BACKGROUND: Higher food intake is implicated in the elevated risk of obesity associated with shorter sleep in children, but the mechanisms driving higher intake are uncertain. Research in adults suggests that acute sleep deprivation affects brain reward systems, which increases responsiveness to palatable foods. However, there have been few studies addressing habitual sleep duration and few in children, among whom the strongest associations with body mass index (BMI) are seen.

OBJECTIVE: The objective of this study is to test the hypothesis that shorter-sleeping children are more food responsive and explores the mediation of the relationship between sleep and weight by food responsiveness (FR).

METHODS: Participants were twin pairs from Gemini, a UK twin birth cohort, who had provided complete information on their children’s sleep and appetite at age 5 years (N = 1008). One child from each twin pair was randomly selected for analysis. Nighttime sleep duration was calculated from parent-reported bedtime and wake time, and categorized as shorter, adequate, or longer according to age-specific reference values. FR was assessed with the Child Eating Behaviour Questionnaire. BMI z-scores (BMI-SDS) were calculated from parent-reported heights and weights using the UK 1990 reference data and were available for 494 children.

RESULTS: There was a significant linear association between shorter sleep and higher FR at age 5 years (P for linear trend = 0.032), which was maintained after adjusting for age, sex, birth weight, maternal education, and BMI-SDS. In the subset with BMI data at age 5 years, shorter sleep was associated with a higher BMI-SDS (P = 0.026) as expected. Testing for mediation by adding FR to the model attenuated the linear relationship to borderline significance (P = 0.049), suggesting partial mediation.

CONCLUSIONS: Shorter sleep in childhood is associated with higher FR, which may partly explain the association between shorter sleep and adiposity in childhood.

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INTRODUCTION
Short sleep in childhood has been shown to significantly raise the risk of overweight and obesity. Evidence to date points to food intake rather than activity as the primary pathway. Studies in healthy adults in which nighttime sleep is restricted show increased energy intake and weight gain. Epidemiological studies in children have identified an inverse relationship between sleep duration and energy intake.

Interest has now turned to the mechanisms through which shorter sleep affects food intake. Neurin aging data show that sleep deprivation increases activity in brain reward centers in response to palatable food, as well as hedonic eating. Experimental studies in adults, in which nighttime sleep is restricted, show increased energy intake and weight gain. Epidemiological studies in children have identified an inverse relationship between sleep duration and energy intake.

Hypothesis has now turned to the mechanisms through which shorter sleep affects food intake. Neuroimaging data show that sleep deprivation increases activity in brain reward centers in response to palatable food, as well as hedonic eating. Experimental studies in adults, in which nighttime sleep is restricted, show increased energy intake and weight gain. Epidemiological studies in children have identified an inverse relationship between sleep duration and energy intake.

In the present study, the primary hypothesis was that habitual short sleep at age 5 years would be associated with higher FR. In the subset of participants with weight data at age 5 years, we tested the hypothesis that FR would partly mediate the association between sleep duration and weight. As a secondary analysis, we also examined whether sleep duration was associated with high energy intake (indexed with hunger responsiveness (HR)) to test whether the sleep-appetite associations was general rather than specific to FR.

SUBJECTS AND METHODS
Participants
Participants were from Gemini, a UK twin birth cohort. The Gemini study has been described in detail previously. All 219 families with twins born in England and Wales between March and December 2007 were contacted through the Office for National Statistics. The baseline sample of 2402 families represented 3% of all live-born twins during this period. The present study used data collected in 2012, when the children were on average 5 years old. Data were from 1008 families who had provided complete information on their children’s sleep and appetite at this age. To avoid clustering effects, one child from each twin pair was randomly selected for the analysis. Mothers who provided complete data were slightly older, more highly educated and more likely to be from a White ethnic background (all P’s < 0.001).

At this age, complete height and weight data were available for 494 (49% of baseline sample). Mothers who provided this information were more likely to be university educated, but there were no differences between the study sample and the sample with body mass index (BMI)
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RESULTS
Participant characteristics are shown in Table 1. In total, 1008 children had complete sleep and appetite data at age 5 years (mean 52 years, SD 0.1). Average nighttime sleep duration was 11.48 h (SD 0.66 h), the average FR score was 2.84 (0.61) and the average SR score was 2.38 (0.76). BMI-SDS information was only available on 494 children, with a mean value of 0.20 (0.96).

Univariate and multivariate associations between sleep and FR are shown in Table 2. There was a significant linear relationship between nighttime sleep duration and FR, such that shorter sleep was associated with higher FR at age 5 years (P for linear trend = 0.026). These associations were retained after adjusting for age, sex, maternal education, birth weight and BM ISDS, the latter adjusting for differences in sample size. There was no significant association between nighttime sleep duration and SR (P for linear trend = 0.074).

The results of analysis of variance and analysis of covariance models predicting BMI ISDS in the subset of the sample with available weight data are given in Table 3. As expected, shorter nighttime sleep was associated with higher BMI ISDS (P for linear trend = 0.026). The linear relationship was strengthened after adjusting for age, sex, birth weight and maternal education. To test the mediation, we added FR into the analysis of covariance model predicting BM ISDS. This model attenuated the linear association between sleep and BM ISDS to borderline significance (P for linear trend = 0.049). The Hayes’ PROCESS add-in for SPSS demonstrated the mediation effect via FR was significant (−0.04 (0.02); 95% confidence interval:−0.09 to−0.01).

DISCUSSION
This study provides strong evidence for an association between habitually shorter nighttime sleep and higher FR in childhood. We also observed that higher FR could partly account for the linear relationship between sleep and BMI ISDS at age 5 years. In contrast, sleep duration showed no association with FR, a measure of homeostatic eating.

These findings support experimental work in adults, which has suggested that acute sleep deprivation in unaccustomed to homeostatic pathways to food consumption. The results are also similar to the previous pediatric study, which found that shorter sleep was associated with higher external eating but not with emotional or restrained eating. External eating has conceptual overlap with FR in that both traits act as the propensity to overconsume in response to palatable food cues. Among adults, one previous study has shown that a tendency to dishibit eating moderates the association between sleep and BMI, with a stronger relationship among adults who had higher disinhibited eating.

Table 1. Participant characteristics given as mean (SD) unless otherwise stated

<table>
<thead>
<tr>
<th>Measure</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth weight</td>
<td>246 (54)</td>
</tr>
<tr>
<td>Maternal education (l)</td>
<td>274/221/505</td>
</tr>
<tr>
<td>Sex (l): m/f/male</td>
<td>494/506</td>
</tr>
<tr>
<td>Nighttime sleep (l)</td>
<td>1148 (66)</td>
</tr>
<tr>
<td>Appetite</td>
<td></td>
</tr>
<tr>
<td>Satiety responsiveness</td>
<td>2.38 (0.76)</td>
</tr>
<tr>
<td>Food responsiveness</td>
<td>2.84 (0.61)</td>
</tr>
<tr>
<td>BMI ISDS</td>
<td>-0.21 (0.96)</td>
</tr>
</tbody>
</table>

Abbreviation: BM ISDS, body mass index sd. st. mean of n = 494.
response to in ages of highly palatable foods but not in response to healthy foods. Together, these findings indicate that suboptimal nighttime sleep may specifically increase the salience of palatable foods, and consequently the drive to consume, within a permissive environment.

Although this study provides evidence that sleep may increase hedonic eating in children, more work is needed to show that this in turn drives overconsumption among shorter sleepers. Studies of dietary intakes have shown that shorter sleeping children have more frequent eating occasions and more unfavourable dietary patterns, in particular a higher intake of energy-dense foods. These patterns of consumption characterize a kind of hedonic overeating, where eating is responsive to food cues rather than due to impaired satiety processes. There is a need for longitudinal research to establish whether the relationship strengthens as children gain increasing autonomy over their eating behaviour and food environment, and whether factors such as food availability, accessibility and rules in the home environment influence susceptibility to weight gain. Given that parents largely control the food environment at this stage, this could have implications for interventions to prevent excess weight gain, for example, controlling exposure and access to palatable foods among children who experience difficulty sleeping, in particular at night when parents may be more inclined to feed-to-soothe.

Limitations
This study has some limitations that should be considered. The cross-sectional design means longer-term follow-up is needed to understand the temporal distribution of the relationship between sleep, FR and weight gain. Our sample included only children with complete data on sleep and appetite, and for the mediation analyses only a smaller sub-sample with BMI data, thereby excluding a considerable proportion of the Gemini sample. Children with complete data had mothers who were more likely to be university educated and from a White ethnic background, which limits the generalisability of the findings; hence, replication in a more diverse sample is required. Gemini children are leaner with respect to the UK 1990 reference values, reflecting the fact that twin tend to be born smaller than singletons. Although this could limit the generalisability of the findings, there is no strong reason to expect that the association between sleep and weight, and the factors that mediate this relationship, should differ between twins and singletons.

Parent-reported nighttime sleep is a limitation, although this method is common in larger population-based studies where objective measures are not feasible. Encouragingly, the mean nighttime sleep duration in this sample is comparable to published reference values for children at age 5 years. Further, one can calculate nighttime sleep from parent-reported bedtime and wake time as has been validated against actigraphy in young children and might provide a better global representation of sleep behaviour than a few nights of objective recording.

CONCLUSION
We show that shorter sleep at age 5 years is associated with higher FR but not with SR. Mediation analysis is consistent with the idea that FR is part of the pathway mediating the effect of shorter sleep on adiposity.

CONFLICT OF INTEREST
The authors declare no conflict of interest.

ACKNOWLEDGEMENTS
We thank the Gemini families who are participating in the study and the Office of National Statistics for their help in recruiting them. Gemini was funded by a grant from Cancer Research UK (C1418/A7974). LMCD is funded by a UK Medical Research Council PhD studentship.

REFERENCES

Table 2. ANOVA and ANCOVA models for associations between appetitive traits and nighttime sleep duration

<table>
<thead>
<tr>
<th>Nighttime sleep duration</th>
<th>o 11 h</th>
<th>11–12 h</th>
<th>4 12 h</th>
<th>P (linear trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Univariate models</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Food responsiveness</td>
<td>2.53 (0.08)</td>
<td>2.36 (0.03)</td>
<td>2.35 (0.04)</td>
<td>0.032*</td>
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<tr>
<td>Satiety responsiveness</td>
<td>2.80 (0.07)</td>
<td>2.89 (0.02)</td>
<td>2.80 (0.03)</td>
<td>0.749</td>
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<tr>
<td><strong>Multivariate models</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Food responsiveness</td>
<td>2.55 (0.07)</td>
<td>2.36 (0.03)</td>
<td>2.35 (0.05)</td>
<td>0.022*</td>
</tr>
<tr>
<td>Satiety responsiveness</td>
<td>2.82 (0.06)</td>
<td>2.88 (0.03)</td>
<td>2.76 (0.04)</td>
<td>0.372</td>
</tr>
</tbody>
</table>

Table 3. ANOVA and ANCOVA models for associations between sleep at BMI-SDS, adjusting for appetite traits FR and SR

<table>
<thead>
<tr>
<th>Nighttime sleep duration</th>
<th>o 11 h</th>
<th>11–12 h</th>
<th>4 12 h</th>
<th>P (linear trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BMI-SDS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Univariate</td>
<td>0.01 (0.15)</td>
<td>-0.15 (0.05)</td>
<td>-0.35 (0.10)</td>
<td>0.026*</td>
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<tr>
<td>Multivariate</td>
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<td></td>
</tr>
<tr>
<td>Model 1</td>
<td>0.05 (0.14)</td>
<td>-0.16 (0.06)</td>
<td>-0.36 (0.09)</td>
<td>0.015*</td>
</tr>
<tr>
<td>Model 2</td>
<td>-0.01 (0.13)</td>
<td>-0.36 (0.06)</td>
<td>-0.33 (0.08)</td>
<td>0.049*</td>
</tr>
</tbody>
</table>

Abbreviations: ANCOVA, analysis of covariance; ANOVA, analysis of variance; BMI-SDS, body mass index s.d. score; FR, food responsiveness; SR, satiety responsiveness. Data given as mean (s.e.). *P = 0.05. Adjusted for age, sex, birth weight and maternal education. Model 2 adjusted for all covariates and BMI-SDS.
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