

insulin-sensitive non-diabetic (Ob_{sen} , BMI > 25 kg/m², HOMA-IR < 1.5, $n=11$), overweight/obese insulin-resistant (Ob_{res} , BMI > 25 kg/m², HOMA-IR > 3.0, $n=20$) and type 2 diabetes mellitus (T2DM, $n=21$). We measured insulin sensitivity by hyperinsulinaemic-euglycaemic clamp, body composition/central abdominal fat by dual energy X-ray absorptiometry, visceral fat area by computed tomography and fasting insulin, adiponectin and BA.

Results: Neither total BA ($r=0.012$, $p=0.91$) nor cholic acid (CA, the predominant primary BA) ($r=0.04$, $p=0.70$) correlated with percent total body fat. However, there were significant associations between BA and CA levels and waist circumference ($r=0.39$, $p=0.0008$; $r=0.34$, $p=0.0035$), central abdominal ($r=0.33$, $p=0.0057$; $r=0.28$, $p=0.01$) and visceral fat ($r=0.26$, $p=0.026$; $r=0.24$, $p=0.045$), respectively. CA, but not total BA, correlated with liver density (an inverse marker of hepatic fat, $r=-0.25$, $p=0.03$). Total BA inversely correlated with insulin sensitivity (glucose infusion rates corrected for fat-free mass [GIR/FFM], $r=-0.35$, $p=0.003$) and adiponectin levels ($r=-0.24$, $p=0.04$). In group comparisons, GIR/FFM was significantly lower, and visceral and liver fat significantly higher, in Ob_{res} compared to lean and Ob_{sen} subjects, despite similar total adiposity in Ob_{res} and Ob_{sen} (data not shown). Consistent with correlation analyses, total BA concentration tended to be higher in Ob_{res} (1.35 + 1.1 mmol/L) versus Ob_{sen} (0.67 + 0.28 mmol/L) ($p=0.057$), but were similar between Ob_{sen} and lean (1.00 + 0.69 mmol/L).

Conclusion: Our data suggest that BA concentrations aligned closely with insulin resistance, central abdominal, visceral and liver fat in human subjects. Whether BA play an aetiological role in insulin resistance is yet to be elucidated.

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Gestational diabetes mellitus among young adult women with PCOS: Association with BMI trajectories over 13 years



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Objective: Meta-analyses indicate a 3X increased risk of gestational diabetes (GDM)

among women with polycystic ovary syndrome (PCOS), however relationships between longitudinal trajectories of body mass index (BMI) and GDM risk in PCOS remain unclear. We aimed to identify BMI trajectory groups, compare GDM prevalence across trajectory groups and assess BMI trajectories impact on GDM risk.

Methods: This is a secondary analysis from the (Australian longitudinal Study on Women's Health) ALSWH with 8200 women aged 18–36 across five surveys (13 years). The main outcome measure was GDM prevalence. We used latent-class growth modelling to identify distinct BMI trajectories and logistic regression to assess GDM risk.

Results: 575 women (7.0%, 95% CI 6.5–7.6%) reported PCOS. Among women with ≥ 1 live pregnancy, 15.1% developed GDM vs. 6.0% controls ($p < 0.001$). Three distinct BMI trajectories were identified over the 13 year follow-up: low-stable (LSG) (63.4% women), moderately-rising (MRG) (29.2%) and high-rising (HRG) (7.5%). These were defined as: LSG-average trajectory remaining within healthy range; MRG-curvilinear trajectory commencing in healthy and terminating in overweight range and HRG-curvilinear trajectory starting and terminating in obese range. Women with PCOS were more likely to belong to MRG and HRG groups (OR 1.8, 95% CI 1.5–2.2 and OR 4.2, 95% CI 3.2–5.4). The GDM prevalence in PCOS differed significantly across trajectory groups (9.4% vs. 20.0% vs. 21.0%, $p=0.02$). After adjusting for BMI trajectories, age and demographic factors, women with PCOS were twice as likely to develop GDM compared to controls (OR 2.3, 95% CI 1.6–3.2).

Conclusion: Women with PCOS have higher rates of weight gain, yet PCOS remains an independent predictor of GDM irrespective of BMI trajectories over reproductive years. This aligns with the non-BMI dependent inherent insulin resistance in PCOS highlighting need for aggressive universal GDM screening in PCOS, independent of BMI and weight gain.

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