



Body surface area at birth and later risk for gestational diabetes mellitus among primiparous women

Senja Masalin^{1,2} · Kristiina Rönö^{2,3} · Hannu Kautiainen^{3,4} · Mika Gissler^{5,6} · Johan G. Eriksson^{1,3,8} · Merja K. Laine^{1,7}

Received: 11 September 2018 / Accepted: 6 November 2018 / Published online: 14 November 2018
© The Author(s) 2018

Abstract

Aims To assess the relationship between body surface area (BSA) at birth and future risk for gestational diabetes mellitus (GDM).

Methods This is an observational cohort study from Vantaa, Finland. The cohort included 1548 Finnish primiparous women, aged 15–28 years, without pre-existing diabetes, who gave birth 2009–2015. All women were born full-term and had complete information about their birth weight and length, from the Finnish Medical Birth Register. Additional data for the study were provided by individual patient health records and Statistics Finland. Study participants were divided into five levels (I–V) according to BSA at birth, based on normal distribution.

Results There was an inverse association between BSA at birth and risk for GDM ($p=0.015$ for linearity, after adjustments for age, educational attainment, pre-pregnancy BMI and smoking). The odds ratio (OR) for GDM in level V, with the largest BSA at birth, compared with level I, with the smallest BSA at birth, was 0.43 [95% confidence interval (CI) 0.22–0.83]; adjusted for age, educational attainment, pre-pregnancy body mass index and smoking. The OR for GDM was 0.8 (95% CI 0.68–0.95, $p=0.009$) for each one standard deviation increase in BSA at birth, adjusted for the same confounders. BSA at birth correlated with adult anthropometry: correlation coefficients were $r=0.16$ (95% CI 0.11–0.21) for weight, $r=0.31$ (95% CI 0.26–0.35) for height, and $r=0.06$ (95% CI 0.01–0.11) for BMI.

Conclusions Body surface area at birth is inversely associated with future risk for GDM in primiparous women.

Keywords Body surface area at birth · Birth length · Birth weight · Gestational diabetes mellitus

Managed by Antonio Secchi.

✉ Senja Masalin
senja.lindholm@helsinki.fi

¹ Department of General Practice and Primary Health Care, University of Helsinki and Helsinki University Hospital, Helsinki, Finland

² Department of Obstetrics and Gynecology, University of Helsinki and Helsinki University Hospital, Helsinki, Finland

³ Folkhälsan Research Center, Helsinki, Finland

⁴ Primary Health Care Unit, Kuopio University Hospital, Kuopio, Finland

⁵ Information Department, National Institute for Health and Welfare, Helsinki, Finland

⁶ Department of Neurobiology, Care Sciences and Society, Karolinska Institute, Stockholm, Sweden

⁷ Vantaa Health Center, Vantaa, Finland

⁸ Department of Chronic Disease Prevention, National Institute for Health and Welfare, Helsinki, Finland

Introduction

Gestational diabetes mellitus (GDM) is a common pregnancy disorder and has long been defined as any degree of abnormal glucose metabolism, first detected or with first onset, during pregnancy [1]. The prevalence of GDM has been rising over the last decade [2, 3], making GDM a serious global health issue today.

Gestational diabetes mellitus has adverse short- and long-term effects on the pregnant woman and her child, both during pregnancy and later in life [4, 5], with maternal obesity augmenting the adverse effects on neonatal outcomes [6]. Some well-known risk factors for GDM include increase in maternal age, genetic predisposition for diabetes mellitus, non-Caucasian ethnicity, as well as maternal obesity [7], possibly also lifestyle and dietary related factors, parental smoking and an early age at menarche [7, 8]. A recent study emphasizes the importance of recognizing GDM as a heterogenic disorder with also non-obese women being

affected—indicating the importance of a large number of risk factors [9]. Further, studies have shown that maternal stature alters glucose metabolism and that short women are at increased risk for GDM [10].

According to the Developmental Origins of Health and Disease (DOHaD) hypothesis prenatal environmental conditions might have long-lasting effects and affect an individual's morbidity in adulthood [11], influencing for example the risk for cardiovascular and metabolic disturbances [12]. In several previous studies, a negative linear association between birth weight and risk for type 2 diabetes (T2D) has been observed [13, 14], although some findings indicating a U-shaped relationship between birth weight and risk of T2D exist [15]. The relationship between birth weight and risk for GDM has also been studied. The results have been conflicting as both studies indicating an inverse association [16–18], as well as a U-shaped relationship have been reported [19–21]. However, birth weight is a very crude measurement of body size at birth. Interestingly, only one recent study has assessed the relationship between maternal birth size using ponderal index (PI) as a measurement of body size at birth and risk for GDM, showing an inverse association [22].

In 2016, we initiated a long-term follow-up study in the city of Vantaa, Finland, to assess both short- and long-term consequences of glucose metabolism on pregnant women and their offspring's health. The relationship between body size at birth and future risk for GDM has previously been studied using primarily birth weight as a measure of body size at birth. Body surface area (BSA) is an anthropometric measurement used to make a more accurate evaluation of metabolic mass and body size. The aim of this study is to evaluate how BSA at birth affects the future risk for GDM.

Materials and methods

Study population

This is an observational cohort study from the city of Vantaa, which is part of the Helsinki metropolitan area and the fourth biggest city in Finland, with around 220,000 inhabitants. During a 7-year follow-up, between January 1st 2009, and December 31st 2015, 13,530 women from Vantaa gave birth. Of these, 1548 primiparous women were Finnish (born in Finland with Finnish or Swedish as native language), without previously diagnosed diabetes mellitus, born at term after the year of 1987 (when the Finnish Medical Birth Register was founded), and aged 15–28 years during the follow-up period. All these women had complete data from the birth register and thereby formed the study cohort.

Data on maternal-fetal characteristics and pregnancy outcomes were obtained from the Finnish Medical Birth

Register, which is administrated by the National Institute for Health and Welfare in Finland and receives the information about all live and stillbirths, from 22 gestational weeks or an offspring weight of at least 500 g onwards, from all Finnish maternity hospitals (<http://www.thl.fi/en/statistics/parturients>). The register started to collect information on deliveries on a nationwide basis from the year 1987. Therefore, only women born 1987 or later were included in this study. The following information about the women was obtained from this source: their own birth weight and birth length, pre-gestational weight in adulthood, adult height, previous pregnancies (miscarriages, induced abortions or ectopic pregnancies), infertility treatments, information about current pregnancies and deliveries, smoking during pregnancy, and hospitalization due to hypertension during pregnancy. Information about GDM, height, and weight was further completed by information from Vantaa Health care patient records.

Since 2008, the nationwide Finnish Current Care Guidelines for GDM recommends screening of all pregnant women during their first pregnancy for GDM using a standard 2-h 75-g oral glucose tolerance test (OGTT), except in women at low risk; that is women with a BMI 18.5–25 kg/m², aged under 25 years and with no family history of diabetes mellitus [23]. The screening is routinely performed between gestational weeks 24 and 28, except in women at high risk; that is women with a pre-pregnancy BMI > 35 kg/m², glucosuria in early pregnancy, family history of diabetes, polycystic ovarian syndrome (PCOS), or use of oral corticosteroids. In these high-risk patients GDM screening is performed earlier in pregnancy between gestational weeks 12 and 16, and if OGTT is normal, it will be repeated between gestational weeks 24 and 28 [23].

GDM was defined according to the Finnish Current Care Guidelines for GDM as one or more pathological glucose values in a standard 2-h 75-g OGTT. The diagnostic thresholds were: fasting plasma glucose ≥ 5.3 mmol/L, 1-h glucose ≥ 10.0 mmol/L, and 2-h glucose ≥ 8.6 mmol/L [23].

Educational attainment was defined according to years of schooling, as obtained from Statistics Finland [Official Statistics of Finland (OSF): http://www.stat.fi/til/vkour/index_en.html].

Body size, taking both weight and height into account, was calculated as BSA, using mathematical formulas. BSA is an anthropometric measurement of interest, in order to make a more accurate evaluation of metabolic mass and body size as a whole. In our study, BSA at birth was calculated according to the *Meban*-BSA formula [24], which in 2008, as evaluated by Ahn et al., has been proved to be the most accurate formula to calculate infant-BSA [25]. Adult pre-pregnancy BSA was calculated according to the commonly used *Mosteller*-BSA formula in adults [26].

Statistical analyses

Data are presented as means with standard deviations (SD) or range, or as counts with percentages. The study population was divided into five levels according to birth BSA levels, based on normal distribution, and corresponding to grades containing 12.5, 25, 25, 25, and 12.5% of the total distribution. Cut-offs for birth BSA levels were: 2011 cm² for level I, 2012–2170 cm² for level II, 2171–2291 cm² for level III, 2292–2450 cm² for level IV, and ≥ 2451 cm² for level V. Statistical significances for the unadjusted hypothesis of linearity across categories of birth BSA levels were investigated by the Cochran–Armitage test for trend and analysis of variance (ANOVA) with an appropriate contrast. Adjusted hypotheses of linearity (orthogonal polynomial) and the association between birth BSA and GDM prevalence were evaluated using logistic models. Models included age, educational attainment, pre-pregnancy body mass index, and smoking as covariates. By using 5-knot-restricted cubic spline regression, a possible nonlinear relationship between prevalence of GDM or maternal birth BSA and PI was assessed. The length of the distribution of knots was located at the 5th, 27.5th, 50th, 72.5th, and 95th percentiles. The relationship between birth BSA and pre-pregnancy weight, height and BMI was assessed using correlation coefficients calculated by the Pearson method. Correlation coefficients less than 0.2 were considered very weak, between 0.2 and 0.4 weak, between 0.4 and 0.6 moderate, between 0.6 and 0.8 strong, and above 0.8 very strong [27]. The normality of the variables was tested using the Shapiro–Wilk *W* test. Statistical significance was set at $p < 0.05$. Stata 15.0 (Stata-Corp LP; College Station, TX, USA) statistical package was used for the analyses.

Results

Characteristics of the study participants

The mean age of the study participants was 22.4 (SD 2.7) years. Baseline characteristics of the 1548 women, divided into five levels, according to body surface area (BSA) at birth is shown in Table 1. Mean BSA at birth was 2231 cm² (SD 191), mean ponderal index was 28.2 kg/m³ (SD 2.5), mean birth weight was 3520 g (SD 472), and mean birth length was 49.9 cm (SD 2.0), respectively. BSA levels at birth showed a positive association with adult pre-pregnancy weight, height and BSA (all $p < 0.001$ for linearity), as well as with pre-pregnancy BMI ($p = 0.004$). There was no significant difference between the different BSA groups at birth and prevalence of hypertensive disorders during pregnancy ($p = 0.20$). Evaluation of the relationship between BSA at birth, and adult pre-pregnancy weight, height and

pre-pregnancy BMI as continuous values showed an overall weak correlation, with the strongest relationship between BSA at birth and adult height. The correlation coefficients were $r = 0.16$ [95% confidence interval (CI) 0.11–0.21] for pre-pregnancy weight, $r = 0.31$ (95% CI 0.26–0.35) for adult height, and $r = 0.06$ (95% CI 0.01–0.11) for pre-pregnancy BMI (Fig. 1).

BSA at birth and prevalence of GDM

Overall GDM prevalence was 12.3% (95% CI 2.3–14.0). BSA at birth was inversely associated with GDM ($p = 0.015$ for linearity, after adjustments for age, educational attainment, pre-pregnancy BMI and smoking). The highest prevalence of GDM, 18.1% (95% CI 12.7–23.5) was observed at level I; whereas, the lowest prevalence of GDM, 9.5% was observed at level V (95% CI 5.7–13.3) (Fig. 2). The odds ratio (OR) for GDM for those with the largest BSA (level V) compared with those with the smallest BSA (level I) was 0.43 (95% CI 0.22–0.83) after adjustments for the same confounders (Fig. 2). Figure 3 illustrates the continuous relationship between BSA at birth, PI at birth and risk for GDM. The OR for GDM was 0.80 (95% CI 0.68–0.95, $p = 0.009$) for each one SD increase in BSA at birth, and the OR for GDM was 0.95 (95% CI 0.80–1.12, $p = 0.53$) for each one SD increase in ponderal index at birth, after adjustment for the same confounders (Fig. 3).

Discussion

According to our study findings, there is a linear inverse association between BSA at birth, and later risk for GDM in primiparous women. The prevalence of GDM was highest (18.1%), among those with the smallest BSA at birth, and lowest (9.5%), among those with the largest BSA at birth. Similarly, the OR for GDM in the group with the largest BSA, compared to the group with the smallest BSA was 0.46. We also used another marker of body size at birth, i.e., ponderal index to evaluate the relationship between body size at birth and risk for GDM. Ponderal index at birth did not predict future risk of GDM. Further, BSA at birth and adult anthropometry correlated, although weakly, with the strongest correlation observed between BSA at birth and adult height.

The GDM prevalence in our study cohort was 12% and the mean age of the primiparous women was 22 years. The mean age of primiparas in Finland during the study period between 2010 and 2015 was 29 years [28]. The nationwide prevalence of GDM in Finland during 2016 was 18% [28], which is higher than the prevalence in the present study. This is probably explained by the fact that the women in our study cohort were primiparas of rather young age, due to

Table 1 Baseline characteristics of 1548 Finnish primiparous women according to body surface area (BSA) at birth

	BSA level at birth					<i>p</i> value for linearity
	I <i>N</i> =183	II <i>N</i> =383	III <i>N</i> =404	IV <i>N</i> =382	V <i>N</i> =186	
Maternal birth characteristics						
BSA (cm ²), mean (range)	1910 (1501–2011)	2097 (2012–2170)	2232 (2171–2291)	2362 (2292–2450)	2547 (2451–2972)	–
Birth length (cm), mean (SD)	47.1 (1.4)	48.8 (1.2)	50.0 (1.1)	51.1 (1.2)	52.5 (1.3)	<0.001
Birth weight (g), mean (SD)	2755 (203)	3185 (117)	3508 (97)	3836 (122)	4321 (266)	<0.001
Ponderal index (kg/m ³), mean (SD)	26.4 (2.2)	27.5 (2.4)	28.1 (2.3)	28.9 (2.2)	30.0 (2.4)	<0.001
Maternal adult characteristics						
Age (years), mean (SD)	22.3 (2.6)	22.3 (2.8)	22.2 (2.6)	22.7 (2.8)	22.7 (2.7)	0.024
Cohabiting, <i>n</i> (%)	125 (68)	270 (71)	285 (71)	266 (68)	132 (71)	0.98
Smoking ^a , <i>n</i> (%)	66 (36)	127 (33)	152 (38)	127 (32)	55 (30)	0.23
Years of education, mean (SD)	11.3 (1.9)	11.2 (2.1)	11.6 (2.1)	11.8 (2.2)	11.9 (2.1)	<0.001
Height (cm), mean (SD)	162 (6)	164 (5)	165 (5)	166 (6)	169 (6)	<0.001
Pre-pregnancy weight (kg), mean (SD)	60.6 (12.2)	63.4 (14.0)	64.2 (12.6)	66.5 (15.0)	68.6 (14.1)	<0.001
Pre-pregnancy BMI (kg/m ²), mean (SD)	23.0 (4.2)	23.6 (4.9)	23.5 (4.5)	24.2 (5.2)	24.1 (4.7)	0.005
BSA (m ²), mean (SD)	1.64 (0.18)	1.69 (0.19)	1.71 (0.17)	1.74 (0.21)	1.78 (0.19)	<0.001
Number of fetuses >1	0 (0)	2 (1)	1 (1)	3 (1)	1 (1)	0.36
Previous pregnancies	49 (27)	75 (20)	70 (17)	72 (18)	33 (18)	0.043
Fertility treatment, <i>n</i> (%)	3 (2)	10 (3)	6 (1)	7 (2)	4 (4)	0.84
Hypertensive disorders ^b , <i>n</i> (%)	16 (9)	24 (6)	16 (4)	21 (5)	11 (6)	0.20

BMI body mass index, *BSA* body surface area, *SD* standard deviation

^aIncluded those who quit smoking during pregnancy

^bHospitalization due to hypertension during pregnancy (including ICD-10 codes O10, O13, and O14)

restrictions of our register-based study setting as the data is limited to women born after 1987, when the Finnish Medical Birth Register was founded.

To the best of our knowledge, this is the first study to investigate the relationship between BSA at birth and later risk for GDM in primiparous women. We aimed to investigate the relationship using BSA at birth as a more accurate estimate for body size at birth than for example birth weight, in order to approximate the metabolic tissue and body size as a whole. Further, we assessed the relationship between both BSA and ponderal index as parameters on risk for GDM. Comparisons between our findings and previous

publications are based on studies that have assessed the relationship between birth weight only or ponderal index, and risk for GDM.

Similar to previous studies that have shown birth weight to be inversely associated with risk for GDM [16–18, 29, 30], and a more recent study from 2017 that reported ponderal index to be inversely associated with GDM [22], we also found an increased risk for GDM in women with a small BSA at birth and a lower risk for women with a large BSA at birth. However, we did not detect a similar statistically significant relationship between ponderal index and risk for GDM. Further, we did not detect a U-shaped relationship

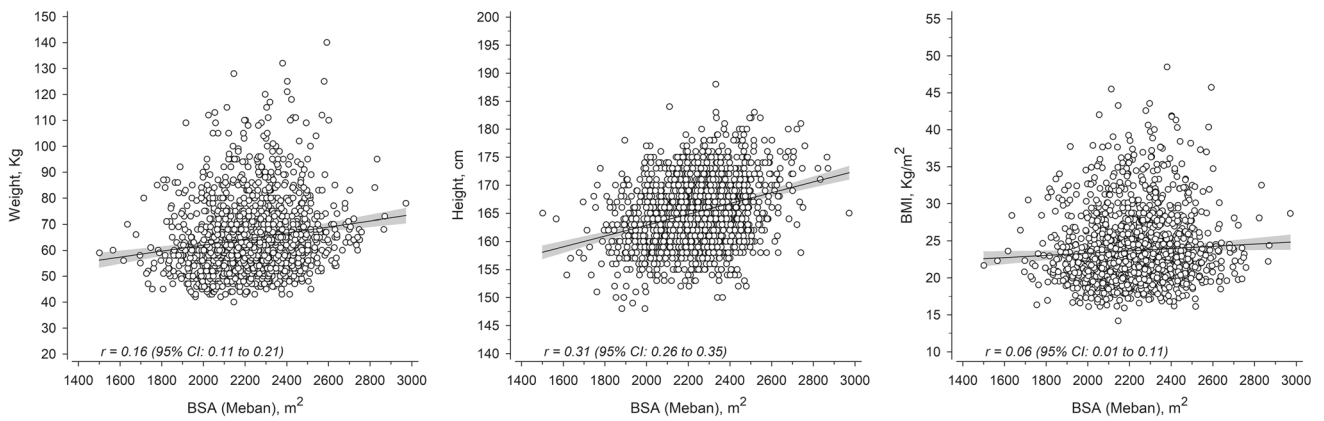


Fig. 1 Relationship between maternal body surface area (BSA) at birth and maternal anthropometry. The relationship between maternal BSA at birth, and maternal adult pre-pregnancy weight, height and BMI shown on scatter plots. *BSA* body surface area, *BMI* body mass index

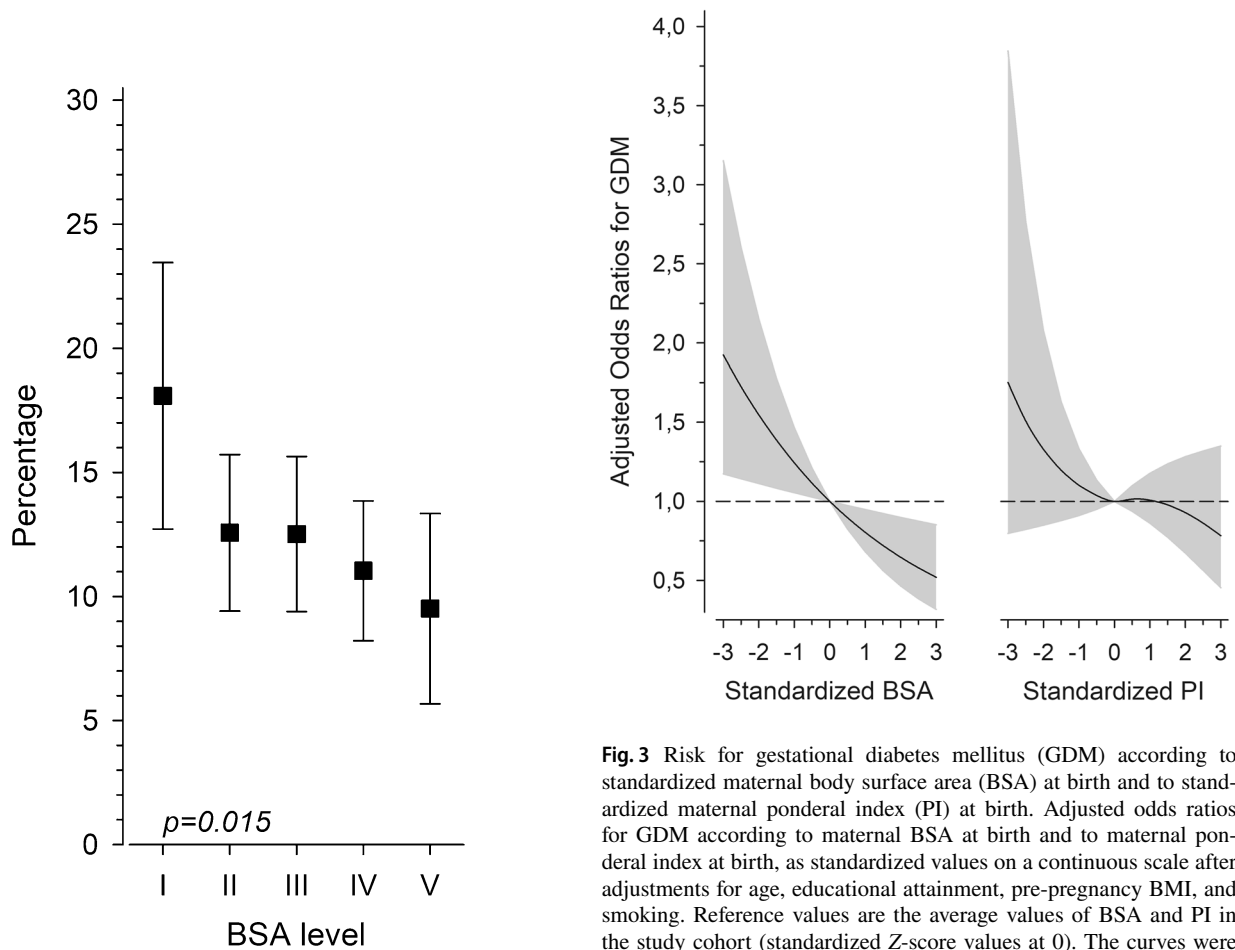


Fig. 2 Prevalence of gestational diabetes mellitus (GDM) according to maternal body surface area (BSA) level at birth. Prevalence of GDM in percentages (%), according to five different maternal BSA levels at birth with the following cut-offs: $2011 \text{ cm}^2 \leq$ for level I, $2012\text{--}2170 \text{ cm}^2$ for level II, $2171\text{--}2291 \text{ cm}^2$ for level III, $2292\text{--}2450 \text{ cm}^2$ for level IV, and $\geq 2451 \text{ cm}^2$ for level V. *p* value is calculated for linearity across the different levels after adjustments for age, educational attainment, pre-pregnancy body mass index, and smoking

Fig. 3 Risk for gestational diabetes mellitus (GDM) according to standardized maternal body surface area (BSA) at birth and to standardized maternal ponderal index (PI) at birth. Adjusted odds ratios for GDM according to maternal BSA at birth and to maternal ponderal index at birth, as standardized values on a continuous scale after adjustments for age, educational attainment, pre-pregnancy BMI, and smoking. Reference values are the average values of BSA and PI in the study cohort (standardized Z-score values at 0). The curves were derived from 5-knot-restricted cubic spline logistic models. Gray areas and whiskers represent 95% confidence intervals. *BMI* body mass index

between small and large infants and risk for GDM, as some previous studies have reported with respect to low and high birth weight and risk for GDM [19–21, 31, 32].

The conflicting results between former studies with regard to a linear inverse versus a U-shaped relationship between birth weight and risk for GDM have, at least to some extent, been thought to reflect differences in study settings, with some studies lacking a big enough comparison group of macrosomic infants. In addition, the relationship has also been explained by ethnic differences; as Williams showed in 1999, women of African-American ethnicity showed a U-shaped relationship between birth weight and risk for GDM, while women of other ethnicities, showed an inverse linear relationship [33]. Moreover, offspring born to pregnant women who have been diagnosed with GDM are more prone to be macrosomic, and maternal GDM has been recognized as a risk factor for future metabolic disturbances in the offspring [5].

There are several factors affecting fetal growth such as gestational age, parity, infant sex, in utero metabolism, and genetic factors [34, 35]. Furthermore, a small body size at birth can be explained by malnutrition during pregnancy [36], or maternal constraints [37], due to limited space in utero as a result of a narrow pelvis.

A small birthweight has been thought to affect morbidity in adulthood and has formed the basis for the DOHaD hypothesis [38]—the concept that the in utero environment, developmental plasticity, and possible epigenetic mechanisms during critical periods of early organ development can have long-lasting effects on health [38]. Moreover, low birth weight has been linked to insulin resistance [39]. In 1991, Hales and Barker proposed in their Hertfordshire study that infants born small have an impaired glucose tolerance in adulthood and that this might be due to impaired development of the endocrine pancreas and result in impaired beta cell function later in life [40]. Further, insulin is recognized to be an important growth factor and studies indicate that there could be genetic alleles that might both reduce fetal growth and cause an impaired insulin secretion and hence, predispose to diabetes [41].

Why did we think it was important to assess the relationship between maternal body size as a whole, also taking birth length into account, and risk for GDM? Compared to birth weight, birth length has been considered to be an even stronger predictor of adult height [42, 43], and height has been shown to influence the risk for GDM [10].

In 2005, Eide et al. concluded that as birth length predicts adult stature and adult stature is associated with several non-communicable disorders—birth length might be a better predictor of adult health than birth weight [42]. Adult weight is to a greater degree influenced by environmental factors and appears to have a weaker hereditary component than height [42]. Therefore, in order to have a more accurate measurement of maternal body size at birth, taking both birth weight and birth length into account and to further dilute the effect of birth weight as the only measurement of birth size in

assessing the risk of disease burden in adulthood, we wanted to evaluate the risk for GDM using both BSA and ponderal index at birth as measurements. According to our results, BSA has a stronger effect on predicting risk for GDM than ponderal index has. Our findings suggest that risk for GDM is inversely associated with BSA at birth. Ponderal index at birth showed no significant effect on risk for GDM.

The strength of the study is that it encompasses all primiparous Finnish women from Vantaa city, the fourth biggest city in Finland, who delivered during a 7-year follow-up period and of whom we had complete data about their own birth length and weight based on register data. To avoid the confounding effect of previous GDM or parity, we included only primiparas in the study. The diagnosis of GDM is based on a standardized 2-h 75-g OGTT and the diagnostic criteria have remained the same during the whole study period. Finally, the Finnish Medical Birth Register is considered to be of high quality [44].

The study also has some limitations. We missed information about some well-known risk factors for GDM; such as family history of diabetes, gestational weight gain, dietary habits, and physical activity. All study participants had Finnish background, and therefore the generalization of the results globally can be restricted. Most importantly, it was only in 1987 when the Finnish Medical Birth Register started to collect information on a nationwide basis, thereby, this cohort consisted only of rather young primiparas and the results cannot be generalized to older pregnant women.

In conclusion, BSA at birth is inversely associated with future risk for GDM in primiparous women. Special attention should be paid to pregnant women who have been born small, in order to follow-up and if possible reduce the risk for GDM. Likewise, to encourage a healthy and nutritious pregnancy diet and to aim for an optimal fetal growth are important in any pregnancy to reduce transgenerational transmission of GDM and to prevent an infant's risk for being born small and thereby at risk for future metabolic disturbances.

Acknowledgements Open access funding provided by University of Helsinki including Helsinki University Central Hospital. Research Foundation for Primary Health Care, Helsinki, Finland; and The Hospital District of Helsinki and Uusimaa, Helsinki, Finland, awarded a research grant for this study. Samfundet Folkhälsan, Helsinki, Finland, funded for the statistical analyses. The funders did not have any role for the design or conduct of the research; nor in the preparation, analyses or review of the article. We acknowledge Marko Raina and Pirjo Penanen for participating in data collection.

Author contributions SM participated in designing the study, literature search, interpretation of study results and writing and editing of the manuscript. KR participated in interpretation of study results, and helped in drafting, writing and editing the manuscript. HK is the statistician of the research group and did, together with SM, MKL and KR the statistical analysis of this study. MG participated in data collection and in editing the manuscript. JGE participated in study plan,

implementation and interpretation of study results, helped in drafting and editing the manuscript. MKL is the principal investigator of the study, and participated in data collection, implementation of the study, study design, analysis of study results and drafting and editing the manuscript. All authors have approved the final version of the article.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval No animal or human studies were carried out by the authors for this article. The study has been approved by the health authority of Vantaa city, and by the ethics committee of the Hospital District of Helsinki and Uusimaa (356/13/03/03/2015, November 2nd 2015), Finland. The National Institute for Health and Welfare, and Statistics Finland gave their permission to use register data for the study.

Informed consent According to the health authority of Vantaa city, and the ethics committee of the Hospital District of Helsinki and Uusimaa, the study participants did not need to provide the Statement of Informed Consent as this is an observational register-based cohort study. For this type of study formal consent is not required.

Data availability The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Open Access This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made.

References

- American Diabetes Association (2004) Gestational diabetes mellitus. *Diabetes Care* 27(Suppl 1):S88–S90
- Anna V, van der Ploeg HP, Cheung NW, Huxley RR, Bauman AE (2008) Sociodemographic correlates of the increasing trend in prevalence of gestational diabetes mellitus in a large population of women between 1995 and 2005. *Diabetes Care* 31(12):2288–2293. <https://doi.org/10.2337/dc08-1038>
- Lavery JA, Friedman AM, Keyes KM, Wright JD, Ananth CV (2017) Gestational diabetes in the United States: temporal changes in prevalence rates between 1979 and 2010. *BJOG* 124(5):804–813. <https://doi.org/10.1111/1471-0528.14236>
- Malcolm J (2012) Through the looking glass: gestational diabetes as a predictor of maternal and offspring long-term health. *Diabetes Metab Res Rev* 28(4):307–311. <https://doi.org/10.1002/dmrr.2275>
- Mitanchez D, Jacqueminet S, Nizard J et al (2015) The offspring of the diabetic mother—short- and long-term implications. *Best Pract Res Clin Obstet Gynaecol* 29(2):256–269. <https://doi.org/10.1016/j.bpobgyn.2014.08.004>
- Pintaudi B, Fresa R, Dalfrà M et al (2018) The risk stratification of adverse neonatal outcomes in women with gestational diabetes (STRONG) study. *Acta Diabetol*. <https://doi.org/10.1007/s00592-018-1208-x> (Epub ahead of print)
- Zhang C, Rawal S, Chong YS (2016) Risk factors for gestational diabetes: is prevention possible? *Diabetologia* 59(7):1385–1390. <https://doi.org/10.1007/s00125-016-3979-3>
- Petry CJ, Ong KK, Dunger DB (2018) Age at menarche and the future risk of gestational diabetes: a systematic review and dose response meta-analysis. *Acta Diabetol*. <https://doi.org/10.1007/s00592-018-1214-z> (Epub ahead of print)
- Huvinen E, Eriksson JG, Stach-Lempinen B, Tiitinen A, Koivusalo SB (2018) Heterogeneity of gestational diabetes (GDM) and challenges in developing a GDM risk score. *Acta Diabetol*. <https://doi.org/10.1007/s00592-018-1224-x> (Epub ahead of print)
- Laine MK, Kautiainen H, Gissler M et al (2018) Short primiparous women are at an increased risk for gestational diabetes mellitus. *Public Health* 156:101–108. <https://doi.org/10.1016/j.puhe.2017.12.020>
- Barker DJ (2007) The origins of the developmental origins theory. *J Intern Med* 261(5):412–417. <https://doi.org/10.1111/j.1365-2796.2007.01809.x>
- Gluckman PD, Hanson MA, Cooper C, Thornburg KL (2008) Effect of in utero and early-life conditions on adult health and disease. *N Engl J Med* 359(1):61–73. <https://doi.org/10.1056/NEJMr0708473>
- Whincup PH, Kaye SJ, Owen CG et al (2008) Birth weight and risk of type 2 diabetes: a systematic review. *JAMA* 300(24):2886–2897. <https://doi.org/10.1001/jama.2008.886>
- Mi D, Fang H, Zhao Y, Zhong L (2017) Birth weight and type 2 diabetes: a meta-analysis. *Exp Ther Med* 14(6):5313–5320. <https://doi.org/10.3892/etm.2017.5234>
- Harder T, Rodekamp E, Schellong K, Dudenhausen JW, Plagemann A (2007) Birth weight and subsequent risk of type 2 diabetes: a meta-analysis. *Am J Epidemiol* 165(8):849–857. <https://doi.org/10.1093/aje/kwk071>
- Ogonowski J, Miazgowski T, Engel K, Celewicz Z (2014) Birth weight predicts the risk of gestational diabetes mellitus and pre-gravid obesity. *Nutrition* 30(1):39–43. <https://doi.org/10.1016/j.nut.2013.05.021>
- Su R, Zhu W, Wei Y et al (2016) Relationship of maternal birth weight on maternal and neonatal outcomes: a multicenter study in Beijing. *J Perinatol* 36(12):1061–1066. <https://doi.org/10.1038/jp.2016.143>
- Yeung EH, Hu FB, Solomon CG et al (2010) Life-course weight characteristics and the risk of gestational diabetes. *Diabetologia* 53(4):668–678. <https://doi.org/10.1007/s00125-009-1634-y>
- Claesson R, Aberg A, Marsal K (2007) Abnormal fetal growth is associated with gestational diabetes mellitus later in life: population-based register study. *Acta Obstet Gynecol Scand* 86(6):652–656. <https://doi.org/10.1080/00016340701207682>
- a Rogvi R, Forman JL, Damm P, Greisen G (2012) Women born preterm or with inappropriate weight for gestational age are at risk of subsequent gestational diabetes and pre-eclampsia. *PLoS One* 7(3):e34001. <https://doi.org/10.1371/journal.pone.0034001>
- Pettitt DJ, Jovanovic L (2007) Low birth weight as a risk factor for gestational diabetes, diabetes, and impaired glucose tolerance during pregnancy. *Diabetes Care* 30(Suppl 2):S147–S149. <https://doi.org/10.2337/dc07-s207>
- Crusell M, Damm P, Hansen T et al (2017) Ponderal index at birth associates with later risk of gestational diabetes mellitus. *Arch Gynecol Obstet* 296(2):249–256. <https://doi.org/10.1007/s00404-017-4427-4>
- Working Group Set up by the Finnish Medical Society Duodecim, the Medical Advisory Board of the Finnish Diabetes Association and the Finnish Gynecological Association (2013) Update on current care guideline: gestational diabetes. *Duodecim* 129(17):1798–1799

24. Meban C (1983) The surface area and volume of the human fetus. *J Anat* 137(Pt 2):271–278
25. Ahn Y, Garruto RM (2008) Estimations of body surface area in newborns. *Acta Paediatr* 97(3):366–370. <https://doi.org/10.1111/j.1651-2227.2008.00666.x>
26. Mosteller RD (1987) Simplified calculation of body-surface area. *N Engl J Med* 317(17):1098
27. Evans JD (1996) Straightforward statistics for the behavioral sciences. Brooks/Cole Publishing, Pacific Grove
28. THL (2017) Perinatal statistics- parturients, deliveries and newborns 2016. <http://www.thl.fi/en/statistics/parturients>. Accessed 17 May 2018
29. Egeland GM, Skjaerven R, Irgens LM (2000) Birth characteristics of women who develop gestational diabetes: population based study. *BMJ* 321(7260):546–547
30. Seghieri G, Anichini R, De Bellis A, Alviggi L, Franconi F, Breschi MC (2002) Relationship between gestational diabetes mellitus and low maternal birth weight. *Diabetes Care* 25(10):1761–1765
31. Innes KE, Byers TE, Marshall JA, Baron A, Orleans M, Hamman RF (2002) Association of a woman's own birth weight with subsequent risk for gestational diabetes. *JAMA* 287(19):2534–2541
32. Savona-Ventura C, Chircop M (2003) Birth weight influence on the subsequent development of gestational diabetes mellitus. *Acta Diabetol* 40(2):101–104
33. Williams MA, Emanuel I, Kimpo C, Leisenring WM, Hale CB (1999) A population-based cohort study of the relation between maternal birthweight and risk of gestational diabetes mellitus in four racial/ethnic groups. *Paediatr Perinat Epidemiol* 13(4):452–465
34. Catalano PM, Drago NM, Amini SB (1995) Factors affecting fetal growth and body composition. *Am J Obstet Gynecol* 172(5):1459–1463
35. Catalano PM, Kirwan JP (2001) Maternal factors that determine neonatal size and body fat. *Curr Diab Rep* 1(1):71–77
36. Papathakis PC, Singh LN, Manary MJ (2016) How maternal malnutrition affects linear growth and development in the offspring. *Mol Cell Endocrinol* 435:40–47. <https://doi.org/10.1016/j.mce.2016.01.024>
37. Camilleri AP (1981) The obstetric significance of short stature. *Eur J Obstet Gynecol Reprod Biol* 12(6):347–356
38. de Boo HA, Harding JE (2006) The developmental origins of adult disease (Barker) hypothesis. *Aust N Z J Obstet Gynaecol* 46(1):4–14. <https://doi.org/10.1111/j.1479-828X.2006.00506.x>
39. Newsome CA, Schiell AW, Fall CH, Phillips DI, Shier R, Law CM (2003) Is birth weight related to later glucose and insulin metabolism?—A systematic review. *Diabet Med* 20(5):339–348
40. Hales CN, Barker DJ, Clark PM et al (1991) Fetal and infant growth and impaired glucose tolerance at age 64. *BMJ* 303(6809):1019–1022
41. Freathy RM, Bennett AJ, Ring SM et al (2009) Type 2 diabetes risk alleles are associated with reduced size at birth. *Diabetes* 58(6):1428–1433. <https://doi.org/10.2337/db08-1739>
42. Eide MG, Oyen N, Skjaerven R et al (2005) Size at birth and gestational age as predictors of adult height and weight. *Epidemiology* 16(2):175–181
43. Pietiläinen KH, Kaprio J, Rasanen M, Winter T, Rissanen A, Rose RJ (2001) Tracking of body size from birth to late adolescence: contributions of birth length, birth weight, duration of gestation, parents' body size, and twinship. *Am J Epidemiol* 154(1):21–29
44. Gissler M, Teperi J, Hemminki E, Meriläinen J (1995) Data quality after restructuring a national medical registry. *Scand J Soc Med* 23(1):75–80