of exact probabilities of Fisher was carried out. A t student test was also done to determine significant defferencies among age averages. The RR was estimated by means of OR in tables 2×2, and its statistic meaning was determined through a CI at 95%, the aproximation of Woolf was used.

Results The age average in infertile women was of 30.4 years and in fertile woman 24.3 years. (4.486 -07), which is a highly significant difference. Bacterial vaginosis was detected in 72% of the infertile women and 30.9 % of pregnant women. *Candida* spp in 20% of infertile women and 65.4% of the controlled group. The presence of a highly significant statistic difference was proved when Fisher Test was applied. This difference was related to the distribution of microrganisms in both groups ($p<0.0001$). *Mycoplasma hominis* was isolated in 17% of infertile women and in 10% of fertile women. In the group of infertile women, *Ureaplasma urealyticum* was obtained in 42.70% of positive cultures; while in pregnant women, 2% was obtained. *U urealyticum* was not isolated in 57.30% of the infertile woman and in 98% of the pregnant ones. OR 83.92. CL for OR at 95% (17.37 to 397.05) statistically significant. In 54.30% of the infertile women and 98% of the pregnant ones, *Chlamydia trachomatis* was not isolated.

Conclusions The presence of bacterial vaginosis, *U urealyticum*, *C trachomatis* resulted to be a risk factor of female infertility.

P1-S3.08 PELVIC INFLAMMATORY DISEASE (PID) IN ADOLESCENTS AFTER TREATMENT FOR CERVICITIS

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Background We did a prospective study of incarcerated adolescents who had been treated for gonorrhoeal and/or chlamydial cervicitis to determine what proportion subsequently developed PID.

Methods We performed the study at the Harris County Juvenile Detention Center, Texas, where PID is relatively common. At the time of their mandated medical assessment, all incarcerated women submitted first-catch urine samples for chlamydia and gonorrhoea testing. We used Gen-Probe NAAT assays. At the time of treatment among those infected, we performed bimanual pelvic examinations to determine if they had PID. We used the PID diagnostic criteria of the US Centers for Disease Control and Prevention: the presence of adnexal or cervical motion or uterine tenderness. The bimanual examinations were performed by one of three experienced practitioners. We treated infected patients with no evidence of PID for chlamydial and gonococcal cervicitis with 1 gm of azithromycin and 400 mg of cefixime, even if they were positive for only one of the two organisms. We treated for both organisms in case that one test was falsely negative. Treatment was observed by clinic staff; if the medicine was vomited, treatment was repeated following the administration of an anti-emetic. We followed all treated women for 30 days or until released, to determine if they developed PID after treatment for cervicitis. During incarceration, they had no opportunity for sexual intercourse.

Results We evaluated 61 adolescents with no evidence of PID, who were treated for chlamydia and gonorrhoea between 29 March 2010 and 27 December 2010. Their mean age was 15.6 (SD 1.2) years; 45% were black, 31% Hispanic, and 24% white. Duration of follow-up after treatment ranged from 6 to 30 days. During follow-up, 8 of 62 (13%) developed lower abdominal pain and had bimanual pelvic examination findings that supported the diagnosis of PID. All but one patient developed PID at least 10 days after cervicitis treatment (range 3–30 days).

Conclusion In incarcerated adolescents treated for gonorrhoeal and/or chlamydial cervicitis, 13% met the criteria for PID in the month subsequent to treatment, even though their therapy was directly observed, and they were not re-exposed to these organisms. Our data suggest that appropriate treatment for cervicitis does not rule out the possibility of subsequent PID even without exposure to gonorrhoea or chlamydia.

P1-S3.09 ESTIMATING THE INCIDENCE OF PID FOLLOWING CHLAMYDIA INFECTION IN SEX WORKERS

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Background There is a lack of consensus on the true incidence of PID following chlamydia infection with few published prospective studies. We have used data from an old prospective cohort of sex workers to investigate the association between *Chlamydia trachomatis* and subsequent pelvic inflammatory disease (PID).

Methods 598 sex workers were recruited between 1985 and 1993 in London. Self-reported exposure to chlamydia and gonorrhoea at enrolment, and diagnoses of chlamydia, gonorrhoea, trichomoniasis, bacterial vaginosis (BV), candida and PID during the study were recorded. Chlamydia was diagnosed by direct immunofluorescence.

Abstract P1-S3.09 Table 1 Crude rate and crude and adjusted HRs of PID for all women and classified by exposure to chlamydia and gonorrhoea

	Number of PID cases	Crude rate of PID, per 100 women per year (95% CI)	Crude HR (95% CI)	p	Adjusted* HR (95% CI) (*incident case of gonorrhoea, history of chlamydia and gonorrhoea at enrolment)	p
All women	38	11.33 (8.22 to 15.56)				
Incident case of chlamydia						
No	26	10.10 (6.88 to 14.83)	reference		reference	
Yes	12	17.20 (9.77 to 30.28)	1.87 (0.92 to 3.79)	0.083	1.49 (0.65 to 3.40)	0.341
Incident case of gonorrhoea						
No	32	11.07 (7.83 to 15.66)	reference		reference	
Yes	6	12.90 (5.80 to 28.72)	1.36 (0.55 to 3.38)	0.503	0.82 (0.28 to 2.39)	0.715
Past history of chlamydia						
No	13	7.58 (4.40 to 13.06)	reference		reference	
Yes	17	16.89 (10.50 to 27.16)	2.19 (1.06 to 4.54)	0.035	2.06 (0.99 to 4.30)	0.054
Past history of gonorrhoea						
No	14	9.19 (5.44 to 15.51)	reference		reference	
Yes	20	13.34 (8.63 to 20.74)	1.53 (0.77 to 3.06)	0.226	1.75 (0.81 to 3.80)	0.154

Incident infections and PID diagnoses were determined in women who attended clinic more than once and did not have PID at enrolment. Crude incidence rates of PID were calculated. Cox proportional hazards regression was used to investigate the association between chlamydia, gonorrhoea and PID.

Results 275 women with 387 person years (py) of follow-up were diagnosed with 85 cases of chlamydia, 34 cases of gonorrhoea, 25 cases of trichomoniasis, 253 cases of BV and 148 cases of candida. Thirty-eight women had at least one episode of PID and of these, 24 had chlamydia concurrently or preceding their PID. Nine women presented with both chlamydia and PID, 1 had concurrent gonorrhoea and PID, and 3 had gonorrhoea during the 8 weeks preceding their PID. The crude incidence rate was 16.03 cases per 100 py (95% CI 12.50 to 20.56) for chlamydia and 6.72 cases per 100 py (95% CI 4.58 to 9.87) for gonorrhoea. The crude rate and crude and adjusted HRs of PID were calculated, with women censored after their first episode of PID, for all women and for women classified according to their exposure to chlamydia and gonorrhoea (Abstract P1-S3.09 table 1).

Conclusions In this cohort chlamydia was a risk factor for PID. After controlling for other factors, exposure to chlamydia at enrolment was a borderline predictor of PID while incident infection was not associated, possibly due to the small number of incident cases or to the reduced risk because of prompt treatment. The absolute risk of PID after an incident case was higher than recent estimates which may be due to the greater sensitivity of current chlamydia tests. This emphasises the importance of using contemporary parameters when modelling the cost-effectiveness of chlamydia screening.

P1-S3.10 THE ROLE OF CHLAMYDIA TRACHOMATIS IN THE DEVELOPMENT OF SYMPTOMATIC PELVIC INFLAMMATORY DISEASE: A MULTI-PARAMETER SYNTHESIS

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Background *Chlamydia trachomatis* (CT) is an important risk factor for the development of Pelvic Inflammatory Disease (PID), but despite much study the extent of its causal role remains unclear. Knowledge of the role of Chlamydia is critical for assessing the cost-effectiveness of screening programmes.

Methods We perform a Bayesian multi-parameter synthesis of evidence from a range of prospective, retrospective, and routine data sources. These were; randomised trials following screened and unscreened women to PID; retrospective studies of Chlamydia in PID cases and controls, from which an etiologic fraction can be estimated; routine data on the number of PID cases diagnosed in England in 1 year; survey data on the probability of diagnosis of PID. We also used information on the incidence and prevalence of CT in England and the clearance rate of asymptomatic infection.

Results We found that the different data sources provided consistent estimates of the critical parameters. We estimate the probability an episode of Chlamydia causes an episode of PID to be 0.09 (0.04 to 0.16) and that the Excess fraction of PID cases due to Chlamydia to be 0.20 (0.09 to 0.35). We estimate that prior to the start of the current screening program in the UK the annual population incidence of PID in women in England was 0.022 (0.016 to 0.028) in women aged 16–24 and 0.013 (0.009 to 0.019) in women aged 25–44 and that 42% (31% to 54%) of these cases were diagnosed.

Conclusions In the absence of direct data, it is possible to statistically combine evidence from different types of data, to check the consistency of the data sources, and to estimate the relation between CT and PID. In considering the effect on PID risk of treating women who screen positive for Chlamydia, it is important to note that this is a survivor population who acquired CT an unknown time previously and who have not developed PID.

Epidemiology poster session 3: Burden of disease: Neonatal herpes

P1-S3.11 FACTORS ASSOCIATED WITH DEATH AMONG INFANTS WITH NEONATAL HERPES REPORTED IN NEW YORK CITY, 2006–2010

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Background Neonatal herpes simplex virus infection (nHSV) has a high case fatality rate. In New York City (NYC), healthcare providers (HCP) and laboratories report nHSV to the Health Department; incidence is highest among infants born to black non-Hispanic (NH), and young women. We analysed nHSV cases to identify factors associated with death.

Methods nHSV cases in infants <60 days were investigated by HCP interview and abstraction of labour and delivery, outpatient, inpatient, vital records. Bivariate and multivariate models estimated associations between death and demographic, clinical, and provider factors.

Results During 2006–2010, 71 laboratory-confirmed nHSV cases were reported; 43/71 (55%) were male, 29/71 (41%) HSV type 1, 28/71 (39%) HSV type 2, and 14/71 (20%) untyped. Among 58 cases with recorded maternal race/ethnicity, 19 (33%) were white NH, 25 (43%) were black NH, and 15 (26%) were Hispanic. Fourteen of the 71 (20%) cases died; 10 of the deaths (71%) were among infants born to black NH women. There was a significant association between death and disseminated disease (vs skin/eye/mucous membrane, central nervous system, or congenital infection) (OR 25.8, 95% CI 4.5 to 148.9), black NH maternal race (OR 10.0, 95% CI 2.4 to 41.0), preterm birth (OR 7.1, 95% CI 1.9 to 26.5), age <7 days at first symptom (OR 5.8, 95% CI 1.5 to 23.3), maternal age <26 (OR 3.02, 95% CI 0.8 to 10.86), vaginal delivery (OR 0.2, 95% CI 0.1 to 0.8), and presence of infant herpes lesions (OR 0.2, 95% CI 0.04 to 0.6). Infant sex, fever, viral type, admitting hospital type (academic vs non-academic), illness within 24 h of birth, and duration rupture of membranes were not associated with death. After adjusting for preterm birth, black NH maternal race remained associated with a significantly higher risk for death (OR 7.2 95% CI 1.6—to 31.4, p=0.009). Disseminated disease also remained significantly associated with death after adjusting for preterm birth, and age at first symptom (OR 26.6, 95% CI 3.6 to 196.5, p=0.001).

Conclusions Our data suggest a significant racial disparity in nHSV fatality, only partly explained by preterm births among black NH mothers. Further investigation should examine if care-seeking or provider characteristics contribute to these disparities. Black NH maternal race may be a marker for recently acquired herpes infection, with a high organism load at delivery. Disseminated disease is strongly, and independently, associated with fatality.