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# Influence of maternal metabolism on neonate eating behaviour

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ABSTRACT

Perinatal maternal metabolism influences metabolic control in the offspring, but its impact on food intake in neonates is not known. We investigated whether ingestive behaviour in the first days of life is modulated by maternal BMI, maternal weight gain during pregnancy and by gestational diabetes (GD). Two weeks after birth, mothers of 21 neonates measured neonate food intake by weighing breast-fed babies before and after feeding, or directly in bottle-fed babies. They also filled in the Baby Eating Behaviour Questionnaire (BEBQ). We compared results between groups defined by preconceptual BMI, by pregnancy-related body weight gain according to Institute of Medicine guidelines and by GD diagnosis, and performed supplementary stepwise regression analyses. Birth weight was generally comparable between maternal groups and averaged 3450 g. BEBQ 'food responsiveness' was rated higher in neonates born to females with increased vs. reduced weight gain during pregnancy; weight gain in the second trimester of pregnancy was of particular relevance for this pattern. Neonates born to mothers with recommended weight gain displayed increased 'satiety responsiveness' compared to neonates of mothers with increased or reduced weight gain. BEBQ 'slowness in eating' was rated higher by mothers with increased vs. those with recommended weight gain in the second trimester and tended to be increased in neonates born to mothers with GD. Eating protocols supported the latter finding but in general did not yield significant group differences. We conclude that dysregulation of maternal metabolism during pregnancy may predispose neonates to dysfunctional eating behaviour at a very early age, with potential ramifications for subsequent body weight trajectories.

## 1. Introduction

During pregnancy, the fetus is exposed to maternal metabolism and, consequently, metabolic dysfunctions and disorders that exist or arise in the mother's organism. According to meta-analyses, maternal obesity at the time of conception increases the child's odds to display overweight or obesity between 1 and 16 years of age by 264 % (Heslehurst et al., 2019). Increased maternal weight gain during pregnancy is associated with a higher risk of the child to be born large for gestational age, and particularly so in women with lower vs. higher preconceptual BMI (Goldstein et al., 2017). The influence both of maternal obesity and pregnancy-related weight gain has been found to increase children's body fat mass up to 6 years of age (Andres et al., 2015; Crozier et al., 2010). Maternal metabolism moreover affects cognitive outcomes in children. Recent meta-analyses indicate that maternal diabetes (both pre-gestational and gestational) increases the risk of neurodevelopmental disorders including lower intelligence scores in children aged up to 18 years (Ye et al., 2025).

While there are not many studies on the impact of maternal metabolism on the child's eating behaviour, there is evidence that maternal BMI before pregnancy is positively related to measures of infant appetite at around 3–5 months of age (Rios et al., 2023; Rising & Lifshitz, 2005;

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Stunkard et al., 2004). Thus, Rios and colleagues (2023) have reported a positive association of pre-pregnancy BMI with the baby's general appetite as assessed with the Baby Eating Behaviour Questionnaire (BEBQ; Llewellyn et al., 2011) at 4 months of age. Maternal dietary restraint according to the Dutch Eating Behaviour Questionnaire restrained eating subscale (Van Strien et al., 1986) was negatively associated with general appetite, but, in a subset of babies, positively related to videotaped hedonic responses to sucrose (Rios et al., 2023). In that and the few related studies (Rising & Lifshitz, 2005; Stunkard et al., 2004), maternal weight gain during pregnancy was not analyzed. Also, to the best of our knowledge, eating behaviour of neonates, i.e., in the first 4 weeks of life, in dependence of maternal metabolism has not been assessed before. In order to investigate whether maternal metabolism impacts eating behaviour – as a crucial determinant of body weight trajectories – at this very early age, we explored the relevance of pre-conceptual BMI (pcBMI), weight gain during pregnancy and gestational diabetes mellitus (GD) for neonate food intake measured 2 weeks postpartum.

## 2. Methods

### 2.1. Participants

Twenty-one expectant mothers and their healthy, mature (gestational age >37 weeks), singleton newborns were examined. Participants gave written informed consent to this prospective observational study, which confirmed to the Declaration of Helsinki and was approved by the local Ethics Committee.

### 2.2. Experimental procedure

Maternal body weight was assessed at the time of conception and of birth using medical records and self-report measures, enabling the categorization according to pcBMI (< vs. ≥ 25 kg/m$^2$) and weight gain during pregnancy (reduced, recommended, increased) according to Institute of Medicine (IOM) guidelines (IOM, 2009), which take into account maternal pcBMI, i.e., underweight, <18.5 kg/m$^2$, normal weight, 18.5–24.9 kg/m$^2$, overweight, 25–29.9 kg/m$^2$, or obese ≥30 kg/m$^2$. Reduced/recommended/increased weight gain according to IOM is defined as, respectively, <12.5/12.5–18/>18 kg for mothers with underweight, <11.5/11.5–16/>16 kg for mothers with normal weight, <7/7–11.5/>11.5 kg for mothers with overweight and <5/5–9/>9 kg for those with obesity. Participants were also asked to journalize their body weight at the start of the second and third trimester of pregnancy, enabling us to perform exploratory analyses of trimester-wise weight gain in a subgroup of responders (n = 17). The IOM guidelines for reduced/recommended/increased weight gain/-trimester are as follows: <5.28/5.28–6.96/>6.96 kg in mothers with preconceptional underweight, <4.2/4.2–6/>6 kg in mothers with normal weight, <2.76/2.76–3.96/>3.96 kg in mothers with overweight and <2.04/2.04–3.24/>3.24 kg in mothers with obesity (IOM, 2009). Fifteen participants underwent an oral glucose tolerance test to diagnose potential GD and in six participants, information on GD status was obtained from the respective general practitioners or gynecologists.

Within 24 h after delivery, the child's birth weight was measured. At 13.81 ± 0.80 days postpartum, on-demand neonatal eating behaviour was assessed by the parents across at least two consecutive days by weighing the newborn with an infant scale immediately before and after each individual bout of feeding (breastfed babies), yielding an estimate of the amount of ingested breast milk, or by recording in writing the type and amount of bottle-feeding. Parents also noted down the duration of each feeding session, enabling the calculation of feeding speed. At the end of the assessment period, the mothers filled in the BEBQ (Llewellyn et al., 2011), which uses 18 dimensional items (never/-rarely/sometimes/often/always) to reflect the baby's ingestive behaviour at a typical daytime feed on five scales. 'Food responsiveness' captures how demanding the baby is with regard to being fed and the level of responsiveness to cues of milk and feeding (e.g., "If allowed to, my baby would take too much milk"). 'Enjoyment of food' captures the infant's perceived liking of milk and feeding in general (e.g., "My baby enjoys feeding time"). 'Satiety responsiveness' measures the baby's fullness threshold (e.g., "My baby gets full easily"). 'Slowness in eating' examines typical feeding speed (e.g., "My baby feeds slowly"). Additionally, 'general appetite' captures overall appetite on a one-item scale ("My baby has a big appetite").

### 2.3. Statistical analyses

Data were analyzed with SPSS (IBM Corp., 2021) and R (R Core Team, 2022) and are presented as means ± SEM. We compared, respectively, groups of mothers with a pcBMI <25 vs. ≥ 25 kg/m$^2$, with reduced vs. recommended vs. increased weight gain during pregnancy, and with vs. without GD diagnosis via analyses of variance (ANOVA) and covariance (ANCOVA) and subsequent independent t-tests. We also performed supplementary multiple stepwise regression analyses with the predictors maternal pcBMI, weight gain during pregnancy, GD diagnosis, the infant's sex and age at the start of eating assessment, maternal age, parity and feeding type (breast- or bottle-fed). In an exploratory fashion, we also ran analyses of the relationship between trimester-wise weight gain and measures of neonate food intake. IOM guidelines for trimester-wise weight gain are available for the second and third trimesters, when it is usually greater than in the first trimester and follows a linear pattern (IOM, 2009); accordingly, trimester-wise analyses in terms of reduced/recommended/increased weight gain were restricted to the second and third pregnancy trimesters. Since neonate age turned out to be the strongest predictor of total food intake per day ($R^2 = 0.26$, $B = 29.26$, $SE = 11.41$, $\beta = 0.51$, $P < 0.02$), it was used as a covariate in the analyses of eating behaviour. (Note that analyses without this covariate yielded essentially comparable results.) A P value < 0.05 was considered significant.

## 3. Results

### 3.1. Participants and neonates

Our sample of pregnant females/mothers comprised 14 nulliparous and 7 multiparous females (second child, n = 3; third child, n = 3; fifth child, n = 1) with a mean age (at birth) of 32.83 ± 1.08 yrs, who delivered 12 female and 9 male babies. Sixteen babies were breastfed, 4 babies were bottle-fed and 1 baby was both breast- and bottle-fed. Table 1 presents pcBMI and weight gain during pregnancy according to group. Maternal pcBMI was significantly lower in the <25 than the ≥25 kg/m$^2$ BMI group; the two groups did not differ in weight gain during pregnancy. The reduced/recommended/increased weight gain groups showed the expected significant pattern in terms of weight gain. They displayed a trend towards a difference in pcBMI that was due to relatively increased pcBMI in the increased vs. recommended group ($P < 0.02$, independent *t*-test). Six participants developed GD. Females with GD had a higher pcBMI than females without GD but displayed comparable weight gain during pregnancy. Maternal age was comparable in all comparisons of groups categorized according to pcBMI, weight gain and diagnosis of GD ($P > 0.12$). Birth weight and length were likewise comparable between groups (all $P > 0.11$) and averaged 3447 ± 131 g and, respectively, 51.21 ± 0.51 cm.

### 3.2. Neonate eating behaviour

Food intake according to weighing protocols averaged 567.91 ± 39.54 g/day across all babies and was in general significantly higher between 8.00 a.m. and 8.00 p.m. than at night, with relative maxima around noon and in the evening (data not shown). The amount of food intake/day, the duration of food intake/day and the speed of food intake

**Table 1**
Participants according to metabolic categories.

| | pcBMI | | *Group* | Weight gain | | | *Group* | GD | | *Group* |
|---|---|---|---|---|---|---|---|---|---|---|
| | <25 | ≥25 | | Red. | Rec. | Inc. | | Yes | No | |
| **BMI [kg/m$^2$]** | 21.9 ± 0.6 | 29.9 ± 1.4 | F (1,19) = 38.35, **P < 0.001** | 23.4 ± 2.1 | 22.0 ± 1.2 | 27.4 ± 1.4 | F (2,18) = 3.17, P = 0.07 | 28.5 ± 2.5 | 23.0 ± 0.8 | F (1,19) = 7.75, **P = 0.01** |
| **Weight gain [kg]** | 13.5 ± 1.3 | 13.7 ± 2.0 | F (1,19) = 0.009, P = 0.93 | 9.5 ± 1.9 | 13.1 ± 1.0 | 17.4 ± 1.0 | F (2,18) = 9.11; **P = 0.002** | 13.1 ± 2.2 | 13.7 ± 1.2 | F (1,19) = 0.07; P = 0.80 |

Data are presented as mean values ± SEM. Participants were categorized according to preconceptual (pc) BMI (<25 kg/m$^2$, n = 14; ≥25 kg/m$^2$, n = 7), weight gain during pregnancy according to IOM guidelines (reduced [Red.], n = 7; recommended [Rec.], n = 6; increased [Inc.], n = 8) and the diagnosis of gestational diabetes mellitus, GD (yes, n = 6; no, n = 15). *Group* column indicates respective ANOVA result.

did not depend on pcBMI and weight gain (all P > 0.46 for ANCOVA comparisons and P > 0.05 for regression results; see Table 2 for detailed results). Babies born to mothers with GD compared to those of healthy mothers tended to spend more time eating (4.26 ± 0.80 vs. 2.95 ± 0.35 h/day; F (1,18) = 3.58, P = 0.075) and, consequently, to eat more slowly (2.69 ± 0.66 vs. 4.46 ± 0.67 g/min; F (1,18) = 3.37, P = 0.083). The amount of food intake/day did not depend on GD diagnosis (P > 0.36 for *Group*).

Analyses of food intake according to BEBQ indicated that pregnancy-related weight gain modulated 'food responsiveness': neonates born to females with increased vs. reduced weight gain were observed to display greater food responsiveness (P = 0.021; F (2,16) = 2.85, P = 0.088 for *Group*; $R^2$ = 0.22, B = 0.80, SE = 0.36, β = 0.47, P = 0.038; Fig. 1). Exploratory analyses of trimester-wise weight gain in the subsample of responders indicated that weight gain in the second trimester explained 68 % of variance ($R^2$ = 0.68, B = 0.38, SE = 0.07, β = 0.82, P < 0.001) and first-trimester weight gain explained 26 % ($R^2$ = 0.26, B = 0.11, SE = 0.05, β = 0.51, P = 0.045). On a descriptive level, increased compared to reduced or recommended weight gain in the second and third trimesters was again associated with increased food responsiveness (P > 0.34 for all comparisons; Fig. 2A/B). There were no systematic effects of pcBMI and GD on food responsiveness (Table 2). Weight gain during pregnancy also affected 'satiety responsiveness' (F (2,16) = 3.68, P = 0.048 for *Group*): mothers with recommended weight gain rated their offspring to display increased 'satiety responsiveness' compared to mothers with increased (P = 0.027) and, respectively, with reduced weight gain (P = 0.046; $R^2$ = 0.56, B = −0.63, SE = 0.23, β = 0.44, P = 0.014; Fig. 1); there was no difference between neonates of mothers with increased and mothers with reduced weight gain (P > 0.74; Table 2). In trimester-wise analyses, this pattern was restricted to the third trimester, albeit on a non-significant level (P > 0.43 for all comparisons; Fig. 2B). Satiety responsiveness was not affected by pcBMI and GD (Table 2). 'Slowness in eating' according to BEBQ appeared to be more pronounced in babies of mothers with GD compared to healthy mothers (3.33 ± 0.35 vs. 2.66 ± 0.17; F (1,17) = 3.56, P = 0.077), whereas pcBMI and weight gain had no effect (Table 2). Supplementary analyses indicated that 'slowness in eating' of the baby was rated higher by mothers with increased vs. recommended weight gain in the second trimester (3.58 ± 0.26 vs. 2.33 ± 0.31; P = 0.013; F (2,12) = 5.82, P = 0.017 for *Group*; Fig. 2A); weight gain in the second trimester explained 38 % of variance

**Table 2**
Neonate eating behaviour.

| | pcBMI | | *Group* | Weight gain | | | *Group* | GD | | *Group* |
|---|---|---|---|---|---|---|---|---|---|---|
| | <25 | ≥25 | | Red. | Rec. | Inc. | | Yes | No | |
| **Food intake according to weighing protocols** | | | | | | | | | | |
| **Amount/day [g]** | 555 ± 52.9 | 597 ± 57.5 | F (1,18) = 0.08, P = 0.78 | 609 ± 78.6 | 525 ± 97.4 | 566 ± 40.0 | F (2,17) = 0.46, P = 0.64 | 533 ± 76.2 | 583 ± 47.4 | F (1,18) = 0.87, P = 0.36 |
| **Duration/day [h]** | 3.16 ± 0.40 | 3.66 ± 0.73 | F (1,18) = 0.57, P = 0.46 | 3.59 ± 0.60 | 2.82 ± 0.68 | 3.47 ± 0.62 | F (2,17) = 0.73, P = 0.50 | 4.26 ± 0.80 | 2.95 ± 0.35 | F (1,18) = 3.58, P = 0.075 |
| **Speed [g/min]** | 3.92 ± 0.62 | 4.02 ± 1.08 | F (1,18) = 0.01, P = 0.94 | 4.03 ± 0.99 | 4.12 ± 1.24 | 3.77 ± 0.76 | F (2,17) = 0.37, P = 0.70 | 2.69 ± 0.66 | 4.46 ± 0.67 | F (1,18) = 3.37, P = 0.083 |
| **Eating behaviour according to Baby Eating Behaviour Questionnaire (BEBQ)** | | | | | | | | | | |
| **Food response.** | 2.18 ± 0.17 | 2.57 ± 0.46 | F (1,17) = 0.82, P = 0.38 | 1.75 ± 0.22 | 2.24 ± 0.32 | 2.8 ± 0.32 | F (1,16) = 2.85, P = 0.088 | 2.77 ± 0.52 | 2.12 ± 0.15 | F (1,17) = 2.33, P = 0.15 |
| **Enjoyment of food** | 4.73 ± 0.10 | 4.67 ± 0.15 | F (1,17) = 0.09, P = 0.77 | 4.78 ± 0.11 | 4.67 ± 0.21 | 4.69 ± 0.13 | F (2,16) = 0.35, P = 0.71 | 4.61 ± 0.17 | 4.75 ± 0.10 | F (1,17) = 0.46, P = 0.51 |
| **Satiety response.** | 2.21 ± 0.18 | 2.02 ± 0.26 | F (1,17) = 0.30, P = 0.59 | 1.97 ± 0.30 | 2.67 ± 0.15 | 1.88 ± 0.20 | F (2,16) = 3.68, **P = 0.048** | 2.25 ± 0.26 | 2.10 ± 0.18 | F (1,17) = 0.21, P = 0.65 |
| **Slowness in eating** | 2.79 ± 0.16 | 3.00 ± 0.39 | F (1,17) = 0.30, P = 0.59 | 2.75 ± 0.10 | 2.54 ± 0.44 | 3.19 ± 0.23 | F (2,16) = 1.25, P = 0.31 | 3.33 ± 0.35 | 2.66 ± 0.16 | F (1,17) = 3.56, P = 0.08 |
| **General appetite** | 4.00 ± 0.19 | 4.33 ± 0.19 | F (1,16) = 1.08, P = 0.31 | 4.00 ± 0.27 | 4.00 ± 0.26 | 4.25 ± 0.27 | F (2,15) = 0.41, P = 0.67 | 4.40 ± 0.22 | 4.00 ± 0.18 | F (1,16) = 1.43, P = 0.25 |

Measures of food intake (mean ± SEM) were analyzed according to preconceptual (pc) BMI (<25 kg/m$^2$, n = 14; ≥25 kg/m$^2$, n = 7), weight gain during pregnancy according to IOM guidelines (reduced [Red.], n = 7; recommended [Rec.], n = 6; increased [Inc.], n = 8) and the diagnosis of gestational diabetes mellitus, GD (yes, n = 6; no, n = 15); Food response., food responsiveness, Satiety response., satiety responsiveness. *Group* columns indicate ANCOVA *Group* terms with the covariate neonate age at measurement.

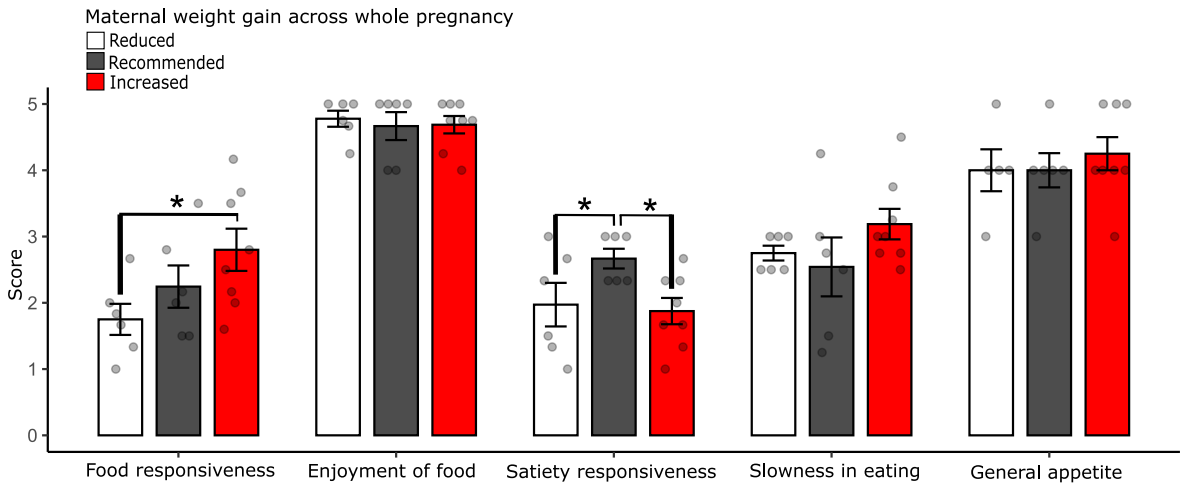


**Fig. 1.** Neonate eating behaviour according to categorized weight gain during the complete pregnancy. Results (mean ± SEM) of the Baby Eating Behaviour Questionnaire (BEBQ) completed by the mothers at the end of the eating protocol period, categorized according to weight gain during the whole pregnancy (IOM guidelines); *P < 0.05 (independent t-tests).

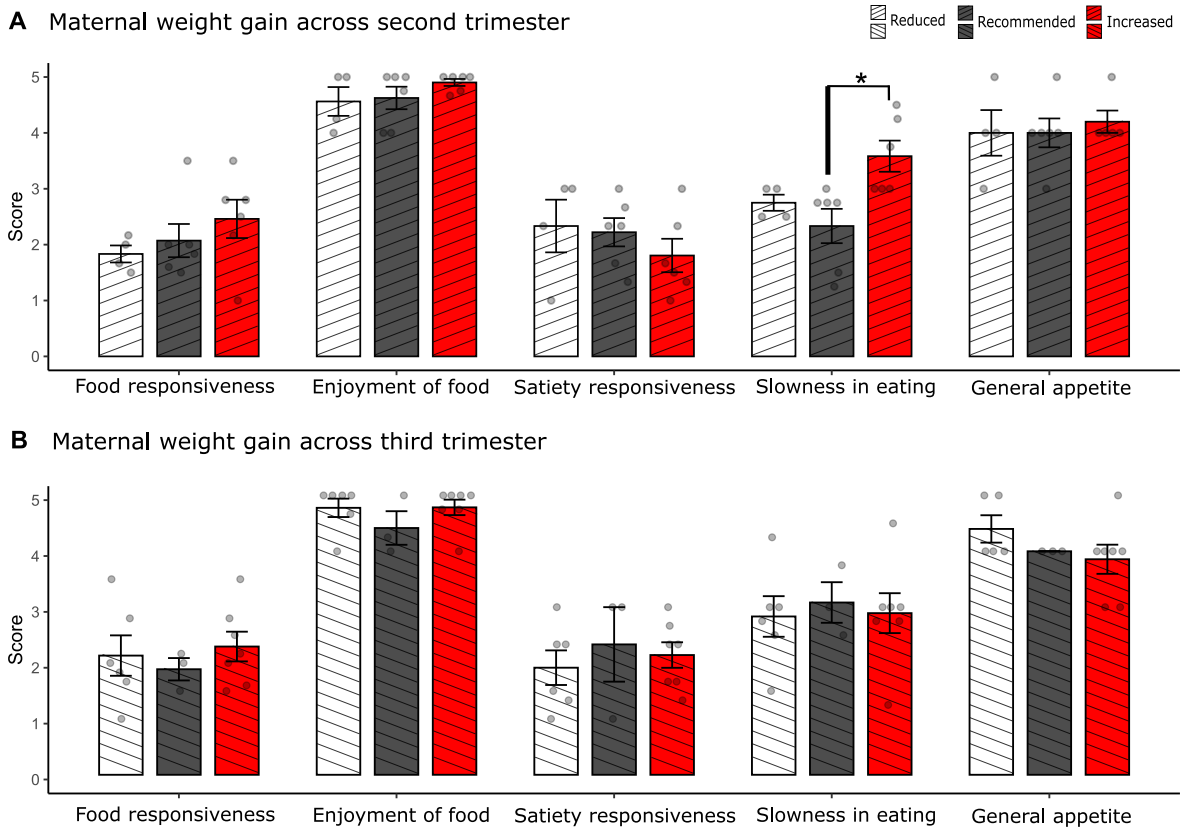


**Fig. 2.** Neonate eating behaviour according to categorized weight gain in the second and third pregnancy trimesters. Results (mean ± SEM) of the Baby Eating Behaviour Questionnaire (BEBQ) completed by the mothers at the end of the eating protocol period, categorized according to weight gain during (A) the second and (B) the third trimester of pregnancy (IOM guidelines); *P < 0.05 (independent *t*-test).

in the slowness score ($R^2 = 0.38$, B = 0.33, SE = 0.11, β = 62, P = 0.02). Across all neonates, the results on the slowness BEBQ scale were negatively related to feeding protocol-assessed ingestion speed (r = −0.52, P = 0.019). BEBQ 'enjoyment of food' and 'general appetite' were not affected by maternal metabolism. Likewise, other trimester-wise analyses of BEBQ results did not yield systematic results (all P > 0.16). Maternal age was positively related to 'general appetite' ($R^2 = 0.22$, B = 0.06, SE = 0.03, β = 0.46, P = 0.045).

## 4. Discussion

The majority of females in the US enter pregnancy with overweight or obesity (Centers for Disease Control and Prevention, 2016), and positive associations between maternal BMI and macrosomia are well known (Creanga et al., 2022). We investigated the relationship between maternal metabolism and food intake behaviour around two weeks after birth and found that weight gain during pregnancy rather than body weight around conception (Rising & Lifshitz, 2005) influences ingestive

behaviour at this very early age. Neonates born to females with increased compared to those with reduced weight gain displayed greater food responsiveness, while satiety responsiveness was most pronounced in neonates born to mothers with recommended weight gain. Increased weight gain in the second trimester was of particular relevance for rated food responsiveness. Moreover, babies of mothers with GD compared to healthy mothers were rated to eat somewhat more slowly, a finding supported by the assessment of actual eating speed. Slower eating of their babies was also reported by mothers with increased vs. those with recommend second-trimester weight gain. We included a relatively small, but well-characterized group of mother-infant dyads. While our results call for broader investigations in larger samples, they suggest a role of pregnancy-related weight gain rather than pcBMI for offspring eating behaviour in early infancy.

Neonatal food intake is very much under the control of the person in charge of feeding. Our finding that neonate food intake as assessed by the mother depends on maternal weight gain during pregnancy raises the question whether hunger and satiety regulation in the baby was directly affected or whether this pattern can be primarily or to some extent be attributed to altered dynamics in the mother-infant dyad. In this context, the ability of the mother to correctly interpret her infant's behavioural signals is critical and can be expected to be influenced by factors such as parity and breast vs. bottle-feeding (Hahn-Holbrook et al., 2021). For 14 of our participants, it was the respective first child, so that feeding the 2 weeks-old baby was a relatively new and potentially challenging experience. Rapid infant weight gain as a risk factor for obesity may be driven by maternal overfeeding because infant cues are misread (Hetherington, 2017). Mothers who are breastfeeding compared to bottle-feeding seem to be more sensitive to infant cues, and prolactin concentrations have been found to be positively correlated with maternal sensitivity (Hahn-Holbrook et al., 2021), suggesting that the majority of breast-feeding mothers in our study (n = 16) were at an advantage in estimating their baby's appetite. Finally, infant eating behaviour also displays a heritably component and might partially reflect maternal traits (Llewellyn & Wardle, 2015).

Regarding the basic validity of our results, the convergence of BEBQ ratings of slowness in eating and of the results of weighing protocols (as an estimate of actual eating speed) in children born to mothers with GD compared to those of healthy mothers suggest that our approach worked as planned, and that weighing the infant before and after eating (Kent et al., 2006; Stunkard et al., 2004) can be a useful tool to assess infant food intake at an early age. Still, keeping reliable weighing protocols across multiple days might be expected to present (first-time) parents with logistical hurdles in everyday life, which might lead to greater variance and measurement errors and could therefore explain why we did not find a clear-cut dependency of directly assessed food intake on maternal metabolism. In general, parental ratings of infant food intake necessarily imply a subjective component and are influenced by implicit or explicit ideas of age-appropriate eating behaviour. Such expectations might also have led to ceiling effects in BEBQ ratings of enjoyment of food and general appetite, the two scales that did not differentiate between any of the study groups.

The mechanisms underlying our observation that maternal weight gain modulates neonate food intake cannot be derived from our study. Many determinants of infant weight gain in the first year are still unknown and additional research into the development of obesity in children younger than 2 years is clearly necessary (Buvinger et al., 2017; Lumeng et al., 2015). Maternal glucose, as the principal substrate driving fetal growth, can cross the placental barrier, with hyperglycaemia causing fetal complications (Bowman et al., 2021). Preconceptual maternal weight and gestational weight gain are known to affect infant birth weight (Creanga et al., 2022; Ludwig & Currie, 2010), which may influence eating behaviour. For example, parents of infants born small for gestational age report more feeding difficulties in early life (Oliveira et al., 2015). However, we did not detect associations between birth weight and maternal pcBMI, pregnancy-related weight gain and GD, maybe because of the relatively small sample size of our study. Moreover, the females investigated in our study, including those diagnosed with GD, were under closer medical supervision than is usual during pregnancy, which might have benefitted their glucose regulation and prevented macrosomia in their offspring.

Developmental changes in metabolically relevant neurocircuitries in dependence of maternal input have been extensively investigated in animal studies (Reynolds et al., 2017). Specifically, changes in the maternal diet can alter neuroendocrine pathways in the hypothalamus that control energy balance and appetite regulation in the offspring (Penfold & Ozanne, 2015). In neonatal mice, insulin action in response to a maternal high fat diet impairs hypothalamic neurocircuit formation (Vogt et al., 2014). Maternal diet during pregnancy moreover alters reward signaling and food preferences in the offspring (Ong & Muhlhausler, 2011). Thus, fat intake is increased in the offspring of rat dams fed a "junk-food" diet in conjunction with alterations in mesolimbic reward pathways (Ong & Muhlhausler, 2011). Neuropeptidergic signals likewise play a role. Maternal BMI during pregnancy is for example positively associated with insulin and leptin concentrations but negatively with insulin-like growth factor binding protein-1 (IGFBP-1) concentrations in the first and third trimesters (Jansson et al., 2008). Leptin concentrations during pregnancy predict maternal weight gain (Stein et al., 1998), and leptin has been shown in animal models to play a key role in reprogramming metabolic outcomes in the offspring by regulating the maturation of metabolic signaling pathways (Vickers & Sloboda, 2012). Consequently, periods of hypo- or hyperleptinaemia accompanying decreased and, respectively, increased weight gain might lead to metabolic adaptations (Vickers & Sloboda, 2012) and potentially also modulate food and satiety responses in the neonate. In this respect, it might be speculated that recommended weight gain implies normoleptinaemia eventually ensuring a balanced response of the baby to food cues and food intake (Reynolds et al., 2017). This scenario would also be in accordance with our finding that weight gain in the second trimester of pregnancy, when diet-induced neuroendocrine changes become manifest, appear to exert the greatest influence on neonate food responsiveness. Hypermethylation of proopiomelanocortin persists in the offspring of high-fat diet-fed dams even when standard chow is given after weaning (Marco et al., 2014). Finally, higher maternal weight gain during pregnancy has been associated with potentially detrimental changes in the infant microbiota (Robinson et al., 2017).

In our study, infants were rated to eat more slowly by mothers with increased vs. those with recommended second-trimester weight gain. Likewise, babies born to mothers with GD tended to eat more slowly than babies born to healthy mothers. Breast milk of mothers with GD compared with that of healthy mothers has been found to contain less protein but the same total energy, lactose and fat content (Manerkar et al., 2020). The composition of breast milk differs from that of healthy mothers also in females with GD who received optimal treatment and showed good control of glucose values (Wen et al., 2019), as was the case in our study. Thus, while psychological effects of GD on maternal feeding behaviour and attitudes cannot be excluded, altered breastmilk composition in GD could be one explanation for slower feeding. However, in an animal model of GD, GD triggered larger and more frequent bouts of feeding and increased energy intake (Fahrenkrog et al., 2004; Manerkar et al., 2020).

Our study has some limitations, first of all the relatively small sample size, which limited statistical power and precluded more fine-grained analyses in terms of, e.g., neonate sex, parity and feeding type. Moreover, the quality and composition of the diet that our pregnant participants consumed was not assessed, mainly because we wanted to restrict the burden of participation at this transitional period of life. The long-term relevance of the observed changes in neonate eating behaviour is likewise unclear, although previous studies suggest that early-life changes in metabolic control determine subsequent body weight trajectories (Giles et al., 2015). Despite the scale of our study, however, we conclude that maternal weight gain during pregnancy is a critical

determinant of calorie intake in the neonate, highlighting the relevance of maternal metabolism for the emergence of food intake routines in the early days of life.

## CRediT authorship contribution statement

**Nina Goll:** Writing – review & editing, Writing – original draft, Visualization, Validation, Formal analysis. **Cornelia Wiechers:** Writing – review & editing, Supervision, Methodology, Investigation, Conceptualization. **Katharina Zinke:** Writing – review & editing, Formal analysis, Data curation. **Hubert Preissl:** Writing – review & editing, Project administration, Methodology, Funding acquisition, Conceptualization. **Andreas Fritsche:** Writing – review & editing, Investigation, Funding acquisition, Conceptualization. **Manfred Hallschmid:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Investigation, Funding acquisition, Formal analysis, Conceptualization.

## Ethical approval

All participants gave written informed consent to the study which was approved by the ethics committee of the University of Tübingen and conformed to the principles of the Declaration of Helsinki.

## Declaration of generative AI and AI-assisted technologies in the writing process

The authors did not use generative AI or AI-assisted technologies in the writing process.

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## Declaration of competing interest

The authors have nothing to declare.

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## Data availability

Data will be made available on request.

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