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LETTER TO EDITOR

Maternal Triglycerides
Underestimated predictors of neonatal birth weight

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الدهون الثلاثية الأُمومِيَّة
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The concept of maternal glucose levels predicting birth weight in diabetic pregnancies is well known and has been extensively investigated. This notion however seems to overshadow the understanding of the effect of other maternal factors on birth weight in non-diabetic pregnancies. Here, we emphasise that, in healthy non-diabetic pregnancies, postprandial and fasting glucose levels at late gestation are in the normal range.[1,2] On the other hand, marked lipemia, mainly hypertriglyceridemia is a marked feature of late gestation.[2] In spite of this fact, maternal triglyceride (TG) levels appear to be underestimated in this debate. Notably, recent studies reported highly significant positive correlations between fasting maternal TG levels at late gestation and birth weight in non-diabetic mothers independent of glucose levels or maternal weight.[3,4] Also, in diabetic mothers, TG levels were shown to be better predictors of birth weight than glucose levels.[5] In spite of these highly significant associations, the number of human studies that describe the effect of increased maternal TG levels on birth weight in normal pregnancies are surprisingly limited. This may be due to the understanding that maternal TG does not cross the maternal-fetal placental barrier unless hydrolysed into fatty acids that have not been proven readily to cross the maternal-fetal placental barrier as glucose.

In this commentary, we bring into focus maternal TG levels as potential independent predictors of birth weight that may contribute to early fetal programming events predisposing to childhood obesity and metabolic derangements later in life. This is of particular importance in light of expanding epigenetic research that may overcome maternal-fetal placental barrier considerations and may therefore contribute to the understanding of the specific effect of maternal TG and dietary fat on the expression of obesity related genes.

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