptomatic	50–70%	95–100%
Females	50–70%	95–100%
Hybridization (Pace2; GenProbe, San Diego, Calif)	92.1–96.4%	98.8–99.1%
Polymerase chain reaction (COBAS; Roche Molecular Diagnostics, Indianapolis, Ind)		
Cervical	92.4%	99.5%
Female urine	64.8%	99.8%
Male urine (symptomatic)	94.1%	99.9%
Strand displacement amplification		
Cervical	96.6%	98.0–100%
Female urine	84.9%	99.3–100%
Male urethral	98.5%	91.9–100%
Male urine	97.9%	92.5–100%
Transcription-mediated amplification		
Cervical	99.2%	98.7%
Female urine	91.3%	99.3%
Male urine	97.1%	99.2%
Male urethral	98.8%	98.2%

Reproduced from: Gaydos CA, Quinn TC. Urine nucleic acid amplification tests for the diagnosis of sexually transmitted infections in clinical practice. *Curr Opin Infect Dis*. 2005;18:59.

of Gram-negative intracellular diplococci on Gram stain is essentially pathognomonic for gonococcal infection. Cultures using chocolate agar or selective media such as Thayer-Martin agar preparations are also very useful, with nearly 100% sensitivity. Nucleic acid amplification using polymerase chain reaction (PCR), ligase chain reaction, or transcription-mediated amplification are less invasive and have good sensitivity and specificity. Although these tests are becoming increasingly accurate, especially on urine samples, they are not approved for diagnosis of rectal or pharyngeal infections. Culture remains the test of choice for most settings and is the only test that permits antimicrobial sensitivity testing. People suspected of having gonorrhea should also be tested for chlamydia, syphilis, hepatitis, and HIV infection.

Patients treated for gonorrhea should also be treated for chlamydia infection unless a highly sensitive method of laboratory testing has ruled it out. In emergency departments, it is standard practice for patients to receive observed antibiotic treatment for both pathogens before discharge, long before most laboratory results are available. Cases rapidly become noninfective following treatment for gonorrhea, but patients should refrain from sexual contact until the chlamydia treatment has been completed or for 7 days, whichever is longer. As with chlamydia and other STI infections, partner management is needed. Unlike chlamydia, follow-up testing to confirm a cure is not recommended if an appropriate treatment regimen is administered. If symptoms persist, a culture for *N gonorrhoeae* should be obtained so that isolated gonococci can be tested for antimicrobial susceptibility.[12]

Increases in resistance to the fluoroquinolone antibiotics used to treat gonococcal infections (eg, ciprofloxacin) have been reported from certain regions. The CDC's Gonococcal Isolate Surveillance Project reported fluoroquinolone resistance in 4.1% of all isolates tested in 2003, although considerable geographic variation was observed, with the highest rates noted in Hawaii and California.[12] In 2003, the prevalence of fluoroquinolone-resistant *N gonorrhoeae* infections continued to increase, particularly among men who have sex with men (MSM); as a result, fluoroquinolones are no longer advised for treatment of gonorrhea in Hawaii or California or for infections among MSM.[12,51]

Syphilis

Epidemiology. Since the discovery of penicillin, syphilis rates in the United States have decreased dramatically, making it a relatively uncommon STI. Syphilis remains important, however, because of the severity of untreated infection and sequelae and the increased risk of HIV transmission associated with active syphilis infection.

From 1990 to 2000, the national primary and secondary (P&S) syphilis rate declined 90%, from 20.34 cases per 100,000 population to an all-time low of 2.12 cases per 100,000. The rate of congenital syphilis has also declined sharply, from a peak of 107.3 per 100,000 live births in 1941 to a rate of 10.3 cases per 100,000 live births in 2003, presumably due to the substantial reduction in the rate of P&S syphilis among women.[35]

These declines, along with the fact that the majority of P&S syphilis occurs in a small number of geographic areas, led to the development of a national plan to eliminate syphilis from the United States, announced by the Surgeon General in October 1999. The low HP 2010 target of 0.2 cases per 100,000 population is consistent with the elimination plan. Unfortunately, the national rate of P&S syphilis has increased each year since 2000. During 2003, a total of 7,177 P&S syphilis cases (2.5 cases per 100,000) were reported.[35]

Despite the decline in infection rates since the 1940s, transient increases in infection rates are documented approximately every 10 years. The consecutive increases from 2000 to 2003 constitute the most recent upsurge. The observed increase in overall syphilis rates during this period was driven by a 62% increase among men despite a 53% decrease among women; the disparity between men and women was observed across all racial and ethnic populations.[35] Outbreaks of syphilis were reported in large urban areas among MSM, indicating that increases in syphilis were being fueled by the MSM population.[52] Rates remain disproportionately high in southern states and among African Americans, but these rates are continuing to decline.[53]

A previous increase in P&S syphilis occured between 1985 and 1993, when 762 cases were recorded, causing rates to soar from 11.4 cases per 100,000 to 20.3 cases per 100,000. This transient rise in infection rates was deemed to be the result of an increase in crack cocaine use among minority groups and an increase in rates of infection within the MSM community.[54] The epidemic struck both military and civilian populations simultaneously; epidemic curves in the two populations were parallel, peaking in 1990 and 1991, with annual incidences of 122.6 cases per 100,000 (military) and 48.0 cases per 100,000 (civilian). In the military, which represented roughly one third of identified P&S infections, cases were primarily among males, African Americans, people under 30, and personnel in the lower enlisted ranks.[50,54,55]

Clinical Manifestations. Primary syphilis, which is characterized by one or several painless, indurated ulcers, develops 2 to 10 weeks after inoculation. Lesions are most commonly found on external genitals but can be present intravaginally or, even less likely, in extragenital locations (eg, anorectum, lips, and fingers).

Regional adenopathy is common. Oral ulcerations are uncommon, but not rare. Without treatment the ulcer usually heals spontaneously within 6 weeks, but the spirochete continues to spread to other sites.[12] Three to six weeks later, untreated primary infections typically progress to secondary syphilis, which is characterized by new lesions and a more generalized reaction; these lesions will resolve within weeks to months even if untreated. Manifestations of secondary syphilis can include a maculopapular rash (commonly on the palms and soles), generalized lymphadenopathy, mucous patches, condyloma lata, and alopecia.[12]

Overall, about one third of people who acquire P&S syphilis are cured without treatment. The remainder of untreated cases enter a latent stage, in which the immune system suppresses but does not cure the infection. These persons have laboratory evidence of infection but no signs or symptoms. About one third of these individuals will develop tertiary syphilis within 1 to 20 years of infection; however, tertiary syphilis is rare because of the widespread availability and use of antibiotics. Tertiary syphilis can cause granulomas to form in many parts of the body, including the skin, bones, and the circulatory system. When the circulatory system is involved, tertiary syphilis can cause aortitis, aneurysms, and valvular abnormalities as well as other problems. Neurosyphilis can occur at any stage of infection and may result in tabes dorsalis and paresis.[12]

Diagnosis and Management. Due to the low number of infections, routine screening for syphilis is not recommended and is no longer required upon entrance into the military, even during accession physical examinations. It has been estimated that the elimination of universal syphilis screening of recruit applicants at MEPS has saved the military $2.5 million per year.[19] The US Preventive Services Task Force currently recommends against routine screening of asymptomatic persons who are not at increased risk for syphilis infection. It also strongly recommends, however, that clinicians screen persons at increased risk for syphilis infection and all pregnant women.

Diagnosing and treating syphilis is complicated by the fact that *T pallidum* cannot be grown on bacteriologic media or in cell cultures. The diagnostic tests currently used have variable sensitivities based on stage of disease and suffer from poor specificity.[56] The tests most commonly used for syphilis detection rely on identification of antibodies and only provide presumptive evidence of infection. These tests are sensitive for secondary infections but may return false negatives during primary infections. The most specific method of diagnosing P&S syphilis is by direct examination using dark field or immunofluorescent antibody microscopy, but high rates of false negative results and the difficulty in detecting advanced disease have led clinicians to favor serologic testing.

Serologic tests for syphilis are divided into nonspecific tests, which use nontreponemal antigens, and specific tests that use treponemal antigens. Because a number of diseases (eg, mononucleosis, hepatitis, varicella, measles, lymphoma, tuberculosis, malaria, endocarditis, and connective tissue disease), as well as pregnancy, can produce a positive result on nontreponemal tests, and treponemes other than *T pallidum* (eg, the organisms causing yaws, pinta, and leptospirosis) can result in false positive results on the treponemal tests, both types of tests must be used for diagnosis.[56]

The most common nontreponemal tests are the rapid plasma reagent test, the venereal disease research laboratory test, and the automated reagin test. These tests are relatively inexpensive, easy to perform, and provide quantitative results. However, they are often negative during primary syphilis and may be falsely negative when extremely high concentrations of antibodies against syphilis are present in the serum.

The treponemal antibody tests include the microhemaglutination test for *T pallidum*, treponemal pallidum particle agglutination, and the fluorescent treponemal antibody absorption test. These tests often remain reactive following resolution of the infection and are therefore not useful for monitoring success of treatment.

Nontreponemal tests are commonly used for screening, with positive results confirmed by a treponemal test. Once treatment begins, a nontreponemal test is used to monitor therapeutic response. Follow-up testing for P&S syphilis is performed at 3, 6, and 12 months; for latent syphilis, testing is done at 3, 6, 12, 18, and 24 months; and, in neurosyphilis cases, testing of cerebral spinal fluid and serum is performed at 6-month intervals. Treatment is considered efficacious if there is a 4-fold decrease in antibody levels; a 4-fold increase is evidence of reinfection.

Vigilant partner management is recommended for sexual contacts of syphilitic patients. Procedures vary based on the stage of infection: anyone who had sex with the infected person within 3 months of the onset of primary syphilis, within 6 months of onset of secondary syphilis, and within 1 year of onset of early latent syphilis should be contacted. All contacts exposed within 90 days should be treated regardless of laboratory testing results.[12]

Nongonococcal Urethritis and Cervicitis

Epidemiology. It is estimated that 3 million new cases of NGU and cervicitis are contracted in the United States each year. Worldwide estimates amount

to 89 million.[57] As with most STIs, young adults (20–24 years of age) such as those that comprise the military recruit population have proved to be at increased risk; MSM are another high-risk group.

Clinical Manifestations. *C trachomatis, Ureaplasma urealyticum, Mycoplasma genitalium,* and *Mycoplasma hominis* are bacteria that have been linked to NGU and cervicitis; *T vaginalis* is also emerging as a causative protozoan agent. Association with other STI organisms has been rare. *C trachomatis* immunotypes D through K have been implicated in up to 50% of NGU and virtually all cervicitis cases; *Ureaplasma* is estimated to cause between 30% and 60%; and *Mycoplasma* and *Trichomonas* have each attributed to approximately 5% of infections.[57,58] In addition to urethritis and cervicitis these bacteria can cause salpingitis, endometritis, chorioamnionitis, prostatitis, and epididymitis; complications such as pyelonephritis and PID may also develop.

U urealyticum bacteria are found in the genital tract of sexually active adults. Colonization occurs in 40% to 80% of women who are asymptomatic and sexually active. *M hominis* has been isolated from cervicovaginal specimens in 21% to 53% of asymptomatic, sexually active women, and may also colonize the throat, eyes, and umbilicus to a lesser extent. These rates are somewhat lower in males.[58]

Diagnosis and Management. Following infection there is a 10- to 20-day incubation period before symptoms appear. Laboratory testing is of limited value since the bacteria colonize large numbers of sexually active adults. Culture requires specific media and special handling. Serologic tests are of little or no value. PCR tests do exist but are not widely available. These infections are best managed as if they were chlamydial. Without treatment these infections usually resolve within 6 months.[58]

Protozoal Infections

Trichomonas

Epidemiology. Trichomoniasis is considered to be the most common nonviral STI among women. It is estimated that approximately 180 million women worldwide are infected. Prevalence estimates vary greatly between populations studied, ranging from 5% to 74% among women and 5% to 29% among men, with the highest rates observed among high-risk populations such as STI clinic patients and prisoners.[59] Within the United States an estimated 7.4 million cases occur among both sexes each year, associated with a cost of approximately $375 million.[12] Prevalence studies among women within the United States indicate that 2% to 3% of the general female population is infected, and up to 60% of high-risk populations (eg, female prison inmates and commercial sex workers) are infected.[12] Estimates of asymptomatic rates in women vary from 10% to 50%.[58] Less is known about both prevalence of infection and asymptomatic rates in men.

Older age, histories of previous STIs, multiple sex partners, pregnancy, and drug use have been associated with trichomoniasis. The association with older age is in contrast with the association between age and other STIs, especially chlamydia.[59]

Clinical Manifestations. Among women, symptoms may include a frothy gray or yellow-green vaginal discharge, pruritus, and cervical petechiae ("strawberry cervix"). *T vaginalis* may also infect Skene's glands and the urethra, areas where the organisms may not be susceptible to topical therapy.[12] Medical opinion has traditionally placed little importance on the role *Trichomonas* has played in health complications in women, and it has been considered a rare infection in men. However, evidence now implicates *T vaginalis* as a contributor to adverse outcomes among both men and women. Multiple studies have shown a link between *Trichomonas* infection and acquisition and transmission of HIV. *Trichomonas* may also be associated with the development of cervical neoplasia, postoperative infections, adverse pregnancy outcomes, and PID in women; among men it has been identified as a cause of NGU and male factor infertility.[59] Concomitant STI diagnosis, specifically coinfection with chlamydia or gonorrhea, is also common.[60]

Diagnosis and Management. As evidence accumulates, consideration is being given to improved diagnostics and screening. At present, routine screening is not recommended. Infection is usually identified in vaginal or urethral secretions using a wet preparation, but this method has relatively poor sensitivity (30%–80%) compared with culture; the sensitivities of culture techniques and wet preparation have been reported as 70% and 36%, respectively.[46] Several PCR assays have been developed for research, but there are currently no commercially available amplified assays; the sensitivity and specificity of the PCR assays studied was 97% and 98%, respectively.[46] These tests hold promise for incorporation into screening programs for detection of multiple pathogens in asymptomatic persons.

Viral Infections

Herpes Simplex Virus

Epidemiology. HSV infection is caused by serotypes HSV-1 and HSV-2. HSV-1 has a predilection for the mouth but also causes around 10% of genital infections;

it is predominantly acquired during childhood. HSV-2 has a predilection for the genital areas but also causes oral disease; the majority of infections are acquired during the third decade of life. Herpes simplex infection is the most common cause of genital ulcer disease and one of the three most common STIs in the United States, with an estimated 500,000 to 1 million new cases of sexually transmitted herpes infections each year.[2,12] Infection is lifelong. Prevalence increases to age 40 and then plateaus. Overall, it is estimated that between 50 million and 65 million Americans are infected, making HSV the most prevalent STI in the United States. It is also estimated that a quarter of Americans older than 30 years have HSV.[34] The National Health and Nutrition Examination Survey (NHANES), which tracks the seroprevalence of HSV, found a seroprevalence of 21.9% during NHANES III (1988–1994), an increase of 30% since NHANES II was conducted in the late 1970s.[2,12,61] Risk factors for infection included female gender, black or white race, and lower socioeconomic status, but the strongest predictor of infection in NHANES III was the lifetime number of sexual partners.[12,50]

Rates of infection and adverse outcomes have been high among military populations. Among US Navy personnel evaluated from 1980 to 1989, the incidence (new hospitalizations) of HSV was 12.1 per 100,000 person-years. Younger age, female gender, and white or black race were all associated with higher rates of infection.[50]

Clinical Manifestations. The majority of cases are subclinical or asymptomatic; only 9% of people who were seropositive in NHANES III reported having genital herpes. In the prototypical case, a person develops pain, itching, burning and/or tingling at the inoculum site within 2 to 12 days after acquisition.[12,58] This is followed by the development of grouped vesicles on an erythematous base that ultimately ulcerates. In the absence of treatment, lesions typically heal within 6 weeks without scarring. Constitutional symptoms (eg, fatigue, fever, myalgias) are not uncommon and last 5 to 10 days.[12] Regional adenopathy can occur. Up to one third of women and one tenth of men with primary infection develop aseptic meningitis. This is most likely to occur during primary infection.

Following primary infection the virus enters the dorsal root ganglia and becomes latent, but asymptomatic viral shedding can occur. Shedding rates are greatest within the first 3 months of primary infection. Recurrence is also most likely to occur soon after infection. During the first 12 months after infection, 80% to 90% of those infected will have a recurrence.[34]

Nonprimary infection occurs when a previously exposed person is infected with another serotype of the virus. Nonprimary disease is much more likely to be mild and of shorter duration than primary infection.

Recurrence occurs when a person with a latent infection experiences a reactivation of the virus due to immune system impairment. This can be a result of immunosuppressive diseases, other viral infections, drugs, malnutrition, fatigue, stress, skin trauma, or exposure to sunlight. Recurrences are usually mild, with a limited number of lesions and occasional regional lymphadenopathy, both of which last for much shorter periods of time than during primary infection.

Diagnosis and Management. Diagnosis of HSV infection can be confirmed with a number of laboratory tests. Many of these tests are expensive or not widely available, which has limited their use. A Tzanck smear from the lesion showing multinucleated giant cells is suggestive of infection, but this test has low sensitivity and specificity. Viral culture during the vesicular and early ulcerative stages has high sensitivity and specificity. During the vesicular stage of primary infections, the sensitivity is approximately 95%; in the ulcerative phase the sensitivity decreases to approximately 70%. Once crusting of the lesions has occurred, sensitivity drops further to approximately 30%.[34] Antibody tests have, in the past, suffered from the inability to differentiate between HSV-1 and HSV-2 infections, but better serologic tests are now available that differentiate between the two. The newer serologic tests detect a 4-fold increase in antibody levels with over 95% specificity and 70% to 90% sensitivity. Unfortunately, it takes several weeks for this rise in antibodies to appear, so the serologic tests are not useful in determining treatment for acute infections. PCR tests are now being used but are not widely available. A drawback to nucleic amplification tests is that people who have genital ulcer disease from another cause may still shed HSV. In one study, asymptomatic infected individuals had HSV-2 nucleic acid detectable by PCR 20% of the time they were tested.[62] Another problem with PCR is that it can detect virus that was shed days or weeks prior to testing. At present, routine screening for HSV is not recommended.[63]

Prevention and reduction of herpes is extremely difficult. Infected persons, who often do not realize they are infected, shed virus for years to decades. Although shedding tends to be highest in the first 3 months, infection is lifelong.[12] Persons in monogamous relationships for years may suddenly present with lesions. Consistent use of condoms can significantly decrease the likelihood of transmission. In general, individuals with lesions should abstain from sexual relations. In the future it is likely that immunization will be the key to controlling herpes. Although there are no vaccines currently licensed for use in the United

States, recent vaccine trials of an HSV-2 glycoprotein D subunit vaccine have demonstrated that the vaccine is safe, with 75% efficacy in women who were seronegative for HSV-1 and HSV-2 before vaccination.[34] Additional studies are underway.

Human Papilloma Virus

Epidemiology. HPV is a deoxyribonucleic acid (DNA) virus that multiplies in the nuclei of infected epithelial cells and can cause genital warts. The virus can also cause changes in the epithelium of the genital tract that may lead to cervical dysplasias and cancers. There are at least 120 different types of HPV that may affect the skin or mucous membranes. Over 30 of these can affect the genital tract, but the most common are types 6, 11, 16, and 18. In most cases, because the infection remains confined to the epidermis, causing warts, temporary squamous changes, or subclinical infections, HPV infection is thought to be benign. Types 6 and 11 are the cause of most visible genital warts. Infections with types 16, 18, 31, 33, and 45 have been found to be associated with neoplastic changes in a small percentage of cases. Types 16 and 18 create the highest risk of cervical and anal cancer.

HPV infection has the highest incidence of any STI in the United States, with approximately 6.2 million new infections every year.[12] Because it is rarely a lifelong infection, HPV is the second most prevalent STI: it is estimated that 10 to 20 million people are infected at any time, and 50% to 80% of sexually active women will be infected at some point during their lives.[12] Some studies have found prevalence rates of 28% to 46% in females less than 25 years old, and the percentage of females infected by 50 years of age may be as high as 80%.[36] High rates have also been documented in female military populations. A study that screened asymptomatic female ROTC cadets found squamous changes in 7.8% of those examined.[64]

Clinical Manifestations. Young age, increasing numbers of sexual partners, immunosuppressive conditions, and lack of circumcision are associated with an increased risk of infection. Unlike many STIs, HPV infection is highly prevalent in all socioeconomic groups.

The incubation period is at least 2 to 3 months, but most infections are transient, subclinical, and unrecognized. Many infections resolve without treatment within 2 years. Incidental lesions are often noted during routine physical examinations. Clinical manifestations include genital warts, cervical cell abnormalities, and other epithelial changes. Warts typically appear on the external genitalia but can also occur on the cervix, vagina, urethra, anus, and mouth. Lesions can vary greatly in appearance, from isolated small papules to cauliflower-like growths. In females, infection can cause cervical squamous epithelial changes and cervical cancer. Other presentations include anal cancer and respiratory tract papillomatosis.

Diagnosis and Management. Approximately one third of infected individuals spontaneously regress within 3 months of developing lesions, and most regress within 2 years, with a mean duration of infection of approximately 8 months. Infections that persist may include asymptomatic shedding and occasional wart development. Diagnosis of HPV infection in males is generally by visual examination for exophytic lesions on the external genitals. Acetic acid is occasionally used for better visualization. Biopsy is employed for pigmented lesions, atypical growths, or growths that do not respond to treatment.

Procedures for diagnosis in females are detailed in chapter 20, Gynecologic and Reproductive Health for the Female Recruit. In addition to the diagnostic advances described in that chapter, a number of promising vaccines have been developed and are being studied for safety and efficacy. Some of the vaccines are aimed at decreasing the length of infection; others are intended to prevent the development of abnormal cellular changes despite infection.[33,65,66] A double-blind, multicenter, randomized, placebo-controlled study assessing the efficacy of bivalent HPV-16/18 L1 VLP vaccine among 1,113 healthy HPV-negative women aged 15 to 25, at facilities in Brazil, Canada, and the United States, indicated an efficacy of 95.2% against HPV-16 ($P < 0.01$), 91.2% against HPV-18 ($P < 0.01$), and 92.9% against HPV-16/18 ($P < 0.01$).[33]

Human Immunodeficiency Virus

Epidemiology. HIV-1 is the most prevalent and most important retrovirus that infects humans. Other related, but far less prevalent retroviruses include HIV-2 (prevalent in West Africa), human T-lymphotropic virus (HTLV) 1 and HTLV-3 (which can also be sexually transmitted and are prevalent worldwide, especially among native populations in Japan, the Caribbean, and the Americas), and the newly identified HTLV types III and IV (which have been recently identified among bushmeat hunters in Cameroon, and for which there is no firm evidence of sexual transmission).[67,68] Within the United States, estimates of the prevalence of this disease have slowly increased to approximately 1.1 million individuals in 2003, with an estimated annual incidence of 40,000 to 45,000. Between 1978 and 2000, 454,058 people died from aquired immunodeficiency syndrome (AIDS).[69] HIV-1 is a virus of particular significance within the US

military because of its potential for transmission to the Department of Defense blood products donation pool. Rate of transmission per act is highest among blood transfusion recipients who receive unscreened blood or blood products (approximately 90%); moderately high among injection drug users who share needles and other drug-using paraphernalia (approximately 1%); intermediate through homosexual contact (approximately 0.5%); and lowest through heterosexual contact (approximately 0.1%).[70] Prevalence and incidence within the military has slowly decreased since the introduction of testing for HIV-1, from 2.59 cases per 1,000 person-years in 1986 to 0.13 per 1,000 person-years in 1999.[68] HIV-1 seroconversions in the Army have been low and stable since the early 1990s. The HIV-1 incidence rate among more than 2 million Army personnel tested between 1985 and 1999 was 0.17 cases per 1,000 person-years (95% CI = 0.16–0.17). HIV was found to disproportionately affect males (relative risk [RR] = 3.1), minorities (black RR = 4.6; Hispanic RR = 2.8), enlisted soldiers (RR = 2.5), unmarried soldiers (RR = 2.0), and soldiers older than 30 (RR = 1.5).[71] Surveillance data from preaccession HIV-1 screening of applicants to the armed services has demonstrated a similar decrease, from 1.98 cases per 1,000 person-years in 1986 to 0.36 per 1,000 person-years in 2000.[72] These figures are represented graphically in Figure 14-1.

Clinical Manifestations. The incubation period of HIV is generally 1 to 3 weeks following exposure to the virus, and acute infection is characterized by nonspecific symptoms common to many viral syndromes: headache, sore throat, chills, fever, malaise, and body aches. Enlarged and tender lymph nodes may be present, resembling those seen in acute mononucleosis. Signs and symptoms may also include an erythematous, maculopapular rash on the trunk and extremities, gastrointestinal disturbances such as diarrhea, esophageal and anal ulcers, and central nervous system disturbances. Many of these symptoms may resolve within several weeks, while others, such as lymphadenopathy, may be present for several months. All symptoms eventually resolve as the patient enters a period of apparent latency, during which the virus continues to infect and destroy CD4+ cells, including CD4 T lymphocytes and macrophages. Loss of CD4+ cells over the course of disease progression results in a gradual reduction in the immune response to various challenges, including many viral and fungal organisms. The disease, if not treated with antiretroviral medications and prophylaxis for opportunistic infections, leads to death.

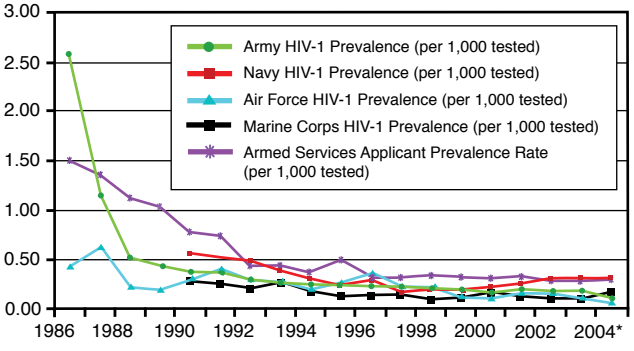

Fig 14-1. HIV-1 prevalence in the US active military branches compared to applicants to all branches, 1986 to 2004.
*The 2004 data is only through June 2004 (therefore not a full year).
Data source: Warren Sateren, Division of Retrovirology, Walter Reed Army Institute of Research, Rockville, Md.

Diagnosis and Management. Although routine screening is not recommended for the general population, military screening is mandatory. US Military Entrance and Processing Command (USMEPCOM) and a majority of Army, Navy, and Marine Corps facilities ship specimens for testing to a contracted testing facility. Standard screening is done with an ELISA. A nonreactive result is recorded as a negative test. A reactive result is retested using the original ELISA and a second ELISA (from another manufacturer) in parallel. Two of three reactive results from this algorithm require confirmation by a Western blot, graded according to recognized criteria. A positive HIV-1 screening result is followed by a request for a confirmatory sample. Indeterminate samples (based on Western blot criteria) are sent for viral load testing; if the test result is negative, a repeat screening is done 6 months later. USMEPCOM makes every attempt to contact the applicant by certified mail and telephone. Active duty and reserve personnel have the second sample retested at the service-specific facility and generally also have nucleic acid testing performed at the same time. If the tested individual is in the window period (ie, the time from exposure to the virus to formation of anti–HIV-1 antibodies, which may last up to 6 months) at the time of sample acquisition, or if the individual has a condition that precludes antibody response to the virus, a false negative test result may occur.

Evolving military HIV policies reflect the increasing knowledge of the HIV-1 epidemic and improvements in diagnostics. USMEPCOM Regulation 40-1 outlines the requirements for preaccession testing. A MEPS physical examination with HIV-1 testing remains valid for 24 months (however, this policy is currently under review). The Assistant Secretary of Defense for Health Affairs, in HA Policy 04-007, directs that routine HIV-1 screening be done biennially.[70] Army

Regulation 600-110, Identification, Surveillance, and Administration of Personnel Infected with Human Immunodeficiency Virus (HIV); Navy NAVMC 290; and Air Force Instruction 48-135 govern the HIV programs within each service.

An individual recruit or trainee found to be HIV-positive will be notified according to post- or base-specific regulations, generally by the individual's commander and the preventive medicine or STI officer, and counseling, contact tracing, and appropriate medical evaluations are initiated. The disposition of each case is determined by a medical review board.

CONCLUSION

There are significant costs associated with STI in terms of morbidity, mortality, and personnel time lost, which affect individual well-being and force readiness. Health care providers must be aware not only of the clinical presentation, diagnostic modalities, and treatment of these conditions, but also of the importance of surveillance and prevention.

Active surveillance has consistently demonstrated increased rates of STI among the military population, particularly in recruits, whose prevalence rates of screened infections such as chlamydia have repeatedly been found to be as high as 1 in 10. With approximately 1.5 million people presently serving on active duty in the US armed forces and more than 240,000 enlisted accessions per year, the magnitude of the problem can not be overstated. While control of STI in the military has been and will remain challenging, prevention campaigns that include mass screening and STI education targeted to high-risk groups have been proved to be both feasible and cost effective.

The large numbers of high-risk people who pass through recruit reception stations in short periods of time create opportunities for efficient and effective intervention. Studies in the Army have shown intervention to be cost effective for high-risk females, even when approximately half of new recruits returned to civilian life within months.[7] Additional cost savings could be gained if military services jointly negotiated better rates for laboratory testing. Still, because much of the long-term benefit will be reaped by the civilian healthcare sector, military leaders may be reluctant to fund intervention programs. Military and civilian cost-sharing should be considered as a solution.

There is ample evidence of STI transmission between civilian and military populations; interaction between these two communities will likely continue and may increase. Therefore, the success of future STI control efforts will depend upon cooperation among federal, state, and local civilian agencies and their military counterparts in the design and execution of education, prevention, and intervention strategies.

Acknowledgments

The authors wish to express gratitude to Colonel (Retired) Joel C. Gaydos and Colonel (Retired) Kelly T. McKee for their contribution to the STI Prevention section and Colonel (Retired) Toti Sanchez for his assistance to the HIV section of this chapter. In addition, this chapter was editorially reviewed by Ms. Kathleen Huycke, Colonel Christine Scott, Colonel (Retired) Joel C. Gaydos, and Captain Amy Millikan.

REFERENCES

1. Weinstock H, Berman S, Cates W Jr. Sexually transmitted diseases among American youth: Incidence and prevalence estimates, 2000. *Perspect on Sexual Reprod Health*. 2004;36:6–10, 22, 59.

2. Handsfield HH. Sex, science and society: A look at sexually transmitted diseases. *Postgrad Med*. 1997;101:268–273, 277–278.

3. Emerson LA. Sexually transmitted disease control in the Armed Forces, past and present. *Mil Med*. 1997;162:87–91.

4. Gaydos CA, Quinn TC, Gaydos JC. The challenge of sexually transmitted diseases in the military: What has changed? *Clin Infect Dis*. 2000;30:719–722.

5. Population Representation in the Military Services, Fiscal Year 2003. Office of the Undersecretary of Defense, Personnel and Readiness Web site. Available at: http://www.dod.mil/prhome/poprep2003. Accessed November 21, 2005.

6. Howell MR, Gaydos JC, McKee KT Jr, et al. Control of *Chlamydia trachomatis* infections in female army recruits: Cost-effective screening and treatment in training cohorts to prevent pelvic inflammatory disease. *Sex Trans Dis*. 1999;25:519–526.

7. Howell MR, McKee KT Jr, Gaydos JC, Quinn TC, Gaydos CA. Point-of-entry screening for *C trachomatis* in female army recruits. Who derives the cost savings? *Am J Prev Med*. 2000;19:160–166.

8. Rein DB, Kassler WJ, Irwin KL, Rabiee L. Direct medical cost of pelvic inflammatory disease and its sequelae: decreasing but still substantial. *Obstet Gynecol*. 2000;95:397–402.

9. Arcari CM, Gaydos JC, Howell MR, McKee KT, Gaydos CA. Feasibility and short-term impact of linked education and urine screening interventions for Chlamydia and gonorrhea in male army recruits. *Sex Transm Dis*. 2004;31:443–447.

10. Boyer CB, Shafer MA. Development of a cognitive-behavioral group randomized control intervention trial to prevent STIs and unplanned pregnancies for young women entering the US military. *J Adolesc Health*. 2002;30:129.

11. Centers for Disease Control and Prevention. Sexually Transmitted Diseases Treatment Guidelines 2002. *MMWR Morb Mortal Wkly Rep*. 2002;51:1–80.

12. Ready To Use STI Curriculum for Clinical Educators. CDC Division of Sexually Transmitted Diseases Training and Health Communication Branch Web site. Available at: http://www2a.cdc.gov/STITraining/Ready-To-Use/userLogin.asp.

13. Arol SO. Determinants of STD epidemics: Implications for phase appropriate intervention strategies. *Sex Transm Infect*. 2002;78(1 suppl):i3–13.

14. D'Souza CM, Shrier LA. Prevention and intervention of sexually transmitted diseases in adolescents. *Curr Opin Pediatr*. 1999;11:287–291.

15. *2002 Department of Defense Survey of Health Related Behaviors Among Military Personnel*. Research Triangle Park, NC: Research Triangle Institute; 2003. RTI/7841/006-FR.

16. Shafer M, Boyer CB, Shaffer RA, et al. Correlates of sexually transmitted diseases in a young male deployed military population. *Mil Med*. 2002;167:496–500.

17. Knapp JS, Fox KK, Trees DL, Whittington WL. Fluoroquinolone resistance in *Neisseria gonorrhoeae*. *Emerg Infect Dis*. 1997;3:33–39.

18. US Department of Defense. *Criteria and Procedure Requirements for Physical Standards for Appointment, Enlistment, or Induction in the Armed Forces.* Washington, DC: DoD; 2005. DoD Instruction 6130.4. Available at: www.dtic.mil/whs/directives/corres/pdf/i61304_011805/i61304p.pdf. Accessed October 13, 2005.

19. Clark KL, Kelley PW, Mahmoud RA, et al. Cost-effective syphilis screening in military recruit applicants. *Mil Med*. 1999;164:580–584.

20. Nagara BE. Completeness and timeliness of reporting hospitalized notifiable conditions, active duty servicemembers, US Army medical treatment facilities 1998–2003. *Med Surveillance Monthly Rep.* 2004;10:9–13.

21. Nagara BE. Completeness and timeliness of reporting hospitalized notifiable conditions, active duty servicemembers, US Navy Medical Treatment Facilities 1998–2003. *Med Surveillance Monthly Rep.* 2004;10:14–17.

22. Nagara BE. Completeness and timeliness of reporting hospitalized notifiable conditions, active duty servicemembers, US Air Force Medical Treatment Facilities 1998–2003. *Med Surveillance Monthly Rep.* 2004;10:18–21.

23. Gaydos CA, Howell MR, Pare B, Clark KL, et al. *Chlamydia trachomatis* infections in female military recruits. *N Engl J Med*. 1998;339:739–744.

24. Brodine S, Shafer M. Combating *Chlamydia* in the military: Why aren't we winning the war? *Sex Transm Dis*. 2003;30:545–548.

25. Clark KL, Howell MR, Li Y, et al. Hospitalization rates in female US Army recruits associated with a screening program for *Chlamydia trachomatis*. *Sex Transm Dis*. 2002;29:1–5.

26. Gaydos CA, Howell MR, Quinn TC, McKee KT Jr, Gaydos JC. Sustained high prevalence of *Chlamydia trachomatis* infections in female army recruits. *Sex Trans Dis*. 2003;30:539–544.

27. Emerson LA. Sexually transmitted disease control in the Armed Forces, past and present. *Mil Med*. 1997;162:87–91.

28. Jenkins PR, Jenkins RA, Nannis ED, et al. Reducing risk of sexually transmitted disease (STD) and human immunodeficiency virus infection in a military STD clinic: Evaluation of a randomized preventive intervention trial. *Clin Infect Dis*. 2000;30:730–735.

29. Minnis AM, Padian NS. Effectiveness of female controlled barrier methods in preventing sexually transmitted infections and HIV: Current evidence and future research directions. *Sex Transm Infect*. 2005;81:193–200.

30. Valappil T, Kelaghan J, Macaluso, et al. Female condom and male condom failure among women at high risk of sexually transmitted diseases. *Sex Transm Dis*. 2005;32:35–43.

31. Phillips DM, Sudol KM, Taylor CL, et al. Lubricants containing N–9 may enhance rectal transmission of HIV and other STIs. *Contraception*. 2004;70:107–110.

32. Bond MM, Yates SW. Sexually transmitted disease screening and reporting practices in a military medical center. *Mil Med*. 2000;165:470–472.

33. Harper DM, Franco EL, Wheeler C, et al. Efficacy of a bivalent L1 virus-like particle vaccine in prevention of infection with human papillomavirus types 16 and 18 in young women: A randomized trial. *Obstet Gynecol Surv*. 2005;60:171–173.

34. Kimberlin DW, Rouse DJ. Genital herpes. *N Engl J Med*. 2004;350:1970–1977.

35. Centers for Disease Control and Prevention. *Sexually Transmitted Disease Surveillance, 2003*. Atlanta, Ga: US Department of Health and Human Services; 2004.

36. Centers for Disease Control and Prevention. *Tracking the Hidden Epidemics 2000: Trends in STDs in the United States.* Atlanta, Ga: US Department of Health and Human Services; 2000.

37. Dambro MR. *Griffith's 5 Minute Clinical Consult*. 13th ed. Philadelphia, Pa: Lippincott, Williams and Wilkins, 2005.

38. Zenilman J, Glass G, Shields T, et al. Geographic epidemiology of gonorrhoea and *Chlamydia* on a large military installation: Application of a GIS system. *Sex Transm Infect*. 2002;78:40–44.

39. Sena AC, Miller WC, Hoffman IF, et al. Trends of gonorrhea and chlamydial infections during 1985–1996 among active duty soldiers at a US Army installation. *Clin Infect Dis*. 2000;30:742–748.

40. Brodine SK, Shafer MA, Shaffer RA, et al. Asymptomatic sexually transmitted disease prevalence in four military populations: Application of DNA amplification assays for *Chlamydia* and gonorrhea screening. *J Infect Dis*. 1998;178:1202–1204.

41. Sutton TL, Martinko T, Hale SP, Fairchok MP. Prevalence and high rate of asymptomatic infection of *Chlamydia trachomatis* in male college reserve officer training corps cadets. *Sex Transm Dis*. 2003;30:901–904.

42. Cecil JA, Howell MR, Tawes JJ, et al. Features of *Chlamydia trachomatis* and *Neisseria gonnorrhea* infection in male Army recruits. *J Infect Dis*. 2001;184:1216–1219.

43. Smith JS, Munoz N, Herrero R, et al. Evidence for *Chlamydia trachomatis* as a human papillomavirus cofactor in the etiology of invasive cervical cancer in Brazil and the Philippines. *J Infect Dis*. 2002;185:324–331.

44. Koskela P, Anttila T, Bjorge T, et al. *Chlamydia trachomatis* infection as a risk factor for invasive cervical cancer. *Int J Cancer*. 2000;85:35–39.

45. Fleming DT, Wasserheit JN. From epidemiological synergy to public health policy and practice: The contribution of other sexually transmitted diseases to sexual transmission of HIV infection. *Sex Transm Infect*. 1999;75:2–17.

46. Gaydos CA, Quinn TC. Urine nucleic acid amplification tests for the diagnosis of sexually transmitted infections in clinical practice. *Curr Opin Infect Dis*. 2005;18:55–66.

47. Birdsong W. Ectopic pregnancy in a military population. *Mil Med*. 1987;152:525–526.

48. Barnett SD, Brundage JF. Incidence of recurrent diagnoses of *Chlamydia trachomatis* genital infections among male and female soldiers of the US Army. *Sex Transm Infect*. 2001;77:33–36.

49. Blythe MJ, Katz BP, Batteiger BE, Ganser JA, Jones RB. Recurrent genitourinary chlamydial infections in sexually active female adolescents. *J Pediatr*. 1992;121:487–493.

50. Gunderson EK, Garland C, Hourani LL. Infectious disease rates in the US Navy, 1980 to 1995. *Mil Med*. 2001;166:544–549.

51. Centers for Disease Control and Prevention. Increases in fluoroquinolone-resistant *Neisseria gonorrhoeae* among men who have sex with men—United States, 2003, and revised recommendations for gonorrhea treatment, 2004. *MMWR Morb Mortal Wkly Rep*. 2004;53:335–338.

52. US Public Health Service, Centers for Disease Control and Prevention, Division of STI Prevention, National Center for HIV, STI and TB Prevention. Primary and secondary syphilis: United States, 1999. *MMWR Morb Mortal Wkly Rep*. 2001;50:113.

53. Centers for Disease Control and Prevention. Summary of notifiable diseases—United States, 2003. *MMWR Morb Mortal Wkly Rep*. 2005;52:1–85.

54. McKee KT Jr, Burns WE, Russell LK, et al. Early syphilis in an active duty military population and the surrounding civilian community, 1985–1993. *Mil Med*. 1998;163:368–376.

55. Thomas RJ, MacDonald MR, Lenart M, et al. Moving toward the eradication of syphilis. *Mil Med*. 2002;167:489–495.

56. Pickering L, ed. *Red Book: 2003 Report of the Committee on Infectious Diseases*. 26th ed. Elk Grove Village, Ill: American Academy of Pediatrics; 2003.

57. Terris MK. *Urethritis*. Available at: http://www.emedicine.com/med/topic2342.htm. Accessed March 15, 2005.

58. Heymnan DL, ed. *Control of Communicable Diseases Manual*. 18th ed. Washington, DC: American Public Health Association; 2000.

59. Soper D. Trichomoniasis: Under control or undercontrolled? *Am J Obstet Gynecol*. 2004;190:281–290.

60. Swygard H, Sena AC, Hobbs MM, Cohen MS. Trichomoniasis: Clinical manifestations, diagnosis and management. *Sex Transm Infect*. 2004;80:91–95.

61. Corey L, Handsfield HH. Genital herpes and public health, addressing a global problem. *JAMA*. 2000;283:791–794.

62. Wald A, Zeh J, Corey L. Virological characteristics of subclinical and symptomatic genital herpes infections. *N Engl J Med*. 1995;333:770–775.

63. US Preventive Services Task Force. *Screening for Genital Herpes: Recommendation Statement*. Rockville, Md: Agency for Healthcare Research and Quality; 2005.

64. Stafford EM, Stewart RS Jr, Teague GR, et al. Detection of human papillomavirus in cervical biopsies of summer camp ROTC cadets with abnormal papanicolaou smears. *J Pediatr Adolesc Gynecol*. 1996;9:119–124.

65. Christensen ND. Emerging human papillomavirus vaccines. *Expert Opin Emerg Drugs*. 2005;10:5–19.

66. Maclean J, Rybicki EP, Williamson AL. Vaccination strategies for the prevention of cervical cancer. *Expert Rev Anticancer Ther*. 2005;5:97–100.

67. Roucoux DF, Wang B, Smith D, et al. A prospective study of sexual transmission of human T lymphotropic virus (HTLV)-I and HTLV-II. *J Infect Dis*. 2005;191(9):1490–1497.

68. Kuehn BM. New human retroviruses discovered: Evidence that cross-species leap not a rare event. *JAMA*. 2005;293(24):2989–2990.

69. Centers for Disease Control, National Center for HIV, STD and TB Prevention, Division of HIV / AIDS Prevention. *Cases of HIV Infection and AIDS in the United States, 2003.* Atlanta, Ga: CDC; 2005. HIV / AIDS Surveillance Report, Vol. 15.

70. CDC. Updated US Public Health Service Guidelines for the Management of Occupational Exposures to HIV and Recommendations for Postexposure Prophylaxis. MMWR Morb Mortal Wkly Rep. 2005; 54 (No. RR–2).

71. Rezullo PO, Sateren WB, Garner RP, et al. HIV-1 seroconversion in the United States Army active duty personnel, 1985–1999. *AIDS*. 2001;15:1569–1574.