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The influence of temperament on stress-induced emotional eating in children

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Summary

Background: Stress-induced emotional eating is a risk factor for overweight and obesity. Previous research proposes both the human serotonin transporter gene (*5-HTTLPR*) and child's reactive temperament are promising candidates to help explain individual differences in stress-induced emotional eating and weight. Understanding the association between specific genotypes, reactive temperament factors, and stress-induced emotional eating may inform the development of personalized and effective treatment for children who may be at risk for overweight and obesity.

Objective: The current study explored the conditional indirect effect of genetic and environmental susceptibility (i.e., the interaction between *5-HTTLPR* and reactive temperament) on weight (as measured by percent body fat) mediated by stress-induced emotional eating.

Method: One hundred and forty-seven children (4 to 6 years old; 50.3% female; 22.4% Hispanic), along with their primary caregiver, completed laboratory tasks and questionnaires that assessed the child's reactive temperament, stress-induced emotional eating, and percent body fat.

Results: The interaction between *5-HTTLPR* and impulsivity as well as with negative affectivity significantly predicted percent body fat. The interaction between *5-HTTLPR* and impulsivity as well as with negative affectivity significantly predicted both total calorie consumption and rate of total calorie consumption. However, the mediation aspect of this statistical model was not supported.

Conclusions: Child reactive temperament is an important indicator of how children approach eating when stressed. Mental health providers may consider prescribing strategies to reduce emotional eating among children with the SL variant and moderate to high impulsivity as well as children with the LL variant and high negative affectivity.

KEYWORDS

5-HTTLPR, biological susceptibility, childhood obesity, emotional eating, temperament, weight

1 | INTRODUCTION

Approximately 23% of children in the United States between the ages of two and five are classified as having overweight or obesity; among school-age children, this figure increases to almost 35%.[1] The health risks associated with having paediatric obesity, such as hypertension, hyperlipidaemia, hyperinsulinaemia, sleep apnoea, Type 2 diabetes, social exclusion, and increased depression, are well established[2] and contribute to high annual public health costs.[3] Modifying weight trajectories is easier before an individual reaches obesity.[4] As such, a wide variety of childhood obesity prevention programmes exist for different settings that focus on dietary changes, activity changes, psychoeducation, behavioural modification, and family involvement.[5] However, extensive research suggests limited efficacy of these programmes.[6–8] This may be due in part to limited ecological validity of certain programmes, specifically, challenges presented in capturing eating under different levels of stress.

During times of stress, eating behaviours can become problematic with some children consuming more food,[9,10] particularly energy dense, sweet, and/or fatty foods.[11–13] Further, such eating patterns are especially prominent among people experiencing overweight/obesity.[14] Thus, stress-induced emotional eating (SEE; i.e., overeating in the absence of hunger in response to stress[15]) is a proposed risk factor for the development and maintenance of childhood overweight/obesity.[16] Numerous factors such as dietary restraint,[9,17] stress reactivity,[9] cortisol reactivity,[18] and physical activity[19] can influence SEE. However, not all individuals exposed to stressful situations emotionally eat or are at risk for obesity. The diversity of eating behaviours during stress and who is most likely to engage in SEE have not been fully explored.[20] Understanding the individual differences in stress vulnerability and engagement in SEE can assist in identifying children for whom targeted childhood obesity prevention programmes may be indicated.

The brain serotonergic system is related to both stress vulnerability and eating, and dysfunction of the serotonin system is associated with the serotonin transporter gene.[21] Gene expression regulates differently based on different forms of the genotype found in the promoter region of the serotonin transporter gene.[21] Specifically, this difference depends on whether individuals are carrying one or two copies of the long (L) and/or short (S) allele of the serotonin transporter linked polymorphic region (*5-HTTLPR*), the S variant being less efficient.[22] Studies suggest that carriers of the S allele (i.e., SS or SL) show greater biological reactivity to stress (i.e., sympathetic reactivity and hypothalamic pituitary adrenal axis reactivity), elevated amygdala reactivity after stressful events and difficulties shifting attention away from stressors.[21]

The *5-HTTLPR* acts as a genetic catalyst for negative affectivity (i.e., the propensity towards feeling and/or expressing negative emotions and distress[23]), a reactive temperament trait that predicts risk for many anxiety- and stress-related disorders.[21] This suggests individuals with SS or SL variants are more vulnerable to stressful stimuli because of their stable trait of negative affectivity. In addition, S-carriers' behavioural and neurophysiological responses to environmental stress are biased towards negative affect.[21] Further, S-carriers show increased risk for experiencing depression, anxiety, and obesity,[24,25] which in turn, may make them vulnerable to SEE. Specifically, homozygous and heterozygous S-carriers (i.e., SS and SL, respectively) were almost twice as likely to have overweight as were homozygous L allele carriers (i.e., LL),[22,26] and adolescents with depressive feelings showed increased rates of emotional eating with SS or SL variants of the *5-HTTLPR*.[27] However, not all S-carriers are at increased risk, suggesting the *5-HTTLPR* is more of a contributory factor.[28,29] The relationship between the *5-HTTLPR* and stress vulnerability may be better captured as an interaction with reactive temperamental traits.

Child reactive temperament traits (i.e., traits that are more emotionally and behaviourally reactive such as negative affectivity and impulsivity) are genetically based and reflect environmental influences.[30] People differ in their sensitivity to the environment, and this differential sensitivity or susceptibility moderates the impact of the environment on developmental outcomes.[31] Past research supports a link between adult females with SS or SL variants of the *5-HTTLPR* and reactive temperament traits of negative affectivity and impulsivity (i.e., an aspect of involuntary or reactive control that involves negative urgency or readiness to experience negative emotions and to approach and act rashly, without giving it much thought[32-35]). For example, smoking cessation participants with one or two S alleles experienced larger increases in negative affect compared with those with two L alleles,[32] whereas infant humans carrying the SS variant of the *5-HTTLPR* showed more distress and had significantly higher negative affectivity scores than those with the SL or LL variants.[33] In addition, findings suggest that women prone to binge eating who carried the homozygous S genotype (i.e., SS) were also more likely to show higher levels of impulsivity,[34] whereas a study showed that adolescent girls carrying one or two S alleles and from homes where their families were less warm and emotionally supportive exhibited higher impulsivity.[35]

Reactive temperament traits are also associated with eating and weight. A recent review found evidence linking negative affectivity (from infancy to early childhood) to obesity later in life,[36] and research suggests that negative affectivity is predictive of binge eating,[37,38] emotional eating,[39] and stress-induced eating[40] in samples with nonoverweight and overweight. Research findings also suggest that impulsivity is linked to externalizing problems in children,[41] higher rates of emotional eating in adults,[42] and that levels of impulsivity tend to be higher in individuals with obesity and those who binge eat.[43] Thus, genetics (e.g., serotonin transporter gene) and reactive temperament traits may be key to understanding individual differences in reactions to stress. Taken together, it stands to reason that those with the S variant of the serotonin transporter gene, in conjunction with increased negative affectivity and/or impulsivity, may show significantly higher levels of stress-related problems such as SEE.

A limitation of the previous research on the serotonin transporter gene, reactive temperament, eating, and weight is that studies have only examined associations between two variables at a time (*5-HTTLPR* and eating, negative affectivity and obesity, etc.). A more comprehensive analysis of the relationships between the serotonin transporter gene and reactive temperament on SEE can determine what combinations of characteristics render children most at risk for obesity. In the current study, a moderated mediation model is proposed between *5-HTTLPR*, reactive temperament traits, and SEE in predicting child's obesity risk (see Figure 1). Specifically, the conditional indirect effect of *5-HTTLPR* and reactive temperament (i.e., negative affectivity and impulsivity) on a child's obesity risk mediated by SEE was explored in a sample of 4- to 6-year-old children. Children with the S variant of the serotonin transporter gene along with increased levels of negative affectivity or impulsivity would have higher percent body fat compared with L allele carriers. Negative affectivity and impulsivity are individually associated with emotional eating; thus, children with a genetic stress vulnerability such as the S-carriers of the *5-HTTLPR* and a reactive temperament may be more susceptible to SEE. The relationship between S-carriers and reactive temperament on child percent body fat would be exclusively among children who engage in SEE.

2 | METHODS

2.1 | Participants

The present study included 147 children (M_{age} = 4.85, SD_{age} = 0.85; 50.3% female). The racial and ethnic composition of the children was 68.7% Caucasian, 20.4% African American, 8.2% Asian, and 2.0% Native American; 22.4% identified as Hispanic. Children were recruited through their primary caregivers using flyers placed within an urban community within south central United States. Primary caregivers (M_{age} = 34.23, SD_{age} = 7.10, 85.7% mothers, 26.5% divorced/separated, 53.1% low income) who were interested in having their children participate, and who met inclusion criteria (i.e., fluent in English and no food allergies related to the food groups provided in this study), were invited to the laboratory.

2.2 | Procedure

Primary caregivers were instructed and reminded to feed their child lunch and then ensure their child did not eat for 2 h before the scheduled laboratory visit. Upon arrival to the lab, parents and children

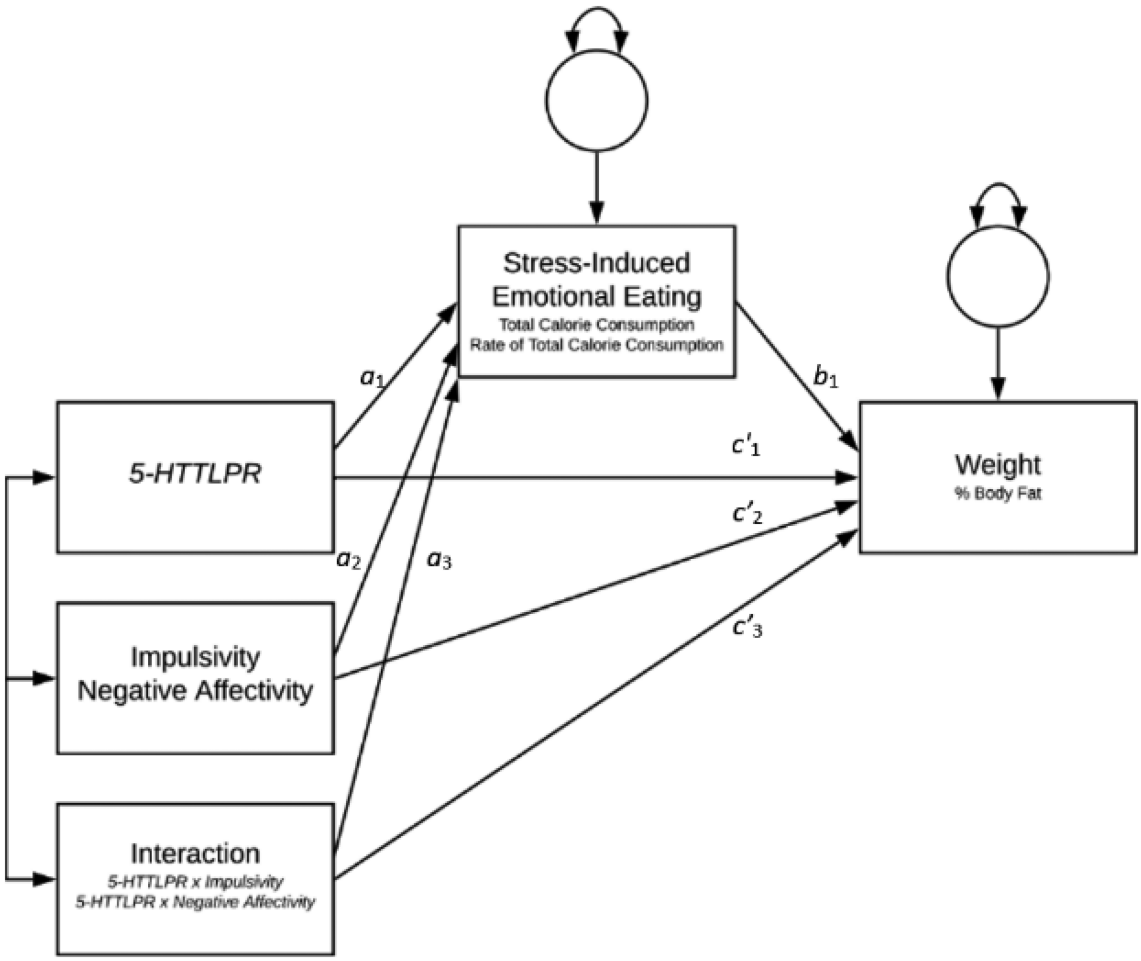

FIGURE 1 The proposed study model

completed parental informed consent or child assent procedures. Next, the child's height, weight, and percent body fat were assessed. The child's saliva was obtained for genetic analysis, and the child was given a standard snack consisting of dry cereal (i.e., Cheerios), bottled water, and a fruit cup (selected based on Food and Drug Administration [FDA]-approved guidelines); the children were instructed to eat as much as they wanted and to eat until they felt full. After the snack, the child participated in a mild stress task alone in a room. Following the mild stressor, children proceeded to the laboratory eating task. Children earned stickers and silly bands throughout the lab visit and a toy from the treasure chest at the end. Children were videotaped during the entire lab visit. While the children completed the tasks, their caregivers completed several questionnaires about the children. Parents were debriefed and compensated $50 for the lab visit.

2.2.1 | Stressor

The puzzle activity, a challenging and stressful task,[43,45] asks children to complete the puzzle using only their sense of touch.[46] Children were told that they would earn stickers towards a prize for completing the task to ensure motivation. A timer was placed on the table that announced how much time remained for children to complete the task. As a manipulation check, children were hooked up to physiological equipment to measure heart rate variability (HRV) during the puzzle task. When compared over multiple stressors, psychometric studies have shown satisfactory temporal stability of HRV; furthermore, HRV results from laboratory stress measures have been shown to translate well to real-world settings.[47] Following a stress-inducing event, individuals who respond well to stress show reductions in HRV when compared with baseline and elevation of HRV represents strong emotional reactivity or poor responses to stress.[48] HRV values at baseline and during the puzzle stress task were compared and revealed that HRV was significantly different during the puzzle task, which suggests that the puzzle task was stressful enough to elicit a change in these physiological measures.

2.3 | Measures

2.3.1 | Stress-induced emotional eating

SEE was measured using an objective free access procedure with foods children would likely be familiar with.[49] After the children completed the challenging puzzle task, they were asked to sit in a room in front of a table that had a bowl of 30 M&Ms and a bowl of 20 red grapes, with each bowl being exactly the same size, shape, and colour. All children in the study reported liking both foods. Children were instructed that they were allowed to eat the food and were left alone in the room for 5 min. Children's behaviours were coded for the type of food chosen, the amount of food eaten, and the pace at which they ate. Total calories consumed were calculated based on the total number of M&Ms and red grapes eaten. Rate of total caloric intake was calculated as total calories consumed divided by total time spent eating (in seconds) with higher numbers indicating more calories consumed over a shorter period of time. M&Ms and grapes were selected to give children an option of an unhealthy and a healthier sweet choice and because a study of children from 1 to 11 years of age revealed that chocolate and grapes were among the 10 foods preferred; no gender differences in terms of likeability existed among these foods.[50] Further, these foods are common snacks for children, and previous studies using SEE have used M&Ms and grapes.[9]

2.3.2 | Serotonin transporter gene (*5-HTTLPR*)

At the lab visit, trained research assistants collected saliva using Salimetrics, Inc. passive drool collection kits for children. Samples were stored in a −80°C freezer until DNA extraction. Samples were sent out to Polymorphic DNA Technologies, Inc. for DNA isolation, amplification, and sequencing. All samples were run in duplicate and passed initial quality control. Participants were genotyped for the 43-bp insertion/deletion polymorphism in the regulatory promoter region of the serotonin transporter gene with standard polymerase chain reaction procedures previously reported in the literature.[51] Participants were coded as either homozygous short (SS), heterozygous short (SL), or homozygous long (LL) polymorphisms of the serotonin transporter gene. Polymerase chain reaction analyses indicated that the serotonin transporter genotype distribution in our sample was as follows: 23% short/short; 55% short/long; and 22% long/long. There was no deviation from Hardy–Weinberg equilibrium ($\chi^2 = 0.89$, $p = .34$).

2.3.3 | Impulsivity and negative affectivity

Child levels of impulsivity and negative affectivity were measured using the parent-report Child Behaviour Questionnaire (CBQ), a widely used measure to assess child temperament.[52] Subscale scores can range from 1 to 7 with higher scores indicating higher levels of each temperament trait. Cronbach's alpha for the CBQ on a nationally representative sample was .76 for impulsivity in samples of 4 to 7 year olds[52]; within this sample, it was .66. A composite for negative affectivity was computed as the average of the anger/frustration, discomfort, and fear subscale scores.[52] The alpha reliability for the CBQ on the same representative sample was .81 for anger/frustration, .70 for discomfort, and .70 for fear[52] and within this sample are .80, .74, and .67, respectively.

2.3.4 | Percent body fat

Body mass index for age is commonly used as an indicator of adiposity.[53–55] However, among diverse (e.g., gender and ethnicity) groups of children, body mass index alone is not an equivalent measure of adiposity.[53,54] In this study, percent body fat was used a proxy indicator of obesity risk because percent body fat better distinguishes

fat from lean body mass or bone when compared with body mass index.[56] Skinfold measurements were used to measure body fat at four sites: biceps, triceps, subscapular, and suprailiac. Assessment was on the right side of the body and was recorded to the nearest 1 mm using standard procedures.[57] All research assistants were extensively trained and conducted over 100 skinfold measurements.

2.4 | Data analysis

Analyses were conducted to explore a moderated mediation model that occurs when the strength of a mediated effect (in this case, the effect of the *5-HTTLPR* on percent body fat through SEE) depends on the level of another variable (in this case, impulsivity or negative affectivity).[58] The interaction between the *5-HTTLPR* and impulsivity/negative affectivity on percent body fat was explored first. Hierarchical regression was used to conduct these analyses, with gender entered as a covariate in step one. Following significant findings, the interaction of the *5-HTTLPR* and impulsivity/negative affectivity on SEE, the mediating variable, was explored. A separate hierarchical regression equation was constructed for each dependent variable (rate of total calorie consumption and total calorie consumption). Finally, the relationship between SEE and percent body fat was examined.

3 | RESULTS

All variables were normally distributed, and sample statistics (mean and standard deviation) and correlations were obtained for all study variables (Table 1). There was a significant correlation between gender and rate of calorie consumption, such that male children consumed their food at a faster rate than female children. Impulsivity was positively related with total calorie consumption and rate of total calorie consumption; children with higher rates of impulsivity consumed more food at a faster pace than other children.

Of the 147 genotyped child participants, one individual (0.7%) failed to respond to the impulsivity items, one individual (0.7%) failed to respond to the negative affectivity items, seven individuals (4.8%) did not have food consumption data, and three individuals (2.0%) did not have their food consumption timed. Analyses were handled in a pairwise manner to address this missing data.

3.1 | The association between *5-HTTLPR* and negative affectivity/impulsivity and percent body fat (c'_3)

The interaction between the serotonin transporter gene and impulsivity (Figure 2A) as well as with negative affectivity (Figure 2B) significantly predicted percent body fat (Table 2). These results show that negative affectivity (f^2 = .038) has a slightly greater impact on percent body fat than does levels of impulsivity (f^2 = .037), although both have small effect sizes. At average impulsivity levels, percent body fat appeared to be similar across genotype; however, at extreme levels of impulsivity (either high or low), percent body fat differentiated across genotype. Specifically, percent body fat was highest for individuals with the SL genotype across low and high levels of impulsivity; percent body fat was lowest for those with SS allele across all levels of impulsivity. Similarly, children with the SS allele had the lowest percent body fat across all levels of negative affectivity. However, at both high and low levels of negative affectivity, LL allele individuals showed the highest levels of body fat compared with the other carriers.

3.2 | The association between *5-HTTLPR* and negative affectivity/impulsivity and SEE (a_3)

The serotonin transporter gene and negative affectivity did not independently predict either measure of SEE above and beyond the covariate; impulsivity significantly predicted only total calorie consumption (Table 3). The interaction between the serotonin transporter gene and

TABLE 1 Correlations and sample statistics of all variables

	1	2	3	4	5	6	7
1. Gender[a]	1.0						
2. Impulsivity	−.124	1.0					
3. Negative affectivity	−.033	.007	1.0				
4. *5-HTTLPR*[b]	−.048	.093	.039	1.0			
5. Total calorie consumption	−.162	.281**	−.006	−.016	1.0		
6. Rate of total calorie consumption	−.172*	.273**	.009	.049	.971**	1.0	
7. Percentbody fat	.084	.118	.141	.122	−.044	−.046	1.0
Mean	1.51	4.59	4.23	2.02	92.27	.31	22.55
(Standard deviation)	(.50)	(1.06)	(.83)	(.73)	(38.19)	(.14)	(5.70)

[a]Child gender: 1.00 = male, 2.00 = female.
[b]Variants of the serotonin transporter gene: 1.00 = SS, 2.00 = SL, 3.00 = LL.
*$p < .05$.
**$p < .01$.

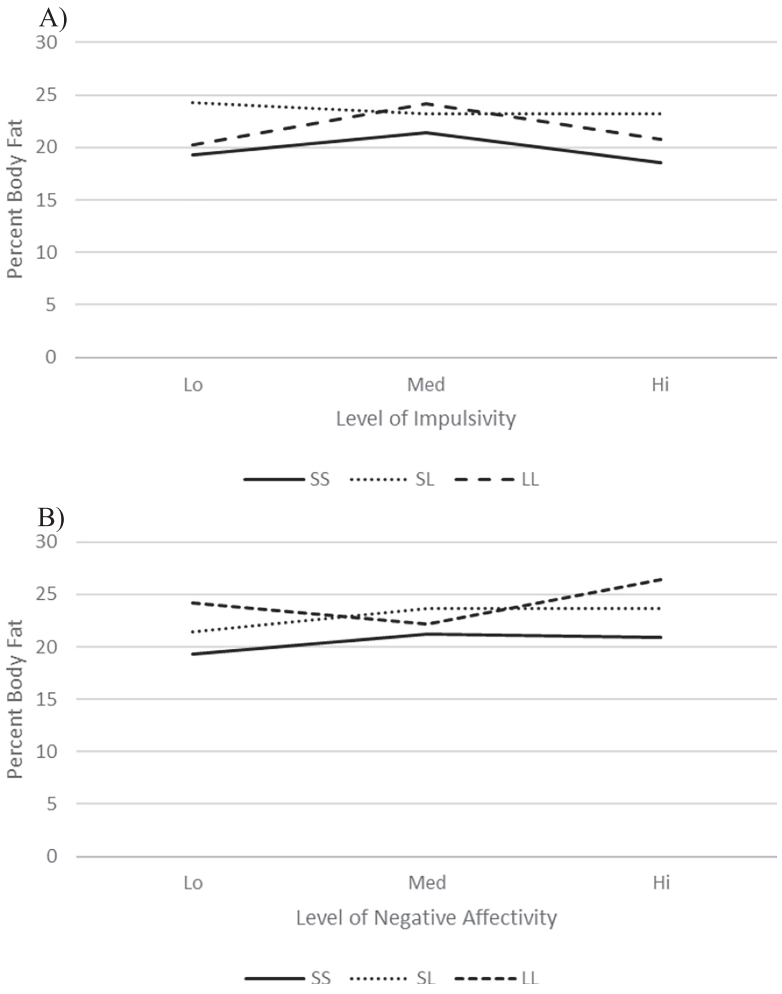

FIGURE 2 Exploration of the interaction between the *5-HTTLPR* and impulsivity (A) as well as negative affectivity (B) on percent body fat

TABLE 2 Interaction predicting percent body fat (c'_3)

	Model summary			Coefficients		
	R^2	Adj. R^2	F (df)	ß	t	p
Impulsivity						
Step 1	.007	.000	1.039 (1, 143)			
Gender				.967	1.019	.310
Step 2	.036	.022	2.670 (2, 142)			
5-HTTLPR × impulsivity				.235	2.068	.040
Negative affectivity						
Step 1	.007	.000	1.039 (1, 143)			
Gender				.967	1.019	.310
Step 2	.037	.023	2.705 (2, 142)			
5-HTTLPR × negative affectivity				.278	2.085	.039

impulsivity significantly predicted both the rate of total calorie consumption and total calorie consumption. For rate of calorie consumption, the interaction between the *5-HTTLPR* and impulsivity suggests that at high levels of impulsivity, all genotypes are consuming more calories in shorter periods of time. At average and low levels of impulsivity, the genotypes differentiate on eating so that those with the SL variant consume more food at a faster speed than the other two groups (Figure 3A). Of note is that both homozygous variants

TABLE 3 Interaction predicting stress-induced emotional eating (a_3)

	Model summary			Coefficients		
	R^2	Adj. R^2	F (df)	ß	t	p
Interaction with impulsivity						
Rate of total calorie consumption						
Step 1	.047	.040	6.610 (1, 135)*			
Gender				−.058	−2.571	.011
Step 2	.069	.048	3.270 (3, 133)*			
5-HTTLPR				.005	.313	.755
Impulsivity				.019	1.712	.089
Step 3	.146	.120	5.630 (4, 132)**			
5-HTTLPR × impulsivity				.050	3.450	.001
Total calorie consumption						
Step 1	.043	.036	6.155 (1, 138)*			
Gender				−14.648	−2.481	.014
Step 2	.073	.053	3.577 (3, 136)*			
5-HTTLPR				−2.008	−.495	.621
Impulsivity				6.043	2.089	.039
Step 3	.101	.075	3.808 (4, 135)**			
5-HTTLPR × impulsivity				8.010	2.061	.041
Interaction with negative affectivity						
Rate of total calorie consumption						
Step 1	.047	.040	6.610 (1, 135)*			
Gender				−.058	−2.571	.011
Step 2	.051	.030	2.398 (3, 133)			
5-HTTLPR				.007	.483	.630
Negative affectivity				−.008	−.665	.507
Step 3	.107	.079	3.934 (4, 132)**			
5-HTTLPR × negative affectivity				.052	2.856	.005
Total calorie consumption						
Step 1	.043	.036	6.155 (1, 138)*			
Gender				−14.648	−2.481	.014
Step 2	.045	.024	2.143 (3, 136)			
5-HTTLPR				−1.203	−.293	.770
Negative affectivity				−1.661	−.500	.618
Step 3	.092	.065	3.428 (4, 135)*			
5-HTTLPR × negative affectivity				12.765	2.646	.009

*p < 05.
**p < .01.

increased rate of calorie consumption as level of impulsivity increased, whereas the SL variant had greater rates of calorie consumption across both medium and high levels of impulsivity and less rates of calorie consumption at low levels of impulsivity. There is a similar pattern of results for total calorie consumption (Figure 3B), such that as levels of impulsivity increase, calorie consumption increases across genotypes. However, those with the SL variant consume more food than the other two groups across levels of impulsivity.

The interaction between the *5-HTTLPR* and negative affectivity significantly predicted both total calorie consumption and rate of total calorie consumption. For total calorie consumptions (Figure 3D), at low levels of negative affectivity, individuals with an S consume more calories than those with the LL genotype. Those with SL stay constant across levels of negative affectivity. However, SS individuals decrease their calorie consumption at average and high levels of negative affectivity. At high levels of negative affectivity, LL individuals show the

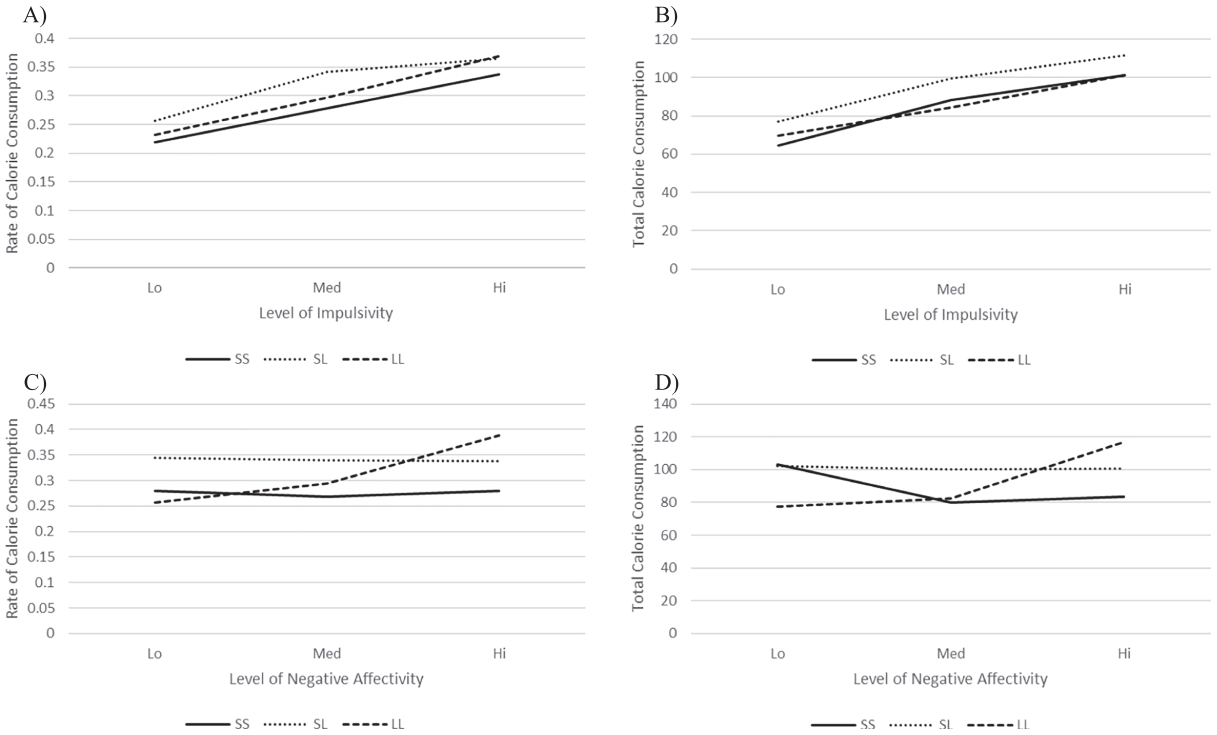

FIGURE 3 Exploration of the interaction between the *5-HTTLPR* and impulsivity on rate of calorie consumption (A) and total calorie consumption (B) as well as the interaction between *5-HTTLPR* and negative affectivity on rate of calorie consumption (C) and total calorie consumption (D)

highest level of calorie consumption. Pattern of results are similar for rate of calorie consumption (Figure 3C), with one exception. At low levels of negative affectivity, SS individuals show lower rates of food consumption than the SL group. This suggests that those with the SS genotype consumed their food at a slower pace compared with their heterozygous counterparts. Looking at effect sizes, levels of negative affectivity and impulsivity appear to have medium effects on rate of food consumption (f^2 = .120 and .171, respectively) and small effects on total food consumption (f^2 = .101 and .112, respectively).

No measures of SEE predicted percent body fat prohibiting the examination of the mediation model.

4 | DISCUSSION

The current study examined a moderated mediation model assessing how *5-HTTLPR*, reactive temperament, and SEE are associated with children's percent body fat. Interestingly, the findings elucidate how the association between two child reactive temperament traits (negative affectivity and impulsivity) with SEE and percent body fat differ by genotype. Across most analyses, children with the SL variant showed more stable rates of total calorie consumption and percent body fat across levels of impulsivity and negative affectivity. The only exception being that children with the SL variant ate quicker at medium and high levels of impulsivity and consumed more food. Based on these findings, mental health providers within a primary care-mental health integration setting may consider prescribing emotion regulation and distraction strategies or other targeted obesity prevention techniques to reduce emotional eating among children with the SL variant and moderate to high levels of impulsivity. This approach may also be appropriate for children with the LL variant and high levels of negative affectivity as they showed the highest percent body fat, consumed the most food, and ate the food the quickest during the eating laboratory task. Thus, children with the LL variant displayed the most SEE in response to high negative affectivity.

In contrast, children with the SS variant tended to score the lowest on percent body fat, rate of calorie consumption, and total calorie consumption across impulsivity and negative affectivity, suggesting children with the SS variant may be at lowest risk for overweight and obesity. Importantly, findings do suggest that child reactive temperament is an important indicator of how children approach eating when stressed. Specifically, across all genotypes, as level of impulsivity increased, so too did rate and amount of food consumption. When considering personalized medicine, this suggests when practitioners are concerned with SEE, they can assess child impulsivity level (using low-cost, easy measures) and recommend programmes that teach children and parents to incorporate skills that reduce impulsivity.

Results for the serotonin transporter gene conflict with original expectations and add to the mixed literature on this topic.[59,60] The children with homozygous L allele carriers (i.e., those with more efficient genetic variants) showed higher weight and more SEE behaviours in the current study. However, the small to medium effects seen

in this study may reflect the effects of other determinants of obesity acting in tandem with biological risks. For example, there is extant literature exploring the impact of additional individual risk factors (i.e., gender and approach to food) as well environmental factors (i.e., parenting style, parent feeding style, and feeding environment) on weight[61–65] that were not explored in the current study. Furthermore, previous studies exploring the relationship between the serotonin transporter gene and weight use adolescent or adult samples. It is possible that these additional factors play a larger role in eating behaviours and weight in preschool than does biological susceptibility.

Although these findings are contrary to the hypotheses, they may still have clinical implications. The children with the LL variant at high levels of negative affectivity had the highest percentage of body fat (from 22.22 to 26.43 or in the 85th to 95th percentile) for both boys and girls.[66] In addition, while children with the LL variant and high levels of negative affectivity consumed the most food and ate the food the quickest during the eating laboratory task, they also consumed the least amount of food and ate the slowest with low levels of negative affectivity. This could indicate a potential differential susceptibility to childhood obesity among children with the LL variant, particularly those with high negative affectivity temperaments as these children are at greatest risk for obesity. Although these findings do lend support for the assessment of *5-HTTLPR* as a way for clinicians to tailor personalized treatments, the literature is mixed, and more research is needed. Furthermore, the findings are from cross-sectional data, and longitudinal designs from childhood to adolescence could reveal potential directionality of effects between biological susceptibility, early SEE behaviours, and future weight.

Although previous research has linked SEE to weight,[16] the current findings are inconsistent with the existing literature on this topic. In our sample of preschool-aged children, SEE was not associated with percent body fat. A key difference between our sample and the existing literature is the age of the children. The majority of the literature has focused on adolescent children and adults,[11,13,15] with one study on preadolescent children from 9 to 12 years old.[12] Thus, it is possible that 4 to 6 years of age is too early for SEE behaviours to predict weight during the early childhood or preschool years. However, the lack of prediction could be promising, in that a universal prevention programme for childhood obesity could teach parents how to change their child's eating behaviours before SEE behaviours produce future, longer term detrimental impacts on weight. Future research should assess at what age the association between SEE and childhood obesity risk emerges in children.

The current study had several limitations worth noting. Although the sample was racially and ethnically diverse, all parents were required to speak and read English fluently in order to complete the self-report questionnaires. Although almost 25% of our sample was Hispanic, our findings may only generalize to English-speaking populations. The eating laboratory task only contained sweet foods. When overeating occurs, sweet foods are preferred,[67] but a more varied food selection may have yielded different consumption patterns. Further, laboratory-based eating studies allow for precise measurement of food intake but at the cost of ecological validity.[49] Thus, future research should aim to replicate the findings of this study using epidemiological and dietary survey data. Rate of food consumption was calculated with total calories consumed, limiting the ability to distinguish speed of eating by food preference. Parents reported the degree of child negative affectivity and impulsivity on surveys, which is subject to parental bias. Some previous research has found a relationship between behavioural measures of temperament and weight and no relationship with parent-report measures of temperament[68–71]; future research should replicate the findings in this study using an emotion regulation or effortful control battery on children. Due to small sample sizes, the analyses controlled for gender, which prohibited the examination of gender differences across the associations. Although skinfold testing is one of the most common assessments of body composition and is particularly useful in children given their small body size, the gold standard for body composition measurement is the dual-energy X-ray absorptiometry.[72] However, dual-energy X-ray absorptiometry is more invasive and expensive, as well as less feasible in medical providers' offices. Nonetheless, the correlation between skinfolds and fat mass as measured by dual-energy X-ray absorptiometry is .87 among children 4 to 9 years old,[72]

indicating that the use of skinfolds testing does have more measurement error in percent body fat. Finally, this study involves a cross-sectional design that limits the ability to establish causal relationships.

Future research should aim to explore this research in a different-aged sample. Not only would this allow for comparison of the model in developmentally different groups, but it could allow for the continued examination of impulsivity, negative affectivity, and biological susceptibility to stress that will continue to tease apart the relationship between these factors and determine for which combination of factors SEE (and therefore obesity risk) increases. Significant findings will inform intervention and prevention programs designed to reduce the risk for SEE in this group. Having a more nuanced understanding of these variables can inform the development of personalized treatment decision making for children at risk for overweight and obesity. Furthermore, findings in this area may expand beyond the domain of eating and obesity research. People who struggle with impulsivity/negative affectivity and contain specific variants of a gene may struggle in areas beyond SEE and/or having overweight. Thus, this research stands to inform a more global prevention effort.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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