Intrauterine Influences on Obesity and Insulin Resistance in Pre-pubertal Children

Oana Maftei
M.D., F.Rom.C.P.

Discipline of Public Health, School of Population Health and Clinical Practice
Discipline of Obstetrics and Gynaecology, School of Paediatrics and Reproductive Health
Faculty of Health Sciences, University of Adelaide
Australia
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A thesis submitted in partial fulfilment of the requirements for the degree of Doctor of Philosophy
“Truth lies all around us, but is only revealed to those who search for it.”

Nicolae Iorga (1871-1940)
This thesis is dedicated to my mother for her immense love, trust, support and encouragement not only through the PhD candidature years, but throughout my entire life. I have learnt from her wisdom that giving anything less than all I could give meant sacrificing a gift.
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Abstract

Within the paradigm of developmental origins of health and disease, an intrauterine environment that stimulates fetal overnutrition has been found to contribute to the risk of subsequent obesity in the offspring. There is compelling epidemiological evidence for a positive association between maternal obesity prior to pregnancy, gestational diabetes (GD) or excessive gestational weight gain, and the development of childhood obesity (as measured by body mass index, BMI). However, the evidence is limited and inconsistent with respect to more specific measures of adiposity (body composition or fat pattern) and insulin resistance in children. Furthermore, the long-term effects of maternal borderline gestational glucose intolerance (BGGI) on the offspring have not been considered.

Therefore, I sought to examine whether maternal obesity prior to pregnancy, gestational glucose intolerance across the entire spectrum, and gestational weight gain have deleterious effects on the development of obesity (both global and specific measures of adiposity) and insulin resistance in pre-pubertal children. These associations are particularly important from a public health perspective as, once identified, they may point towards potential windows for prevention of childhood obesity and related metabolic disorders.

This project entailed a follow-up of an existing representative, prospective birth cohort study (Generation 1 Study, n=557) in Adelaide, South Australia, recruited during 1998-2000. At the 9-10 year follow-up, rigorous anthropometric measurements were conducted in 443 children (80% of the original cohort), of whom 163 consented to provide a fasting blood sample for the estimation of insulin resistance based on homeostasis model assessment (HOMA-IR). Information on intrauterine exposures and confounders was collected from the antenatal interviews and hospital records. Maternal age, parity, smoking, pregnancy-induced hypertension, and education at the time of pregnancy were considered as potential confounders for all the associations of interest, and child current BMI z-score as a potential mediator on the pathway between the intrauterine exposures and child insulin resistance. Data were analysed using multiple linear regression and generalized linear models.
Maternal pre-pregnancy BMI was positively associated with all three obesity-related measures considered in the 9-10 year-old children (BMI z-score, percentage body fat estimated by bioelectrical impedance analysis, and waist-to-height ratio); these relationships were robust to adjustment for potential confounders (adjusted coefficients for each one kg/m\(^2\) increase in maternal pre-pregnancy BMI were 0.08 (95% confidence interval 0.06, 0.10) for child BMI z-score, 0.44 (95% CI 0.31, 0.58) for percentage body fat and 0.002 (95% CI 0.002, 0.003) for waist-to-height ratio). There was no association between maternal pre-pregnancy BMI and HOMA-IR in children (with or without adjustment); however, when child current BMI z-score was included as a mediating variable, the relationship between maternal pre-pregnancy BMI and child HOMA-IR was inverse and significant (adjusted change in child HOMA-IR for each one kg/m\(^2\) increase in maternal pre-pregnancy BMI was -0.83% (95% CI -1.63, -0.02)).

Intrauterine exposure to glucose intolerance during pregnancy (either BGGI or GD) was not associated with any of the three obesity-related measures in children at 9-10 years. Children of mothers who developed GD during the index pregnancy had a higher HOMA-IR; this relationship was robust to adjustment for potential confounders (adjusted change in child HOMA-IR if exposed to maternal GD was 42.9% (95% CI 20.9, 68.9)) and partly mediated by child current BMI z-score. No association was found between exposure to maternal BGGI and child HOMA-IR (with or without confounder adjustment); however, when child current BMI z-score was added as a potential mediator, exposure to BGGI was associated with a reduction in child HOMA-IR (adjusted change in child HOMA-IR if exposed to maternal BGGI was -17.9% (95% CI -29.9, -3.96)).

There were no significant associations between maternal gestational weight gain and any of the outcome measures of interest in unadjusted models. However, adjustment for pre-pregnancy BMI led to a positive association between gestational weight gain and child BMI z-score (adjusted changes in child BMI z-score for each one kg increase in maternal gestational weight gain was 0.032 (95% CI 0.007, 0.057)). Gestational weight gain was not associated with child insulin resistance, and this did not change when child current BMI z-score was included as a potential mediator on the pathway between gestational weight gain and child insulin resistance.

Potential two-way interactions between the main exposures were investigated in relation to all outcomes of interest. Two significant interactions were identified: maternal pre-pregnancy BMI and glucose tolerance status, and maternal pre-pregnancy BMI and gestational weight gain, with a synergistic effect on child waist-to-height ratio.
These results suggest that childhood obesity and insulin resistance have origins, at least in part, in intrauterine life, particularly in relation to maternal obesity at the time of pregnancy and GD. Further research to differentiate between genetic, environmental and intrauterine programming is recommended. That said, maternal pre-pregnancy BMI was the strongest predictor of child BMI z-score, while GD appeared to have an independent effect on child insulin resistance, and both clinical and public health actions to address these maternal factors are warranted for a range of reasons.
Declaration

I, Oana Maftei certify that this work contains no material which has been accepted for the award of any other degree or diploma in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text.

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Oana Maftei

Date: 21 December 2011
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### Abbreviations and acronyms

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<th>Description</th>
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<tr>
<td>%BF</td>
<td>percentage body fat</td>
</tr>
<tr>
<td>ACHOIS</td>
<td>Australasian Carbohydrate Intolerance Study in Pregnancy</td>
</tr>
<tr>
<td>ACOG</td>
<td>American College of Obstetricians and Gynecologists</td>
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<tr>
<td>ADA</td>
<td>American Diabetes Association</td>
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<tr>
<td>ADIPS</td>
<td>Australasian Diabetes in Pregnancy Society</td>
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<tr>
<td>ALSPAC</td>
<td>Avon Longitudinal Study of Parents and Children</td>
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<tr>
<td>BGGI</td>
<td>borderline gestational glucose intolerance</td>
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<tr>
<td>BIA</td>
<td>bioelectrical impedance analysis</td>
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<td>BMI</td>
<td>body mass index</td>
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<td>BW</td>
<td>birth weight</td>
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<td>CDC</td>
<td>Centers for Disease Control and Prevention</td>
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<tr>
<td>CI</td>
<td>confidence interval</td>
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<tr>
<td>CT</td>
<td>computed tomography</td>
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<td>DXA</td>
<td>dual energy X-ray absorptiometry</td>
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<tr>
<td>EPOCH</td>
<td>Exploring Perinatal Outcomes among Children</td>
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<tr>
<td>FFM</td>
<td>fat-free mass</td>
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<tr>
<td>FGIR</td>
<td>fasting glucose-to-insulin ratio</td>
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<tr>
<td>FM</td>
<td>fat mass</td>
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<td>FSIVGTT</td>
<td>frequently sampled intravenous glucose tolerance test</td>
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<td>FTO</td>
<td>fat mass and obesity associated gene</td>
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<tr>
<td>GD</td>
<td>gestational diabetes</td>
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<tr>
<td>GLM</td>
<td>generalised linear model</td>
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<td>GWG</td>
<td>gestational weight gain</td>
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<tr>
<td>HAPO</td>
<td>Hyperglycemia and Adverse Pregnancy Outcomes</td>
</tr>
<tr>
<td>HbA1c</td>
<td>haemoglobin A1c</td>
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<tr>
<td>HDL</td>
<td>high-density lipoprotein</td>
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<tr>
<td>HOMA-IR</td>
<td>homeostasis model assessment of insulin resistance</td>
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<tr>
<td>IGF</td>
<td>insulin-like growth factor</td>
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<td>IGT</td>
<td>impaired glucose intolerance</td>
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<tr>
<td>IL</td>
<td>interleukin</td>
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<tr>
<td>Abbreviation</td>
<td>Full Form</td>
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<tr>
<td>IOM</td>
<td>Institute of Medicine</td>
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<td>IOTF</td>
<td>International Obesity Task Force</td>
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<tr>
<td>IQR</td>
<td>interquartile range</td>
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<td>IR</td>
<td>insulin resistance</td>
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<td>LDL</td>
<td>low-density lipoprotein</td>
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<td>LMS</td>
<td>lambda-mu-sigma</td>
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<td>MRI</td>
<td>magnetic resonance imaging</td>
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<td>NDDG</td>
<td>National Diabetes Data Group</td>
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<td>NGT</td>
<td>normal glucose tolerance</td>
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<td>NZSSD</td>
<td>New Zealand Society for the Study of Diabetes</td>
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<td>OGCT</td>
<td>oral glucose challenge test</td>
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<td>OGTT</td>
<td>oral glucose tolerance test</td>
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<td>OR</td>
<td>odds ratio</td>
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<td>PAR</td>
<td>population attributable risk</td>
</tr>
<tr>
<td>PCOS</td>
<td>polycystic ovarian syndrome</td>
</tr>
<tr>
<td>QUICKI</td>
<td>quantitative insulin sensitivity check index</td>
</tr>
<tr>
<td>r</td>
<td>Pearson’s correlation coefficient</td>
</tr>
<tr>
<td>RCT</td>
<td>randomised clinical trial</td>
</tr>
<tr>
<td>RR</td>
<td>relative risk</td>
</tr>
<tr>
<td>SD</td>
<td>standard deviation</td>
</tr>
<tr>
<td>SES</td>
<td>socio-economic status</td>
</tr>
<tr>
<td>SFT</td>
<td>skinfold thickness</td>
</tr>
<tr>
<td>TBW</td>
<td>total body water</td>
</tr>
<tr>
<td>TNF</td>
<td>tumour necrosis factor</td>
</tr>
<tr>
<td>TAFE</td>
<td>Training and Further Education</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
</tr>
<tr>
<td>WHtR</td>
<td>waist-to-height ratio</td>
</tr>
</tbody>
</table>
Conference presentations

1. Maftei O, Whitrow MJ, Moore VM, Davies MJ. *Intrauterine influences on offspring obesity in prepubertal children* - oral presentation, 7th World Congress on Developmental Origins of Health and Disease, Portland, Oregon, USA, 18-21 September 2011

2. Maftei O, Whitrow MJ, Moore VM, Davies MJ. *Intrauterine influences on insulin resistance in prepubertal children* - oral presentation (ranked in top 10%), 7th World Congress on Developmental Origins of Health and Disease, Portland, Oregon, USA, 18-21 September 2011