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. . . But the Bugs Bounce Back: Simple Transmission Models and 'Failure' of Bacterial STD Control Programs

Genital tract infections caused by Neisseria gonorrhoeae and Chlamydia trachomatis are a major cause of sexually transmitted disease. Although infections caused by these pathogens differ in important respects, there is overlap in the spectrum of associated illness, the limited duration of immunity conferred by infection (and hence the possibility of re-infection), and in the focus of control efforts on identification and treatment of infectious individuals. For both pathogens surveillance data suggest that incidence has increased after initially falling in the face of intensified control efforts. We evaluate the likely mechanisms behind such rebound using simple compartmental "susceptible-infectious-recovered-susceptible" (SIRS) models (Chlamydia) or SIS models (gonorrhoea), and explore the implications of such rebound for disease control practice. In the case of Chlamydia, we model apparent rebound in prevalence in the Philadelphia High School STD Screening Program. We show that the degree of rebound observed could be explained by truncation of transient immunity through treatment, through "risk compensation" (increased sexual risk-taking), or through reduced participation in screening programs, but that neither the presence of rebound, nor the underlying mechanism leading to rebound, substantially diminishes the economic attractiveness of Chlamydia screening. For gonorrhoea, we explore SIS models that include and exclude the possibility of antibiotic resistance. When antibiotic resistance is not possible, strategies that focus on treatment of highest risk individuals (the so-called "core group") result in collapse of disease transmission; however, when antimicrobial resistance exists, a focus on the core group causes rebound in incidence, with maximal dissemination of antibiotic resistance.