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Metabolism Clinical and Experimental 55 (2006) 561–562

Metabolism
Clinical and Experimental

www.elsevier.com/locate/metabol

Preliminary report

The interrelation of birth weight and regional lipid deposition: a twins study

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Received 10 May 2005; accepted 18 January 2006

Abstract

This study examined the hypothesis that low birth weight is associated with changes in regional lipid deposition as well as insulin sensitivity in adult twins. Eleven adult female twin pairs were studied by magnetic resonance to determine regional adiposity. Their insulin sensitivity was assessed by the homeostasis model assessment. There were significant associations between birth weight and current homeostasis model assessment value ($r = -0.528$, $P = .012$), abdominal visceral ($r = -0.581$, $P = .005$), and subcutaneous fat volumes ($r = -0.638$, $P = .001$) if the group of 22 subjects were analyzed as individuals. There were no significant associations of the intratwin pair difference in birth weight and differences between adult twins in these same variables possibly because of limited patient numbers. Reduced birth weight does confer an increased risk of abdominal adiposity as well as insulin resistance in twin populations as it does in the general population.

1. Introduction

Low birth weight is associated in epidemiologic studies with cardiovascular disease, central adiposity, and the metabolic syndrome [1]. It has been suggested that this is caused by a programming effect of intrauterine malnutrition. The reduced fetal growth during gestation in twin pregnancies may have its origin outside inadequate maternal nutrient supply and might therefore have different long-term metabolic consequences [2]. Most twin studies report an association between low birth weight and insulin resistance in adult life and that association is supported by within twin pair analysis [3,4]. Recent studies suggested that twinning itself is associated with increased insulin resistance and, among twins, low birth weight may not impart increased risk [5,6]. These latter studies may be in the minority, but the question of an association between abdominal adiposity and low birth weight in twins remains unclear [7]. In this study of adult twins we examine the hypothesis that low birth weight is associated with changes in regional lipid deposition as well as insulin sensitivity.

2. Research design and methods

We examined 6 pairs of monozygotic and 5 pairs of dizygotic female twins. Subjects were examined for body fatness by bioelectrical impedance analysis, for intramyocellular lipid by magnetic resonance spectroscopy (1.5 T magnet, Philips, Best, Netherlands) of the soleus muscle, for abdominal adiposity by magnetic resonance imaging in 5 axial slices centered at the L4 vertebral body, and insulin sensitivity was assessed by the homeostatic model assessment (HOMA) method [8].

These variables were compared within twin pairs according to relative birth weight using paired t tests. Correlations were measured between birth weight and current metabolic variables for the 22 subjects individually to assess whether the associations reported in the general population also exist in this nonrandomly selected population of twins. We sought correlation (Pearson coefficient) of the intrapair difference in birth weight with current differences in insulin sensitivity, intramyocellular lipid, and abdominal subcutaneous and visceral adiposity.

3. Results

The average age of the study population was 41.2 years (SD, 8.7 years; range, 30.4-63.0 years). Mean body mass

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doi:10.1016/j.metabol.2006.01.007

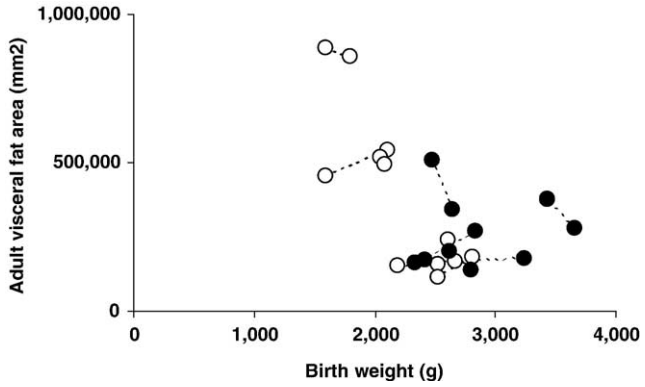

Fig. 1. The relation of birth weight to current visceral fat area assessed by magnetic resonance imaging in 22 twins. The monozygotic twins are represented by open circles and the dizygotic twins by closed circles. Each twin pair is linked by a broken line. There is a significant inverse association (n = 22, $r = -0.581$, $P = .005$).

index was 24.8 kg/m^2 (SD, 5.1; range, 20.5-37.7 years) and percentage of body fatness was 35.0% (SD, 5.9; range, 27.6-47.9 years). Paired t test comparison of the lighter twin at birth to their heavier sibling showed no significant difference in current body fatness ($P = .23$), intramyocellular lipid ($P = .70$), visceral adiposity ($P = .50$), abdominal subcutaneous adiposity ($P = .85$), or HOMA ($P = .17$). Assessing the group of 22 subjects separately, we found significant correlations between birth weight and current HOMA ($r = -0.528$, $P = .012$), current visceral ($r = -0.581$, $P = .005$) (Fig. 1) and subcutaneous ($r = -0.638$, $P = .001$) adiposity, but not with current intramyocellular lipid levels ($r = 0.044$, $P = .847$). There were significant associations between birth weight and current HOMA ($r = -0.586$, $P = .045$), current visceral ($r = -0.824$, $P = .001$) and subcutaneous ($r = -0.822$, $P = .001$) adiposity, but not with current intramyocellular lipid levels ($r = -0.262$, $P = .410$), when assessing just the monozygotic twin pairs. There were no significant associations when assessing just the dizygotic twin pairs.

The intrapair difference in twins' birth weight ranged to 900 g, body mass index difference to 4.1 kg/m^2, subcutaneous fat volume to 506 000 mm^3, and visceral fat volume to 165 000 mm^3. Bivariate correlation of the difference in birth weight with the difference in current insulin sensitivity assessed by HOMA or current differences in body mass index, intramyocellular lipid, visceral or subcutaneous adiposity showed no significant relationship. No relationship was present if the dizygotic or monozygotic twin pairs were analyzed separately.

4. Conclusions

Examination of this population of twin subjects demonstrates that, across the cohort, there are similar significant metabolic associations with birth weight as those previously seen in the general population. Our data extend these associations to abdominal adiposity both visceral and subcutaneous.

The combination of the dizygotic and monozygotic twins in analysis allows only for an examination of the effect of twinned pregnancy upon metabolic characteristics. Combining the data thus does obscure an effect of similar genetic makeup that would be clearer if only monozygotic twins were studied. However, separation of the monozygotic and dizygotic twins has its greatest value if a difference in birth weight is compared with a difference in metabolic parameters in adult life. There were no such associations in the monozygotic group, the dizygotic group, or in the combined group.

The sample size was too small to permit multiple regression analysis and the etiology of this relationship may presumably be similar to that present in the general population. Reduced birth weight adversely affects fat distribution as well as insulin sensitivity in adult life in twinned and in singleton pregnancies.

Acknowledgment

This work was funded by the National Health and Medical Research Council of Australia.

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