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### Service provides; the adolescent decides: weight loss outcomes in a tertiary obesity service

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**Background:** Management of adolescent obesity requires a range of evidence –based dietary interventions [1]. This study explored the outcomes of a multidisciplinary weight management program for adolescents with obesity in a tertiary setting. The 6 month dietetic and medical led program offers adolescents the choice of 3 dietary interventions; a Very Low Energy Diet (VLED), Intermittent Energy Restriction (IER) and Higher Protein Diet (HPD).

**Methods:** A retrospective medical chart review of the Adolescent Weight Management Program at The Children's Hospital at Westmead between October 2014 to December 2017 was conducted. Participants ($n = 57$) aged 14–17 y who engaged with the program were identified, and anthropometric data extracted for index calculations. Statistical analysis was intention-to-treat, and sub-group analysis was completed to compare sex, dietary interventions and attendance between engagers ($n = 47$) and program completers ($n = 10$).

**Results:** Weight- related outcomes significantly improved from baseline until last contact with the clinic: BMI *z*-score ($-0.05 \pm 0.45$, $p < 0.001$), weight ($-2.32 \pm 6.49$ kg, $p = 0.021$), waist circumference (WC; $-2.3 \pm 6.49$ cm, $p = 0.03$), BMI ($-1.06 \pm 2.08$ kg/m$^2$, $p = 0.001$), BMI expressed as a percentage of the 95th percentile (BMI95; $-7.89 \pm 12.19\%$, $p < 0.001$) and Waist-to-Height ratio (WtHR; $-0.02 \pm 0.16$, $p = 0.01$). Attendance decreased over time. Increased contact with the clinic significantly improved all weight-related outcomes. Adolescents who completed the program ($n = 10$) had significant reductions in weight related outcomes, including BMI *z*-score ($-0.15 \pm 0.13$, $p = 0.021$); and BMI95 ($-7.63 \pm 7.46\%$, $p = 0.043$). Girls ($n = 6$) had greater reductions in BMI *z*-score and centile ($-0.11 \pm 0.06$, $p = 0.02$ and $-0.35 \pm 0.17$, $p = 0.017$) compared to boys. Changes in weight-related outcomes were greatest for adolescents following a VLED; ($n = 16$), particularly for BMI *z*-score ($-0.12 \pm 0.11$) and BMI95 ($-5.44 \pm 6.19\%$), in comparison to the IER ($-0.09 \pm 0.13$, $-3.49 \pm 4.68\%$ respectively) and HPD ($-0.05 \pm 0.13$, $-1.12 \pm 5.01\%$ respectively).

**Conclusions:** The results demonstrate the effectiveness of real-world clinical practice for management of adolescent obesity. VLEDs produce greater weight reductions overall. Increased attendance at clinic lead to a significant improvement in all weight related outcomes.

### Reference

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### Early adolescent weight gain and its relationship to the tempo and timing of puberty

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**Introduction:** Cross-sectional studies show an association between obesity and earlier timing of puberty. However, few studies have considered how the tempo of puberty potentially impacts upon weight and adiposity. This study assessed longitudinal associations for pubertal timing and tempo to weight-related outcomes in adolescents.

**Methods:** 226 healthy adolescents (46% female; 10–13 y at recruitment) from the ARCHER cohort study [1] of puberty hormones were followed over a 3-year period. Annual anthropometric and body composition (via bio-electrical impedance) measures were analysed against pubertal timing (earlier/later) and tempo (fast/slower) groups derived from non-linear growth curve modelling of annual self-reported Tanner stage.

**Results:** Earlier pubertal timing was significantly associated with higher absolute levels of adiposity (including weight, BMI *z*-score, waist circumference, waist-to-height ratio and fat mass) in girls, and more rapid increases in adiposity (including weight, BMI *z*-score and fat mass) over 3 years among boys (all $p < 0.05$). Faster pubertal tempo predicted more rapid increases in weight and BMI *z*-score over 3 years among girls (both $p < 0.05$), but paradoxically, less rapid increases in central adiposity (including waist circumference and waist-to-height ratio; both $p < 0.01$) among boys. Adjustment for baseline BMI *z*-score attenuated the pubertal timing associations observed in girls, but other results remained largely unchanged.

**Conclusions:** Pubertal timing and tempo showed sex-specific relationships to weight-related outcomes. This study identified pubertal tempo as a potentially relevant risk factor for weight and central adiposity in girls and boys, respectively. Findings for pubertal timing align with the cross-sectional evidence. However, our results in girls showed a stronger association for pubertal timing to pre-existing adiposity than weight gain during adolescence. The reverse was apparent in boys. These associations may assist in early identification of adolescents at risk of excess weight gain during adolescence. Findings also highlight the importance

of sex- and developmentally-appropriate weight management advice/strategies for youth.

### Reference

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## Early clinical markers of overweight/obesity onset and resolution by adolescence: Longitudinal Study of Australian Children

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**Background:** Clinical prediction tools could assist with prevention and treatment of adolescent overweight/obesity. We examined how combinations of clinical indicators at various ages predict overweight/obesity development, as well as resolution, by 10–11 and 14–15 years of age.

**Methods:** Data were derived from Birth ($N = 3469$) and Kinder ($N = 3276$) cohorts of the Longitudinal Study of Australian Children, followed from ages 2–3 and 4–5 years, respectively. Potential obesity-relevant clinical indicators included six historic factors (eg mode of delivery, birthweight $z$-score) and 18 concurrent factors (eg sleep problems, BMI, sugary drink consumption) that were assessed every two years (Birth cohort: 2–3, 4–5, 6–7, 8–9 years of age; Kinder cohort: 4–5, 6–7, 8–9, 10–11, 12–13 years of age). Overweight/obesity was defined using International Obesity Taskforce cutpoints at 10–11 years and 14–15 years.

**Results:** In both cohorts, only three factors consistently predicted development or resolution of overweight/obesity in multivariable models. Among normal weight children, an increased odds of developing overweight/obesity was associated with higher child (odds ratio (OR) 1.7–3.8 across all study waves) and maternal (OR 1.05–1.09) BMI, and inversely with higher maternal education (OR 0.60–0.62, when assessed at age 2–7 years). Among overweight/obesity children, a lower odds of resolving to normal weight was associated with higher child (OR 0.51–0.79 across all study waves) and maternal (OR 0.89–0.95) BMI, and inversely with maternal education (OR 1.6–1.9, when assessed at age 2–5 years). The prevalence of overweight/obesity at the age of 14–15 years was 13% among children with none of these risk factors at age 6–7 years, compared with 71% among those with all three risk factors ($P < 0.001$).

**Conclusions:** From early childhood onwards, child and maternal BMI and maternal education predict overweight/obesity onset and resolution by adolescence. These risk factors, easily available to clinicians, could help better target treatment or prevention of child overweight/obesity.

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## Longitudinal association of a body mass index (BMI) genetic risk score with growth and BMI changes across the lifecourse

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**Background:** Genome-wide associations studies have identified genetic variants associated with obesity. However, the effect of genetic variants on body mass index (BMI) at different periods in life remains poorly understood. We examined the association between validated BMI-associated single nucleotide polymorphisms (SNPs) and BMI growth trajectories from childhood to mid-adulthood.

**Methods:** Data were from 2631 participants aged 6–18 years at baseline in the Cardiovascular Risk in Young Finns Study who had up to 8 BMI measures collected over 31 years follow-up. A genetic risk score (GRS) was generated from 98 BMI-associated SNPs. Mixed effect and Latent Class Growth Mixture modelling were used to model individual growth parameters, and identity 6 distinct long-term BMI trajectory groups. Associations between the GRS, growth parameters, BMI trajectory groups was examined using hierarchical linear and multinomial logit regression.

**Results:** The GRS associated with higher BMI at 9 years (females = 0.017 kg/m$^2$ per allele, males = 0.013 kg/m$^2$ per allele) and faster BMI growth in childhood in both sexes (age*GRS interactions $p < 0.01$), resulting in separation of BMI trajectories between participants with high and low GRS throughout childhood. The GRS was also associated with higher weight gain after age 25 years (age*GRS interaction $p < 0.01$, with each unit increase in GRS associated with a 0.12 kg/m$^2$ increase in BMI/year). The relative risk for belonging to the progressively overweight, incident obese, and increasing obese categories vs. stable normal BMI trajectory ranged between 0.3 and 0.9 per unit increase in GRS.

**Conclusions:** Genetic determinants of BMI effect early childhood growth and weight gain in adulthood. These data shed light on biological pathways of genetic predisposition for obesity and provide insight into the timing of BMI changes that lead to the development of adult obesity.

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