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Maternal obesity and vitamin D status influence adiponectin concentrations in pregnancy

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Maternal obesity increases the risk of both low vitamin D status (25(OH)D) during pregnancy and of infants being born with 25(OH)D deficiency[(1)]. Adiponectin has been inversely related to numerous conditions including insulin resistance, atherosclerosis, type 2 diabetes, and dyslipidaemia[(2)]. Vitamin D and adiponectin have been shown to be positively correlated, but little is known about their interrelationship in pregnancy and the influence of maternal weight status on this relationship.

The aim of this investigation was to examine the influence of maternal weight status and vitamin D supplementation on adiponectin concentrations across pregnancy.

The MO-VITD study[(1)] was a 2-arm parallel double-blind randomised trial with 240 pregnant women recruited throughout the year in Northern Ireland. Women were stratified to receive 10 or 20μg of vitamin D^3/day from 12 gestational weeks (GW) until delivery. Data on maternal anthropometry and body composition, alongside blood samples, were obtained at 12, 28, and 36 GW. Blood samples were analysed for total serum 25(OH)D and total serum adiponectin. Data were analysed using t-tests and linear regression, using IBM SPSS version 28 and considered significant at $P < 0.05$.

Adiponectin concentrations did not differ according to vitamin D treatment group (10 vs 20 μg) at any timepoint in pregnancy. Linear regression showed that adiponectin in early pregnancy was a positive predictor of 25(OH)D at 36 GW (β 13.22, $P < 0.001$). BMI in early pregnancy was a negative predictor of adiponectin at all timepoints ($P < 0.001$). At 28 GW, 25(OH)D was a positive predictor of adiponectin concentrations (β 0.275, $P < 0.001$). Women with obesity had lower adiponectin concentrations at 12 and 28 GW compared to women of normal weight ($P < 0.05$ at both timepoints). Adiponectin concentrations significantly decreased across pregnancy, with women of normal weight shown to have the largest decrease when compared to with women with overweight or obesity (-8.6 ± 12.3 μg/mL vs -3.4 ± 13.7 μg/mL, -1.6 ± 14.0 μg/mL; $P < 0.05$, respectively). Women of normal weight were also observed to have the highest gestational weight gain (11.3 ± 2.8 kg vs 7.2 ± 4.8 kg, $P < 0.001$) and increase in fat mass across pregnancy (7.1 ± 4.7 kg vs 3.2 ± 5.0, $P < 0.001$), when compared to women with obesity. Fat mass change was significantly and negatively correlated with total adiponectin change (r -0.310, $P < 0.001$). Gestational weight gain did not predict adiponectin concentrations.

Despite positive associations between 25(OH)D and adiponectin, BMI in early pregnancy and fat mass increase throughout pregnancy were consistent negative predictors of a low adiponectin status. The more precise measure of fat mass change rather than gestational weight gain has demonstrated an important negative influence on adiponectin. Body composition may be a more useful tool than body weight in the prediction of adverse markers of maternal health.

References

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