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Adipose Tissue Architecture and Gestational Weight Gain in Normoglycemic Pregnancies

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Objective: To investigate histologic architecture of subcutaneous (SQAT) and visceral adipose tissue (VAT) growth in relationship to gestational weight gain (GWG). Epidemiological data suggest that SQAT expansion may be protective of obesity related co-morbidities, whereas VAT expansion is associated with Type-2 diabetes risk. We hypothesized that in normal gravidas, GWG would be associated with hypertrophy of SQAT and not VAT.

Methods: A subset of subjects enrolled in the Pregnancy & Postpartum Observational Dietary Study (PPODS) and undergoing Cesarean delivery had SQAT (midline superior edge Pfannenstiel incision) and VAT (inferior omental periphery) biopsies after neonatal delivery, uterine closure and hemostasis achievement. Excised tissues were fixed and stained. Average adipocyte size and capillary density were assessed in 10 independent sections per AT depot per subject. GWG determined by the difference of weight at first visit and immediately postpartum (1-4 days post-op). GWG plotted vs mean SQAT or VAT adipocyte size.

Results: Table illustrates general clinical characteristics of the 5 subjects. Figure A demonstrates SQAT and VAT mean adipocyte size with representative sections from patient E depicted above bar graphs representing means and SEM from 5 patients. SQ adipocytes were significantly larger than those from VAT. Significant positive correlation was noted between GWG and SQAT adipocyte size (Figure B), but not VAT adipocyte size (Figure C).

Discussion: Preliminary results reveal that in normal pregnancies, GWG is associated with changes in SQAT but not VAT architecture, which reflects lipid accumulation. These results are consistent with the model that SQAT is specifically adapted for healthy lipid storage, and provides a basis for comparison between normal gravidas and those with GDM.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Non-fasting Glucose @ GDM Screen (mg/dL)</th>
<th>VAT @ 1st Study Visit (pre-21wks) (Kg)</th>
<th>Weight gain (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>131</td>
<td>88.076</td>
<td>6.3916</td>
</tr>
<tr>
<td>B</td>
<td>157</td>
<td>101.242</td>
<td>8.0812</td>
</tr>
<tr>
<td>C</td>
<td>105</td>
<td>61.744</td>
<td>8.3536</td>
</tr>
<tr>
<td>D</td>
<td>159</td>
<td>64.922</td>
<td>10.9868</td>
</tr>
<tr>
<td>E</td>
<td>119</td>
<td>86.714</td>
<td>20.43</td>
</tr>
</tbody>
</table>

Table 1: Characteristics of 5 representative patients without GDM