In the group with BMI ⩾ 30 (07 pregnancies), facility of captation/stability of signal was 85%, instead of when BMI ⩽ 30 was 42%.

Conclusions: We have not identified any differences regarding the quality of analysis fetal vitality between the methods. The easy achievement/continuity of record by AN24 monitor in maternal obesity may indicate that this resource is particularly valuable for this group. Additional studies may increase the information for this research.

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[177-POS]

Seasonality of pregnancy induced hypertensive disorders in South Australia – A retrospective population study 2007–2011

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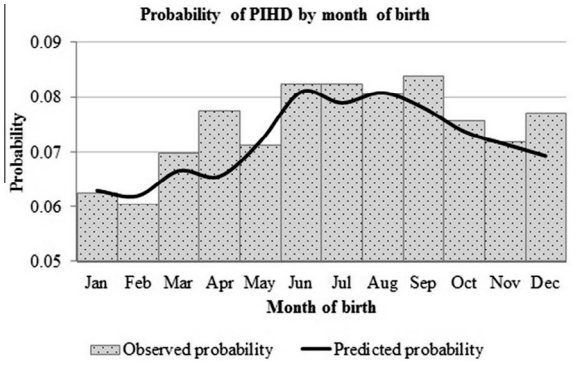
Objectives: To assess the seasonal variation of pregnancy induced hypertensive disorders (PIHD) in an Australian population.

Methods: Retrospective study of 59,993 South Australian singleton live born births, for whom a body mass index (BMI) of the mother and sex of the baby were recorded, during 2007–2011 in the South Australian Perinatal Statistics Collection. The incidence of PIHD in relation to birth date was assessed. Fourier series analysis was used to model seasonal trends.

Results: Of a total of 59,993 births recorded during the study period 4252 (7.1%) women were diagnosed with PIHD. Seasonal modelling showed a strong relation between PIHD and date of birth ($p < 0.000$). When adjusted for confounders (age, BMI, race, smoking during second half of pregnancy, parity and gestational diabetes) the model still showed a strong relation between PIHD and date of birth ($p < 0.000$). The peak prevalence occurred among births in Winter (Jun/Jul/Aug), with a trough in pregnancies with birth in (late-) Summer (Jan/Feb).

Conclusions: These epidemiological data support seasonal periodicity for PIHD in an Australian population. The highest incidence of PIHD was associated with birth in the Winter months (Jun/Jul/Aug). The etiology of PIHD is still elusive, but theories include genetic and immune mechanisms, abnormal placentation, and cardiovascular maladaptation to pregnancy, nutritional, hormonal and angiogenetic factors and enhanced systemic inflammatory response.

Recent studies found a relation between both infection and low maternal vitamin D levels and pre-eclamspia. These conditions could explain the detected seasonality for PIHD. Further investigation into the biological mechanism(s) for this finding should be undertaken to identify additional risk factors, so PIHD can be prevented in the clinic.



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[178-POS]

Maternal and placental leptin levels are increased in patients with pre-eclampsia

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Objectives: A hypoxic placenta may release factors that result in maternal endothelial dysfunction, among which, leptin seems to participate. The aim of this study was evaluate the association of leptin levels in placenta, maternal and umbilical cord plasma between normotensive controls and pre-eclamptic women.

Methods: Placental biopsies, maternal and umbilical cord plasma were taken from 67 normotensive and 50 pre-eclamptic women. Leptin levels were quantified using MagPlexTH-C – microspheres system. The leptin concentration was analyzed by ANCOVA adjusted by BMI, gestational age and maternal age. To estimate the difference between groups, mean ratio (MR) and confidence interval (CI) of 95% was calculated. Analysis between leptin levels and maternal/fetal variables were made by Pearson correlation. The null hypothesis was rejected when $p < 0.05$.

Results: Higher levels of leptin were found in maternal plasma (MR = 1.40; 95%CI: 1.00–1.97, $p = 0.049$) and placenta (MR = 1.82; 95%CI: 1.11–2.98, $p = 0.019$) in patients with pre-eclampsia. A positive correlation between gestational age, birth weight vs. fetal leptin levels in pre-eclamptic group was found ($r = 0.416$, $r = 0.618$; $p < 0.001$, for both), respectively. Also, a positive correlation was found

between placental leptin and maternal plasma levels in entire group ($r = 0.36$, $p < 0.001$) and in the normotensive group ($r = 0.344$, $p = 0.021$). No correlation was found between placenta vs. fetal plasma or fetal plasma vs. maternal plasma.

Conclusions: Leptin values in patients with pre-eclampsia were significantly increased in maternal plasma and placental tissue. Besides that, the positive correlation not observed between placentae and maternal leptin levels in pre-eclamptic group may be due to a loose of this regulation in pre-eclampsia, and should be considered for future work. Finally, a strong positive correlation in relation to clinical data and fetal leptin concentration, in pre-eclamptic group, intensify the possibility of leptin being also involved in preterm birth and birth weight, when pre-eclampsia is presented.

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[179-POS]

First trimester screening for early onset preeclampsia is a cost effective approach in prenatal care

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Objectives: To determine if first trimester screening for early-onset preeclampsia can be implemented in a cost effective manner.

Methods: We used decision tree analysis to determine the breakeven price and the cost-effectiveness price at which the incremental cost effectiveness ratio was $100,000 per quality-adjusted life-year (QALY). A recent review article (Poon and Nicolaides, 2014) demonstrated detection rates of 60% and 93% for biochemistry (PlGF and PAPP-A) and combined biochemistry and biophysical (mean arterial pressure and uterine artery Doppler) screening, respectively. A metaanalysis (Roberge et al., 2012) of the effectiveness of administration of aspirin prior to 16 weeks demonstrated a reduction in incidence of early-onset preeclampsia of 89%, with the lower end of the 95% confidence interval of 67%. These parameters were incorporated into 4 scenarios along with assumptions for aspirin treatment compliance (93%), the rate of disability and neonatal death and the cost of care associated with the gestational age of delivery. Utility values of 0, 0.61 and 1.00 were used for death, disability and healthy and discounted 3% per year for an average lifespan of 76 years. Costs were based on 2013 dollars.

Results: The table shows QALYs gained per 100,000, the breakeven price and the breakeven cost effectiveness price based on screening performance and aspirin effectiveness.

Conclusions: The data indicate that reasonable prices can be offered for both biochemical and biophysical testing so that the cost of first trimester early-onset preeclampsia screening will be offset by the savings and improved outcomes resulting from reduced numbers of early-onset preeclampsia. Inclusion of additional biochemical markers and the ability to identify some cases of late onset preeclampsia may provide further advantages.

Detection (%)	Aspirin effectiveness (%)	QALYs gained per 100 K	Breakeven price	Breakeven cost effectiveness price
60	67	299	$164	$463
93	67	463	$255	$718
60	89	397	$219	$615
93	89	615	$339	$954

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[180-POS]

Is superimposed pre-eclampsia different from de-novo pre-eclampsia?

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Objectives: Women with EH are at high risk of developing PE but it is not clear whether PE in this setting is worse than in the absence of EH. We therefore compared outcomes of women with pre-eclampsia superimposed upon essential hypertension (PE/EH) with those of women with de-novo pre-eclampsia (PE).

Methods: We have gathered data on pre-eclamptic pregnancies since 1991. We compared maternal outcomes (severe hypertension – BP >170/110 mmHg; cerebral, renal or hepatic dysfunction or thrombocytopenia) and fetal outcomes (SGA <10% and perinatal mortality [PNM]) of women with de novo PE to those with PE/EH. PE was defined according to ISSHP 2014 criteria and required de novo proteinuria, renal, liver, platelet or neurological problems.

Results: 1947 women developed PE and 143 women had PE/EH. Women with PE/EH were older than those with PE (33 vs. 30 years, $p < 0.05$). 1st trimester BP was higher in the PE/EH group (137/84 vs. 115/70 mmHg, $p < 0.05$). Fewer women with PE/EH were nulliparous (52% vs. 69%, $p < 0.05$). Severe HT was more frequent in the PE/EH group (43% vs. 33%, $p < 0.005$) and these women were more likely to present with non-proteinuric pre-eclampsia (50% vs. 28%, $p < 0.01$). Women with PE/EH had less thrombocytopenia (17% vs. 11% $p < 0.05$) but CNS, renal and liver complication rates were similar between groups.

Delivery before 37 weeks was higher in PE/EH (39% vs. 30% $p < 0.05$) but the SGA rate was lower in the PE/EH group (6 vs. 13% $p < 0.05$). PNM rates were 7/1000 in the PE/EH group and 16/1000 in the PE group (NS).