The metabolic consequences of pregnancy for mother and child

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Solvay Healthcare sponsored the 2007 Northern Lipid Forum, held on 31 October 2007 at Lumley Castle Hotel, Chester-le-Street, Co. Durham.

Chairing the meeting was Dr Dermot Neely, Consultant Clinical Biochemist at the Royal Victoria Infirmary, Newcastle- upon-Tyne and member of H·E·A·R·T UK's Medical, Scientific & Research Committee.

The programme, entitled "Pregnancy – the metabolic consequences for mother and child" featured presentations by Dr Dilys Freeman, Senior Lecturer in Vascular Biology, Dept of Obstetrics & Gynaecology, Glasgow Royal Infirmary; and Mr Jason Waugh, Consultant in Obstetrics & Maternal Medicine, Royal Victoria Infirmary, Newcastle. Mr Waugh spoke about hypertension in pregnancy: short, medium & long-term cardiovascular risk. Dr Freeman's presentation is summarised below.

THE METABOLIC CONSEQUENCES OF PREGNANCY FOR MOTHER AND CHILD

Healthy pregnancy is associated with metabolic change including increased insulin resistance, hyperlipidaemia, increased inflammatory status and enhanced coagulation. In particular there is a hypertriglyceridaemia of pregnancy characterised by an increase in both VLDL1 and VLDL 2 subfractions and a decrease in LPL mass. This results in increased levels of circulating small, dense LDLIII. Specific fatty acids, particularly the essential long chain omega 3 fatty acids, are mobilised by the mother for transfer via the placenta to the developing fetus.

Maternal obesity is an increasing clinical problem with a doubling of the proportion of women with BMI >30kg/m2 over the last decade.

Obesity results in an exaggeration of the metabolic changes of healthy pregnancy and results in changes in the quality of plasma metabolites such as an increased proportion of LDLIII and reduced mobilisation of palmitoleic acid. Obese pregnant women have decreased endothelial dependent microvascular function that is apparently explained by increased inflammatory status rather than hyperlipidaemia.

Obese pregnancy is associated with increased complications of pregnancy such as hypertension, gestational diabetes mellitus and preeclampsia. Preeclampsia, a disease characterised by maternal endothelial dysfunction, demonstrates a further exaggeration and change in quality of the metabolic response to pregnancy.

Metabolic syndrome is apparent and atherosis of the maternal spiral arteries is observed. Preeclampsia has a two step aetiology with a primary placental defect leading to release of a factor(s) resulting in a secondary maternal metabolic response. It has been proposed that the unknown placental trigger may lead to increased adipocyte lipolysis. This is proposed to result in an increased circulating atherogenic lipoprotein pool which, in the presence of oxidative stress due to reduced placental perfusion, produces oxidised lipid mediators that trigger endothelial dysfunction.

Markers of oxidative stress are increased in preeclampsia and a pilot study indicated that treatment with the antioxidant vitamins C and E might prevent preeclampsia. However a larger multi-centre intervention trial (Vitamins in Pregnancy [VIP]) demonstrated no prevention of preeclampsia with antioxidant vitamins and suggested that treatment might result in poorer fetal outcome.

Many of the metabolic changes associated with pregnancy occur in the first 8 weeks of pregnancy and also prior metabolic syndrome is associated with an increased risk of placental dysfunction. Thus study of early pregnancy metabolic changes and the impact of obesity is warranted. Maternal metabolic profile has an influence on the neonate.

Maternal glucose levels are associated with offspring birth weight and fat mass. Cord blood leptin levels, a marker of fetal fat mass, is associated with an increase in cord blood inflammatory markers. Cord blood hyperlipidaemia is associated with pregnancies complicated by preeclampsia and gestational diabetes mellitus and is also correlated with neonatal inflammatory status. Thus maternal in utero metabolic environment may influence offspring risk of future vascular disease via chronic inflammation.

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