

## Brief Report

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# Maternal Dietary Fat Intake During Pregnancy Is Associated With Infant Temperament

**ABSTRACT:** Research with rodents and nonhuman primates suggests that maternal prenatal dietary fat intake is associated with offspring behavioral functioning indicative of risk for psychopathology. The extent to which these findings extend to humans remains unknown. The current study administered the Automated Self-Administered 24 hr Dietary Recall Questionnaire three times in pregnancy ( $n = 48$ ) to examine women's dietary fat intake in relation to infant temperament assessed using the Infant Behavior Questionnaire at 4-months old. The amount of saturated fat that the mother consumed was considered as a moderator of the association between total fat intake and child temperament. Results from a series of multiple linear regressions indicate that greater total fat intake was associated with poorer infant regulation and lower surgency. However, this second effect was moderated by maternal saturated fat intake, such that total fat intake was only related to infant surgency when mothers consumed above the daily recommended allowance of saturated fat. Under conditions of high total fat and high saturated fat, infants were rated as lower on surgency; under conditions of low total fat yet high saturated fat, infants were rated as higher on surgency. There were no associations between maternal prenatal fat intake and infant negative reactivity. These findings provide preliminary evidence that pregnant women's dietary fat intake is associated with infants' behavioral development, though future research is needed to address this report's limitations: a relatively small sample size, the use of self-report measures, and a lack of consideration of maternal and infant postnatal diet. © 2015 Wiley Periodicals, Inc. *Dev Psychobiol* 58:528–535, 2016.

## INTRODUCTION

Despite plateauing rates in recent years (Ogden, Carroll, Kit, & Flegal, 2014), the obesity epidemic in the US has increased substantially over the past several decades (Mokdad et al., 1999; Wang, McPherson, Marsh, Gortmaker, & Brown, 2011). An estimated 65% of women over the age of 20 were obese or overweight in 2009–2012 (National Center for Health Statistics, 2014) and about one third of pregnant women in the

US are obese (King, 2006). This rise in obesity is a consequence, in part, of unhealthy eating habits that include consuming diets high in fat. Adequate amounts of dietary fat are important for healthy functioning (e.g., fat is necessary for the absorption of certain vitamins); however, in excess, dietary fat can exert negative effects. This may be particularly true for pregnant women, as dietary fat intake during pregnancy influences both maternal health (Meyer, Kushi, Jacobs, & Folsom, 2001; Oh, Hu, Manson, Stampfer, & Willett, 2005) and fetal growth and development (Georgieff, Brunette, & Tran, 2015; Wainwright, 1992).

Based largely on experiments with rodents and nonhuman primates, a growing body of research demonstrates that higher prenatal fat intake predicts poorly adapted behavioral functioning in the next generation. Offspring of animals fed diets that are high in total fat (TF) as well as saturated fat (SF) display

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behaviors indicative of anxiety or depression and have been shown to exhibit greater stress responses (Bilbo & Tsang, 2010; D'Asti et al., 2010; Peleg-Raibstein, Luca, & Wolfrum, 2012). For example, Sullivan et al. (2010), in their study of Japanese macaques, found that exposure to a high fat versus control diet was associated with greater anxiety-like behaviors in young offspring. These researchers—and others (e.g., Bilbo & Tsang, 2010)—suggest that inflammatory cytokines secondary to dietary fat cross the placenta and change the *in utero* environment, thereby influencing the development of areas of the fetal brain that are related to behavioral and psychological functioning (e.g., the serotonin system; Sullivan et al., 2010). This research has yet to be extended to humans.

Although these animal studies give significant insight into the association between dietary fat and offspring behavioral development, several questions remain. First, as indicated, it is not clear whether these findings extend to human populations. Though there are many physiological similarities between humans and nonhuman primates (Phillips et al., 2014), findings from animal models often are not replicated or are shown to vary when attempted with humans (Hackam & Redelmeier, 2006). Second, the high fat diets typically used in animal studies are frequently not ecologically valid; rather, they include a higher percentage of fat than the average pregnant woman consumes. Last, few of the animal studies on prenatal dietary fat intake have considered the proportion of SF relative to TF. Rather, they have compared a diet high in *both* TF and SF versus a control diet. Testing, in an exploratory fashion, whether varying combinations of SF and TF are associated with different offspring adaptations is an important extension of this animal research.

To characterize infant behavioral outcomes in relation to prenatal exposure to maternal fat intake, the current study utilized measures of infant temperament. Infant temperament—defined as individual differences in reactivity and self-regulation that are believed to have a biological basis (Rothbart & Derryberry, 1981)—was selected because dimensions of temperament (a) emerge early in life, can be measured reliably during early infancy, and have been shown to exhibit relative stability across the lifespan (Gartstein & Rothbart, 2003; Rothbart, 1991); (b) have previously been shown to be influenced by alterations in the *in utero* environment (Davis et al., 2007; Werner et al., 2007), though not in relation to prenatal dietary fat; and (c) have been shown to be related to risk of psychopathology (Nigg, 2006; Rothbart, Posner, & Hershey, 1995), making them a promising analogue to the behaviors observed in the animal studies. For example, young children who have difficulty regulating

their behavior and emotions have been shown to be at greater risk for later psychological difficulties including anxiety and depression (Cicchetti, Ackerman, & Izard, 1995). Similarly, high negative reactivity is a risk factor for later anxiety (Muris & Ollendick, 2005). Temperamental surgency at either end of the spectrum also reflects risk for psychopathology: children low on surgency (i.e., introversion) are at risk for depression (Clark, Watson, & Mineka, 1994) while high surgency (i.e., extraversion or exuberance) is predictive of psychological difficulties including lower social competence and increased internalizing and externalizing behaviors (Dollar & Stifter, 2012; Stifter, Putnam, & Jahromi, 2008).

The overall goal of the current study was to examine if prenatal dietary fat intake was associated with these three dimensions of infant temperament. First, we tested the following research question: (1) *Is maternal prenatal dietary fat intake associated with infant temperament at 4 months of age?* Second, as an exploratory aim, we examined: (2) *Is the effect of TF on temperament moderated by the amount of SF the mother consumed?*

Past research has identified correlates of maternal dietary intake and infant temperament that may be important to consider when testing these research questions. Specifically, family income (Drewnowski & Specter, 2004) and perinatal psychological distress (Macht, 2008) have been associated with dietary choices as well as infant behavioral development; maternal diet has been associated with fetal growth (Godfrey, Robinson, Barker, Osmond, & Cox, 1996), and thus infant birthweight and gestational age at birth may be important covariates; maternal prenatal depression (Davis et al., 2007), age (Werner et al., 2013), and child sex (Gartstein & Rothbart, 2003) have been associated with infant temperament. Though animal studies that use isocaloric diets that differ only in the amount of fat have found that prenatal fat intake is associated with offspring outcomes (e.g., Franco et al., 2012), we also considered whether maternal caloric intake or gestational weight gain should be included as a covariate. To strengthen our ability to interpret differences that we observe as the results of prenatal fat intake and not about adiposity prior to conception, we controlled for maternal pre-pregnancy body mass index (BMI) in all analyses.

## METHODS

### Participants

Participants in the current study were part of an ongoing longitudinal study examining epigenetic modifications associated with maternal prenatal stress ( $N = 129$ ). Pregnant women ages 18–45 were recruited through Columbia University

Medical Center. All had a healthy pregnancy at the time of recruitment. Participants were excluded if they acknowledged smoking tobacco or use of recreational drugs, or lacked fluency in English. Participants also were excluded on the basis of frequent use of nitrates, steroids, beta blockers, triptans, and psychiatric medications. Recruitment procedures allowed for participant enrollment in either the 1st or 2nd trimester of pregnancy.

The overarching study protocol did not involve postnatal data collection sessions, however, beginning in the second year of the study, families were invited to participate in an optional data collection session when the child was 4 months old. All families enrolled in the study were eligible, though only 51% ( $n=66$ ) did. The reasons that families did not participate were inability to reach the participant ( $n=27$ ), withdrawal from the study prior to birth ( $n=11$ ), no show for the 4-month appointment ( $n=10$ ), because the family had moved out of the area ( $n=3$ ), and inability of the study coordinator to offer an appointment ( $n=13$ ). Assessment of infant temperament was not included in the first 15 4-month sessions. Participants missing infant temperament data and those who did not contribute any nutritional data during pregnancy ( $n=2$ ) were not included in the current analyses. These inclusion criteria yielded a final analytic sample of  $n=48$ . Women included in the current analyses were on average 30.05 years ( $SD=6.20$ ) at enrollment; 62.50% self-identified as Latina. The median household income was \$51,000–\$100,000 and 50% of the fetuses were female. Women included in the current analyses were not significantly different from the complete sample on any of these demographic variables. Because of limited study funding for these postnatal sessions, information about postnatal child and maternal dietary intake was not collected.

### Procedures

Participants completed laboratory sessions when they were 13–16, 24–27, and 34–37 weeks pregnant. At each session, women completed self-report and clinician-administered measures. They also reported on household demographic variables and their medical history. Medical records were culled for relevant medical information. All procedures were approved by the Institutional Review Board of the New York State Psychiatric Institute.

### Measures

**Maternal Dietary Intake.** At the 13–16, 24–27, and 34–37 week assessment sessions, nutrition information was acquired via the Automated Self-Administered 24 hr Dietary Recall (ASA24). The ASA24 is an internet-based questionnaire provided by the National Cancer Institute (Subar et al., 2012) that asks participants to recall food intake over the preceding 24 hr using detailed probes and portion-size food images. The ASA24 estimates TF, SF, and the total calories consumed using three databases: the USDA's Food and Nutrient Database for Dietary Surveys, MyPyramid Equivalents Database (MPED), and the Center for Nutrition Policy and Promotion's MPED Addendum. To take advantage of

the current study's multiple measures of maternal dietary intake—and to more comprehensively capture women's dietary intake across pregnancy—we averaged women's reports from the three assessments to create composite variables of the average TF, SF, and calories consumed across pregnancy. Research indicates that maternal TF and SF intake does not change significantly across the three trimesters of pregnancy (Al, Badart-Smook, Von Houwelingen, Hasaart, & Hornstra, 1996).

**Infant Temperament.** When children were 4 months old, mothers completed the Revised Infant Behavior Questionnaire (IBQ-R; Gartstein & Rothbart, 2003), a 191-item measure that assesses infant temperament. Mothers are asked to rate on a 7-point Likert-type scale (where 1 = *never* and 7 = *always*) how often their child completed various behaviors; querying about specific, concrete behaviors is meant to reduce the influence of maternal bias. Following standard scoring procedure, this scale's 14 subscales were composited to create three factors: Regulation (the mean of Low Intensity Pleasure, Cuddliness, Duration of Orienting, and Soothability;  $\alpha = .90$  for this subsample), Surgency (the mean of Approach, Vocal Reactivity, High Intensity Pleasure, Smiling and Laughter, Activity Level, and Perceptual Sensitivity;  $\alpha = .93$ ), and Negative Affectivity (the mean of Sadness, Distress to Limitations, Fear, and reverse scored Falling Reactivity;  $\alpha = .77$ ); these three broad factors were used in the current analyses. This measure has been shown to have good reliability and validity (Parade & Leerkes, 2008).

**Demographic Covariates.** At enrollment, women reported their age (in years) and ethnicity (0 = *Not Latina*, 1 = *Latina*). At the 34–37 week assessment women reported the family's annual income (1 = *\$0–\$15,000* and 6 = *Above \$250,000*). The child's sex (0 = *Female*, 1 = *Male*) and gestational age (weeks) and weight (grams) at birth were culled from medical records.

**Maternal BMI and Gestational Weight Gain.** At enrollment, a trained research assistant measured participant height and women reported on their pre-pregnancy weight in pounds. Using this information, standard calculation procedures were used to calculate maternal pre-pregnancy BMI (Garrow & Webster, 1984). Women also self-reported their weight at the 3rd trimester session, which—in conjunction with self-reported pre-pregnancy weight—was used to calculate maternal gestational weight gain.

**Maternal Depressive Symptoms.** When women were 34–37 weeks pregnant they underwent the clinician administered Hamilton Rating Scales for Depression (HRSD). This 15–20 min measure indexes depressive symptoms over the previous 2 weeks (Williams, 1988). HRSD values of 8–13 indicate mild depression, 14–18 indicate moderate depression, and 19–22 indicate severe depression (Hamilton, 1960). The validity and reliability of the HRSD is well established (Ramos-Brieva & Cordero-Villafafila, 1988; Trajković et al., 2011).

### Analytic Plan

Our research questions were tested using a series of multiple linear regressions. To test our first hypothesis, three multiple linear regressions were estimated—one for each of the dimensions of temperament. Specifically, maternal TF intake and pre-pregnancy BMI were used as predictors of infant regulation, surgency, and negative affectivity. Covariates shown to be significantly related to fat intake, infant temperament, or pre-pregnancy BMI during bivariate analyses (described below) were also included in these models. To test our exploratory aim, the total amount of SF and an interaction term that captures the interaction between TF and SF was entered into the models. That is, three additional models were estimated, each of which used the mother's TF intake, SF intake, pre-pregnancy BMI, and the interaction of TF and SF intake to predict infant regulation, surgency, and negative affectivity. All continuous variables were mean-centered prior to model estimation. Significant interaction effects were probed following Preacher, Curran, and Bauer (2006).

## RESULTS

### Descriptive Statistics

Descriptive statistics and bivariate correlations among all study variables appear in Table 1. Women, on average, had a pre-pregnancy BMI of 26.89 (SD = 6.13; range = 16.73–43.20), which is considered overweight by the standards outlined by the Centers for Disease Control and Prevention (2011). Women ate on average 70.22 g of TF per Day (SD = 29.70 g; range = 19.82–

160.29 g) and 23.36 g of SF per Day (SD = 9.86 g; range = 7.28–46.79 g); these values are within the daily recommended amount of TF (61.11–85.56 g) and SF (less than 24 g) for someone consuming 2,200 calories per day, the recommendation for women in their third trimester of pregnancy (US Department of Agriculture and US Department of Health and Human Services, 2010).

### Covariate Selection

Prior to model building, we examined whether a number of child-, mother-, and family-level variables were associated with child temperament, and thus should be included in the regression analyses. Specifically, we examined whether infant regulation, surgency, and negative affectivity were significantly correlated with infant weight and gestational age at birth, child sex, maternal age, maternal prenatal depressive symptoms, family income, average caloric intake, and maternal gestational weight gain. None of these variables were significantly associated with infant temperament, and thus were not included in the regression models.

### Main Effects Model

A series of multiple regressions (Table 2) show that greater amounts of TF intake were associated with less infant regulation ( $\beta = -.38, p < .05$ ) and surgency ( $\beta = -.53, p < .01$ ). These effects were observed above and beyond the influence of maternal pre-pregnancy

**Table 1. Descriptive Statistics and Bivariate Correlations Among Study Variables ( $N = 48$ )**

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Total fat	—												
2. Saturated fat	.90**	—											
3. Total calories	.86**	.75**	—										
4. Pre-pregnancy BMI	-.09	-.03	-.26	—									
5. Infant regulation	-.37*	-.28	-.31*	-.10	—								
6. Infant surgency	-.52**	-.38**	-.41**	-.04	.60**	—							
7. Infant negative reactivity	-.02	-.06	-.01	-.21	-.05	-.04	—						
8. Child sex <sup>a</sup>	.03	-.01	-.07	-.01	-.25	-.23	-.07	—					
9. Maternal age	.18	.14	-.03	.07	-.26	-.22	-.10	.19	—				
10. Maternal prenatal depression	.17	.14	.28	.01	-.09	-.06	-.02	-.20	-.10	—			
11. Family income	-.01	.02	-.15	-.05	-.21	-.15	.13	.15	.45**	-.10	—		
12. Infant birthweight	.44**	.49**	.27	.06	-.22	.02	.17	-.09	.13	.09	.15	—	
13. Gestational age at birth	-.19	-.27	-.13	.08	.10	.17	-.16	-.15	.12	.06	-.03	.07	—
Mean	70.22	23.36	1,922.11	26.89	4.36	4.01	3.01	—	30.05	7.40	3.54	3,331.74	38.41
Standard deviation	29.70	9.86	559.96	6.13	0.66	0.71	0.46	—	6.20	4.75	1.41	495.19	5.86
Minimum	19.82	7.28	946.63	16.73	3.24	1.66	2	—	20.26	0	1	1,995	0
Maximum	160.29	46.79	3,352.93	43.20	6.07	5.69	4.41	—	43.08	22	6	4,470	41.14

Note: BMI = body mass index; <sup>a</sup>0, male; 1, female. In contrast to those used in the regression models, the values presented here are not mean centered.

\* $p < .05$ , \*\* $p < .01$ .

**Table 2. Results of Multiple Regression Analyses Used to Predict Infant Temperament at Four Months of Age**

Variable	Regulation				Surgency				Negative Reactivity			
	Model 1		Model 2		Model 1		Model 2		Model 1		Model 2	
	$\beta$	SE	$\beta$	SE	$\beta$	SE	$\beta$	SE	$\beta$	SE	$\beta$	SE
Total fat	-.38*	.00	-.60 <sup>+</sup>	.01	-.53**	.00	-.78**	.01	-.04	.07	.12	.01
Pre-pregnancy BMI	-.15	.02	-.14	.02	-.13	.02	-.06	.02	-.20	.00	-.18	.01
Saturated fat	—	—	.28	.02	—	—	.43	.02	—	—	-.15	.01
Total fat saturated fat interaction*	—	—	-.07	.00	—	—	-.30*	.00	—	—	-.06	.00
R <sup>2</sup>	.16		.18		.28		.39		.04		.05	
Model F	F (2,42) = 1.58*		F (4,40) = 2.12		F (2,42) = 8.32**		F (4,40) = 6.27**		F (2,42) = .88		F (4,40) = .49	

Note: All independent variables were mean centered before model estimation.

\* $p < .05$ , \*\* $p < .01$ , <sup>+</sup> $p < .01$ .

BMI. TF was not significantly associated with infant negative reactivity ( $\beta = -.04$ ,  $p = .78$ ).

### Interaction Models

To test our exploratory aim, two additional variables were added to the models: a variable that captures the amount of SF consumed and an interaction term of TF and SF intake. Results indicate that the amount of SF consumed moderated the effect of TF on infant surgency ( $p < .05$ ); SF did not moderate the effect of TF on infant regulation ( $p = .68$ ), or infant negative reactivity ( $p = .76$ ).

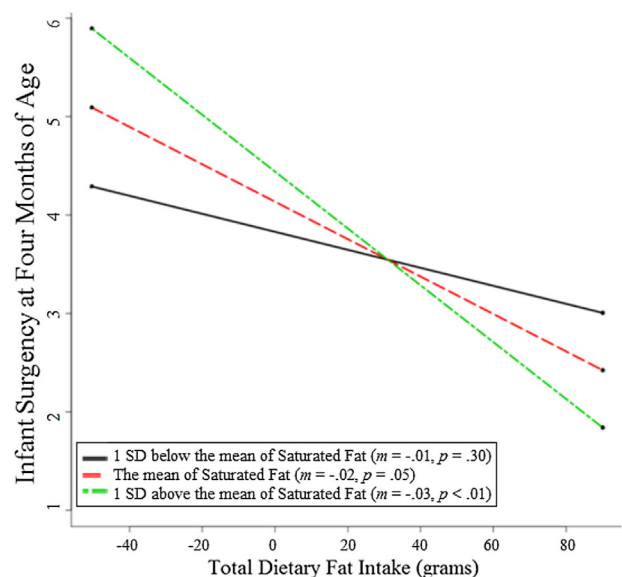
As can be seen in Figure 1, for women who consumed low ( $m = -.01$ ,  $p = .30$ ) and average ( $m = -.02$ ,  $p = .05$ ) levels of SF, there was no significant association between TF and infant surgency. However, for women who consumed high levels of SF, there was a significant negative effect of TF on infant surgency,  $m = -.03$ ,  $p < .01$ . For women who consumed low TF yet high SF, infants were rated higher on surgency (i.e., greater extraversion); under conditions of high TF and high SF, infants exhibited lower levels of surgency (i.e., greater introversion). The region of significance tests provided by Preacher et al. (2006) online calculator indicates that the association between TF and surgency was significant only for women who consumed more than 23.72 g of SF, a value that is very close to the daily recommended level of SF during the 3rd trimester of pregnancy, 24 g.

### DISCUSSION

Using data from a small sample of pregnant women consuming levels of TF and SF that are typical of pregnant American women, the current study is the first to use data from humans and to find associations between maternal prenatal dietary fat intake and

offspring behavioral functioning. In the context of high obesity rates in the US, in part a result of high fat diets, these results indicate that the amount of dietary fat that women consume during pregnancy may be affecting their children's neurobehavioral development.

This study found that maternal TF intake was associated with multiple dimensions of infant temperament at four months old. Specifically, women's TF intake was inversely associated with infant regulation. Higher TF intake also was associated with lower infant



**FIGURE 1** Visual depiction of the interaction between total dietary fat intake and saturated fat intake. Note: SD = standard deviation. Although saturated fat was treated continuously in all analyses, we selected three values of saturated fat to create this visualization: (1) one standard deviation below the mean of saturated fat; (2) the mean of saturated fat; and (3) one standard deviation above the mean of saturated fat. All independent variables were mean centered before model estimation, however, the values that were plotted are equivalent to 13.50, 23.36, and 33.22 g of saturated fat, respectively.

surgency, however, maternal SF levels moderated this association. Probing this interaction produced two findings: (1) the negative association between TF and infant surgency was only significant for mothers who consumed at or above the daily recommended limit for SF in 3rd trimester of pregnancy; and (2) the direction of this association depended on women's TF consumption: under conditions of high TF, high SF was associated with low levels of surgency (i.e., a child that is inhibited, introverted); under conditions of low TF yet high SF, infants were high on surgency (i.e., impulsive, active, enjoys highly stimulating activities). Both very high and very low surgency may place children at risk for later psychopathology and other psychosocial difficulties (Polak-Toste & Gunnar, 2006).

Contrary to expectation, dietary fat intake was not significantly associated with infant negative reactivity, a dimension of temperament that has been consistently associated with internalizing symptomatology (Degnan & Fox, 2007; Kagan, Snidman, Zentner, & Peterson, 1999), and therefore may be most similar to the anxiety-like behaviors reported in animal studies. This lack of an effect may reflect differences in the way that fat influences animal and human development (or differences in the behavioral manifestations of similar effects), or they may simply be due to lack of power. Given the relatively small sample size used in this preliminary study, we hesitate to draw further conclusions about the nature of the association between maternal dietary fat intake and infant negative reactivity.

Although this study's findings further our understanding of the impact of dietary fat on offspring behavioral development, the mechanisms through which this occurs remain underexplored, particularly as they relate to our finding that, among women eating above average amounts of SF, low levels of TF are associated with greater surgency and high levels of TF are associated with less surgency. Elevated levels of inflammatory cytokines secondary to dietary fat and adipose tissue are thought to influence the *in utero* environment, thereby influencing offspring behavioral functioning. Given that diets high in SF versus those high in other fats have been associated with increased neuropeptide-y expression (Wang, Storlien, & Huang, 2002) and that neuropeptide-y binds to Leptin, it is possible that differences in the ratio of SF to non-SF (and thus the ratio of neuropeptide-y to Leptin) results in differences in the intrauterine milieu, which result in different fetal adaptations.

The current study had a number of strengths. Most notably, this is the first study to bring to human research what has been identified in animal models. Given that many associations established in animal studies do not replicate or prove to operate differently

in human populations (Hackam & Redelmeier, 2006), these findings point to the potential for translational research on the effects of prenatal exposure to maternal diet. Another strength of the study is the number of potential covariates considered for inclusion in our models. Demonstrating that these concomitant psychosocial, dietary, and health-related variables do not account for the associations between dietary fat intake and infant temperament allows us to more confidently attribute our results to maternal dietary fat. Last, the current study utilized multiple versus single assessments of dietary fat, collected at various stages during pregnancy.

Despite these strengths, this study also had a number of limitations. Most obviously, the sample used in the current analyses was not large and may have lacked sufficient power to detect some effects. Additionally, our measures of maternal dietary intake and child temperament both relied on maternal report, though they were not completed at the same data collection sessions. Nevertheless, we may have a common reporter bias; replication of these findings using multiple reporters or more objective measurement of diet or temperament is needed. In particular, the inclusion of more direct measures of maternal fat intake (e.g., biochemical analysis of erythrocyte plasma membrane fluidity) and infant temperament (e.g., behavioral or biological measures of infant reactivity and regulation) may provide additional insight into the nature of these associations. To take full advantage of our multiple assessments of maternal dietary fat intake (and because women's daily TF and SF consumption typically does not change across pregnancy; Al et al., 1996), we averaged mothers' report of her daily fat intake at the three assessment sessions. This approach allowed us to more comprehensively capture dietary intake across pregnancy, however, it did not allow us to examine whether the timing or chronicity of the fat intake moderated the associations reported here. Furthermore, we did not consider specific types of unsaturated fat in these analyses. Last, the current study did not collect measures of postnatal dietary fat intake, and thus we are unable to eliminate the possibility that postnatal nutrition may also play a role in these associations. Future research should examine the extent to which maternal postnatal diet and infant feeding practices (e.g., breastfeeding duration) influence infant temperament.

## NOTES

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## REFERENCES

- Al, M. D., Badart-Smook, A., Von Houwelingen, A. C., Hasaart, T. H., & Hornstra, G. (1996). Fat intake of women during normal pregnancy: Relationship with maternal and neonatal essential fatty acid status. *Journal of the American College of Nutrition*, 15(1), 49–55.
- Bilbo, S. D., & Tsang, V. (2010). Enduring consequences of maternal obesity for brain inflammation and behavior of offspring. *The FASEB Journal*, 24(6), 2104–2115. doi: 10.1096/fj.09-144014
- Centers for Disease Control and Prevention. (2011). PNNS Health Indicators. Retrieved from [http://www.cdc.gov/pednss/what\\_is/pnns\\_health\\_indicators.htm#Maternal%20Health%20Indicator](http://www.cdc.gov/pednss/what_is/pnns_health_indicators.htm#Maternal%20Health%20Indicator)
- Cicchetti, D., Ackerman, B. P., & Izard, C. E. (1995). Emotions and emotion regulation in developmental psychopathology. *Development and Psychopathology*, 7(01), 1–10.
- Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, 103(1), 103. doi: 10.1037/0021-843X.103.1.103
- D'Asti, E., Long, H., Tremblay-Mercier, J., Grajzer, M., Cunnane, S. C., Di Marzo, V., & Walker, C.-D. (2010). Maternal dietary fat determines metabolic profile and the magnitude of endocannabinoid inhibition of the stress response in neonatal rat offspring. *Endocrinology*, 151(4), 1685–1694. doi: 10.1210/en.2009-1092
- Davis, E. P., Glynn, L. M., Schetter, C. D., Hobel, C., Chicz-Demet, A., & Sandman, C. A. (2007). Prenatal exposure to maternal depression and cortisol influences infant temperament. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46(6), 737–746. doi: 10.1097/chi.0b013e318047b775
- Degnan, K. A., & Fox, N. A. (2007). Behavioral inhibition and anxiety disorders: Multiple levels of a resilience process. *Development and Psychopathology*, 19(03), 729–746. doi: 10.1017/S0954579407000363
- Dollar, J. M., & Stifter, C. A. (2012). Temperamental surgency and emotion regulation as predictors of childhood social competence. *Journal of Experimental Child Psychology*, 112(2), 178–194. doi: 10.1016/j.jecp.2012.02.004
- Drewnowski, A., & Specter, S. (2004). Poverty and obesity: The role of energy density and energy costs. *The American Journal of Clinical Nutrition*, 79(1), 6–16.
- Franco, J., Fernandes, T., Rocha, C., Calvino, C., Pazos-Moura, C., Lisboa, P., . . . Trenzoli, I. (2012). Maternal high-fat diet induces obesity and adrenal and thyroid dysfunction in male rat offspring at weaning. *The Journal of Physiology*, 590(21), 5503–5518. doi: 10.1113/jphysiol.2012.240655
- Garrow, J. S., & Webster, J. (1984). Quetelet's index (W/H<sup>2</sup>) as a measure of fatness. *International Journal of Obesity*, 9(2), 147–153.
- Gartstein, M. A., & Rothbart, M. K. (2003). Studying infant temperament via the revised infant behavior questionnaire. *Infant Behavior and Development*, 26(1), 64–86. doi: 10.1016/S0163-6383(02)00169-8
- Georgieff, M. K., Brunette, K. E., & Tran, P. V. (2015). Early life nutrition and neural plasticity. *Development and Psychopathology*, 27(02), 411–423. doi: 10.1017/S0954579415000061
- Godfrey, K., Robinson, S., Barker, D., Osmond, C., & Cox, V. (1996). Maternal nutrition in early and late pregnancy in relation to placental and fetal growth. *BMJ*, 312(7028), 410–414.
- Hackam, D. G., & Redelmeier, D. A. (2006). Translation of research evidence from animals to humans. *JAMA*, 296(14), 1727–1732. doi: 10.1001/jama.296.14.1731
- Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry*, 23(1), 56–62.
- Kagan, J., Snidman, N., Zentner, M., & Peterson, E. (1999). Infant temperament and anxious symptoms in school age children. *Development and Psychopathology*, 11(02), 209–224.
- King, J. C. (2006). Maternal obesity, metabolism, and pregnancy outcomes. *Annual Review of Nutrition*, 26, 271–291. doi: 10.1146/annurev.nutr.24.012003.132249
- Macht, M. (2008). How emotions affect eating: A five-way model. *Appetite*, 50(1), 1–11. doi: 10.1016/j.appet.2007.07.002
- Meyer, K. A., Kushi, L. H., Jacobs, D. R., & Folsom, A. R. (2001). Dietary fat and incidence of type 2 diabetes in older Iowa women. *Diabetes Care*, 24(9), 1528–1535.
- Mokdad, A. H., Serdula, M. K., Dietz, W. H., Bowman, B. A., Marks, J. S., & Koplan, J. P. (1999). The spread of the obesity epidemic in the United States, 1991–1998. *JAMA*, 282(16), 1519–1522.
- Muris, P., & Ollendick, T. H. (2005). The role of temperament in the etiology of child psychopathology. *Clinical Child and Family Psychology Review*, 8(4), 271–289. doi: 10.1007/s10567-005-8809-y
- National Center for Health Statistics. (2014). Healthy weight, overweight, and obesity among adults aged 20 and over, by selected characteristics: United States, selected years 1988–1994 through 2009–2012. Retrieved from <http://www.cdc.gov/nchs/data/abus/2014/064.pdf>
- Nigg, J. T. (2006). Temperament and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 47(3–4), 395–422.
- Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2014). Prevalence of childhood and adult obesity in the United States, 2011–2012. *JAMA*, 311(8), 806–814.
- Oh, K., Hu, F. B., Manson, J. E., Stampfer, M. J., & Willett, W. C. (2005). Dietary fat intake and risk of coronary heart disease in women: 20 years of follow-up of the nurses' health study. *American Journal of Epidemiology*, 161(7), 672–679. doi: 10.1093/aje/kwi085
- Parade, S. H., & Leerkes, E. M. (2008). The reliability and validity of the infant behavior questionnaire-revised. *Infant Behavior and Development*, 31(4), 637–646. doi: 10.1016/j.infbeh.2008.07.009
- Peleg-Raibstein, D., Luca, E., & Wolfrum, C. (2012). Maternal high-fat diet in mice programs emotional behavior in adulthood. *Behavioural Brain Research*, 233(2), 398–404. doi: 10.1016/j.bbr.2012.05.027
- Phillips, K. A., Bales, K. L., Capitanio, J. P., Conley, A., Czoty, P. W., 't Hart, B. A., . . . Voytko, M. L. (2014).

- Why primate models matter. *American Journal of Primatology*, 76(9), 801–827. doi: 10.1002/ajp.22281
- Polak-Toste, C. P., & Gunnar, M. R. (2006). Temperamental exuberance: Correlates and consequences. In P. J. Marshall & N. A. Fox (Eds.), *The development of social engagement: Neurobiological perspectives* (pp. 19–45). New York, NY: Oxford University Press.
- Preacher, K. J., Curran, P. J., & Bauer, D. J. (2006). Computational tools for probing interactions in multiple linear regression, multilevel modeling, and latent curve analysis. *Journal of Educational and Behavioral Statistics*, 31(4), 437–448. doi: 10.3102/10769986031004437
- Ramos-Brieva, J., & Cordero-Villafafila, A. (1988). A new validation of the hamilton rating scale for depression. *Journal of Psychiatric Research*, 22(1), 21–28.
- Rothbart, M. K. (1991). Temperament: A developmental framework. In J. Strelau & A. Angleitner (Eds.), *Explorations in temperament* (pp. 61–74). New York, NY: Springer.
- Rothbart, M. K., & Derryberry, D. (1981). Development of individual differences in temperament. In M. E. Lamb & A. L. Brown (Eds.), *Advances in developmental psychology* (Vol. 1). Hillsdale, NJ: Edlbaum.
- Rothbart, M. K., Posner, M. I., & Hershey, K. L. (1995). Temperament, attention, and developmental psychopathology. In D. J. Cohen (Ed.), *Developmental psychopathology* (Vol. 2, pp. 465–501). New York, NY: Wiley.
- Stifter, C. A., Putnam, S., & Jahromi, L. (2008). Exuberant and inhibited toddlers: Stability of temperament and risk for problem behavior. *Development and Psychopathology*, 20(02), 401–421. doi: 10.1017/S0954579408000199
- Subar, A. F., Kirkpatrick, S. I., Mittl, B., Zimmerman, T. P., Thompson, F. E., Bingley, C., . . . Potischman, N. (2012). The automated self-administered 24-hour dietary recall (ASA24): A resource for researchers, clinicians, and educators from the National Cancer Institute. *Journal of the Academy of Nutrition and Dietetics*, 112(8), 1134–1137. doi: 10.1016/j.jand.2012.04.016
- Sullivan, E. L., Grayson, B., Takahashi, D., Robertson, N., Maier, A., Bethea, C. L., . . . Grove, K. L. (2010). Chronic consumption of a high-fat diet during pregnancy causes perturbations in the serotonergic system and increased anxiety-like behavior in nonhuman primate offspring. *The Journal of Neuroscience*, 30(10), 3826–3830. doi: 10.1523/JNEUROSCI.5560-09.2010
- Trajković, G., Starčević, V., Latas, M., Leštarević, M., Ille, T., Bukumirić, Z., & Marinković, J. (2011). Reliability of the Hamilton rating scale for depression: A meta-analysis over a period of 49 years. *Psychiatry Research*, 189(1), 1–9.
- US Department of Agriculture and US Department of Health and Human Services. (2010). *Dietary guidelines for Americans, 2010 (7th Edition)*. Washington, DC: US Government Printing Office.
- Wainwright, P. E. (1992). Do essential fatty acids play a role in brain and behavioral development? *Neuroscience & Biobehavioral Reviews*, 16(2), 193–205. doi: 10.1016/S0149-7634(05)80180-0
- Wang, H., Storlien, L. H., & Huang, X.-F. (2002). Effects of dietary fat types on body fatness, leptin, and ARC leptin receptor, NPY, and AgRP mRNA expression. *American Journal of Physiology-Endocrinology and Metabolism*, 282(6), E1352–E1359. doi: 10.1152/ajpendo.00230.2001
- Wang, Y. C., McPherson, K., Marsh, T., Gortmaker, S. L., & Brown, M. (2011). Health and economic burden of the projected obesity trends in the USA and the UK. *The Lancet*, 378(9793), 815–825.
- Werner, E., Zhao, Y., Evans, L., Kinsella, M., Kurzius, L., Altincatal, A., . . . Monk, C. (2013). Higher maternal prenatal cortisol and younger age predict greater infant reactivity to novelty at 4 months: An observation-based study. *Developmental Psychobiology*, 55(7), 707–718. doi: 10.1002/dev.21066
- Werner, E. A., Myers, M. M., Fifer, W. P., Cheng, B., Fang, Y., Allen, R., & Monk, C. (2007). Prenatal predictors of infant temperament. *Developmental Psychobiology*, 49(5), 474–484. doi: 10.1002/dev.20232
- Williams, J. B. (1988). A structured interview guide for the Hamilton depression rating scale. *Archives of General Psychiatry*, 45(8), 742–747. doi: 10.1001/archpsyc.1988.01800320058007