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Analysis of *Chlamydia trachomatis* serovar distribution changes in the Netherlands (1986–2002)

Up to 19 different *Chlamydia trachomatis* (CT) serovars which are pathogenic predominantly for the urogenital tract and numerous CT variants have been identified.[1 2] An increasing number of isolates are typed worldwide and provide a wealth of information on the epidemiology of CT infections, a sexually transmitted disease (STD) for which screening has been proposed.[3–5] Recent studies have demonstrated an association between CT serovar G and squamous cell carcinoma.[6] A possible shift in the serovar distribution over time in a region or country could reveal information on changes in the epidemiology of CT infections and could potentially have clinical implications.

We therefore determined the CT serovar distribution in a large STD population in Amsterdam in 2000–2 and compared it together with all published serovar distributions since 1986 in the Netherlands to assess if serovar distribution shifts over time occurred.

Of people attending the STD outpatient clinic in Amsterdam from 2000–2, those found CT positive (n = 407) by LCx (Abbott Laboratories, Chicago, IL, USA) were genotyped as described previously.[1] This is the largest STD population typed to date in The Netherlands. The following serovar distribution was found: B = 1%; D = 12%; Da = 0.2%; D- = 1%: E = 33%; F = 23%; G = 4%; Ga = 5%; H = 8%; I = 6%; Ia = 1%; J = 3%; K = 2%.

Literature searches identified eight serovar distribution studies in the Netherlands, of which the first was performed in 1986. With the inclusion of the present study, 2204 serovars were available for analyses. In the serovar distributions comparison, we (1) did not distinguish between male and female participants, (2) did not distinguish between serovar distributions based on serotyping or genotyping techniques, (3) excluded serovars B/Ba because of the low numbers, (4) excluded double infections, (5) excluded variants, and (6) classified CT serovars in the three phylogenetically based serogroups: the B group (serovars D, Da, D-, E), the intermediate serogroup (serovars F, G, Ga), and the C group (serovars I, Ia, J, Jv, and K).

Results are shown in figure 1. In general, no statistical significant serovar distribution trends in time were observed between 1986 and 2002 when all studies were taken together. Of the nine studies, 1 and 6 represent serovar distributions from STD populations in Rotterdam and show no significant changes in general or over time (mean: C group: 30%; Int group: 21%; B group: 49%). Studies 2, 3, 4, and 9 represent serovar distributions from STD populations in Amsterdam and show no significant changes (mean: C group: 20%; Int group: 31%; B group: 49%). Studies 5, 7, and 8 represent serovar distributions from mixed symptomatic and asymptomatic infected people (5 and 7) and asymptomatically infected populations in Amsterdam. They show no significant changes in general, over time, or compared to the Amsterdam STD based serovar distribution (C group: 17%; Int group: 30%; B group: 53%).

However, when the two geographically derived serovar distributions were compared to each other, (1) serogroup C was found more frequently in Rotterdam: 30 *v* 19% ($p<0.0001$; OR 1.8 (95% CI: 1.4 to 2.3)), the most prominent serovar difference was serovar K (10.6 *v* 3.2%, $p<0.0001$; OR 3.6 (95% CI 2.4 to 5.3)); (2) the intermediate serogroup was found less frequently in Rotterdam: 21 *v* 31% ($p = 0.0002$; OR 1.6 (95% CI: 1.2 to 2.0)), the most prominent serovar difference was serovar F (15 *v* 22%, $p = 0.0018$; OR 1.6 (95% CI: 1.2 to 2.1)), and serogroup B was stable (49% *v* 50%).

In conclusion, no changes in serovar distribution differences were found over time in the Netherlands in general or within the two different geographic areas. However, the Rotterdam population differed significantly from the Amsterdam populations in having a larger incidence of C group serovars and a lower incidence of the intermediate group serovars, albeit an identical B group serovar distribution. The findings could be the result of different ethnic compositions of the studied cohorts or other confounding factors between Rotterdam and Amsterdam, a subject that warrants further study.

Key messages

- No statistically significant serovar distribution shifts were observed between 1986 and 2002 in the Netherlands
- The type of cohort did not influence the analyses: STD based, asymptomatically screenings based, mixed cohorts
- Geographical serovar distribution differences were observed between Rotterdam and Amsterdam but these were stable in time:
  - serogroup C was found more frequently in Rotterdam: 30 *v* 20%, $p<0.0001$, most prominent serovar difference was serovar K (10.6 *v* 3.2%, $p<0.0001$)
  - the Intermediate serogroup was found less frequently: 21 *v* 31%, $p=0.0002$, most prominent serovar difference was serovar F (15 *v* 22%, $p=0.0018$)
  - serogroup B was stable (49% *v* 50%)

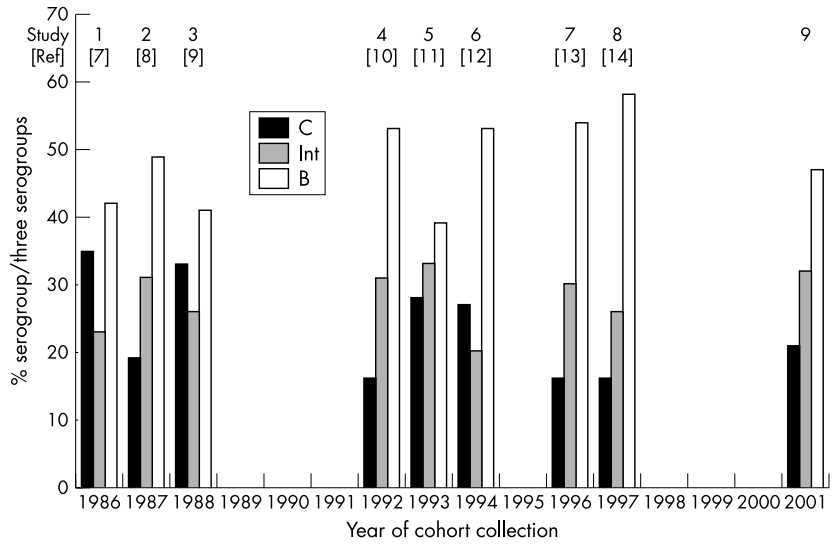

Figure 1 Serovar distribution studies in the Netherlands from 1986 to 2002. The time of cohort collections is shown since the year of publication can be different from the year of cohort collection. Differences in serovar distributions in time were analysed. Each study is indicated by first author, year of publication, and number of isolates included: 1, Wagenvoort, 1998, n = 190; 2, vd Laar, 1996, n = 372; 3, Morré, 1998, n = 90; 4, Ossewaarde, 1994, n = 289; 5, Lan, 1995, n = 51; 6, v Duynhoven, 1998, n = 305; 7, Morré, 2000, n = 426; Morré, 1998, n = 74; 9, Spaargaren, this study, n = 407. C = serogroup C (serovars H, I, Ia/I′, J, Jv, K); Int = intermediate serogroup (serovars F, G, Ga); B = serogroup B (serovars D, Da, D-, E).

Contributors

JS working on *Chlamydia trachomatis* infections, database management, writing of the manuscript; CS responsible for the statistical analyses; IV and SM, technicians performing all chlamydia typing experiments (culture and PCR based RFLP typing) and sample database management; HSAF, in charge of the STD outpatient clinic in Amsterdam, responsible for the logistics of the sample collection, critically reviewing the manuscript; ASP and RAC, providing the setting for the work performed, guidance of JS on this topic, and critically reading the manuscript; SAM, responsible for the study design, direct guidance of JS, critically reading the manuscript.

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Surveillance of sexually transmitted infections in primary care

Surveillance for sexually transmitted infections must respond to increases in the provision of sexual health services outside genitourinary clinics. Simms *et al*[1] propose repeated panel surveys in general practices to improve surveillance in primary care, monitor changes in prevalence over time, and address the current lack of behavioural data.

There are some limitations to this approach. Firstly, prevalence surveys will not measure actual diagnostic activity in primary care and other clinical settings. This is essential for determining whether proposals from the National Strategy for Sexual Health[2] are being implemented effectively. Secondly, periodic surveys in different areas could not readily identify outbreaks. In the Bristol area, for example, most cases in an ongoing outbreak of sexually transmitted hepatitis B infection have presented to general practitioners.[3] Although genitourinary medicine clinics are the main setting for detecting outbreaks their impact in primary care should be monitored. Thirdly, the validity of panel surveys will depend on a high response rate and postal invitations often have low uptake.[4]

A single system cannot fulfil all the requirements for infectious disease surveillance. Laboratory reporting remains incomplete[5] and denominator data need to be available for infections other than chlamydia for appropriate interpretation of time trends. Routine collection of data on laboratory diagnosed sexually transmitted infections from all clinical settings and linkage to demographic data could complement current proposals.

The Avon Surveillance System for Sexually Transmitted Infections (ASSIST) integrates person based genitourinary clinic and laboratory data to provide information for action at local level and to inform national initiatives.[6] Data on positive and negative tests for laboratory diagnosed infections taken in any clinical setting are collected from the Health Protection Agency and trust laboratories. Postcode information for geographical mapping and small area analysis is obtained by matching pseudoanonymised data with GP registration databases. These data are also matched to disaggregate data from genitourinary and Brook clinics to identify duplicate tests and obtain geographic data for infections diagnosed in these settings.

ASSIST project data can be used to estimate the population burden of diagnosed infections and explore associations with demographic and socioeconomic characteristics over time. Automating regular data downloads and reporting will improve the timeliness of data collection to facilitate identification and monitoring of outbreaks. The wide coverage of the system can guide local service development and clinical practice and monitor the impact of the Sexual Health Strategy. For example, in 2001 half of all chlamydia tests and 44% of positive results came from GP, family planning, or Brook clinics. Nearly two thirds (62%) of those tested in general practice were over 25 years old in whom the positivity rate was 4% compared with 11% for under 25 year olds.

We propose that, while behavioural data obtained from panel surveys in primary care provide depth, sentinel surveillance of laboratory diagnosed infections in all clinical settings provides breadth, and both are needed for effective surveillance.

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Comparison of the serological response to treatment of early syphilis in HIV positive versus HIV negative individuals

The effectiveness of treatment for syphilis is evaluated by demonstrating declining titres of the non-treponemal antibody tests—for example, the rapid plasma reagin (RPR). The serological response in HIV co-infected individuals has been the subject of debate, with some studies reporting a similar serological response[1 2] and others a delayed response in HIV positive patients.[3 4]

A resurgence of infectious syphilis has occurred in Manchester, United Kingdom, in recent years.[5] From January 1999 to August 2002, 379 cases of early syphilis were