Maternal vitamin D status in pregnancy is associated with adiposity in the offspring: findings from the Southampton Women’s Survey1–4

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ABSTRACT
Background: Low vitamin D status has been linked to adiposity, but little is known of the effects of low status in pregnancy on offspring body composition.
Objective: The objective was to determine how maternal vitamin D status relates to lean and fat mass of the offspring.
Design: The offspring of 977 pregnant women, who had serum 25-hydroxyvitamin D [25(OH)D] measured at 34 wk gestation, were followed up within 3 wk of birth and at 4 and 6 y of age for dual-energy X-ray absorptiometry assessment of lean and fat mass.
Results: The median maternal serum 25(OH)D concentration was 62 nmol/L (IQR: 43–89 nmol/L); 35% of the women studied had values <50 nmol/L. Lower vitamin D status was associated with lower fat mass in the offspring at birth but with greater fat mass at ages 4 and 6 y. It was not associated with lean mass at any of the ages studied. The opposing associations seen between maternal 25(OH)D (SDs) and fat mass (SDs) in the offspring at birth and at age 6 y were robust to adjustment for a range of confounding factors, including maternal BMI and weight gain in pregnancy [β (95% CI): 0.08 (0.02, 0.15) and −0.10 (−0.17, −0.02), respectively]. The key independent predictors of higher maternal vitamin D status were season of assessment and use of vitamin D supplements.
Conclusions: Lower maternal vitamin D status may be linked to programmed differences in offspring fat mass. The findings require replication but add to a growing evidence base for a role of vitamin D in the origins of adiposity. Am J Clin Nutr 2012;96:57–63.

INTRODUCTION

There is a growing recognition of the diversity of the metabolic roles played by vitamin D, beyond its effects on bone health. Low vitamin D status has been linked to impaired glucose tolerance, insulin resistance, and the metabolic syndrome in adults (1). It has also been proposed that vitamin D insufficiency may be causally associated with adiposity in both adults and children (2–4); consistent with this proposition, vitamin D receptor gene polymorphisms have recently been linked to adiposity phenotypes (5).

Vitamin D insufficiency is common in the United Kingdom (6). A particular concern is that almost a third of young women have low vitamin D status (6). Although women are recommended to take an additional 10 μg vitamin D/d in pregnancy, supplementation is not routine in the United Kingdom, and low status is prevalent (7). The long-term effects on children born to mothers who have low vitamin D status in pregnancy have not been widely studied. Maternal vitamin D insufficiency has been linked to insulin resistance and low muscle mass (8) and to poorer bone mineral accrual (9) in the offspring; little is known about effects on adiposity (10). The primary determinant of vitamin D status is sunshine exposure (11). Adult BMI has been shown to differ with season of birth (12), and it is possible that higher BMI, indicating greater adiposity, among adults born in winter and spring could be linked to lower maternal vitamin D status in the winter months. Recent findings from a follow-up study in Indian children support this possibility, because low maternal vitamin D status in pregnancy was associated with greater adiposity (8). However, in an earlier study in a smaller cohort of UK children, body composition was not related to maternal vitamin D status (13).

In the context of current concerns about the high prevalence of vitamin D insufficiency in young women, and increasing rates of childhood obesity, the metabolic consequences for children born to mothers with low maternal status require further investigation. We therefore examined how maternal vitamin D status in late pregnancy related to the body composition of the offspring at birth and at ages 4 and 6 y in a prospective cohort of 977 mothers who were studied in detail throughout pregnancy.

SUBJECTS AND METHODS

Study sample

The Southampton Women’s Survey (SWS)5 is a prospective cohort study that has assessed the diet, body composition, and other factors, including maternal vitamin D status, during pregnancy and in the offspring. The SWS study group has collaborated closely with the Southampton NIHR Biomedical Research Unit in Nutrition, Diet, and Lifestyle (University of Southampton and Southampton University Hospitals NHS Trust (KMG), Southampton General Hospital, Southampton, United Kingdom) and the Southampton Women’s Survey (SWS) Study Group.

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3 Supported by the Medical Research Council, the British Heart Foundation, the Food Standards Agency, and Arthritis Research UK. KMG is supported by the National Institute for Health Research (NIHR) through the Southampton NIHR Biomedical Research Unit in Nutrition, Diet, and Lifestyle.
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5 Abbreviations used: DXA, dual-energy X-ray absorptiometry; IOM, Institute of Medicine; SWS, Southampton Women’s Survey; 25(OH)D, 25-hydroxyvitamin D.
physical activity, and social circumstances of a large group of nonpregnant women aged 20–34 y living in the city of Southampton, United Kingdom. Comprehensive details of the study have been published previously (14). Women were recruited through General Practices across the city between April 1998 and December 2002. Each woman was invited to take part by letter, followed by a telephone call when an interview date was arranged. A total of 12,583 women agreed to take part, 75% of all women who were contacted. Trained research nurses visited the women at home and collected information about their health, diet, and lifestyles and performed anthropometric measurements. Women who subsequently became pregnant were followed up at 11, 19, and 34 wk gestation, and their offspring were studied in infancy and childhood.

The SWS was approved by the Southampton and South West Hampshire Local Research Ethics Committee (ethics permissions granted during study: 307/97, 153/99w, 005/03/t, and 06/Q1702/104), and written informed consent was obtained from all participating mothers.

Maternal data

Details of mothers’ parity, educational attainment (defined in 6 groups according to highest academic qualification), and social class were obtained at the prepregnancy interview, and height and weight were measured. Among women who became pregnant, smoking status in pregnancy was ascertained at the 11- and 34-wk interviews. At 34 wk, a research nurse obtained a venous blood sample, and an aliquot of maternal serum was frozen at −80°C. Serum 25-hydroxyvitamin D [25(OH)D] concentrations were analyzed by radioimmunoassay (Diasorin). This assay measures both 25(OH)D$_2$ and 25(OH)D$_3$. The assay met the requirements for models with outcomes at age 4 or 6 y (19) to a normally distributed variable with a mean of 0 and an SD of 1. Although fat-free mass variables were normally distributed, they were similarly Fisher-Yates transformed so that all outcome variables were on the same scale of measurement. Maternal serum 25(OH)D concentrations were not normally distributed and were transformed with the use of Fisher-Yates normal scores, thus were transformed with the use of Fisher-Yates normal scores.

Statistical analysis

All children’s fat mass variables were positively skewed and thus were transformed with the use of Fisher-Yates normal scores to a normally distributed variable with a mean of 0 and an SD of 1. Although fat-free mass variables were normally distributed, they were similarly Fisher-Yates transformed so that all outcome variables were on the same scale of measurement. Maternal serum 25(OH)D concentrations were not normally distributed and were transformed with the use of Fisher-Yates normal scores, as were other variables that were nonnormal (maternal pre-pregnancy BMI, 3-y vitamin D intake, and 3-y physical activity). Linear regression models were fitted with body-composition variables as the outcomes and with maternal serum 25(OH)D concentration as the predictor. Outcomes at birth were adjusted for sex, gestation, age at measurement, age squared, and crown-heel length. All outcomes at ages 4 and 6 y were adjusted for sex, age at measurement, and childhood height; by including length or height as covariates, any associations with body composition were independent of the child’s stature. Additional adjustments were made for potential confounding factors. Maternal factors were as follows: educational attainment, smoking in pregnancy, prepregnancy BMI, height, parity, social class, and IOM weight-gain category. Child factors were breastfeeding duration, vitamin D intake at age 3 y, and physical activity at age 3 y (the latter only for models with outcomes at age 4 or 6 y). A cubic spline model was fitted to describe the association between vitamin D status and 6-y fat mass in more detail.

A form of Fourier analysis was used to model the association between vitamin D status and date of sample to take account of...
the cyclical variation in 25(OH)D concentration by season. A variable was derived that described the number of years the sample was taken after the first sample in the study and multiplied by $2\pi$ to give a value $\theta$, in radians. Maternal serum 25(OH)D concentration was regressed on $\cos \theta$ and $\sin \theta$ (representing one cycle per annum), on $\cos 2\theta$ and $\sin 2\theta$ (representing 2 cycles per annum), and on $\cos 3\theta$ and $\sin 3\theta$ (representing 3 cycles per annum). The most parsimonious model was the regression of maternal serum 25(OH)D on $\cos \theta$, $\sin \theta$, $\cos 2\theta$, and $\sin 2\theta$.

Maternal height, age, parity, educational qualifications, social class, smoking in pregnancy, IOM weight-gain category, pre-pregnancy BMI, supplementary vitamin D intake in late pregnancy, and vitamin D intake from food in late pregnancy, together with the predicted seasonal component of vitamin D (as described by the Fourier analysis model), were considered as predictors of vitamin D status. Predictors with a $P$ value of $<0.2$ in univariate linear regression models were entered into a multivariate linear regression model, and a backward stepwise procedure was used to identify significant predictors at the 5% level. Statistical analysis was performed with the use of Stata 11.1 (20).

**RESULTS**

The characteristics of the mothers studied and their children are shown in **Table 1**. Vitamin D status was assessed at a mean (±SD) of 34.6 ± 0.7 wk gestation. Maternal serum 25(OH)D concentrations were not associated with gestational age ($P = 0.51$).

Compared with the 875 participants who were eligible for the analyses but who did not have a measure of maternal vitamin D status or did not have DXA scans at any time point, the 977 participants in the current study were better educated ($P < 0.0001$), less likely to smoke during pregnancy ($P = 0.0004$), older ($P = 0.0006$), slightly taller ($P = 0.02$), and more likely to be from a white ethnic group ($P = 0.0002$). However, there was no difference in prepregnancy BMI between the 2 groups ($P = 0.86$).

We examined associations between maternal 25(OH)D in late pregnancy and offspring fat mass and fat-free mass at birth and at ages 4 and 6 y (Table 2). The unadjusted results indicate associations between lower vitamin D status and greater fat mass at both ages 4 and 6 y. After adjustment for maternal educational attainment, smoking in pregnancy, prepregnancy BMI, height, parity, social class, IOM weight-gain category, child’s breastfeeding duration, and child’s vitamin D intake and physical activity at 3 y, the association between maternal 25(OH)D and 4- to 6-y fat mass became nonsignificant. After adjustment for these confounders, there was a positive association between maternal 25(OH)D and fat mass at birth ($P = 0.02$), whereas the inverse association with 6-y fat mass remained ($P = 0.01$). There was no association between maternal 25(OH)D and fat-free mass at birth or at ages 4 or 6 y, either before or after adjustment for confounders.

The associations between maternal vitamin D status in late pregnancy and offspring fat mass at birth and at ages 4 and 6 y are shown in Figure 1. In comparison with the offspring of mothers with low serum 25(OH)D concentrations (≤50 nmol/L), fat mass at birth was 8% (95% CI: 1%, 16%) greater among those born to mothers whose 25(OH)D concentrations were between 50 and 75 nmol/L, and 10% (95% CI: 3%, 17%) greater among those whose mothers had a 25(OH)D concentration $>75$ nmol/L. In comparison with mothers who had a low vitamin D status

### Table 1

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
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<tbody>
<tr>
<td>Mother</td>
<td></td>
</tr>
<tr>
<td>Prepregnancy BMI (kg/m²)</td>
<td>24.3 (22.2–27.6)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.64 ± 0.64</td>
</tr>
<tr>
<td>Degree qualification or above (%)</td>
<td>23.8</td>
</tr>
<tr>
<td>Smoked in pregnancy (%)</td>
<td>14.6</td>
</tr>
<tr>
<td>Age at child’s birth (y)</td>
<td>30.4 ± 3.8</td>
</tr>
<tr>
<td>Primiparous (%)</td>
<td>45.9</td>
</tr>
<tr>
<td>Total vitamin D intake in late pregnancy (µg/d)</td>
<td>3.9 (2.7–5.7)</td>
</tr>
<tr>
<td>Use of vitamin D supplements in late pregnancy (%)</td>
<td>22.2</td>
</tr>
<tr>
<td>Use of ≥10 µg vitamin D supplement/d in late pregnancy (%)</td>
<td>8.5</td>
</tr>
<tr>
<td>Serum 25(OH)D concentration (nmol/L)</td>
<td>62 (43–89)</td>
</tr>
<tr>
<td>&lt;50 nmol/L (%)</td>
<td>35.1</td>
</tr>
<tr>
<td>50 to ≤75 nmol/L (%)</td>
<td>28.3</td>
</tr>
<tr>
<td>&gt;75 nmol/L (%)</td>
<td>36.6</td>
</tr>
<tr>
<td>Child</td>
<td></td>
</tr>
<tr>
<td>Male (%)</td>
<td>52.3</td>
</tr>
<tr>
<td>Fat mass at birth (kg)</td>
<td>0.53 (0.40–0.68)</td>
</tr>
<tr>
<td>Fat-free mass at birth (kg)</td>
<td>3.01 ± 0.35</td>
</tr>
<tr>
<td>Fat mass at age 4 y (kg)</td>
<td>4.65 (4.07–5.55)</td>
</tr>
<tr>
<td>Fat-free mass at age 4 y (kg)</td>
<td>13.20 ± 1.59</td>
</tr>
<tr>
<td>Fat mass at age 6 y (kg)</td>
<td>5.21 (4.26–6.53)</td>
</tr>
<tr>
<td>Fat-free mass at age 6 y (kg)</td>
<td>18.07 ± 2.20</td>
</tr>
</tbody>
</table>

1 Median; IQR in parentheses (all such values)
2 Mean ± SD (all such values).
3 25(OH)D, 25-hydroxyvitamin D.
(≤50 nmol/L), 6-y fat mass was 8% (95% CI: 2%, 14%) lower among mothers who had a vitamin D status between 50 and 75 nmol/L, and 6% (95% CI: 0, 12%) lower among mothers who had a vitamin D status >75 nmol/L.

It appeared that there might be a threshold effect of vitamin D status on 6-y fat mass. A cubic spline model fitted to the data indicated a plateauing of the effect at ≥64 nmol/L. Among mothers whose serum 25(OH)D concentrations were <64 nmol/L, there was a significant negative association between vitamin D status and 6-y fat mass ($\beta = -0.24; 95\% \text{ CI}: -0.41, -0.07; P = 0.006$), whereas among mothers with higher 25(OH)D there was no association ($\beta = 0.00; 95\% \text{ CI}: -0.19, 0.18; P = 0.99$), after adjustment for confounders. This interaction was significant ($P = 0.03$).

We examined a number of potential influences on vitamin D status in pregnancy. The multivariate linear regression model of predictors of maternal 25(OH)D is presented in Table 3. The most important predictor of higher vitamin D status was season of sampling, as described by the pattern in Figure 2. Among women whose samples were taken in July–September, 84% had 25(OH)D concentrations >64 nmol/L, whereas among women whose samples were taken in December–April, only 23% had values >64 nmol/L. The second most important predictor was taking vitamin D in the form of dietary supplements in late pregnancy. Of the 8.5% of women who followed the UK recommendation to take an additional 10 μg vitamin D in pregnancy, 94% had serum 25(OH)D concentrations >64 nmol/L, whereas among the rest of the cohort only 44% had values >64 nmol/L. Higher late-pregnancy vitamin D intake from food and not smoking in pregnancy were less important predictors of higher maternal vitamin D status. After all of these variables were included in the multivariate model, maternal prepregnancy BMI did not make an additional contribution, and it was not significant ($P = 0.11$).

**DISCUSSION**

We have shown that maternal vitamin D status measured in late pregnancy is associated with differences in DXA-assessed adiposity in the offspring. However the pattern of association

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Unadjusted</th>
<th>Adjusted for confounders</th>
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</thead>
<tbody>
<tr>
<td>Birth fat mass (SD)</td>
<td>$0.06 (-0.01, 0.12)$</td>
<td>$0.08 (0.02, 0.15)$</td>
</tr>
<tr>
<td>Birth fat-free mass (SD)</td>
<td>$0.02 (-0.03, 0.07)$</td>
<td>$0.04 (-0.02, 0.09)$</td>
</tr>
<tr>
<td>4-y fat mass (SD)</td>
<td>$-0.09 (-0.16, -0.02)$</td>
<td>$-0.01 (-0.08, -0.07)$</td>
</tr>
<tr>
<td>4-y fat-free mass (SD)</td>
<td>$0.03 (-0.02, 0.08)$</td>
<td>$0.03 (-0.02, 0.08)$</td>
</tr>
<tr>
<td>6-y fat mass (SD)</td>
<td>$-0.16 (-0.23, -0.08)$</td>
<td>$-0.10 (-0.17, -0.02)$</td>
</tr>
<tr>
<td>6-y fat-free mass (SD)</td>
<td>$0.01 (-0.04, 0.06)$</td>
<td>$0.02 (-0.03, 0.07)$</td>
</tr>
</tbody>
</table>

1 Birth outcomes adjusted for sex, gestation, age at measurement, age squared, and length; childhood outcomes adjusted for sex, age, and height. 25(OH)D, 25-hydroxyvitamin D.

2 Regression coefficient $\beta$ from multiple linear regression of offspring body composition measurement on maternal 25(OH)D at 34 wk gestation (per SD increase).

3 Confounders were maternal educational attainment, smoking in pregnancy, prepregnancy BMI, height, parity, social class, and Institute of Medicine weight-gain category and, for childhood outcomes, breastfeeding duration, vitamin D intake at age 3 y, and physical activity at age 3 y.
differed at birth from that in later childhood; lower vitamin D status was associated with lower fat mass assessed at birth and with higher fat mass assessed at age 6 y. These associations were robust to adjustment for a range of confounding factors, including childhood vitamin D intake and known predictors of adiposity in children. To our knowledge, these associations between maternal vitamin D status and adiposity in the offspring at birth and in later childhood have not been previously described. In contrast to the relations observed with adiposity, we found no association between maternal vitamin D status and fat-free mass in these children.

Although the potential effects of maternal vitamin D deficiency on offspring body composition have been recognized (10), there are few published studies with which we can compare our findings directly. We found that lower vitamin D status was associated with lower adiposity at birth (Figure 1); the significance of variations in fat mass at birth measured directly by using DXA, as in the current study, is not yet fully understood (21). We have previously described limited “tracking” of DXA-assessed fat mass between birth and age 4 y in the SWS cohort (r = 0.24), whereas the correspondence between ages 4 and 6 y is much stronger (r = 0.86) (16). This suggests that a lower fat mass at birth is not necessarily associated with lower adiposity in later childhood.

By the age of 4 y, the pattern of association between maternal vitamin D status and fat mass in the SWS children had reversed, such that lower status in late pregnancy was predictive of greater adiposity at age 6 y, and this persisted after taking into account a wide range of confounders. There are 2 studies that are relevant to our findings. First, in a recent study in Indian children aged 9.5 y (8), percentage fat determined by using bioimpedance analysis was greater in offspring of mothers with a lower vitamin D status assessed at 28–32 wk gestation, although this was of borderline significance after adjustment for the effects of maternal covariates. Unlike our study, among the Indian children, there were positive associations between maternal vitamin D status and a measure of fat-free mass (arm-muscle area). There are methodologic differences between the studies that may be important: for example, we assessed body composition directly by using DXA. However, it may also be difficult to compare the findings because so many maternal characteristics differ, including body composition and vitamin D status. For example, 67% of Indian women studied had low 25(OH)D (<50 nmol/L), compared with 35% in the present study.

A study that allows a more direct comparison with our current data included 178 children aged 9 y from a previous Southampton birth cohort, whose body composition was also assessed with the use of DXA (13). In contrast with the present findings, maternal vitamin D status in late pregnancy was not related to offspring fat mass in the earlier study. It is not clear why these findings differ. Apart from differences in the size of the cohorts, there are other differences between the cohorts that may be relevant. Among these, the children in the previous study were approaching puberty, and the greater use of vitamin D supplements (22% compared with 7%) and higher maternal vitamin D status in late pregnancy [median 25(OH)D: 62 compared with 50 nmol/L] in the SWS may be important. In our continued follow-up of the SWS children we will be able to reassess the relation between maternal vitamin D status and body composition in later childhood, but the differences between the findings of the studies are currently not explained.

Strengths and weaknesses

The SWS provides data from a contemporary cohort of women and their offspring from a wide range of sociodemographic backgrounds. Vitamin D status was measured in a large number of participants, and offspring outcomes were assessed at 3 different ages. We used DXA to provide measurements of fat mass...
and fat-free mass, and by including length or height measures in the models we were able to describe effects independent of infant or childhood stature. Because we have detailed data on this cohort we were able to control for a large number of other potential confounding factors, including important predictors of childhood fat mass such as maternal education, prepregnancy BMI, smoking in pregnancy, and duration of breastfeeding. For example, although we have previously shown that excessive weight gain in pregnancy is associated with offspring adiposity (16), we found that the association between maternal vitamin D status and offspring fat mass at age 6 y was independent of this effect (Table 2).

A limitation is that DXA measurements and maternal vitamin D status were not available for the whole SWS cohort but for a subset of 977 children and mothers. When compared with a similar group without measurements, these mothers were better educated, less likely to smoke in pregnancy, older, taller, and more likely to be from a white ethnic group. However, unless the associations between maternal vitamin D status and offspring body composition are different in the remainder of the cohort, it is unlikely that selection bias could explain our findings. A further consideration is that of reverse causality, because increased storage of vitamin D in women who have a greater fat mass results in lower 25(OH)D (22–24). An apparent association between lower maternal vitamin D status and adiposity in the offspring could therefore be attributed to the effects of shared diet and lifestyle, rather than causally related to variations in status. Although we cannot exclude this possibility, the association between vitamin D status and 6-y fat mass was independent of maternal prepregnancy BMI in our study (Table 2); we therefore do not think this would explain the pattern of associations we observed.

Although the use of DXA is well validated in adults, it is important to recognize potential problems with neonates and young children because of their low absolute bone mineral content and tendency to move. However, we used specific pediatric software, and movement artifact was modest; the few individuals with excessive movement at each time point were excluded from our analyses. The accuracy of DXA for the assessment of body composition in small animals has been shown in piglets (25, 26).

Implications of the findings

We will be able to address some of the implications of the findings of the present study in our continued follow-up of these children. Although the findings require extension and replication, the mechanisms that link vitamin D insufficiency to a lower fat mass in infants at birth but greater adiposity in later childhood also need to be elucidated. The role of vitamin D in adipocyte metabolism is complex (4, 5), and the mechanisms that link vitamin D status to adiposity are currently unknown. One interpretation of our data is that there are programmed effects on the fetus that arise from maternal vitamin D insufficiency that remain with the individual and that may predispose him or her to gain excess body fat in later childhood. We speculate that there may be different routes to childhood obesity arising in prenatal life because greater adiposity in childhood has been associated with both insufficient maternal nutrition [vitamin D insufficiency or inadequate pregnancy weight gain (16)] as well as excess nutrition [maternal adiposity and excessive pregnancy weight gain (16)]. Although further studies are needed, the present findings add weight to current concerns about the prevalence of low vitamin D status among women of reproductive age (6). Future analyses of an ongoing UK trial of maternal vitamin D supplementation will therefore provide important evidence of the role of vitamin D insufficiency in determining offspring body composition (27).

We thank the general practitioners and midwives in Southampton for their support. We are grateful to the research nurses and other staff of the Southampton Women’s Survey for all of their work in recruiting and interviewing the participants and in processing the data and samples. We also thank the women of Southampton and their children who gave their time to take part in the study. Zoe Cole collected the 6-y outcome data. Rami Swaminathan was responsible for the vitamin D assays.

The authors’ responsibilities were as follows—SRC: performed the statistical analysis and drafted the manuscript; SMR: designed the research, conceived the analyses, and revised the manuscript; NCH: collected the 4-y outcome data and revised the manuscript; and HMI, KMG, and CC: designed the research and revised the manuscript. All authors contributed to the interpretation of the data and approved the final version of the manuscript. KMG has acted as a consultant to Abbott Nutrition and Nestlé Nutrition and has received reimbursement for speaking at an Abbott Nutrition Conference on Pregnancy Nutrition and Later Health Outcomes and at a Nestlé Nutrition Institute Workshop; he is part of an academic consortium that has received research funding from Abbott Nutrition, Nestec, and Danone. None of the other authors had any potential conflicts of interest.

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