Perinatal Transmission of Bacterial Sexually Transmitted Diseases

Part II: Group B Streptococcus and *Chlamydia trachomatis*

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Sexually transmitted diseases (STDs) have reached epidemic proportion in the United States and have captured the attention of both laypersons and health care professionals. Of special concern is that most STDs can be transmitted vertically to the offspring of infected mothers. Since the advent of acquired immunodeficiency syndrome, other STDs have been at risk of being relatively disregarded. This paper, the second of two parts, reviews issues of prevalence, morbidity, mortality, diagnosis, prevention, and treatment of group B streptococcal and chlamydial infections as they affect the maternal-fetal dyad. **J Fam Pract 1990; 30:689-696.**

A striking increase in the incidence of four major sexually transmitted diseases (STDs) is having a significant impact on a growing population of sexually active young persons. Two of the four STDs, syphilis and gonorrhea, were the subjects of the first part of this article.[1] Group B streptococcus and *Chlamydia trachomatis* are considered in this concluding part.

GROUP B STREPTOCOCCUS

The group B streptococcus, or *Streptococcus agalactiae*, is currently the most common cause of sepsis and meningitis in the neonate and young infant.[2] Its meteoric rise in importance since its delineation as a neonatal pathogen by Hood and coworkers[3] in 1961 has not been explained, but it is possible that some of the β-hemolytic streptococcal infections of mothers and infants ostensibly thought to be due to Lancefield group A earlier in the century may have been due to *S agalactiae*.

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Epidemiology and Spectrum of Disease

Group B streptococcus may be a sexually transmitted organism, and thus is included in this paper. The question has been raised, however, as to whether infection with the organism might not simply be a zoonosis.[4] This bacterium is a cause of mastitis and other infections in animals and might present a situation similar to that of *Listeria monocytogenes*, in which human cases are linked with animals through the food chain, especially dairy products.[5] Perhaps food is contaminated with group B streptococcus, which colonizes the gut, from which the vagina is secondarily colonized in women and the urethra in both sexes. Culturing both vagina and rectum has been shown to be important in identifying colonized women.[6] Group B streptococcus has also been demonstrated to be a urinary pathogen in both men and women.[7]

Sexual transmission of group B streptococcus is consistent with the finding of frequent urethral colonization among male consorts of culture-positive women.[8] Risk factors for colonization in women include the presence of other STDs such as trichomoniasis.[9] Group B streptococcus is also clearly a pathogen in antepartum urinary tract infections,[10] postpartum endometritis and puerperal sepsis,[11,12] and chorioamnionitis.[13] Early work suggested that antenatal antimicrobial treatment of both colonized mother and consort was efficacious,[8] but more recent data suggest such treatment is ineffective.[6] Thus, although broad consensus exists that "there is a strong relationship

between sexual activity and an increased frequency of GBS [group B streptococcus] colonization,"[14] much remains to be learned about the epidemiology of the sexually transmitted pathogenesis of this organism.

As described by Baker and others[15] in the early 1970s, neonatal group B streptococcal infections encompass two distinct clinical syndromes. Early-onset disease presents within 48 hours (mean age 20 hours) after birth and is associated with maternal factors that include premature rupture of membranes, premature labor and birth, multiple births, high group B streptococcal inoculum, and bacteriuria.[8,10] Late-onset disease occurs beyond the initial 7-day period (mean age of onset 24 days) in infants whose mothers did not typically have obstetric complications.[10] The most common manifestations of early-onset disease are septicemia, pneumonia, and meningitis, whereas meningitis is the most common clinical expression of late-onset disease, but septic arthritis, osteomyelitis, cellulitis-adenitis, pneumonia, pleural empyema, endocarditis, urinary tract infection, and endophthalmitis may also be seen.[10]

The incidence of early-onset disease is approximately 2 cases (range 1.3 to 3.7) per 1000 live births; the incidence of late-onset disease is about 0.7 to 1.0 per 1000. Approximately 15% of early cases are fatal, whereas late-onset disease is associated with about a 7% to 10% mortality. Some 50% of survivors of group B streptococcal neonatal disease will suffer permanent neurodevelopmental defects.[2,16]

Early-onset infection is acquired by the infant either just before delivery (through intact or ruptured membranes) or during birth passage.[10] Infection has been shown to be well established in the fetus before the first hour of life.[17] From 15% to 35% of pregnant women are colonized at delivery.[2] Colonization may be intermittent and is more common in younger women.[14] The transmission rate from untreated culture-positive women to their offspring is estimated to be from 45% to 75%.[10,14] Overall, early-onset disease occurs in 1 infant per 100 to 200 colonized women.[18]

Diagnosis

The standard method of establishing the diagnosis of neonatal group B streptococcal disease is from cultures of blood, cerebrospinal fluid, or other suppurative foci. Isolation of the organism from surface sites in the absence of local inflammation, or from mucous membranes in the absence of bacteremia, is clinically insignificant. Rapid diagnostic tests (eg, latex particle agglutination) may provide a presumptive diagnosis based upon detection of polysaccharide antigens. These tests may be diagnostically useful even after antimicrobial therapy has been initiated.[18]

Treatment and Prevention

Current recommendations for initial treatment of neonatal group B streptococcal infection include high-dose penicillin or ampicillin plus an aminoglycoside for presumptive life-threatening infection. Many authorities recommend continuation of both antibiotics until the group B streptococcus isolate is demonstrated to be penicillin-sensitive. Treatment is for 10 to 14 days in infants with bacteremia and no focus and 14 to 21 days in infants with meningitis. A second lumbar puncture should be performed 24 hours after initiation of treatment for meningitis to document bacteriologic cure. A longer period of treatment is advised for musculoskeletal infections, depending on the clinical severity, response to treatment, and complications.[18]

In view of the risk of neonatal disease, a number of clinical trials have attempted to interdict the vertical transmission of group B streptococcus from the colonized mother to infant by the administration of antibiotics. A study of low birthweight infants from Chicago showed that a single injection of penicillin G given to neonates within 60 minutes of birth did not prevent early-onset disease, nor did it reduce associated excess mortality.[17] A more recent investigation found a group B streptococcus carriage rate of 23% among 13,831 pregnant women.[19] The study selected culture-positive women at high risk for group B streptococcal transmission because of preterm labor or premature rupture of membranes and randomized about one half of mothers and their babies to treatment with ampicillin and one half to no antibiotic. Treated newborns had fewer positive surface cultures and no positive blood cultures compared with untreated babies (who had 5 of 79 positive blood cultures). In a smaller study, Teres et al[9] confirmed these findings.

The American Academy of Pediatrics has recommended that group B streptococcal chemoprophylaxis be considered on an individual case or hospital basis. Their expert committee notes that parenteral administration of ampicillin to high-risk, colonized pregnant women throughout labor has resulted in decreased transmission of group B streptococcus and disease in offspring.[18] Minkoff and Mead[20] have recommended culturing for group B streptococcus all patients admitted for preterm labor or premature rupture of membranes. All those who are culture-positive are treated with ampicillin; those whose labor cannot be inhibited are treated presumptively, pending culture results. Yet, while this approach may be helpful for high-risk colonized women who have relatively more babies with group B streptococcus disease, as Baker and Edwards[10] have pointed out, such a plan is not helpful for full-term infants, who make up 70% to 80% of group B streptococcal cases. Intrapartum as opposed to prenatal screening with latex agglutination antigen detection has

been proposed,[21] but the cost and suboptimal sensitivity of these tests remain obstacles.

One promising approach to the prevention of group B streptococcal disease supplants reliance upon antibiotics with the potential of immunoprophylaxis. A recent study by Baker et al[22] has shown that maternal prenatal immunization for group B streptococcus is feasible and can provide passive immunity against systemic infection with the type III organism in the majority of newborns. Although these preliminary results demonstrated suboptimal immunogenicity, immunoprophylaxis shows definite promise. Primary care physicians involved in the care of pregnant women or their infants will need to stay abreast of evolving recommendations for the management of the pregnant patient colonized with group B streptococcus.

CHLAMYDIA TRACHOMATIS

From 3 to 5 million Americans are newly infected with *Chlamydia trachomatis* each year.[23,24] The annual cost, direct and indirect, of treating mothers and babies infected with *C trachomatis* in the United States has been estimated to approach $1.5 billion.[23] Between 1 in 10 and 1 in 20 US women will have chlamydial endocervical infection documented during pregnancy,[24] resulting in about 155,000 infants born to infected mothers each year.[23] This microorganism is now generally considered to be the most common sexually transmitted pathogen in western societies,[25,26] and is said to infect 10% to 20% of sexually active adolescent girls.[23] Chlamydial infection may coexist in up to 45% of patients presenting for treatment of gonorrhea.[27,28]

Epidemiology and Transmission

Chlamydiae, previously termed *Bedsonia,* are peculiar bacteria that are obligate intracellular parasites. *C trachomatis* seems to be a specific pathogen for humans,[29] and 15 serotypes have been recognized.[30] Long before modern laboratory study, it was epidemiologically observed that nongonococcal urethritis in men was related to cervical infection in women, which in turn was related to ophthalmia neonatorum in infants born vaginally.[29]

Vertical transmission of chlamydia from a seronegative mother to her infant has not been reported in the United States, and in utero transmission is not known to occur when membranes are intact. The transmission is intrapartum; infants born by cesarean section are not at risk for *C trachomatis* infection unless membranes have ruptured prematurely.[31]

Reported prevalence of chlamydial infection varies among subpopulations, depends upon how the organism is sought diagnostically, and is associated with certain high-risk factors. One very large study in a San Francisco obstetrics clinic revealed a prevalence of about 5%.[32] A New Jersey study of 205 prepaid health plan patients at a family practice center yielded a prevalence of 5.4%.[33] A Kansas City family practice clinic reported prevalence figures of 12% among 54 men and 28% among 282 women who reported for evaluation of urogenital symptoms or an annual examination.[34] From an urban Baltimore clinic an average prevalence of 26% (35% in men, 27% in pregnant women, and 23% in nonpregnant women) was reported among sexually active adolescents.[35] Investigators from Ohio State University reported a prevalence of *C trachomatis* of 19% among sexually active girls and 2% among girls virginal by history.[36]

Chlamydial infection apparent at birth, resulting from ascending infection associated with premature rupture of membranes, is quite rare.[31] Underestimated in the past, *C trachomatis* is now known to be a common cause of infections in the first 6 months of life. Estimates of vertical transmission rates in the United States, ranging from 18% to 74%, have been hampered by the nonspecific nature of urogenital symptoms in women with chlamydial infections and the fact that many endocervical infections are asymptomatic.[23,29,37] In addition, laboratory methods for detection of chlamydia are only recently widely available.

Attack rates of neonatal chlamydial disease have now been relatively well defined. Perhaps 60% to 70% of progeny exposed to chlamydia will show serologic evidence of transmission, but only a subset of these will actually manifest overt infectious disease.[32] Overall, about one third of infants born to infected mothers will themselves be infected at some epithelial site.[31] Conjunctivitis will develop in about 30% to 40% of exposed infants, and pneumonia will occur in approximately 10% to 20% of exposed neonates.[23] In addition, from 15% to 20% of the infants will develop nasopharyngeal infection.[31]

Pathogenesis

C trachomatis has been isolated from the human salpinx,[38] and the organism is linked to ectopic pregnancy.[39] Chlamydia has been shown to be a pathogen of both the upper and lower genital tracts.[40] Chlamydial amnionitis and untoward outcomes of pregnancy are theoretically possible because amniotic cells have been shown to support growth of the organism quite well.[41]

Several studies have indeed suggested an association between *C trachomatis* and untoward outcomes of pregnancy. Gravett et al[42] followed 534 gravid women prospectively and found that cervical infection with *C trachomatis* was associated with premature rupture of membranes, preterm labor, and low birthweight. Yet in a larger study, Harrison et al[43] found that cervical infection

with *C trachomatis* did not predict low birthweight, abortion, stillbirth, premature rupture of membranes, or premature delivery. Evidence from other studies is very suggestive that such a relationship exists, if it can only be clarified.[44,45] Furthermore, it has been shown that *Mycoplasma hominis* and *Ureaplasma urealyticum* infections often coexist with *C trachomatis*; the precise relationship of one or interrelationship of all of these organisms to untoward outcomes also remains unclear.[31]

New infections may be more significant than old. Two studies have suggested that a subset of immunoglobulin M (IgM)-seropositive chlamydia-infected women had a higher incidence of premature rupture of membranes[43] and more low birthweight infants[43,46] than IgM-seronegative or uninfected women. This evidence, together with that of Kass et al,[47] may indicate that women with recent chlamydial infections may suffer more untoward outcomes of pregnancy than women with chronic chlamydial infection.[46]

Clinical Spectrum of Disease

Among adults, *C trachomatis* has been reported to cause cervicitis, salpingitis, endometritis, bartholinitis, prepubertal vaginitis, peritonitis, perihepatitis (Fitz-Hugh–Curtis syndrome: probably more commonly than *Neisseria gonorrhoeae*), Reiter syndrome, arthritis, endocarditis, epididymitis, urethral syndrome, and urethritis.[31,48,49] It is estimated that among women who attend gynecology clinics and who are infected with *N gonorrhoeae, C trachomatis* may be concomitantly recovered up to 62% of the time, and that women culture-positive for *C trachomatis* may be asymptomatic from 44% to 77% of the time.[23,50] Evidence indicates that pelvic infection with *C trachomatis* may be associated with a greater likelihood of infertility than infection with *N gonorrhoeae*.[39,51]

C trachomatis previously has been implicated as the etiologic pathogen in about one third of cases of neonatal conjunctivitis.[52] Incidence varies, however, with the maternal population, and *C trachomatis* was recently documented in nearly 50% of cases of ophthalmia neonatorum in urban Baltimore.[53] The infection has a usual incubation period of 5 to 14 days (up to 19 days) postpartum. Bilateral in about one half of cases,[54] the disease manifests as thickened, erythematous conjunctivae with lid edema and a watery discharge that subsequently becomes mucopurulent.[29] Untreated, *C trachomatis* conjunctivitis tends toward spontaneous resolution in several months. Loss of vision is rare (except in world regions where trachoma is endemic).

C trachomatis pneumonitis, described originally by Schachter et al[55] and Beem and Saxon,[56] is one of the most common pneumonias seen in infancy, and is usually recognized between the 4th and 11th weeks of life. A recent prospective study of 205 infants hospitalized for pneumonitis when younger than 3 months of age documented *C trachomatis* as the cause in about one third of cases.[57] The infected baby usually has minimal or no fever and presents with nasal obstruction, abnormal tympanic membranes, a paroxysmal cough, rales, and, occasionally, apneic spells. Wheezing is uncommon. Chest radiography typically shows hyperinflation with bilateral interstitial infiltrates. The white cell count is typically normal with an increased proportion of eosinophils.[29,56] Conjunctivitis will be present in from 10%[57] to about 50%[58] of cases of pneumonia. Untreated pneumonia may persist for many weeks and be associated with irritability and weight loss, but mortality is rare, and the usual course is recovery without sequelae[32]; however, recent studies have shown abnormal pulmonary function and respiratory symptoms in some children followed for years after recovery from chlamydial pneumonia.[57,59]

Bronchiolitis, enteritis, and otitis media in infancy are infectious syndromes less firmly associated with *C trachomatis*. The significance of vaginal and rectal colonization of babies by *C trachomatis* is unclear.[31]

Diagnosis

Diagnosis of *C trachomatis* may be made by several means. Giemsa staining may reveal intraepithelial inclusions. This technique is quite sensitive for conjunctivitis but accurate only about 20% of the time for the diagnosis of cervicitis.[31] Serodiagnosis and visualization of inclusions on routine Papanicolaou smears are generally ineffective diagnostic methods for adult genital infections.[60] Urinary dipstick testing for leukocyte esterase may be helpful to detect chlamydial (or gonococcal) urethritis in males.[61]

The most established technique for diagnosis is growth of the organism on tissue culture; however, cell culture methodology is slow, rather expensive ($54 per culture at Medical College of Georgia Hospital, 1990), and rather complicated. Because of these difficulties, alternative noncultural antigen detection tests for the diagnosis of chlamydia have become available in recent years. The two "rapid" techniques that have been most studied are the direct monoclonal fluorescent-antibody staining (DFA) and enzyme-linked immunoassay (ELISA) tests. Both identify chlamydial antigen and thus obviate the need to transport viable organisms in specimens on dry ice.

Published experience with antigen detection (noncultural) methods is growing (Table 1). In general, the ELISA test evaluated yielded a higher sensitivity while the DFA test showed greater specificity. Stamm[60] has recommended culture as the best method for screening low-risk populations; in groups where the prevalence ex-

TABLE 1. NONCULTURAL ANTIGEN DETECTION COMPARED WITH CELL CULTURE DETECTION OF CHLAMYDIA TRACHOMATIS IN WOMEN

Study	Type of Test	Prevalence of *Chlamydia* in Population	Sensitivity (%)	Specificity (%)	Positive PV* (%)	Negative PV (%)
Stamm[60] (review of 27 studies	DFA†	High (15%–26%)	90	95	90	98
	ELISA‡	High	89	95	80	98
	DFA	Intermediate (9%–11%)	77	97	79	98
	ELISA	Intermediate	85	97	70	98
Hipp et al[62]	DFA	14.7%	73	99	95	96
	ELISA		83	98	84	98
Binns et al[63]	DFA	10.8%	89	99	78	99
	ELISA		96	95	69	99.5

*PV—Predictive value
†DFA—Direct fluorescent antibody
‡ELISA—Enzyme-linked immunoassay

ceeds 10%, both DFA and ELISA tests offer acceptable diagnostic efficacy.

There are significant problems, however, with all diagnostic methods for chlamydia.[28] Proper cold transport of specimens is crucial for reliable cell culture, and this technique is sometimes undependable as a reference standard. The DFA technique requires an expensive fluorescence microscope, obtaining an adequate cell sample is problematic, and the results can be easily misread by an inexperienced microscopist.[60,63] ELISA tests provide advantages for laboratories performing larger numbers of tests and generally generate more objective endpoints that may be read spectrophotometrically (Chlamydiazyme) or macroscopically. Certain ELISA tests (eg, Test Pack, Sure Cell) can be run in less than 30 minutes. The Chlamydiazyme test, however, requires several hours to complete and involves pipetting techniques that can be troublesome,[62] while other tests (eg, Sure Cell), in the experience of the authors, yield a macroscopic endpoint that can be very difficult to objectify. In addition, ELISA tests are potentially problematic because sample adequacy cannot be assessed. Costs of antigen detection tests are generally less than cell culture but vary with the number of specimens processed. There is hope for further refinement of existing noncultural diagnostic techniques, and newer tests for rapid diagnosis of chlamydia, such as deoxyribonucleic acid (DNA) hybridization,[64] are forthcoming.

Serodiagnosis of *C trachomatis* in adults is generally impaired by lack of distinction between remote and recent infection. For neonates, serologic examination is unreliable for the diagnosis of conjunctivitis, but elevated, specific IgM titers (>1:64) are reliably present with pneumonia. Practically, newborns are usually diagnosed indirectly by assessment of chest radiography, presence of conjunctivitis, eosinophilia, and increased nonspecific immunoglobulins (IgG, IgA, IgM) in the setting of suggestive maternal history.[29]

Treatment and Prevention

Chlamydial ophthalmia neonatorum may respond to careful topical treatment with erythromycin or tetracycline or sulfonamide preparations used four times daily for 2 weeks. Failure rates of 50% have been reported, however, and parental compliance is a problem. Thus, oral erythromycin (40 to 50 mg/kg daily) is recommended in four divided doses for 2 weeks. Topical therapy may be used adjunctively.[29,65]

There is convincing evidence that erythromycin (40 to 50 mg/kg daily, orally or intravenously) shortens the course of pneumonia and decreases nasopharyngeal shedding of organisms in infants.[29,65] Sulfisoxazole (100 to 150 mg/kg daily, orally or intravenously, used after 1 month of age) was as effective as erythromycin in one study.[66] Oxygen and ventilatory support are required in only a minority of cases.[29]

Newborn ocular prophylaxis against *C trachomatis* remains an area of controversy. Many hospitals have changed from use of silver nitrate to topical erythromycin

TABLE 2. TREATMENT OF CHLAMYDIAL INFECTIONS DURING PREGNANCY

Drug	Status	Dose Given 4 Times Daily (mg)	Length of Therapy (d)
Erythromycin base	Recommended	500	7
Erythromycin ethylsuccinate	Alternative	800	7
Erythromycin base	Alternative	250	14
Erythromycin ethylsuccinate	Alternative	400	14
Sulfisoxazole*	Alternative	500	7–10

From MMWR[28] *and* Medical Letter.[65]
NOTE: Erythromycin estolate should be avoided during pregnancy because of potential hepatotoxicity.
**Should not be used near term.*

or topical tetracycline because of data showing silver nitrate to be ineffective against chlamydia.[23] A large study demonstrated 100% efficacy of erythromycin ointment for prevention of chlamydial ophthalmia, whereas among infants treated with silver nitrate, conjunctivitis developed in one third of babies born to culture-positive mothers.[67] Another recent, larger study, however, demonstrated that neither topical erythromycin nor topical tetracycline significantly reduced the incidence of chlamydial ophthalmia as compared with silver nitrate.[68] Such failure may occur because of the long incubation period of *C trachomatis,* which results in "late-onset" (beyond 1 month) conjunctivitis.[31] It has also been shown that ophthalmic erythromycin does not prevent nasopharyngeal *C trachomatis* infection and subsequent pneumonia[67]; it has been suggested that nasopharyngeal seeding may also give rise to late-onset conjunctivitis.[23] Moreover, at least one report has documented the persistence of chlamydial conjunctivitis in about one fifth of babies treated for 2 weeks with oral erythromycin.[53] Experts have, therefore, suggested the key to better neonatal *C trachomatis* prophylaxis is the eradication of maternal infections.[23,68]

Pregnant women infected with *C trachomatis* should be treated with oral erythromycin (Table 2). With compliance, cure rates in excess of 95% may be expected.[23]

Indeed, it has been suggested that if an effective screening strategy for detecting *C trachomatis* were widely available, neonatal chlamydial disease would best be prevented by treating all infected gravid women with erythromycin.[23,31] Schachter et al[69] have reported a study in which 184 women were offered treatment for *C trachomatis* during the third trimester. Only 60 women finished the protocol; 92% of these had negative follow-up cultures. Infection with *Chlamydia* developed in 7% of infants of the treated mothers, compared with 50% of controls. Three percent of women stopped treatment because of drug intolerance. It was concluded that such a regimen was successful, albeit not ideal. It has been suggested that the benefit of the treatment exceeds its cost at a prevalence somewhere between 6% and 8%,[70,71] or in a population at roughly moderate risk for chlamydial urogenital infections.

Screening all pregnant women for *C trachomatis* remains controversial, although it has recently been recommended by the Centers for Disease Control.[28] Phillips et al[72] have suggested three factors—lower educational level, a sexual partner who has had other partners during the preceding 3 months, and endocervical bleeding upon swabbing—as simplified criteria for selection of women for testing in a low-prevalence population. Others would add as additional screening criteria the presence of mucopurulent cervicitis, age 24 years or less, no contraception or a nonbarrier method, a history of STD, a recent new sexual partner, unmarried status, and presence of inflammation on Papanicolaou smear.[24,28,73]

Other general approaches to the prevention of chlamydial infection in neonates include interventions to reduce the infectious reservoir. Physicians should initiate empiric treatment (possibly preceded by diagnostic testing) in both the mother and father of an infected infant and should treat pregnant consorts of men with nongonococcal urethritis.[28] Currently recommended adjunctive therapy for gonorrhea with tetracycline or erythromycin should theoretically reduce the general prevalence of *C trachomatis*. Condoms have been shown to be an effective mechanical barrier, and the spermicide nonoxynol-9 is a partially effective chemical barrier to chlamydia.[74,75]

CONCLUSIONS

There are now about 30 pathogens and perhaps 50 diseases and syndromes classified as sexually transmitted. According to some experts,[76] these STDs account for 13 million cases, 7000 deaths, and $4 billion in costs of treatment annually in the United States.

Sexually transmitted diseases often impose a cruel double jeopardy: What begins as an adult problem becomes a significant and even mortal problem for the offspring of infected parents. While rates of transmission, morbidity, and mortality will be revised as new data are collected and reported, one principle will remain unchanging: Fetal or neonatal infections with *Treponema pallidum, Neisseria gonorrhoeae, Chlamydia trachomatis,* and possibly group B streptococcus are preventable. As a larger percentage of teenagers are initiating sexual intercourse at younger

ages, and as only an estimated 10% of primary care providers regularly assess the sexual behaviors of their patients,[26] sober consideration of some of the difficulties that accompany attempts at treating these infections should cause family physicians to ask how they may effectively engage themselves in, as Bayer[77] has written, "the long, hard task of fostering a culture of sexual responsibility in the face of [STD]."

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