Results: Forty-eight former preterm infants (GA 30.1±2.08 weeks, BW 1372±386g, 51% female) were assessed at age 12.5±1.29 years, and compared to 80 term controls (39.5±1.70wks, 3308±455g, 42% female) at 12.3±1.11 years. 3 children in the preterm and 10 in the term group were small for GA. BMI was similar in both groups (19.1±2.97kg/m² vs 18.6±3.08kg/m²). Systolic and diastolic BP were similar in both groups: 108.5±8.4 and 66.5±5.9mmHg in the preterm, 107.3±11.34 and 65.0±8.98mmHg in the term group (p=0.21 for systolic, p=0.64 for diastolic BP). One boy each in the preterm and term group had systolic BP >99th centile for age and height centile, and were overweight. All other study participants did not have evidence of high blood pressure. In the preterm group, no association could be found between systolic or diastolic BP and GA, BW, days of ventilation, use of NSAR, aminoglycosides, inotropes during neonatal hospitalisation.

Conclusions: No difference in systolic or diastolic BP was found in this study comparing adolescent former preterm infants with term controls. BP in the preterm group did not correlate with neonatal risk factors for adverse renal outcomes.

PLACENTAL INFLAMMATION IS ASSOCIATED WITH ALTERED FETAL IMMUNE RESPONSES AT BIRTH
Stinson LF1, Payne MS1, Keelan JA1
1Division of Obstetrics and Gynaecology, The University of Western Australia, Perth, Australia
Email: lisa.stinson@uwa.edu.au

Background: Intrauterine inflammation has been associated with preterm birth and poor neonatal outcomes, with some evidence of immunomodulation in exposed fetuses. In this study we sought to test the hypothesis that fetal immune maturation and sensitivity is likely to be altered in pregnancies with evidence of feto-placental inflammation.

Methods: Umbilical cord blood was collected from Caesarean section deliveries delivered 34-42 weeks gestation (n=44). Placentas were classified by histopathology as having inflammation in the fetal tissue (n=13), inflammation in the maternal tissue (n=10), or no inflammation (n=23). None of the pregnancies had clinical evidence of intrauterine infection. Cord blood was exposed shortly after delivery (< 2 hours) to a panel of immune stimuli [R848, LPS, PGN, poly (I:C), cGAMP, and 5’ppp-dsDNA] and incubated for 24 h at 37°C. Plasma was harvested and subsequently concentrations of G-CSF, IFN-γ, IL-1β, IL-6, IL-8, IL-10, and TNF-α were determined by multiplex assay to generate immune response profiles.

Results: Fetuses with inflammation in the fetal regions of the placenta had a significantly increased IL-6 responses to cGAMP and 5’ppp-dsRNA (ligands for STING and RIG-I, respectively) compared to fetuses with maternal inflammation or no inflammation (p<0.05). On the other hand, IL-8, IL-10, and G-CSF responses to poly (I:C) (a TLR3 agonist) were significantly suppressed in the fetal inflammation group (p<0.05).

Conclusions: Our study suggests that fetal immune responsivity is altered in pregnancies with sub-clinical feto-placental inflammation. This may be associated with perturbations in fetal immune development and altered infection risk.

DO WE OVERESTIMATE THE INCIDENCE OF PULMONARY HYPERTENSION IN PRETERM INFANTS WITH BRONCHOPULMONARY DYSPLASIA?
Stoecklin B1, Swedenkранs J2, Pillow JJ1, Gill A1
1University of Western Australia, Perth, WA, Australia, 2Karolinska Institutet, Stockholm, Sweden
Email: ben.stoecklin@uwa.edu.au

Background: Our aim was to assess the right ventricular function as a marker for pulmonary hypertension (PH) in very preterm infants with and without bronchopulmonary dysplasia (BPD) against the published incidence of 15 - 25%.

Methods: Preterm infants born <32 weeks gestational age (GA) were studied. Echocardiographic assessments of right ventricular function at 36 weeks postmenstrual age (PMA) included tricuspid regurgitant jet velocity (TRJV), septal shape, tricuspid annular plane systolic excursion (TAPSE), right ventricular output (RVO) and right myocardial performance index (MPI). BPD was categorised using the NICHD criteria. Pulmonary gas exchange capacity was measured using shift of the SpO2/PaO2 curve.