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

Petra E. Verburg[a], Graeme Tucker[b], Wendy Scheil[b], Jan Jaap H. Erwich[a], Claire T. Roberts[c], Gus A. Dekker[c] ([a]University Medical Centre Groningen, Groningen, Netherlands, [b]Epidemiology Branch, SA Health, Adelaide, Australia, [c]School of Pediatrics and Reproductive Health, Robinson Research Institute, University of Adelaide, Adelaide, Australia)

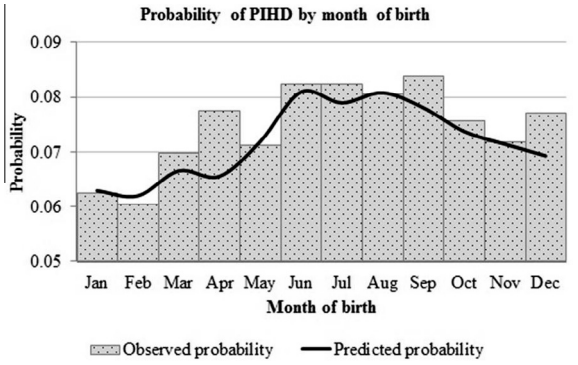
Objectives: To assess the seasonal variation of pregnancy induced hypertensive disorders (PIHD) in an Australian population.

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

Marta R. Hentschke, Adriana B. Comparsi, Luiza S. Lucas, Fernando Sontag, Giovani Gadonski, Letícia G. Paula, Carlos E. Poli-de-Figueiredo, Bartira E. Pinheiro da Costa (Pontifícia Universidade Católica do Rio Grande do Sul, Porto Alegre, Brazil)

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