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Maternal vitamin D status during pregnancy and body composition and cardiovascular risk markers in Indian children: the Mysore Parthenon study

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Abstract

Background—Metabolic consequences of vitamin D deficiency have become a recent research focus. Maternal vitamin D status is thought to influence musculo-skeletal health in children, but its relationship with offspring metabolic risk is not known.

Objective—We aimed to examine the association between maternal vitamin D status and anthropometry, body composition and cardiovascular risk markers in Indian children.

Design—Serum 25-hydroxy D (25(OH)D) concentrations were measured at 28-32 weeks gestation in 568 women who delivered at Holdsworth Memorial Hospital, Mysore. Anthropometry, glucose and insulin concentrations, blood pressure (BP) and fasting lipid concentrations were measured in the offspring at 5 and 9.5 years of age. Muscle-grip strength was measured using a hand held dynamometer at 9.5 years. Arm-muscle-area was calculated as a measure of muscle mass. Fasting insulin resistance was calculated using the HOMA equation.

Results—67% of women had vitamin D deficiency (serum 25(OH)D concentration <50 nmol/l). At 5 and 9.5 years, children born to vitamin D deficient mothers had smaller arm-muscle-area compared to children born to mothers without deficiency ($P<0.05$). There was no difference in grip strength between offspring of women with and without vitamin D deficiency. At 9.5 years, children of vitamin D deficient mothers had higher fasting insulin resistance than children of non-deficient women ($P=0.04$). There were no associations between maternal vitamin D status and other offspring risk factors at either age.

Conclusions—Intra-uterine exposure to low 25(OH)D concentrations is associated with lower muscle mass and higher insulin resistance in children.

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Introduction

Vitamin D is an important nutritional component required for growth and maintenance of the skeletal system and for the regulation of calcium homeostasis (1). Recently, there has been growing interest in other physiological functions of vitamin D including its role in muscle function, regulation of insulin secretion (1,2) and insulin resistance (3,4). It has also been suggested that vitamin D deficiency may be associated with obesity and the metabolic syndrome (5,6), though the causality of association with adiposity/obesity is still debatable (7).

Studies in animals and in vitro studies show that vitamin D influences cell development and/or differentiation in a wide range of tissues including adipocytes, and muscle cells (8,9). Fetal vitamin D concentrations are mainly dependent on maternal levels, and maternal deficiency may lead to adverse outcomes in the offspring (10,11). A few studies have observed that maternal vitamin D concentrations are related to offspring birth size and growth during the postnatal years. Lower maternal vitamin D status was associated with shorter length, reduced lean tissue (mid-upper arm circumference/ MUAC) (12), and lower bone mineral concentrations in the newborn (13). Maternal vitamin D supplementation increased birth weight and length in one human study (14). Maternal vitamin D status was also related to bone mineral density in UK children aged 9 years (15). However, no studies have explored the association between maternal vitamin D status and cardiovascular risk markers in offspring.

People of Indian origin, especially pregnant women, are known to have a high prevalence of vitamin D deficiency (16,17). We had earlier assessed maternal vitamin D status by measuring serum 25-hydroxy D (25(OH)D) in stored serum samples collected during a study of pregnant women in south India (18). More than 60% of the women had low 25(OH)D concentrations (<50 nmol/L) at 30 weeks gestation. There were no associations between maternal vitamin D status and offspring birth size. The current paper examines associations between maternal vitamin D status and offspring anthropometry and features of metabolic syndrome (glucose tolerance, insulin resistance, lipid concentrations and blood pressure) in this cohort of offspring at 5 and 9.5 years of age.

Subjects and methods

During 1997-1998, 830 women booking consecutively into the antenatal clinic of the Holdsworth Memorial Hospital (HMH) in Mysore and matching our eligibility criteria (no known history of diabetes, intention to deliver at HMH, singleton pregnancy) had an oral glucose tolerance test (OGTT) at 28 to 32 weeks gestation. Forty-nine women were diagnosed with gestational diabetes (GDM) using the Carpenter-Coustan criteria (19).

Offspring follow-up

Six-hundred and sixty-three women delivered live, normal babies at HMH. Excluding 25 deaths and 8 children with major medical conditions, all available children had detailed anthropometry annually until 5 years of age, and 6 monthly thereafter. A 2-hour OGTT was performed in 585 children (35 offspring of diabetic mothers, ODM) at 5-year follow-up, as described elsewhere, when blood pressure (BP) and fasting insulin and lipid concentrations were measured (20).

Five-hundred and thirty-nine children were available for follow-up at 9.5 years of age (35 ODM). Detailed anthropometry was carried out in all children including the measurement of weight (Salter, Tonbridge, Kent, UK), height (Microtoise, CMS instruments), MUAC and waist circumference (anthropometric tape) and triceps and subscapular skinfold thickness

measurements (Harpenden callipers, CMS instruments). Arm-muscle-area (AMA) was calculated as a measure of muscle mass using the following formula (21): (MUAC – π triceps skinfold)^2/4π. Fat mass, fat free mass and percentage body fat (fat%) were measured using bioimpedance (Bodystat, Quadscan 4000, Isle of Mann, UK). This method has been validated using the double-labelled water method in a sub sample of the study children, and has been found to be useful for group level comparisons (22). Grip strength was measured to the nearest 0.5 kg by one of 6 trained fieldworkers, using a Jamar dynamometer (Model J00105, Lafayette Instrument Company, Loughborough, UK) as described previously (23). Systolic and diastolic BP were measured in the left arm using an automatic BP monitor (Dinamap8100, Criticon, FL, USA), using appropriate-sized cuffs based on the MUAC. Measurers were standardised by regular intra- and inter-observer variation studies. The socio-economic status (SES) of the family was determined by the Standard of Living Index designed by the National Family Health Survey-2 (24).

Plasma glucose and insulin concentrations were measured in all children from a 2-hour OGTT after an overnight fast. An intravenous cannula was inserted after anaesthetising the skin with EMLA cream. Blood samples were collected for measurement of plasma glucose and insulin concentrations fasting, and 30 and 120 minutes after a 1.75 g/kg body weight load of anhydrous glucose in 150 ml of water. Fasting samples were used for measuring plasma total cholesterol, triglyceride and HDL-cholesterol concentrations. These laboratory assays were carried out at the Diabetes Research Centre, KEM Hospital, Pune, India, whose laboratory is a member of the UK (NEQAS) quality control programme for insulin assays. Plasma glucose and lipid concentrations were measured on an autoanalyzer (Alcyon 300, Abbott laboratories, Abbott Park, IL, USA) by standard enzymatic methods. Insulin was measured using a specific time-resolved, fluoroimmunoassay (Delfia, Wallac QY, Turku, Finland). Inter-assay co-efficients of variations were 12.5% at <45 pmol/l, 9.6% at 45–90 pmol/l and 4.3% at >90 pmol/l. Fasting Insulin resistance was estimated using the Homeostasis Model Assessment equation (HOMA) (25). 30-minute insulin increment was calculated as a measure of insulin secretion using the formula: (30-minute insulin-fasting insulin)/30-minute glucose (26).

All the procedures were followed in accordance with the ethical standards of the institution on human experimentation. The hospital ethical committee approved the study, and informed written consent/ assent was obtained from the parents and children.

Vitamin D status assessment

Vitamin D status was assessed using serum samples [stored at −80′C] from the 663 mothers who delivered at HMH. Adequate samples were available for 568 mothers (86%). Maternal serum 25(OH)D concentrations were measured using radioimmunoassay (IDS Immunodiagnostics Ltd, Boldon, Tyne and Wear UK; intra- and inter-assay coefficients of variation 8.8% and 10.8% respectively), standardised against Nichols & Incstar methodology; each assay run providing data within international vitamin D external quality assurance scheme [DEQAS] requirements (27).

Mothers were defined as having vitamin D deficiency at serum concentrations of 25(OH)D <50 nmol/l (16,28). As conventional deficiency definition was based mainly on skeletal actions of vitamin D (11), we also defined a range of cut-offs (<20, <30, <40, <50, <60 and <70 nmol/l) to examine whether non-skeletal associations emerge at different maternal 25(OH)D thresholds.

Statistical analysis

The distributions of maternal 25(OH)D concentrations, offspring skinfold thicknesses, insulin and fasting triglyceride concentrations, HOMA and 30-minute insulin increment were skewed; these data were log-transformed to normalise them for analysis. Differences between the offspring of mothers with normal and deficient vitamin D status were assessed using t-tests. Multivariable least squares regression was used to examine the associations of maternal vitamin D deficiency, and 25(OH)D concentrations as a continuous variable, with the various outcomes in the children at 5 and 9.5 years of age. Models were initially adjusted for age and sex, and then including data for maternal parity, religion, BMI, gestational diabetes status and socio-economic score. These were designated as co-variates, *a priori,* as being likely to be associated with maternal 25(OH)D concentrations and/or offspring characteristics. The models were further adjusted for season of assessment of offspring characters (Summer: March-June; Rainy: July-October and Winter: November-February) as a proxy for their vitamin D status, as the exposure to sunlight tends to differ in these three seasons. Differences in associations between boys and girls were assessed using interaction terms. All analyses were performed using Stata version 11 and SPSS version 16.

Results

Characteristics of the mothers during pregnancy, and the children studied at 5 years (N=506) and 9.5 years (N=469) of age are shown in Table 1. The median (IQR) 25(OH)D concentrations in 568 mothers who delivered normal, live babies at birth was 39.0 nmol/l (24.0,58.0); 379 (67%) mothers had vitamin D deficiency. There was no difference in the distribution of boys and girls in the vitamin D deficient and non-deficient groups (P=0.5).

Maternal vitamin D status (normal v deficiency) and offspring anthropometry, bioimpedance and grip strength

At both 5 and 9.5 years, children born to mothers with vitamin D deficiency had significantly smaller arm-muscle-area (AMA) compared to children born to mothers with normal vitamin D status (Table 2). These associations were little changed after adjusting for maternal co-variates, and/or season of assessment of offspring characteristics (Table 3). There were no differences between offspring born to mothers with and without vitamin D deficiency in any other anthropometric measurements or in fat or fat-free mass or 9.5-year grip strength. Boys, but not girls, born to mothers with vitamin D deficiency had higher fat percentage and lower fat-free percentage at 5 years but not at 9.5 years old **(Table 2)**. Statistically significant sex interactions (maternal vitamin D deficiency x sex) were observed for both these measurements (P=0.006 for both).

When 25(OH)D was treated as a continuous variable in regression models, lower maternal 25(OH)D concentration was associated with smaller AMA in the child at 5 years (p=0.04 adjusted for age and sex, p=0.03 additionally adjusted for maternal co-variates) and 9.5 years (p=0.02, p=0.009). Maternal 25(OH)D was negatively associated with 9.5 year fat percentage and a positively with fat-free percentage (P=0.02 for both, P=0.06 after adjusting). There was no association with the children's grip strength at 9.5 years with or without adjustment for age, gender, season of assessment and child's weight and height.

Maternal vitamin D status and cardiovascular risk markers at 5 and 9.5 years

At 5 years old, there were no differences in cardiovascular risk markers between children born to mothers with or without vitamin D deficiency (Table 2). At 9.5 years old, children of vitamin D deficient mothers had higher fasting insulin concentrations and insulin resistance (HOMA) than children of non-deficient women (Table 2).

In regression analyses, the association of lower maternal vitamin D status with higher 9.5-year insulin resistance in the child remained statistically significant after adjustment for maternal co-variates (Table 3), and/or season of assessment, and after additionally adjusting for either children's current BMI (P=0.01), AMA (P=0.001), waist circumference (P=0.02), sum of skinfolds, fat percentage or fat-free percentage (P=0.02). There was a statistically significant sex interaction (vitamin D deficiency x sex) for HDL-cholesterol (P=0.03). Among boys, but not girls, maternal vitamin D deficiency was associated with higher HDL-cholesterol concentrations (Table 2, Table 3). The association remained significant even after adjusting for the children's current BMI and season of assessment (P=0.01).

When maternal 25(OH)D was treated as a continuous variable, the inverse association with 9.5 year HOMA was not statistically significant ($\beta=-0.05$, P=0.2 with and without adjusting for maternal co-variates). Neither maternal vitamin D deficiency nor 25(OH)D concentrations as a continuous variable was associated with other cardiometabolic risk markers including 30-minute insulin increment, glucose concentrations and 30-minute and 120-minute and insulin concentrations at 5 and 9.5 year of age.

Maternal vitamin D status at different 25(OH)D thresholds and offspring outcomes at 9.5 years

Offspring AMA at 9.5 years consistently showed positive associations with higher maternal vitamin D status at all levels of 25(OH)D, though not statistically significant at all thresholds (Table 4). We also observed that insulin increment at 9.5 years was significantly lower in children of mothers with 25(OH)D concentrations <70 nmol/l (P=0.04, adjusted for all co-variates). There was no association with insulin resistance or other risk markers at these cut-offs.

There were no difference in associations between boys and girls, and there were no significant sex interactions.

Discussion

In a large birth cohort in India maternal vitamin D deficiency predicted smaller muscle size (AMA) at 5 and 9.5 years of age, and higher insulin resistance at 9.5 years. Maternal vitamin D deficiency, defined at a higher 25(OH)D threshold, also predicted reduced insulin secretion. As far as we know, this is the first study to examine associations between maternal vitamin D status and cardiometabolic risk factors in children.

Major strengths of the study are detailed anthropometry and cardiovascular risk factors measured at two time points in a large group of healthy children, and prior measurements of maternal 25(OH)D status. Limitations were the lack of good data on maternal vitamin D supplement use, and no data on children's serum 25(OH)D concentrations. Maternal lifestyle habits (diet, sun exposure) that could influence her own as well as her offspring's vitamin D status and offspring outcomes were not measured. We used bioimpedance for body composition assessment and HOMA index for insulin resistance, which are not gold standard measurements. However, these methods are widely used and correlate well with more valid methods (29,30).

Two recent studies have reported associations between maternal 25(OH)D concentrations and postnatal bone growth in the children. In the UK, lower maternal serum 25(OH)D concentrations in late pregnancy were associated with reduced bone mineral content in the children at 9 years of age (15). In Lebanon, maternal veiling, a proxy for low maternal vitamin D status, was associated with lower bone mineral content and density in adolescent boys (31). Neither study showed an association with height.

As in these studies, maternal vitamin D status was not related to height in the Mysore children. At 5 and 9.5 years of age, children of mothers without vitamin D deficiency had larger AMA. These associations were also continuous across the range of maternal 25(OH)D concentrations. There was no effect on grip-strength at 9.5 years. One explanation for this is that the association seen with AMA may be related to its bone component. Alternatively, intrauterine vitamin D status may influence muscle size but not function. Current vitamin D status is more likely to be associated with muscle performance (2,32,33). We also observed an association between maternal vitamin D status and lean body mass (fat-free percentage) similar to a UK study that observed a trend towards higher lean body mass in children of mothers with higher vitamin D status (34). Maternal vitamin D may have long-term effects on offspring muscle growth postnatally. Alternatively, these offspring may have similar lifestyle habits as their mothers, and thus better vitamin D status. Studies in animals suggest that vitamin D stimulates growth and differentiation of muscle tissue (9). Bone growth may also be compromised in maternal deficiency due to intrauterine programming of endocrine mechanisms affecting calcium homeostasis (15).

The association between low maternal vitamin D status and higher offspring fat percentage was alarming. Sex differences were a new finding. This may be related to more homogenous lifestyle habits in girls in this population (less out-door exposure) masking an association with maternal vitamin D status. This area needs further exploration.

Vitamin D is known to influence insulin secretion (35), while hypovitaminosis D predicts insulin resistance, glucose intolerance and features of metabolic syndrome in normoglycaemic subjects (5, 36-38). We did not find an association in these Mysore mothers between vitamin D status and their own insulin resistance or risk of gestational diabetes (18). There are no previous studies reporting long-term effects of maternal vitamin D status on risk of type 2 diabetes or metabolic syndrome in the offspring. We found that low concentrations of 25(OH)D in the pregnant mothers were associated with higher insulin resistance in their children at 9.5 years of age. Though the changes observed in our children are small and not currently clinically relevant, childhood HOMA tends to track to adulthood (39). There were no associations with insulin secretion (30-minute increment). However, it was lower in children whose mothers had 25(OH)D concentrations <70 nmol/l. Seasonality of birth (a proxy for intrauterine vitamin D status) as well as maternal 25(OH)D concentrations have been shown to be associated with the prevalence of offspring type 1 diabetes in some studies (40,41). However, a direct association between maternal vitamin D status and offspring β-cell functioning has not been reported. A recent study showed reduced β-cell secretion (HOMA-β) in adults with higher finger print ridge counts, which themselves were predicted by seasonality of conception (42). There is still debate as to the optimal 25(OH)D concentrations for defining vitamin D deficiency, especially in pregnancy (11). Current practice is based on the skeletal actions of the vitamin, and may not be applicable for its non-classic actions. Our study suggests that levels may differ for different parameters.

The mechanism by which maternal vitamin D predicts insulin resistance in late childhood is speculative. Our study suggests that neither fat nor muscle size are mediating factors in this association. The association was significant only at 9.5 years. Age related factors such as puberty and sedentary behaviours may have reduced the threshold for the risk factors to emerge in compromised children. The concept that maternal nutritional status influences the risk of chronic disorders in the offspring has attracted interest over the past 2 decades (43). However, very few studies have been in a position to examine this association directly in humans. Recently, lower maternal vitamin B12 and higher folate status were shown to predict insulin resistance in Indian children (44). Against the background of a high prevalence of vitamin D deficiency among Asian Indians, especially during pregnancy, this

may be one factor that, through fetal programming, contributes to the rise of type 2 diabetes in the region.

An unexpected finding in our study was the association between maternal vitamin D deficiency and increased HDL-cholesterol in the male offspring. We do not have an explanation for this phenomenon. Past studies have reported an increase in serum LDL-cholesterol concentrations in post-menopausal women supplemented with long-term vitamin D3 (45). More studies on this association are needed to understand the significance of our finding.

In conclusion, maternal vitamin D status may be an important micro-nutritional factor for post-natal musculo-skeletal development and glucose homeostasis in human offspring. This is alarming considering the high prevalence of vitamin D deficiency among pregnant women globally. This observation, therefore, warrants replication and further research to determine whether the association is causal and whether these adverse effects of maternal hypovitaminosis D on non-bony tissues in offspring can be prevented by vitamin D supplementation in pregnancy.

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Table 1

Characteristics of the mothers and children at birth 5 years and 9.5 years in the Mysore Parthenon cohort

Variable	Boys N=244			Girls N=267		
	N	Mean / [%]	(SD)	N	Mean / [%]	(SD)
Maternal						
25(OH)D (nmol/l) *	244	38.0	(23.5, 53.75)	267	40.5	(23.75, 61.5)
Age (years)	244	24.0	(4.3)	267	23.9	(4.3)
5 years						
Body mass index (kg/m^2)	243	13.6	(1.1)	263	13.6	(1.2)
Height (cm)	243	106.6	(4.3)	263	105.1	(4.4)
Arm muscle area (cm^2)	243	13.7	(1.8)	263	12.9	(1.8)
Subscapular skinfold (mm) *	243	5.4	(4.8, 6.2)	263	6.0	(5.1, 7.7)
Triceps skinfold (mm) *	243	7.2	(6.3, 8.5)	263	8.0	(6.9, 9.8)
Waist (cm)	243	46.0	(2.8)	262	46.0	(2.8)
Fat mass (kg)	242	3.6	(0.9)	263	4.1	(1.1)
Fat percentage	242	23.2	(4.9)	263	27.4	(5.1)
Fat-free mass (kg)	242	11.9	(1.6)	263	10.9	(1.4)
Fat-free percentage	242	76.9	(4.9)	263	72.6	(5.1)
Fasting glucose (mmol/l)	241	4.8	(0.5)	261	4.8	(0.5)
Fasting insulin (pmol/l) *	241	17.8	(11.3, 28.2)	261	22.5	(15.0, 34.7)
Insulin resistance (HOMA) *	241	0.6	(0.4, 1.0)	260	0.8	(0.5, 1.2)
30-minute insulin increment *	239	15.0	(9.1, 26.0)	257	19.8	(12.9, 27.8)

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Variable		Boys N=244			Girls N=267	
	N	Mean / [%]	(SD)	N	Mean / [%]	(SD)
Triglyceride (mmol/l) *	242	0.8	(0.6, 1.1)	261	0.8	(0.6, 1.1)
Total cholesterol (mmol/l)	242	3.5	(0.6)	261	3.5	(0.8)
HDL cholesterol (mmol/l)	242	1.1	(0.3)	261	1.1	(0.4)
Systolic blood pressure (mmHg)	243	97.7	(8.5)	261	95.9	(8.1)
Diastolic blood pressure (mmHg)	243	58.6	(6.7)	261	57.7	(6.8)
9.5 years						
Body mass index (kg/m^2)	225	14.7	(1.8)	244	14.7	(2.0)
Height (cm)	225	131.6	(5.5)	244	130.6	(6.0)
Arm muscle area (cm^2)	225	18.5	(3.1)	244	17.3	(3.3)
Subscapular skinfold (mm) *	225	6.3	(5.4, 8.1)	243	8.0	(6.3, 10.4)
Triceps skinfold (mm) *	225	8.4	(7.0, 10.2)	244	10.4	(8.6, 13.3)
Waist (cm)	225	54.5	(5.3)	243	54.7	(5.9)
Fat mass (kg)	225	6.3	(2.3)	244	7.7	(2.7)
Fat percentage	225	24.4	(6.2)	244	30.1	(5.9)
Fat-free mass (kg)	225	19.2	(3.0)	244	17.5	(3.0)
Fat-free percentage	225	75.6	(6.2)	244	69.9	(5.9)
Grip strength (kg)	225	12.5	(2.2)	244	10.8	(2.0)
Fasting glucose (mmol/l)	221	4.7	(0.4)	240	4.7	(0.4)
Fasting insulin (pmol/l) *	221	19.8	(12.0, 28.8)	240	27.0	(17.7, 36.3)

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Variable	Boys N=244			Girls N=267		
	N	Mean / [%]	(SD)	N	Mean / [%]	(SD)
Insulin resistance (HOMA) *	221	0.7	(0.4, 1.0)	240	0.9	(0.6, 1.2)
30-minute insulin increment *	213	26.2	(16.8, 45.5)	236	35.8	(21.6, 50.5)
Triglyceride (mmol/l) *	221	0.8	(0.6, 1.0)	240	0.9	(0.7, 1.2)
Total cholesterol (mmol/l)	221	3.8	(0.6)	240	3.9	(0.7)
HDL cholesterol (mmol/l)	221	1.1	(0.2)	240	1.1	(0.2)
Systolic blood pressure (mmHg)	225	102.4	(8.5)	244	100.1	(8.5)
Diastolic blood pressure (mmHg)	225	58.7	(7.0)	244	58.2	(6.4)

* Median (IQR)

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Table 2

Comparison of outcomes between children of mothers with and without vitamin D deficiency at 5 and 9.5 years

	Deficient mothers (Vitamin D < 50 nmol/l)			Non-deficient mothers (Vitamin D ≥ 50 nmol/l)			
Variable	**N=379**	**mean**	**(SD)**	**N=189**	**mean**	**(SD)**	**p-value**
5 years							
Body mass index (kg/m^2)	339	13.5	(1.1)	167	13.7	(1.2)	0.06
Height (cm)	339	106.0	(4.4)	167	105.6	(4.4)	0.33
Arm muscle area (cm^2)	339	13.1	(1.9)	167	13.5	(1.9)	0.03
Subscapular skinfold (mm) *	339	5.6	(5.0, 7.0)	167	5.7	(4.9, 7.2)	0.83
Triceps skinfold (mm) *	339	7.7	(6.5, 8.9)	167	7.8	(6.6, 9.3)	0.59
Waist (cm)	339	46.0	(2.9)	166	46.0	(3.1)	0.80
Fat mass (kg)	338	3.9	(1.0)	167	3.9	(1.2)	0.95
Fat percentage	338	25.4	(5.1)	167	25.2	(6.0)	0.61
Boys	165	23.7	(4.8)	77	22.0	(4.9)	0.01
Girls	173	27.1	(4.9)	90	27.9	(5.5)	0.22
Fat-free mass (kg)	338	11.4	(1.6)	167	11.5	(1.6)	0.52
Fat-free percentage	338	74.6	(5.1)	167	74.8	(6.0)	0.64
Boys	165	76.3	(4.8)	77	78.0	(5.0)	0.01
Girls	173	72.9	(4.9)	90	72.1	(5.5)	0.22
Fasting glucose (mmol/l)	337	4.8	(0.5)	165	4.8	(0.4)	0.47
Fasting insulin (pmol/l) *	336	19.8	(12.3, 29.7)	166	21.6	(13.6, 36.9)	0.13
Insulin resistance (HOMA) *	336	0.7	(0.4, 1.1)	165	0.8	(0.5, 1.2)	0.14
30-minute insulin increment *	334	17.1	(10.8, 26.7)	162	19.1	(10.1, 27.0)	0.68
Triglyceride (mmol/l) *	336	0.8	(0.6, 1.1)	167	0.8	(0.6, 1.1)	0.44
Total cholesterol (mmol/l)	336	3.5	(0.7)	167	3.5	(0.6)	0.69
HDL cholesterol (mmol/l)	336	1.1	(0.4)	167	1.0	(0.2)	0.13
Systolic blood pressure (mmHg)	338	96.7	(8.4)	166	97.0	(8.1)	0.67
Diastolic blood pressure (mmHg)	338	58.3	(6.8)	166	57.9	(6.6)	0.54
9.5 years							

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Variable	Deficient mothers (Vitamin D < 50 nmol/l) N=379	mean	(SD)	Non-deficient mothers (Vitamin D ≥ 50 nmol/l) N=189	mean	(SD)	p-value
Body mass index (kg/m2)	312	14.6	(1.9)	157	14.7	(2.1)	0.62
Height (cm)	312	131.1	(5.8)	157	131.0	(5.9)	0.92
Arm muscle area (cm^2)	312	17.6	(2.9)	157	18.3	(3.8)	0.02
Subscapular skin-fold (mm) *	311	7.2	(5.8, 9.2)	157	7.2	(5.6, 9.1)	0.97
Triceps skinfold (mm) *	312	9.4	(7.7, 12.0)	157	9.4	(7.8, 11.8)	0.80
Waist circumference (cm)	311	54.5	(5.4)	157	54.8	(6.1)	0.61
Fat mass (kg)	312	7.1	(2.5)	157	7.0	(2.9)	0.80
Fat percentage	312	27.6	(6.7)	157	27.0	(6.7)	0.36
Fat-free mass (kg)	312	18.2	(3.1)	157	18.4	(3.0)	0.51
Fat-free percentage	312	72.4	(6.7)	157	73.0	(6.7)	0.36
Grip strength (kg)	312	11.7	(2.2)	157	11.5	(2.4)	0.49
Fasting glucose (mmol/l)	306	4.7	(0.4)	155	4.7	(0.4)	0.48
Fasting insulin (pmol/l) *	306	23.4	(15.6, 36.0)	155	20.4	(12.6, 33.0)	0.04
Insulin resistance (HOMA) *	306	0.8	(0.5, 1.2)	155	0.7	(0.4, 1.2)	0.04
30-minute insulin increment *	299	31.2	(19.2, 47.1)	150	30.9	(17.7, 51.8)	0.45
Triglyceride (mmol/l) *	306	0.8	(0.7, 1.1)	155	0.9	(0.7, 1.2)	0.3
Total cholesterol (mmol/l)	306	3.9	(0.7)	155	3.9	(0.6)	0.8
HDL cholesterol (mmol/l)	306	1.1	(0.2)	155	1.1	(0.2)	0.08
Boys	149	1.1	(0.2)	72	1.0	(0.2)	0.006
Girls	157	1.1	(0.2)	83	1.1	(0.2)	0.71
Systolic blood pressure (mmHg)	312	101.6	(8.7)	157	100.5	(8.3)	0.2
Diastolic blood pressure (mmHg)	312	58.3	(6.5)	157	58.7	(7.2)	0.5

P-values were derived using t-tests comparing children of women with and without vitamin D deficiency.

* log transformed variables, values are given as median (IQR). Sexes are reported separately where there are significant differences in associations between boys and girls.

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Table 3

Multiple regression analyses examining associations between maternal vitamin D status (deficient=0, versus non-deficient=1) and outcomes in the children at 5 and 9.5 years

Model	Adjusted by child sex and age				Adjusted by child sex, age and mothers BMI, gestational diabetes, socio-economic score, parity and religion			
	N	Beta	95% C.I.	p-value	N	Beta	95% C.I.	p-value
5 years								
Body mass index (kg/m^2)	506	0.2	(−0.00,0.42)	0.05	473	0.2	(0.01,0.43)	0.04
Height (cm)	506	−0.3	(−1.12,0.49)	0.44	473	−0.3	(−1.15,0.48)	0.43
Arm muscle area (cm^2)	506	0.4	(0.08,0.76)	0.02	473	0.4	(0.10,0.78)	0.01
Subscapular skinfold (mm) *	506	0.002	(−0.05,0.05)	0.94	473	0.004	(−0.04,0.05)	0.86
Triceps skinfold (mm) *	506	0.01	(−0.03,0.06)	0.63	473	0.01	(−0.03,0.06)	0.55
Waist circumference (cm)	505	0.08	(−0.48,0.63)	0.79	472	0.07	(−0.50,0.64)	0.81
Fat mass (kg)	505	−0.02	(−0.22,0.17)	0.83	472	−0.01	(−0.21,0.19)	0.92
Fat percentage	505	−0.4	(−1.35,0.52)	0.38	472	−0.4	(−1.32,0.62)	0.48
Boys	242	−1.7	(−3.06,−0.41)	0.01	229	−1.5	(−2.92,−0.20)	0.02
Girls	263	0.7	(−0.56,2.06)	0.26	243	0.9	(−0.51,2.29)	0.21
Fat-free mass (kg)	505	0.1	(−0.14,0.43)	0.31	472	0.1	(−0.14,0.43)	0.33
Fat-free percentage	505	0.4	(−0.55,1.33)	0.41	472	0.3	(−0.65,1.30)	0.51
Boys	242	1.7	(0.35,3.01)	0.01	229	1.5	(0.15,2.88)	0.03
Girls	263	−0.7	(−2.05,0.57)	0.27	243	−0.8	(−2.29,0.52)	0.22
Fasting glucose (mmol/l)	502	0.04	(−0.05,0.13)	0.44	469	0.04	(−0.05,0.13)	0.42
Fasting insulin (pmol/l) *	502	0.08	(−0.04,0.21)	0.19	469	0.07	(−0.06,0.19)	0.31
Insulin resistance (HOMA) *	501	0.09	(−0.04,0.21)	0.19	468	0.07	(−0.06,0.20)	0.31
30-minute insulin increment *	496	0.004	(−0.02,0.03)	0.71	463	0.006	(−0.02,0.03)	0.65
Triglyceride (mmol/l) *	503	0.03	(−0.05,0.11)	0.49	470	0.04	(−0.04,0.12)	0.33
Total cholesterol (mmol/l)	503	−0.03	(−0.16,0.11)	0.70	470	−0.02	(−0.16,0.11)	0.75
HDL cholesterol (mmol/l)	503	−0.05	(−0.11,0.02)	0.14	470	−0.06	(−0.12,0.01)	0.09
Systolic blood pressure (mmHg)	504	0.4	(−1.18,1.92)	0.64	472	0.3	(−1.32,1.89)	0.72
Diastolic blood pressure (mmHg)	504	−0.3	(−1.59,0.93)	0.61	472	−0.3	(−1.67,0.98)	0.61

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Model	Adjusted by child sex and age				Adjusted by child sex, age and mothers BMI, gestational diabetes, socio-economic score, parity and religion			
	N	Beta	95% C.I.	p-value	N	Beta	95% C.I.	p-value
9.5 years								
Body mass index (kg/m^2)	469	0.07	(−0.31,0.45)	0.72	465	0.1	(−0.24,0.46)	0.54
Height (cm)	469	−0.2	(−1.29,0.94)	0.76	465	−0.2	(−1.29,0.91)	0.74
Arm muscle area (cm^2)	469	0.7	(0.07,1.29)	0.03	465	0.7	(0.12,1.29)	0.02
Subscapular skinfold (mm) *	468	−0.01	(−0.08,0.06)	0.76	464	−0.009	(−0.08,0.06)	0.80
Triceps skinfold (mm) *	469	−0.0005	(−0.06,0.06)	0.99	465	0.004	(−0.05,0.06)	0.88
Waist circumference (cm)	468	0.2	(−0.91,1.26)	0.75	464	0.3	(−0.78,1.30)	0.62
Fat mass (kg)	469	−0.09	(−0.59,0.40)	0.71	465	−0.07	(−0.54,0.40)	0.77
Fat percentage	469	−0.6	(−1.75,0.58)	0.32	465	−0.6	(−1.70,0.58)	0.34
Fat-free mass (kg)	469	0.1	(−0.43,0.72)	0.63	465	0.2	(−0.38,0.74)	0.52
Fat-free percentage	469	0.6	(−0.57,1.76)	0.32	465	0.6	(−0.57,1.71)	0.33
Grip strength (kg)	469	−0.2	(−0.56,0.25)	0.46	465	−0.1	(−0.54,0.27)	0.52
Fasting glucose (mmol/l)	461	−0.02	(−0.10,0.05)	0.53	457	−0.03	(−0.10,0.04)	0.44
Fasting insulin (pmol/l) *	461	−0.1	(−0.25,−0.02)	0.02	457	−0.1	(−0.26,−0.03)	0.01
Insulin resistance (HOMA) *	461	−0.1	(−0.26,−0.02)	0.02	457	−0.1	(−0.27,−0.03)	0.01
30-minute insulin increment *	449	0.01	(−0.02,0.04)	0.56	445	0.008	(−0.02,0.04)	0.64
Triglyceride (mmol/l) *	461	0.03	(−0.04,0.10)	0.34	457	0.03	(−0.04,0.10)	0.42
Total cholesterol (mmol/l)	461	0.03	(−0.10,0.16)	0.67	457	0.01	(−0.12,0.14)	0.84
HDL cholesterol (mmol/l)	461	−0.03	(−0.07,0.01)	0.18	457	−0.03	(−0.07,0.01)	0.11
Boys	221	−0.08	(−0.14,−0.02)	0.01	218	−0.09	(−0.15,−0.03)	0.006
Girls	240	0.02	(−0.04,0.07)	0.50	239	0.0009	(−0.05,0.05)	0.97
Systolic blood pressure (mmHg)	469	−1.2	(−2.80,0.49)	0.17	465	−1.2	(−2.87,0.42)	0.15
Diastolic blood pressure (mmHg)	469	0.4	(−0.88,1.73)	0.52	465	0.4	(−0.90,1.74)	0.53

P-values were derived using linear regression to compare the children of women with and without vitamin D deficiency.

* Log transformed variables. Sexes are reported separately where there are significant differences in associations between boys and girls.

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Table 4

Comparing multiple cut-offs of 25(OH)D status for outcomes in the children at 9.5 years (deficient=0, versus non-deficient=1).

Level at which 25(OH)D deficient	**<20 nmol/l**		**<30 nmol/l**		**<40 nmol/l**		**<50 nmol/l**		**<60 nmol/l**		**<70 nmol/l**	
N Max=465	**Non-deficient N=381 Deficient N=84**		**Non-deficient N=298 Deficient N=167**		**Non-deficient N=228 Deficient N=237**		**Non-deficient N=157 Deficient N=308**		**Non-deficient N=112 Deficient N=353**		**Non-deficient N=93 Deficient N=372**	
	Beta (95% CI)	**P-value**	**Beta (95% CI)**	**P-value**	**Beta (95% CI)**	**P-value**	**Beta (95% CI)**	**P-value**	**Beta (95% CI)**	**P-value**	**Beta (95% CI)**	**P-value**
Arm muscle area (cm^2)	0.7 (−0.02,1.42)	0.06	0.6 (0.03,1.18)	0.04	0.5 (−0.05,1.07)	0.08	0.7 (0.12,1.29)	0.02	0.5 (−0.12,1.18)	0.11	0.8 (0.09,1.47)	0.03
Fat percentage	−0.6 (−2.01,0.79)	0.39	−1.4 (−2.48,−0.24)	0.02	−0.9 (−2.02,0.15)	0.09	−0.6 (−1.70,0.58)	0.34	−0.4 (−1.67,0.86)	0.53	0.1 (−1.21,1.48)	0.85
Fat-free percentage	0.6 (−0.79,2.02)	0.39	1.4 (0.25,0.49)	0.02	0.9 (−0.15,2.02)	0.09	0.6 (−0.57,1.71)	0.33	0.4 (−0.85,1.68)	0.52	−0.1 (−1.47,1.22)	0.85
Grip strength (kg)	0.2 (−0.33,0.67)	0.50	0.3 (−0.06,0.73)	0.10	0.2 (−0.17,0.61)	0.26	−0.1 (−0.54,0.27)	0.52	−0.2 (−0.64,0.26)	0.40	−0.06(−0.54, 0.41)	0.79
Fasting insulin (pmol/l) *	−0.03 (−0.16,0.11)	0.72	−0.08 (−0.19,0.03)	0.15	−0.1 (−0.21,0.01)	0.06	−0.1 (−0.26,−0.03)	0.01	−0.02 (−0.15,0.10)	0.70	−0.07 (−0.20,0.07)	0.34
Insulin resistance (HOMA) *	−0.02 (−0.17,0.12)	0.75	−0.09 (−0.20,0.03)	0.14	−0.1 (−0.22,0.01)	0.07	−0.1 (−0.27,−0.03)	0.01	−0.03 (−0.16,0.10)	0.66	−0.08 (−0.22,0.06)	0.28
30-minute insulin increment *	−0.007 (−0.05,0.03)	0.71	−0.02 (−0.05,0.01)	0.18	−0.002 (−0.03,0.03)	0.88	0.008 (−0.02,0.04)	0.64	0.02 (−0.01,0.06)	0.20	0.04 (0.00,0.08)	0.04

P-values were derived using linear regression to compare the children of women with and without vitamin D deficiency.

Models are adjusted by child sex, age and mothers BMI, gestational diabetes, socio-economic score, parity and religion

* Log transformed variables

N denotes the maximum frequencies of deficient and non-deficient mothers; frequencies are fewer in outcomes of fasting insulin, HOMA and 30-minute insulin increment