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REDUCED POSTPARTUM URINARY STEROID HORMONE SYNTHESIS IN WOMEN WHO HAVE EXPERIENCED GESTATIONAL HYPERTENSION OR PRE-ECLAMPSIA DURING PREGNANCY

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Introduction and Aims

- Endocrine dysfunction has been described in postpartum women following pregnancy accompanied by significant proteinuria (pre-eclampsia; PE) and non-proteinuric new hypertension (gestational hypertension; GH).
- We hypothesised that adrenal hormone synthesis, clearly affected during hypertensive pregnancy, is also compromised for an extended time period thereafter.
- Thus, we aimed to determine steroid hormone metabolomics at 6-weeks postpartum in women who had PE or GH during pregnancy compared with normotensive controls (NC).

Methods

- Women attending the antenatal clinic between 2008 and 2013, gave informed, written consent. NC women (n=45), women with PE (n=57) and GH (n=28) were recruited.
- Urinary steroid metabolites were measured by gas chromatography-mass spectrometry (GC-MS); creatinine was measured using a standard clinical laboratory assay for normalisation.

Results

- Mean (+SD) blood pressures for the 3 groups, 6 weeks postpartum are shown in Table 1

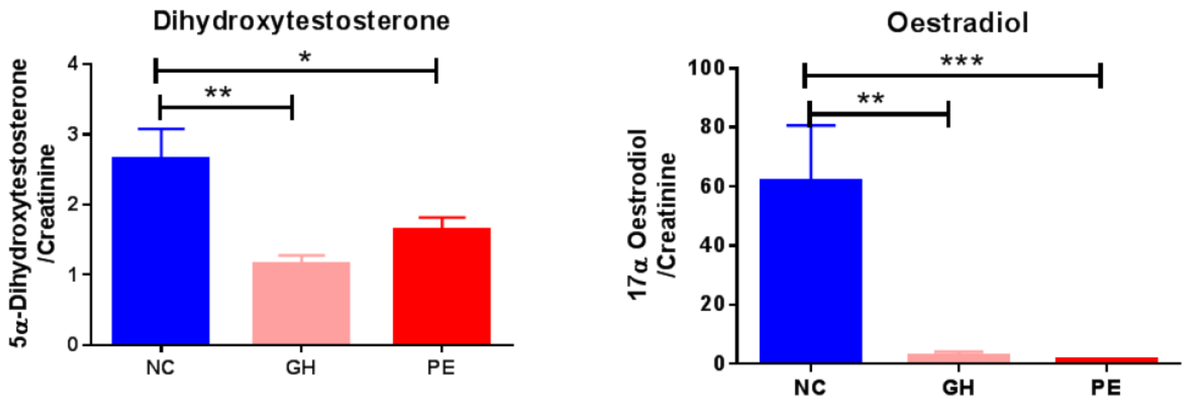
Table 1: Blood pressures, 6 weeks postpartum

Blood Pressure (mm Hg; Mean ± SD)	NC	GH	PE
Systolic	106±12	127±12	121±12
Diastolic	70±11	85±11	80±9

- Overall steroid hormone synthesis was reduced in women who had suffered from PE or GH.
- The active androgens (dihydroxytestosterone) and oestradiol were reduced (P<0.05 for both PE and GH vs. NC; Fig. 1), but there were no differences between DHEA and cortisol production (P>0.05).

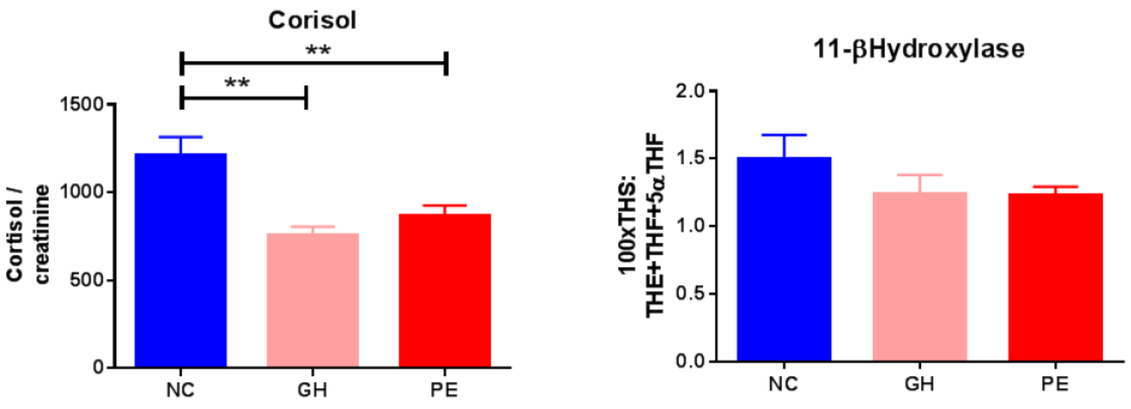
Results

Figure 1: Urine active androgens (mean± SEM); *P<0.05; **P<0.001; ***P<0.0001



- Total cortisol production was reduced in both PE and GH (P≤0.001 for both PE and GH vs. NC), however, the 11-hydroxylase activity was maintained (P>0.05; Fig. 2).

Figure 2: Urine total cortisol and 11β-hydroxylases (mean± SEM); **P<0.001



- In the corticosterone pathway both precursors and products were reduced in PE and GH, in which reduced synthesis of aldosterone (P=0.02 for both PE and GH vs. NC) and its precursors was also demonstrated (Table 2).

Table 2: Urine corticosterone metabolites (mean± SEM); *P<0.05 between NC and both GH and PE

Metabolite	NC	GH	PE
Tetrahydrocorticosterone	23.11±2.40*	17.58±1.08	17.58±1.59
5α-Tetrahydrocorticosterone	34.39±4.02*	24.11±3.51	27.79±2.38
Tetrahydro-11-dehydrocorticosterone	19.48±3.34*	10.63±0.85	11.65±0.89
Tetrahydroaldosterone	4.04±0.65*	2.97±0.45	2.18±0.28

Conclusions

- Hypertension during pregnancy has a sustained impact on the regulation of adrenal steroid hormones.
- As aldosterone production is primarily driven by the renin/angiotensin II axis, inappropriate adrenal responses might favour enhanced renin-driven vasoconstriction, conversely oestradiol-dependent vasorelaxation is probably hampered.
- The temporal consequences of this impact on future pregnancies, on blood pressure regulation and the renal function will have to be addressed.

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