**Effects of an Antenatal Dietary Intervention on Maternal Anthropometric Measures in Pregnant Women with Obesity**

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**Objective:** The effect of providing antenatal dietary and lifestyle advice on secondary measures of maternal anthropometry was evaluated and their correlation with both gestational weight gain and infant birth weight was assessed.

**Methods:** In a multicenter, randomized controlled trial, pregnant women with BMI of $\geq 25$ kg/m$^2$ received either Lifestyle Advice or Standard Care. Maternal anthropometric outcomes included arm circumference, biceps, triceps, and subscapular skinfold thickness measurements (SFTM), percentage body fat (BF), gestational weight gain, and infant birth weight. The intention to treat principles were utilized by the analyses.

**Results:** The measurements were obtained from 807 (74.7%) women in the Lifestyle Advice Group and 775 (72.3%) women in the Standard Care Group. There were no statistically significant differences identified between the treatment groups with regards to arm circumference, biceps, triceps, and subscapular SFTM, or percentage BF at 36-week gestation. Maternal anthropometric measurements were not significantly correlated with either gestational weight gain or infant birth weight.

**Conclusions:** Among pregnant women with a BMI of $\geq 25$ kg/m$^2$, maternal SFTM were not modified by an antenatal dietary and lifestyle intervention. Furthermore, maternal SFTM correlate poorly with both gestational weight gain and infant birth weight.

**Introduction**

Overweight and obesity represent a considerable disease burden during pregnancy, with half of all women having a body mass index (BMI) of $\geq 25$ kg/m$^2$ on entering pregnancy (1,2). The literature highlights the associations between maternal obesity and an increased risk of adverse outcomes for both women and their infants, all of which increase as maternal BMI increases (3,4). Additionally, there is evidence of a persisting longer-term health legacy, maternal obesity being associated with an increased risk of high infant birth weight (4) and, in turn, subsequent obesity (5,6).

The US Institute of Medicine has summarized the observational literature relating to maternal gestational weight gain, reporting average total weight gains between 10 and 15 kg (7). However, there was considerable interindividual variation with many women,
particular those with a BMI of \( \geq 25 \) kg/m\(^2\), exceeding this average (7). Nevertheless, current recommendations suggest a weight gain of 7.0-11.5 kg for women with a BMI of 25-29.9 kg/m\(^2\) and 5.0-9.0 kg for women with a BMI of 30 kg/m\(^2\) or more (7).

In nonpregnant individuals, there is a clear relationship between measured weight gain and adiposity gain. In contrast, gestational weight gain while used as a surrogate marker of adiposity (8) in fact reflects a combination of maternal fat deposition, pregnancy-related plasma volume expansion, peripheral edema, placental mass, fetal mass, and amniotic fluid volume (9). Furthermore, from a practical perspective, determining the relative contribution of each of these individual components is extremely difficult (9).

Although a number of techniques have been proposed to differentiate between the various components and more accurately determine maternal adiposity, all have limitations, particularly during pregnancy. Although BMI correlates well with the markers of adult health and disease risk, it does not differentiate between adipose and lean tissue mass or between maternal and fetal contributions to weight gain. Bioimpedance analysis, dual-energy X-ray absorptiometry, computed tomography, magnetic resonance imaging, and three or four compartment model assessments all more accurately determine adipose and lean tissues. Limitations include the lack of differentiation between maternal and fetal tissues, exposure to ionizing radiation (9,10), and lack of portability and expense (9,11,12), all of which make these assessments impractical to carry out on a large scale.

In contrast, skinfold thickness measurements (SFTM) have been proposed for use in pregnancy as they are reproducible with specific training and adherence to defined protocols, and through the measurement of between three and seven body sites, they allow estimation of body fat (BF) percentage (9,13). In particular, use of the biceps, triceps, and subscapular sites allows the evaluation of pregnancy-related changes in adipose tissue that are more independent of fetal growth (9,14). Although some have suggested that SFTM lack reproducibility (15), we have documented acceptable interobserver variability from a population of pregnant women with a BMI of \( \geq 25 \) kg/m\(^2\) (16).

Observational studies involving lean pregnant women suggest deposition of adipose tissue in the second trimester of pregnancy with subsequent mobilization in late gestation (17-20). It is unclear if this is also the case among women with a BMI of \( \geq 25 \) kg/m\(^2\), who have increased reliance on lipid metabolism during pregnancy (21). Similarly, the relationship between changes in adipose tissue deposition and SFTM, particularly among women with a BMI of \( \geq 25 \) kg/m\(^2\) (19,22), and any correlation with either gestational weight gain or infant birth weight (23,24) remain to be determined.

We have reported the primary findings of the LIMIT randomized trial evaluating the provision of antenatal dietary and lifestyle advice to women with a BMI of \( \geq 25 \) kg/m\(^2\), which indicate a significant 18% relative risk reduction in infant birth weight of \( \geq 4 \) kg (25), mediated by improvements in maternal diet and physical activity (26). The aims of this study were twofold. First, we report the effect of providing antenatal dietary and lifestyle advice on the measures of maternal SFTM; and second, we assess the correlation between maternal SFTM and both gestational weight gain and infant birth weight.

### Methods

#### Study design

We conducted a multicenter, randomized trial involving three maternity hospitals across Adelaide, South Australia. The methods (27) and primary findings (25,26,28) from the LIMIT randomized trial have been reported, and the trial registered on the Australian and New Zealand Clinical Trials Registry (ACTRN12607000161426).

#### Inclusion and exclusion criteria

Women with BMI of \( \geq 25 \) kg/m\(^2\) and singleton pregnancy at 10\(^+0\). 20\(^+0\) weeks gestation were eligible.

#### Trial entry

All women presenting for antenatal care had their height and weight measured, and BMI calculated at the first antenatal appointment. Eligible women were presented with written information, and all women provided written informed consent.

#### Randomization, masking, and group allocation

Randomization occurred by telephoning the central randomization service, which used a computer-generated schedule, with balanced variable blocks. Stratification occurred for parity (0 vs. 1/more), BMI at antenatal booking (25-29.9 vs. \( \geq 30 \) kg/m\(^2\)), and collaborating center. Women were randomized to either “Lifestyle Advice” or “Standard Care.”

#### Treatment schedules

**Lifestyle Advice Group.** Women randomized to receive Lifestyle Advice participated in a comprehensive dietary and lifestyle intervention over the course of pregnancy that included a combination of dietary, physical activity, and behavioral strategies, delivered by a research dietician and trained research assistants (25,26). Within 2 weeks of randomization, women attended a planning session with a research dietician, during which a detailed dietary and physical activity history was obtained (25,26). Women were provided with individualized dietary advice consistent with current Australian standards, to maintain a balance of carbohydrates, fat, and protein, to reduce intake of foods high in refined carbohydrates and saturated fats, while increasing intake of fiber, and promoting consumption of two servings of fruit, five servings of vegetables, and three servings of dairy each day. Physical activity advice focussed on the benefits of exercise in pregnancy, and tips to increase incidental activity and walking. Women were encouraged to set achievable goals for change, and to self-monitor their progress. This information was reinforced during subsequent inputs provided by the research dietician (at 28-week gestation) and trained research assistants (via telephone call at 22-, 24-, and 32-week gestation and a face-to-face visit at 36-week gestation).

**Standard Care Group.** Women receiving Standard Care continued their pregnancy care according to local hospital guidelines, which did not include routine provision of advice related to diet, exercise, or gestational weight gain.

#### Study end points: maternal anthropometry

At trial entry and 36-week gestation, maternal anthropometric measurements were obtained (16), using a standardized protocol on the right-hand side of the woman’s body (13).
Arm circumference. With the woman standing with her arms relaxed at her side, the midpoint between the most superior and the lateral point of the acromion border and the most proximal and lateral border of the head of the radius was identified and the circumference measured using the crosshand technique, with the reading taken at eye level with constant tension applied to the tape (13,16). Two separate marks were made on the woman’s arm on the most anterior point of the biceps and the most posterior point of the triceps, to assist in locating these skinfold landmarks (13,16).

Skinfold thickness measurements. SFTM were obtained using Harpenden Callipers, with a dial graduation of 0.2 mm and measuring range 0-80 mm, which was viewed at 90° to avoid errors of parallax. Two measurements were taken, with a third measurement obtained if the difference was >7.5% (13,16). The measurements were recorded to the nearest 0.1 mm and reported as the average of two measurements or the median of 3 (13,16).

Biceps. With the woman standing relaxed, with arms at her side, the biceps skinfold was visualized, and the site located on the anterior surface of the arm, in line with the mid-arm point (as marked when the arm circumference was measured) and parallel to the long axis (13,16).

Triceps. With the woman standing relaxed and her arm slightly pronated, the triceps skinfold was visualized from the posterior surface of the arm, in line with the mid-arm point in the horizontal plane (as marked when the arm circumference was measured), and parallel to the long axis (13,16).

Subscapular. The inferior angle of the scapula was palpated, and a site marked 2 cm and 45° inferiorly (13,16). If required, the woman was asked to reach behind her back with her right arm to better expose the scapula (13,16).

BF percentage. Using the following equation for use with pregnant women with a BMI of ≥25 kg/m², percentage BF was calculated, where SFTM were reported in millimeters, and arm circumference and height were reported in centimeters (16).
Gestational weight gain. Gestational weight gain was calculated as the difference in measured weight by subtracting the weight obtained at trial entry from the weight obtained at 36 weeks of gestation. Weight was obtained with the woman in light clothing and without shoes, using digital scales, and recorded to the nearest 0.1 kg. Height was obtained without shoes, using a wall-mounted stadiometer, and recorded to the nearest 0.5 cm.

Analysis and reporting of results

The analyses were performed on an intention to treat basis, with women analyzed according to their randomized treatment group. Women were included in the analysis if they consented to anthropometric measurements being taken, and did not withdraw consent to use their data or have a miscarriage, or termination of pregnancy. The outcomes considered were the anthropometric measurements taken at 36-week gestation, and both the total and the average weekly change in anthropometric measurements between trial entry and 36 weeks. The analyses were performed using linear regression models to determine the effect of treatment group on each outcome, adjusting for center, parity, BMI category, age, socioeconomic status, the anthropometric measurement at trial entry, and either the actual gestational age at measurement (for anthropometric measurements at 36 weeks), or the time between measurements (for changes in anthropometric measurements). Exploratory analyses were conducted to assess whether the effect of treatment varied by BMI category (overweight vs. obese), by including an interaction between treatment group and BMI category in the regression model.

### TABLE 1 Baseline demographic and anthropometric measurements at trial entry

| Characteristic | Lifestyle Advice, N = 872<sup>a</sup> | Standard Care, N = 835<sup>a</sup> | Total, N = 1,707<sup>a</sup> |
|---------------|-----------------------------------|---------------------------------|-------------------------------|
| Maternal age (years)<sup>b</sup> | 29.6 (5.3) | 29.6 (5.5) | 29.6 (5.4) |
| Gestational age at entry (weeks)<sup>c</sup> | 14.1 (12.0-16.9) | 14.3 (12.0-17.0) | 14.1 (12.0-17.0) |
| BMI (kg/m<sup>2</sup>)<sup>c</sup> | 31.0 (28.0-35.9) | 31.1 (27.6-35.8) | 31.0 (27.8-35.8) |
| **BMI category** | | | |
| 25.0-29.9 | 365 (41.9) | 360 (43.1) | 725 (42.5) |
| 30.0-34.9 | 249 (28.6) | 238 (28.5) | 487 (28.5) |
| 35.0-39.9 | 167 (19.2) | 135 (16.2) | 302 (17.7) |
| ≥40.0 | 91 (10.4) | 102 (12.2) | 193 (11.3) |
| Public patient<sup>d</sup> | 855 (98.1) | 814 (97.5) | 1669 (97.8) |
| Weight (kg)<sup>b</sup> | 88.4 (17.0) | 88.3 (17.6) | 88.3 (17.3) |
| Height (cm)<sup>b</sup> | 164.9 (6.6) | 164.9 (6.5) | 164.9 (6.5) |
| **Race**<sup>d</sup> | | | |
| Caucasian | 789 (90.5) | 764 (91.5) | 1553 (91.0) |
| Asian | 19 (2.2) | 21 (2.5) | 40 (2.3) |
| Indian | 30 (3.4) | 27 (3.2) | 57 (3.3) |
| Other | 34 (3.9) | 23 (2.8) | 57 (3.3) |
| Smoker<sup>d</sup> | 110 (12.6) | 93 (11.1) | 203 (11.9) |
| Nulliparous<sup>d</sup> | 354 (40.6) | 358 (42.9) | 712 (41.7) |
| **Index of socio economic disadvantage**<sup>e</sup> | | | |
| Unknown | 1 (0.1) | 1 (0.1) | 2 (0.1) |
| Quintile 1 (most disadvantaged) | 262 (30.0) | 240 (28.7) | 502 (29.4) |
| Quintile 2 | 207 (23.7) | 212 (25.4) | 419 (24.5) |
| Quintile 3 | 133 (15.3) | 125 (15.0) | 258 (15.1) |
| Quintile 4 | 126 (14.4) | 133 (15.9) | 259 (15.2) |
| Quintile 5 (least disadvantaged) | 143 (16.4) | 124 (14.9) | 267 (15.6) |
| Arm girth at trial entry (cm)<sup>b</sup> | 35.5 (4.4) | 35.4 (4.4) | 35.5 (4.4) |
| Biceps SFTM at trial entry (mm)<sup>b</sup> | 18.2 (7.5) | 17.4 (6.9) | 17.8 (7.2) |
| Triceps SFTM at trial entry (mm)<sup>b</sup> | 27.8 (7.3) | 28.4 (7.4) | 28.1 (7.4) |
| Subscapular SFTM at trial entry (mm)<sup>b</sup> | 28.8 (9.1) | 29.4 (9.7) | 29.1 (8.4) |
| Percent BF at trial entry<sup>b</sup> | 37.4 (7.2) | 37.7 (7.5) | 37.6 (7.3) |
| GA at trial entry measurement (weeks)<sup>c</sup> | 17.0 (14.3-19.9) | 18.9 (14.9-20.4) | 17.6 (14.6-20.3) |

<sup>a</sup>Includes all women randomized who did not withdraw consent to use their data and who had anthropometric measurements taken at baseline.

<sup>b</sup>Mean and standard deviation.

<sup>c</sup>Median and interquartile range.

<sup>d</sup>Number and percentage.

<sup>e</sup>Socio economic index as measured by SEIFA.

BF% = 12.7 ± 0.457 (Triceps SFTM) + 0.352 (Subscapular SFTM) + 0.103 (Biceps SFTM) − 0.057 (Height) + 0.265 (Arm circumference).
### TABLE 2 Maternal anthropometric outcomes by treatment group\textsuperscript{a}

| Outcome                        | Lifestyle Advice Group | Standard Care Group | Unadjusted treatment effect (95% CI) | Unadjusted P-value | Adjusted treatment effect (95% CI) | Adjusted P-value |
|--------------------------------|------------------------|----------------------|-------------------------------------|--------------------|-----------------------------------|------------------|
| Arm girth at 36 weeks (cm)     | 35.61 (4.13)           | 35.47 (4.24)         | 0.14 (−0.27, 0.56)                  | 0.4909             | −0.03 (−0.19, 0.12)               | 0.6709           |
| Total change in arm girth (cm) | 0.06 (1.66)            | 0.14 (1.61)          | −0.08 (−0.25, 0.09)                 | 0.3474             | −0.03 (−0.19, 0.13)               | 0.7188           |
| Average weekly change in arm girth (cm) | 0.00 (0.09) | 0.01 (0.09) | −0.00 (−0.01, 0.00) | 0.3199 | −0.00 (−0.01, 0.01) | 0.6952 |
| Biceps skinfold at 36 weeks (mm) | 16.32 (6.26) | 15.95 (5.97) | 0.38 (−0.23, 0.98) | 0.2230 | 0.06 (−0.46, 0.58) | 0.8124 |
| Total change in biceps skinfold (mm) | −1.88 (6.54) | −1.38 (6.55) | −0.50 (−1.17, 0.17) | 0.1409 | 0.02 (−0.50, 0.55) | 0.9289 |
| Average weekly change in biceps skinfold (mm) | −0.10 (0.35) | −0.08 (0.36) | −0.03 (−0.06, 0.01) | 0.1356 | −0.00 (−0.03, 0.03) | 0.8673 |
| Triceps skinfold at 36 weeks (mm) | 27.91 (7.60) | 27.44 (7.29) | 0.48 (−0.26, 1.21) | 0.2032 | 0.58 (−0.07, 1.23) | 0.0790 |
| Total change in triceps skinfold (mm) | −0.00 (7.71) | −0.83 (7.42) | 0.83 (0.06, 1.61) | 0.0346 | 0.53 (−0.13, 1.18) | 0.1149 |
| Average weekly change in triceps skinfold (mm) | −0.00 (0.42) | −0.05 (0.42) | 0.05 (0.01, 0.09) | 0.0203 | 0.03 (−0.00, 0.07) | 0.0806 |
| Subscapular skinfold at 36 weeks (mm) | 29.91 (8.82) | 29.66 (8.98) | 0.25 (−0.63, 1.14) | 0.5773 | 0.55 (−0.12, 1.23) | 0.1052 |
| Total change in subscapular skinfold (mm) | 0.90 (7.74) | 0.05 (7.24) | 0.85 (0.07, 1.62) | 0.0325 | 0.50 (−0.17, 1.18) | 0.1429 |
| Average weekly change in subscapular skinfold (mm) | 0.05 (0.43) | −0.00 (0.42) | 0.05 (0.01, 0.09) | 0.0290 | 0.03 (−0.01, 0.07) | 0.1244 |
| Percent BF at 36 weeks          | 37.66 (7.12)           | 37.25 (7.02)         | 0.40 (−0.30, 1.11)                  | 0.2619             | 0.48 (−0.04, 1.00)                | 0.0711           |
| Total change in percent BF      | 0.12 (5.84)            | −0.45 (5.65)         | 0.56 (−0.03, 1.16)                  | 0.0650             | 0.43 (−0.09, 0.95)                | 0.1082           |
| Average weekly change in percent BF | 0.00 (0.32) | −0.03 (0.32) | 0.03 (0.00, 0.07) | 0.0448 | 0.03 (−0.00, 0.06) | 0.0821 |

*Values are mean (SD) and treatment effects are differences in means. Adjusted for center, parity, BMI category, age, socioeconomic status quintile, smoking status, baseline measurement, and either GA at measurement (for 36-week measurement outcomes) or time between measurements (for total and average weekly change). Includes all women randomized who had anthropometric data collected, did not have a miscarriage or termination, and did not withdraw consent to use their data.*
Statistical significance was assessed at the two-sided \( P < 0.05 \) level and no adjustment was made for multiple comparisons. Pearson’s correlation coefficients were calculated to measure the strength of association between anthropometric measurements, total gestational weight gain, and infant birth weight. All analyses were performed using SAS v9.3 (Cary, NC, USA).

**Sample size**

The sample size of 2,180 women was predetermined based on the primary outcome, large for gestational age infant (25).

**Ethics**

Ethics approval was provided by the Women’s and Children’s Local Health Network Human Research and Ethics Committee at the Women’s and Children’s Hospital, the Central Northern Adelaide Health Service Ethics of Human Research Committee (Lyell McEwin Hospital) and the Flinders Clinical Research Ethics Committee (Flinders Medical Centre).

**Results**

Between June 2008 and December 2011, 2,212 women were randomized, with 1,108 allocated to receive Lifestyle Advice, and 1,104 Standard Care. There were 2,154 women (1,080 Lifestyle Advice; 1,072 Standard Care) available for inclusion in the analyses, after excluding women who withdrew consent to use their data (ten women) or had a miscarriage, or termination of pregnancy (50 women) (25). The measurements were obtained from 872 (80.7%) women at trial entry, and 807 (74.7%) at 36 weeks of gestation in the Lifestyle Advice Group, and 835 (77.9%) women at trial entry, and 775 (72.3%) at 36 weeks of gestation in the Standard Care Group, who were included in the analyses (Figure 1). The median time between trial entry and 36-week gestation measurements was 18.4 weeks (interquartile range, 16.0-21.6 weeks). Baseline demographic and anthropometric measures of the women who had their measurements obtained were similar between treatment groups (Table 1) and their clinical characteristics similar to the full randomized groups (data not shown) (25).

There were no statistically significant differences between those women receiving Lifestyle Advice and those receiving Standard Care, with regards to arm circumference, and biceps, triceps, and subscapular SFTM obtained at 36-week gestation (Table 2). There was no difference observed between groups in the change in these measurements from baseline to 36 weeks, when expressed either as total change, or as an average weekly change to account for variation in gestational age at measurement. The calculated percentage BF at 36-week gestation did not significantly differ between the two treatment groups (37.66 ± 7.12% Lifestyle Advice vs. 37.25 ± 7.02% Standard Care; adjusted mean difference 0.48; 95% CI −0.01 to 1.00; \( P = 0.07 \)), with no difference observed in the change in percentage BF expressed either as total change across gestation, or as average weekly change. There was no evidence to suggest that the intervention effect was modified by maternal BMI category (data not shown).

All SFTM and arm circumference measurements were not significantly correlated with either gestational weight gain or infant birth weight, correlation coefficients ranging between −0.239 and 0.111 (Table 3).

**Discussion**

Our randomized trial is the largest reported to date evaluating the effects of an antenatal lifestyle intervention for women with a BMI of \( \geq 25 \) kg/m\(^2\) during pregnancy on maternal anthropometric measures, and it utilized robust methodology. Our findings indicate that providing an intervention during pregnancy was not effective in reducing gestational weight gain (25) or maternal measures of peripheral adipose tissue deposition. Nor was there any effect on maternal percentage BF, with no evidence to suggest this differed among women with a BMI of 25–29.9 kg/m\(^2\) and \( \geq 30 \) kg/m\(^2\). Furthermore, SFTM and anthropometric measures were not significantly correlated with either gestational weight gain or infant birth weight.

Our study has a number of strengths, including the large sample size of pregnant women with a BMI of \( \geq 25 \) kg/m\(^2\), which has been a
limitation of the literature to date. Although there are limitations to the use of skinfold measurements as an accurate predictor of adiposity, we adhered to a validated protocol, with acceptable reported interobserver variability (16), allowing comparisons between treatment groups to be made. Use of alternative methods of the assessment of maternal adiposity may have yielded more accurate estimates of the relative proportions of adipose and lean tissue mass although these were not considered feasible for use on such a large scale.

It is widely assumed that women deposit adipose tissue during pregnancy, with mobilization of peripheral stores as a fuel source for fetal growth in late gestation and during lactation (29). A number of observational studies suggest that there is maximal subcutaneous fat accumulation as measured by skinfold thickness, during the second trimester of pregnancy, followed by a decline in the third trimester as an indicator of tissue mobilization (17-20). However, these studies have largely been confined to relatively lean pregnant women, with the mean reported early pregnancy BMI within the normal range (17-20).

In contrast, description of the subcutaneous accumulation of fat stores across gestation among women with a BMI of $\geq 25$ kg/m$^2$ is less conclusive. Although some suggest that there is a decrease in the measures of subcutaneous adipose tissue over pregnancy (19), others have reported relative stability in SFTM, particularly involving the triceps and subscapular sites (22). However, changes in SFTM across pregnancy appear to be site specific, with increases in central adipose tissue deposition reported by some, as measured at the suprailiac, subscapular (30), and thigh skinfold sites (22). Importantly, there appears to be considerable interindividual variation, with pregnant women with high BMI reported to have a greater variation in the change in fat mass over time when compared with women of normal BMI (31,32). Although we assessed upper body measures only, our findings are consistent with those reported by Sidebottom et al. (22), indicating relatively little change in SFTM and subcutaneous adipose tissue accumulation in the second and third trimesters of pregnancy. Additional assessment of maternal SFTM at 28 weeks of gestation may have allowed greater assessment of mobilization of peripheral adipose tissue stores during gestation although it was not considered feasible in the context of this study, where participant burden was considerable.

Although measured weight gain in nonpregnant individuals is more closely reflective of adiposity gain, measuring gestational weight gain in pregnancy is a relatively poor surrogate for maternal adiposity (8), highlighting the considerable variability which exists across individual women in the composition of that weight gain (11,12). Among relatively lean pregnant women, subcutaneous measures of adipose tissue deposition have been demonstrated to correlate poorly with gestational weight gain (14,22) although this observation is not universal, with others suggesting a positive association (32,33). Furthermore, measured gains in maternal adipose tissue appear to be poorly predictive of infant birth weight (32-34), conferring little advantage over traditional assessment of prepregnancy or early pregnancy BMI (14,20), perhaps with the exception of women with low prepregnancy weight, where there appears to be a negative correlation with low infant birth weight (35,36).

To date, associations between measured maternal adiposity, gestational weight gain, and infant birth weight among women with a BMI of $\geq 25$ kg/m$^2$ have been poorly investigated. Although observational data indicate women with obesity have greater gain in central adiposity (compared with women of normal BMI), there is poor correlation with infant birth weight (23,24). These findings are consistent with ours. From a metabolic perspective, changes which occur during pregnancy are highly complex as it is their relationship to maternal BMI, adiposity gain, gestational weight gain, and infant birth weight. It is therefore not surprising that although these measures may be associated with each other, changes in one may not necessarily correspond to observable or measurable changes in another.

As we have reported previously, providing antenatal lifestyle advice among women with a BMI of $\geq 25$ kg/m$^2$ was associated with an $18\%$ relative risk reduction in infant birth weight of $>4$ kg (25), an effect mediated by changes in maternal diet quality and physical activity (26) despite the fact that both maternal gestational weight gain (25) and total energy intake during pregnancy (26) did not differ significantly between the randomized groups. Our intervention consisted of three face-to-face sessions and three telephone contacts over the course of pregnancy and was of similar intensity to those utilized in the previously published studies (37-39). Although increasing the intensity of the intervention may have been associated with greater effects on maternal gestational weight gain and, in turn, maternal SFTM, it was not considered feasible to implement within current maternity care models. Furthermore, our findings suggest that focusing attention solely on gestational weight gain, rather than achievable, albeit modest changes in dietary intake and physical activity, may reduce important opportunities to significantly impact clinically relevant infant outcomes (25,28).

Conclusion

As indicated previously, the use of gestational weight gain is a surrogate measure of maternal adiposity (8,9). Although both gestational weight gain and maternal adiposity as assessed by peripheral SFTM are relatively simple measures to obtain, they were not impacted by our antenatal lifestyle intervention. Furthermore, among women with a BMI of $\geq 25$ kg/m$^2$, maternal SFTM and measures of anthropometry correlated poorly with both gestational weight gain and infant birth weight. Further mechanistic information is required to elucidate the specific changes in maternal metabolic pathways over the course of pregnancy and their relationship, if any, to peripheral measures of adiposity.

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References

1. Cho SY, Kim SY, Bish CL. Prepregnancy obesity prevalence in the United States, 2004–2005. Maternal Child Health J 2008;13(5):614–620.
2. National Health and Medical Research Council. NHMRC Strategic Plan 2013-2015. Report no. NH160. Canberra, Australia: Commonwealth of Australia; 2012.
3. Callaway LK, Prins JB, Chang AM, McIntyre HD. The prevalence and impact of overweight and obesity in an Australian obstetric population. Med J Aust 2006;184:56–59.
4. Dodd JM, Grivell RM, Nguyen A-M, Chan A, Robinson JS. Maternal and perinatal health outcomes by body mass index category. Austr N Z J Obstet Gynaecol 2011;51:136–140.
5. Mollberg M, Hagberg H, Bager B, Lilja H, Ladfors L. High birthweight and shoulder dystocia: the strongest risk factors for obstetrical brachial plexus palsy in a Swedish population-based study. Acta Obstet Gynecol Scand 2005;84:654–659.
6. Winter JD, Langenberg P, Krugman SD. Newborn adiposity by body mass index predicts childhood overweight. Clin Pediatr 2010;49:866–870.
7. Institute of Medicine and National Research Council. Weight Gain During Pregnancy: Reexamining the Guidelines. Washington D.C.: The National Academies Press; 2009.
8. van der Wijden CL, Delemarre-van der Waal HA, van Mechelen W, van Poppel MN. The concurrent validity between leptin, BMI and skin folds during pregnancy and the year after. Nutr Diab 2013;3:86.
9. Widen EM, Gallagher D. Body composition changes in pregnancy: measurement, predictors and outcomes. Eur J Clin Nutr 2014;68:643–652.
10. Fatiah C, Farah N, Barry S, O’Connor N, Stuart B, Turner MJ. The measurement of maternal adiposity. J Obstet Gynaecol 2009;29:686–689.
11. Hopkinson JM, Butte NF, Ellis KJ, Wong WW, Puaya MR, Smith EO. Body fat estimation in late pregnancy and early postpartum: comparison of two-, three-, and four-component models. Am J Obstet Gynecol 1997;175:432–438.
12. Kopp-Hoolihan LE, van Loun MD, Wong WW, King JC. Fat mass deposition during pregnancy using a four-component model. J Appl Physiol 1999;87:196–202.
13. Marfell-Jones M, Olds T, Stewart A, Carter L. International Standards for Anthropometric Assessment. The International Society for the Advancement of Kinanthropometry; 2006.
14.Anderson GD, Bildner IN, Mc Clement S, Sinclair JC. Determinants of size at birth in a Canadian population. Am J Obstet Gynecol 1984;150:236–244.
15. Hu FB. Obesity Epidemiology. Oxford: Oxford University Press; 2008. p 53–83.
16. Kannieapapan LM, Deussen AR, Grivell RM, Yelland LN. Dodd JM. Developing a tool for obtaining maternal skinfold thickness measurements and assessing inter-observer variability among pregnant women who are overweight and obese. BMC Pregnancy Childbirth 2014;13:42.
17. Hediger ML, Scholl TO, Schall JI, Healey MF, Fischer RL. Changes in maternal upper arm fat stores are predictors of variation in infant birth weight. J Nutr 1994;124:24–30.
18. Sadaranski A, Kabir N, Wager J, Forsum E. Energy metabolism, body composition, and milk production in healthy Swedish women during lactation. Am J Clin Nutr 1988;48:44–49.
19. Straughen JK, Trudeau S, Misra VK. Changes in adipose tissue distribution during pregnancy in overweight and obese compared with normal weight women. Nutr Diab 2013;3:84.
20. Villar J, Cogswell M, Kestler E, Castillo P, Menendez R, Repke JT. Effect of fat and fat-free mass deposition during pregnancy on birth weight. Am J Obstet Gynecol 1992;167:1344–1352.
21. Okereke NC, Huston-Presley L, Ammini SB, Kalhan S, Catalano PM. Longitudinal changes in energy expenditure and body composition in obese women with normal and impaired glucose tolerance. Am J Physiol Endocrinol Metab 2004;287:E472–E479.
22. Sidebottom AC, Brown JE, Jacobs DR Jr. Pregnancy-related changes in body fat. Eur J Obstet Gynecol Reprod Biol 2001;94:216–223.
23. Farah N, Stuart B, Donnelly V, Kennelly MM, Turner MJ. The influence of maternal body composition on birth weight. Eur J Obstet Gynecol Reprod Biol 2011;157:14–17.
24. Kent E, O’Dwyer V, Fatiah C, Farah N, O’Connor C, Turner MJ. Correlation between birth weight and maternal body composition. Obstet Gynecol 2013;121:46–50.
25. Dodd JM, Turnbull DA, McPhee AJ, et al. Antenatal lifestyle advice for women who are overweight or obese: the LIMIT randomised trial. Br Med J 2014;348:g1285.
26. Dodd JM, Cramp CS, Sui Z, et al. The effects of antenatal lifestyle advice for women who are overweight or obese on maternal diet and physical activity: the LIMIT randomised trial. BMC Med 2014;12:161.
27. Dodd JM, Turnbull DA, McPhee AJ, Wittet G, Robinson JS. Limiting weight gain in overweight and obese women during pregnancy to improve health outcomes: the LIMIT randomised controlled trial. BMC Pregnancy Childbirth 2011;11:79.
28. Plagemann AG, Jelovšek M, Drong G. Maternal diet and fat-free mass deposition during pregnancy on birth weight. Eur J Obstet Gynecol Reprod Biol 2011;154:192–196.
29. van Raaij JM, Schonk CM, Vermaat-Miedema SH, Peak ME, Hautvast JG. Body fat mass and basal metabolic rate in Dutch women before, during, and after pregnancy: a reappraisal of energy cost of pregnancy. Am J Clin Nutr 1989;49:765–772.
30. Ehrenberg HM, Huston-Presley L, Catalano PM. The influence of obesity and gestational diabetes mellitus on accretion and the distribution of adipose tissue in pregnancy. Am J Obstet Gynecol 2003;189:944–948.
31. Soltani H, Fraser RB. A longitudinal study of maternal anthropometric changes in normal weight, overweight and obese women during pregnancy and postpartum. Br J Nutr 2000;84:95–101.
32. Maple-Brown LJ, Roman NM, Thomas A, Pesley LH, Catalano PM. Perinatal factors relating to changes in maternal body fat in late gestation. J Perinatol 2013;33:934–938.
33. Langhoff-Roos J, Lindmark G, Grebe-Medhin B. Maternal fat stores and fat accretion during pregnancy in relation to infant birth weight. Br J Obstet Gynaecol 1987;94:1170–1177.
34. Forsum E, Sadaranski A, Wager J. Resting metabolic rate and body composition of healthy Swedish women during pregnancy. Am J Nutr 1988;47:942–947.
35. Kelly A, Kevany J, de Onis M, Shah FM. A WHO collaborative study of maternal anthropometry and pregnancy outcomes. Int J Gynaecol Obstet 1996;53:219–233.
36. Sanin Aguirre L, Reza-Lopez S, Levario-Carrillo M. Relation to maternal body composition and birth weight. J Pediatr 2004;86:55–62.
37. Dodd JM, Grivell RM, Crowther CA, Robinson JS. Antenatal interventions for overweight or obese pregnant women: a systematic review of randomised trials. Br J Obstet Gynaecol 2011;117:1316–1326.
38. Oteng-Ntim E, Varma R, Croker H, Poston L, Doyle P. Lifestyle interventions for overweight and obese pregnant women to improve pregnancy outcome: systematic review and meta-analysis. BMC Med 2012;10:47.
39. Thangaratinam S, Rogozinska E, Jolly K, et al. Effects of interventions in pregnancy on maternal weight and obstetric outcomes: meta-analysis of randomised evidence. Br Med J 2012;344:e2088.