Results End1/E6E7 HBD1 expression did not respond to stimulation. HBD1 secretion by Ect1/E6E7 cells more than doubled in response to IL-1β (p = 0.0002) and IFNγ (p = 0.02), and was suppressed by progesterone (p = 0.02). End1/E6E7 HBD2 release almost doubled in response to LPS (p = 0.02) and progesterone (p = 0.01). HBD2 expression by Ect1/E6E7 cells doubled in response to LPS (p = 0.005) and halved after stimulation with progesterone (p = 0.006). End1/E6E7 HBD3 expression increased in response to LPS (p = 0.0009), and almost doubled after stimulation with PGN (p = 0.0003) and IFNγ (p = 0.027). End1/E6E7 HBD3 secretion almost tripled in response to incubation with progesterone (p = 0.003).
Conclusion Bacterial products elicited differential expression of HBDs in End1/E6E7 and Ect1/E6E7 cells. Progesterone mediated an increase in HBD2 and HBD3 secretion by endo-cervical cells but suppressed HBD1 and HBD2 secretion by ecto-cervical cells; this effect may augment cervical host defence, representing a novel mechanism by which progesterone may contribute to delaying the onset of preterm labour.

PP.15 MORTALITY, CONGENITAL ANOMALY, & MATERNAL RISK FACTORS ACROSS ETHNIC GROUPS IN BIRMINGHAM

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Background Stillbirth and infant mortality rates in Birmingham remain consistently above those seen in the West Midlands and England & Wales. Over half of the maternity population are from minority ethnic groups.
Methods Numerator data 2006–2010 (deaths/anomalies) were selected from the regional, population-based, anomaly and mortality registers, covering a birth cohort of 85,734**.** Denominator data for 2010, including ethnic group, consanguinity, and folate use, were available within the regional denominator database (www.pi.nhs.uk/peer/peerdata_collection.htm).
Results Deaths from congenital anomaly comprise 29.3% of stillbirths and infant deaths in Birmingham. Mortality rates were significantly higher in Pakistani (odds ratio 3.0) and Bangladeshi mothers (odds ratio 2.1) compared to White Europeans. Pakistani mothers had significantly higher mortality rates from metabolic disorders, neural tube defects, and renal anomalies. In terms or primary and secondary screening, the prevalence of antenatal folate use was low in most minority ethnic groups. 49.4% of stillbirths and infant deaths had at least one anomaly that was amenable to detection by fetal anomaly screening programmes. However, in 5.1% of Birmingham births, the first booking appointment took place at 20 weeks or later. Pakistani mothers have the highest rates of consanguineous unions (49.9%, CI 48.1–51.7, compared to 15.9% (CI 15.3–16) for all ethnic groups combined.
Conclusion Ethnic groups in Birmingham have an excess of perinatal mortality due to congenital anomalies. Interventions for these groups need to focus on improved folate uptake, timely access to screening services, and referral for genetic risk assessment.

PP.16 PREGNANCY OUTCOMES IN WOMEN WITH KNOWN SICKLE CELL DISEASE: A CASE CONTROL STUDY FROM A DEDICATED TERTIARY JOINT OBSTETRIC SICKLE CELL CLINIC

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Introduction Sickle cell disease (SCD) in pregnancy has been associated with adverse pregnancy outcomes. However, with a multidisciplinary approach and surveillance, there is a suggestion of improvement in the overall outcome. The aim of this study was to evaluate the pregnancy outcomes in women with known SCD in a singleton pregnancy seen in our clinic, and to assess whether the presence of SCD alters the maternal serum analytes (Free B-human chorionic gonadotrophin and Pregnancy-associated plasma protein - A) at combined screening.
Methods Case-control study of all pregnant women with SCD undergoing combined screening from 01/01/2008 to 31/12/2011. Each case was matched with 3 non-SCD controls. Pregnancy outcomes in the two groups were compared.

Abstract PP.16 Table

Results	SCD (n = 54)	Controls (n = 165)
Pre-eclampsia, n (%)	7 (12.9) *	3(1.8)
Chronic hypertension, n (%)	1 (1.8)	4 (2.4)
Live birth	53 (98.1)	164 (99.3)
CS, n (%)	20 (37.7)	42 (25.6)
Vaginal, n (%)	33 (62.3)*	122 (74.4)
Gestation at delivery, wks, median (IQR)	38(37–39)	39(38–40)
Birth weight (grammes), median (IQR)	3060* (2698–3280)	AB3345 (3000–3563)
B-HCG MoM, median (IQR)	1.2 (0.65–1.85)*	1.05 (0.62–1.63)
PAPP-A MoM, median (IQR)	0.86 (0.56- 1.17)*	1.08 (0.74–1.54)

*P < 0.05

Conclusion Women with SCD, when followed closely from the first trimester, have successful pregnancy outcomes. However, our data suggests an increased incidence of pre-eclampsia and lower birth weights compared to non-SCD controls. There was also a significant difference noted in the maternal serum analytes. Larger prospective studies are therefore required to assess the impact of SCD on combined screening.

PP.17 PRE-GESTATIONAL DIABETES AND THE RISKS OF FETAL AND INFANT DEATH IN NORMALLY-FORMED OFFSPRING

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Background Pre-gestational diabetes is associated with substantially increased risks of congenital anomalies, but the impact on normally-formed offspring is less well explored. This study explored the risks of fetal and infant death, examining the influence of HbA1c, in normally-formed offspring of women with pre-gestational diabetes.
Methods All normally-formed singleton pregnancies in Northern England delivered during 1996–2008 were identified from the Northern Diabetes in Pregnancy Survey. Fetal (≥20 weeks gestation) and infant deaths were identified from the Northern Perinatal Morbidity and Mortality Survey. Relative risks were estimated by comparing the prevalence rates between those with and without diabetes. The associations between peri-conception and third trimester HbA1c with each outcome were examined by logistic regression.
Results 400,158 normally-formed singletons were registered during the study period, including 1548 in women with pre-gestational diabetes. There were 46 fetal and 10 infant deaths following pregnancies in women with diabetes.