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Women with gestational diabetes mellitus controlled for their plasma glucose levels, exhibit dyslipidaemia that may contribute to offspring obesity and the risk of future gestational diabetes mellitus

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Hyperglycaemia and hypertriglyceridaemia are well known characteristics in women with Gestational Diabetes Mellitus (GDM). However, women with tight glucose control can still have babies with adiposity. The aims of this study were to determine 1) if the triglyceride content/enrichment of maternal lipoproteins in women with GDM treated for blood glucose levels, could potentially provide more fatty acids to the placenta compared to normoglycaemic pregnant women and 2) if there was any evidence of foetal lipid dyslipidaemia that may contribute to increased adiposity. Pregnant women were recruited from the Royal Brisbane and Women's Hospital Queensland, Australia and National Health Service Greater Glasgow and Clyde maternity units, Scotland. Fasted blood samples were collected at trimesters 2 (T2) and 3 (T3) and cord bloods were obtained at delivery. GDM was diagnosed using standard institutional clinical criteria of the time. Lipoprotein fractions were isolated from plasma via sequential ultracentrifugation⁽¹⁾ at the following densities: VLDL < 1.006 g/mL, IDL 1.006-1.019 g/mL, LDL1.019-1.063 g/mL and HDL1.063-1.21 g/mL.⁽²⁾ Statistical modelling included the outcome (GDM status); Trimesters (T2 and T3); and GDM status and outcome*trimesters interaction, for all outcome variables. The model also included gestation at blood sampling as a covariate to correct for the difference at T2 between GDM status groups. Cord plasma means were compared using t-tests. All statistical analyses were conducted using JMP Pro 16.1.0 SAS Institute Inc and significant levels were set at p < 0.05. Plasma glucose did not differ between normoglycaemic women and women with GDM who were treated by diet and/or medication to lower blood glucose levels. Plasma VLDL- and IDL- protein, total cholesterol and phospholipid were significantly higher (25-47%) in GDM compared to normoglycaemic women at T2 and reached a plateau by T3 for all women, suggesting this is related to increased insulin resistance in women with GDM. Plasma triglycerides were higher in GDM and increased from T2 to T3 in all women. VLDL triglyceride enrichment and IDL triglyceride enrichment was 5-26% lower in GDM compared to normoglycaemic women. HDL triglyceride per HDL protein was 40% lower in GDM, possibly due to reduced cholesteryl ester transfer protein activity in GDM.⁽³⁾ This may be the result of increased transport of maternal VLDL-, IDL-triglyceride across the placenta to the foetus. Certainly, cord blood VLDL, IDL and LDL from women with GDM were triglyceride enriched, double that compared to normoglycaemic women. In conclusion, despite normal blood glucose levels in women with GDM, the offspring had double the triglyceride load in their lipoproteins that may contribute to the risk of obesity and future GDM.

References

- Havel RJ, Eder HA & Bragdon JH (1955) J Clin Investig 34, 1345–1353.
 Sattar N, Greer IA, Louden J, et al. (1997) J Clin Endocrinol Metab 82, 2483–2491.
 Liao Y, Xu GF, Jiang Y, et al. (2018) Med 97, e12232.