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The Impact of Between-Session Habituation, Within-Session Habituation, and Weight Gain on Response to Food Exposure for Adolescents with Eating Disorders

Jamal H. Essayli, PhD[1,2], **Lauren N. Forrest, PhD**[2], **Hana F. Zickgraf, PhD**[3], **Emily C. Stefano, PhD**[4], **Kathleen L. Keller, PhD**[5,6], **Susan E. Lane-Loney, PhD**[1,2]

[1]Pennsylvania State University College of Medicine, Department of Pediatrics, 500 University Drive, Hershey, PA, 17033

[2]Pennsylvania State University College of Medicine, Department of Psychiatry and Behavioral Health, 500 University Drive, Hershey, PA, 17033

[3]Emory University School of Medicine, Department of Psychiatry and Behavioral Sciences, 2015 Uppergate Dr., Atlanta, GA 30307

[4]Wake Forest University School of Medicine, Bariatric and Weight Management Center, 4614 Country Club Rd., Winston-Salem, NC 27104

[5]Pennsylvania State University, Department of Nutritional Sciences, 321 Chandlee Lab, University Park, PA 16802

[6]Pennsylvania State University, Department of Food Science, 321 Chandlee Lab, University Park, PA 16802

Abstract

Objective: Exposure therapy is a promising treatment for eating disorders (EDs). However, questions remain about the effectiveness of exposure to feared foods during the weight restoration phase of treatment, and the importance of between-session and within-session habituation.

Method: We recruited 54 adolescents from a partial hospitalization program (PHP) for EDs which included daily food exposure. Throughout treatment, participants provided subjective units of distress (SUDS) ratings before and after eating a feared food, and completed measures of ED symptomatology.

Results: Multilevel models found that pre-exposure SUDS decreased over time, providing some evidence that between-session habituation occurred. In contrast, the difference between pre-exposure and post-exposure SUDS did not decrease over time, indicating that within-session habituation did not occur. Weight gain predicted greater between-session habituation to feared foods, but did not predict within-session habituation. Between-session habituation, but not

corresponding author: jessayli@pennstatehealth.psu.edu, (717) 531-0003 x320191.

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within-session habituation, predicted favorable treatment outcomes, including weight gain and improvements on the Children's Eating Attitudes Test and Fear of Food Measure.

Discussion: PHPs that include daily exposure to feared foods may be effective at decreasing anxiety about foods for adolescents with EDs who are experiencing weight restoration. Further research is warranted to replicate our findings challenging the importance of within-session habituation, and to better understand between-session habituation and inhibitory learning as mechanisms of change when conducting food exposure for EDs.

Keywords

eating disorders; anorexia nervosa; exposure therapy; food; habituation

Exposure therapy is the most effective psychological intervention for anxiety disorders (Abramowitz, 2013), and holds promise as a treatment for eating disorders (EDs). Anxiety disorders and EDs are highly comorbid (Pallister & Waller, 2008), and anxiety is a prominent feature of EDs (Schaumberg et al., 2020). Given the apparent role of anxiety in the development and maintenance of EDs, effective treatment might be achieved by targeting anxious cognitions and avoidance behaviors in patients with EDs via exposure therapy.

According to Foa and Kozak's (1986) emotional processing theory, exposure therapy works by activating a "fear structure" using in vivo, imaginal, or interoceptive exposure to the feared stimulus (e.g., eating chocolate). Repeated exposure results in the development of safe, non-threatening associations with the feared stimulus, a process that is often referred to as *extinction learning* (Abramowitz, 2013). During exposure trials, extinction learning is theorized to be evidenced by *habituation* – or decreases in fear – both *within* and *between* exposure sessions.

Despite its initial widespread acceptance, a growing body of research suggests that habituation of fear may not be the operative process through which exposure therapy facilitates extinction learning (Craske et al., 2008). Prior studies have not consistently supported the hypothesis that greater within-session habituation during exposure therapy is associated with greater reductions of fear at follow-up (Peterman et al., 2019). Additionally, while between-session habituation is positively associated with exposure outcomes in some studies, other studies have failed to find this relationship (Knowles & Olatunji, 2019).

Little is known about the relationship between habituation and outcomes in individuals with EDs, and whether clinicians should emphasize or disregard fear reduction when providing exposure therapy for EDs. Studies that have measured changes in subjective units of distress (SUDS) ratings during food exposure for EDs found that individuals commonly report *higher* SUDS during and immediately after exposure sessions (Boutelle, 1998; Carter et al., 2002; Levinson et al., 2019). While SUDS tend to progressively reduce over the two hours following food exposure (Gray & Hoage, 1990), it is unclear if this decrease in distress is evidence of extinction learning. During this time, some patients may in fact be incorporating information that is incompatible with their feared beliefs about food (e.g., "eating chocolate was not as terrible as I thought it would be"). For other patients, however, reductions in SUDS hours after food exposure may serve to reinforce fears about food (e.g.,

"I feel much better when I'm not eating fatty foods like chocolate"). Research in the field of EDs is needed to understand the underlying mechanisms involved in exposure therapy's effectiveness, and whether habituation facilitates extinction learning.

The lack of within-session habituation during food exposure may be related to the longer-term fears often reported by patients with EDs, particularly the belief that feared foods will lead to uncontrollable weight gain over time (Murray et al., 2016). Food exposure may be most effective when patients repeatedly eat feared foods and experience no or modest weight gain, which can be the case when treating patients with bulimia nervosa (BN) (Bulik et al., 2012) and binge-eating disorder (BED) (Grilo et al., 2011). On the other hand, patients who are underweight or weight suppressed – which include all patients with AN and many with other EDs – are *expected* to gain weight as they modify their eating behaviors. Accordingly, some have suggested that exposure to feared foods during the weight restoration phase of treatment may reinforce the belief that consumption of these foods results in weight gain (Murray et al., 2016).

While research dating back to the 1980s support the efficacy of food exposure for EDs, these studies focused on non-underweight patients with BN (Carter & Bulik, 1994). More recently, investigators have started to evaluate the efficacy of exposure therapy for individuals with AN (Reilly et al., 2017). Food exposure has resulted in increases in calorie intake and reductions in anxiety in weight-restored adults with AN (Steinglass et al., 2014), and significant decreases in ED psychopathology and weight gain in underweight adults with AN (Cardi et al., 2019). Others studies support the effectiveness of exposure therapy when integrated into family-based treatment (FBT) for AN (Hildebrandt et al., 2013), a partial hospitalization program (PHP) for adolescents with EDs (Iniesta Sepulveda et al., 2017), a PHP for adults with EDs (Farrell et al., 2019a), and an inpatient hospital for adults with EDs (Farrell et al., 2019b).

While three of the aforementioned studies investigated food exposure while participants were gaining weight, sample sizes were small (Hildebrandt et al., 2013; Iniesta Sepulveda et al., 2017) and/or the degree of weight gain was modest (Cardi et al., 2019). Additional research with a larger sample of adolescents with EDs undergoing weight restoration is needed to better understand the effectiveness of food exposure and the impact of weight gain on response to food exposure.

Present Study and Hypotheses

In this open trial, adolescents undergoing weight restoration while receiving treatment at a PHP for EDs were repeatedly exposed to a feared food every day. Participants provided SUDS ratings immediately before and after each food exposure, and completed a battery of measures at baseline and every three weeks.

Although debate continues about whether between-session habituation is an important *mechanism* of change, research supports that between-session habituation is, at the very least, an encouraging *marker* of change (Maples-Keller & Rauch, 2020). Thus, we hypothesized that participants would experience between-session habituation, or decreases

in daily *pre-exposure* SUDS ratings for exposure foods, over the course of treatment. Despite concerns that weight gain could limit the effectiveness of food exposure (Murray et al., 2016), prior studies suggest that exposure therapy can be effective while patients with EDs are gaining weight (Hildebrandt et al., 2013; Iniesta Sepulveda et al., 2017). Thus, we predicted that weight gain would *not* impact between-session habituation.

In contrast to findings about between-session habituation, research suggests that individuals with EDs do not reliably report decreases in anxiety within food exposure trials. Thus, we hypothesized that participants would *not* experience within-session habituation throughout treatment. Additionally, we hypothesized that between-session habituation, but not within-session habitation, would predict improvements in body weight and ED symptomatology over the course of treatment.

Method

Participants

We recruited participants for an open trial study (i.e., no control condition) from a PHP for adolescents with EDs located at an academic medical center. Patients who were identified by a medical provider as requiring an inpatient or residential level of care (e.g., due to risk for refeeding syndrome) would not be accepted into the PHP, and were not included in our pool of potential participants. All patients at this PHP with a diagnosis of AN, BN, BED, and other specified feeding or eating disorder were eligible to participate. Patients with avoidant/restrictive food intake disorder were not eligible.

We approached 84 patients for this study, and obtained parental consent and adolescent assent from 73 patients (86.9% of recruitment pool). We excluded 19 participants (26.0% of initial sample) from analyses, as they did not complete at least two self-reported data collections. All 19 of these excluded participants spent less than one week in the PHP before being discharged, typically to an inpatient facility.

The final sample consisted of 54 participants. Participants were predominately female (n = 44, 81.5%) and White (n = 48, 88.9%). Mean age was 14.38 (SD = 1.64) years. Individuals included those diagnosed with AN (n = 39), atypical AN (n = 13), and BN (n = 2). Reflecting the characteristics of real-world clinical samples, comorbid diagnoses were common: 66.7% of participants were diagnosed with a comorbid anxiety disorder; 53.7% with a depressive disorder; 13.0% with obsessive-compulsive disorder; 11.1% with post-traumatic stress disorder; 3.7% with attention-deficit/hyperactivity disorder; and 1.9% with a psychotic disorder. The majority of participants (68.5%) were taking at least one psychotropic medication during treatment, including: anti-depressants (68.5% of participants), anti-anxiety medication (20.4%), anti-psychotics (13.0%), anticonvulsants (14.8%), stimulants (1.9%), and other psychiatric medications (1.9%). Length of stay in the program varied from two to 16 weeks, with a mean of 7.89 (SD = 3.03) weeks. Participants and their parent or legal guardian provided consent to participate upon admission to the PHP. This study was approved by the Penn State University intuitional review board.

Treatment

Upon admission to our PHP, participants were evaluated by a psychiatrist and/or clinical psychologist to determine ED and other psychiatric diagnoses. The PHP ran five days per week, and integrated components of CBT (Fairburn, 2008; Waller et al., 2007) and FBT (Lock & Le Grange, 2015). Similar to FBT (Lock & Le Grange, 2015), adolescents' parents were trained to exert external control to assist their child with meeting "treatment non-negotiables" (Geller & Srikameswaran, 2006) such as adhering to a prescribed meal plan. At the same time, treatment integrated the CBT principles of collaborative empiricism, individual decision-making, and corrective learning as much as possible within the confines of these non-negotiables (Essayli & Vitousek, 2020).

Each day, participants ate an "exposure challenge" food for lunch. Participants were given a full-sized candy bar on Mondays, a baked good (e.g., cookies) on Tuesdays, pizza on Wednesdays, a dessert from the cafeteria on Thursdays, and a challenge breakfast item (e.g., pancakes) on Fridays. These exposure challenges were processed in weekly therapy sessions, which included discussion of participants' feared predictions about eating these foods.

Measures

Challenge Food Questionnaire—On the Challenge Food Questionnaire, which was created for the present study, participants were asked to provide a SUDS rating immediately before and after each food exposure session on a scale from 0 (*no distress*) to 10 (*extremely high distress*). The SUDS scale is commonly used to assess subjective distress, including in individuals with EDs (e.g., Gray & Hoage, 1990; Hildebrandt et al., 2013; Steinglass et al., 2014). Participants' daily pre-exposure SUDS ratings on this measure were used to determine whether they experienced between-session habituation. Differences between daily pre-exposure and post-exposure SUDS ratings on this measure were used to determine whether participants experienced within-session habituation.

Percent of Median Body Mass Index (%MBMI)—Patients were weighed by program staff at admission, discharge, and at least once per week throughout treatment. %MBMI was calculated according to the 50^{th} percentile BMI-for-age using the 2000 Centers for Disease Control and Prevention growth charts (www.cdc.gov/growthcharts).

Children's Eating Attitudes Test (ChEAT)—The ChEAT (Maloney et al., 1988) is a 26-item scale that assesses ED attitudes and behaviors in children. The ChEAT was administered at admission, every three weeks, and discharge.

Fear of Food Measure (FOFM)—The FOFM (Levinson & Byrne, 2014) is a 23-item self-report instrument with three subscales: anxiety about eating, food avoidance behaviors, and feared concerns related to eating. The FOFM was administered at admission, every three weeks, and discharge.

Data Analyses

Multilevel models were used to test all hypotheses. Observations (Level 1) were nested within participants (Level 2). Time was modeled as the number of days since admission. Time was centered at day = 0, such that the effect of time was relative to participants' admission date.

All models were estimated in three steps. First, we ran empty models to obtain the intraclass correlation coefficients. Second, we ran random intercept only models. Third, we ran random intercept and random slope models. Given that the random intercept only models yielded the best fit, we present results from these models.

All analyses were performed in the R software environment (R Core Team, 2021). We used the dplyr package (Wickham, Françios, Henry, & Müller, 2021) for data restructuring, lmer (Bates, Maechler, Bolker, & Walker, 2015) and lmerTest (Kuznetsova, Brockhoff, & Christensen, 2017) for multilevel model estimation and interpretation, and ggplot2 (Wickham, 2016) for constructing figures.

Between-Session Habituation to Daily Food Exposure—To determine whether pre-exposure SUDS decreased over time, we estimated a multilevel model where observations were nested within participants, pre-exposure SUDS was the outcome, time was the predictor, and %MBMI was the covariate. %MBMI was participant-mean centered. Participant-level mean %MBMI was also included as a covariate at Level 2 to disaggregate within-person versus between-person effects of %MBMI.

Within-Session Habituation to Daily Food Exposure—To determine whether the difference between pre-exposure and post-exposure SUDS changed over time, we estimated another multilevel model where observations were nested within participants. The SUDS difference was the outcome, time was the predictor, and %MBMI was the covariate. We also performed a supplementary analysis where we computed each participant's average pre-exposure SUDS and post-exposure SUDS, and used a paired samples *t*-test to determine whether post-exposure SUDS were, on average, lower than pre-exposure SUDS.

Participants completed between 4 and 52 daily food exposures throughout treatment; the average number of food exposures was 22.54 (*SD* = 10.82; *median* = 21). Given Level 2 and Level 1 sample sizes, at a target level of power ≥ 80%, we are powered to detect effect sizes ≥ approximately .15 (Arend & Schäfer, 2019).

Between-Session and Within-Session Habituation as Predictors of Improvements in Weight and ED Symptomatology—Five models were estimated to investigate between-session habituation and within-session habituation as predictors, with a separate model for each of the following outcomes: %MBMI, ChEAT total score, and the three FOFM subscale scores. The %MBMI model included the average of the pre-exposure SUDS and SUDS difference for all program days prior to and including the date of that day's weight. The ChEAT and FOFM models included the average of the pre-exposure SUDS and SUDS difference for all program days over the three weeks prior to and including the date of the assessment administration.

Participants completed between two and six symptom assessments throughout the course of treatment; the average number of assessments was 3.68 (*SD* = 0.93, *median* = 4). Given Level 2 and Level 1 sample sizes, at a target level of power ≥ 80%, we are powered to detect effect sizes ≥ approximately .23 – .26 (Arend & Schäfer, 2019).

Results

Between-Session Habituation to Daily Food Exposure

As shown in Table 1, the fixed effect of time was negatively predictive of pre-exposure SUDS. This indicates that pre-exposure SUDS significantly decreased between sessions over time. On average, participants' SUDS decreased from a 6.93 before their first exposure to a 4.56 before their last exposure.

In contrast to expectations, the Level 1 effect of %MBMI on pre-exposure SUDS was significant and negative, indicating that, for a given participant, higher than average %MBMI (i.e., being further along in the weight restoration process) was associated with *lower* pre-exposure SUDS (i.e., greater between-session habituation).

Within-Session Habituation to Daily Food Exposure

The fixed effect of time did not significantly predict the difference between pre- versus post-exposure SUDS (see Table 1), suggesting that within-session habituation did not occur. The Level 1 effect of %MBMI on SUDS difference was not significant, indicating that weight restoration did not impact within-session habituation.

The supplemental paired samples *t*-test revealed that the average pre-exposure SUDS (*mean* = 6.19) and average post-exposure SUDS (*mean* = 6.02) did not significantly differ, [$t(53) = 1.78$, $p = .08$].

Between-Session and Within-Session Habituation as Predictors of Weight Gain and Improvements in ED Symptomatology

As shown in Table 2, time in treatment was independently associated with significant %MBMI increases and significant ChEAT and FOFM decreases. On average, participants' %MBMI increased from 92.14% to 101.07% over the course of treatment. Participants with AN experienced a %MBMI increase from 86.56% to 96.89%.

In line with hypotheses, the Level 1 effect of pre-exposure SUDS was significant and negatively related to %MBMI over time. Additionally, the Level 1 effect of pre-exposure SUDS was significant and positively predictive of ChEAT and FOFM scores over time.

Consistent with hypotheses, the Level 1 effect of SUDS difference was not significantly associated with any treatment outcome.

Discussion

Results from this open trial generally supported our hypotheses. Analyses revealed significant decreases in pre-exposure SUDS over time, providing preliminary evidence for

between-session habituation across daily exposure sessions. Also in line with hypotheses, participants did not experience within-session habituation, defined as decreases from pre-exposure SUDS to post-exposure SUDS during food exposure trials. These findings add to a growing body of research indicating that individuals with EDs do not reliably experience within-session habituation during food exposure (e.g., Cardi et al., 2019; Carter et al., 2002; Levinson et al., 2019).

Results from analyses investigating treatment outcomes converge with prior research from our PHP (Bryson et al., 2018; Ornstein et al., 2017), and indicated that our exposure-oriented treatment program resulted in improvements for adolescents with AN and other EDs, including weight gain, and decreases in fear of food and other ED symptomatology. Adding to this prior evidence, we found that between-session habituation predicted improvements in these treatment outcomes. Within-session habituation, on the other hand, did not significantly predict any of these outcomes. These findings appear to challenge at least one tenet of the original emotional processing theory (Foa & Kozak, 1986) – the importance of within-session habituation – which may not be a critical mechanism of food exposure when treating adolescents with EDs, at least when within-session habituation is defined as change in SUDS from before eating a feared food to after eating a feared food. In contrast, our findings suggest that between-session habituation across food exposure trials may be a mechanism involved in extinction learning. However, given that this study was an open trial, we cannot conclude that food exposure is driving decreases in SUDS ratings across time. To answer this question, further research should use randomized controlled designs that compare exposure therapy to an alternative treatment.

Likewise, it is unclear whether between-session habituation is a *mechanism* of change, or merely a *marker* of extinction learning. Inhibitory learning, which is an alternative to the fear habituation model, suggests that violating expectancies about a feared stimulus and tolerating fear are more important than fear reduction (Craske et al., 2008). Preliminary evidence supports the effectiveness of treatments for EDs that adhere to principles of inhibitory learning, such as the ten-session version of CBT for EDs, which aims to maximize patients' anxiety rather than achieve habituation using a gradual exposure hierarchy (Waller et al., 2019; Waller & Raykos, 2019). To better understand habituation and inhibitory learning as mechanisms of change in exposure therapy for EDs, future research should measure and compare these putative mechanisms. For example, participants could be asked to report the following across food exposure trials: (1) SUDS ratings to assess between-session habituation; (2) the likelihood that a feared outcome will occur on a 0 – 100 scale to assess expectancy violation; and (3) confidence in one's ability to tolerate exposure on a 0 – 100 scale to assess fear tolerance (Deacon et al., 2013).

In contrast to expectations, %MBMI significantly predicted reductions in daily pre-exposure SUDS, indicating that weight gain in treatment was associated with *greater* between-session habituation. These findings suggest that weight gain does not interfere with extinction learning, and may in fact *improve* learning during food exposure. There are several possible explanations for this finding. Malnutrition and semi-starvation in individuals with AN and other EDs are associated with neurocognitive impairment (Pender et al., 2014; Zwipp et al., 2014) and low estrogen availability (Graham & Milad, 2013; Shufelt et al., 2017), both of

which may limit the effectiveness of exposure therapy and other psychological treatments (Schaumberg et al., 2021). Consequently, exposure therapy may have been more effective for participants who gained more weight in treatment. In view of research supporting weight gain early in treatment as a predictor of positive outcomes (Linardon et al., 2016), there may be other variables driving both weight gain and decreases in self-reported SUDS, such as high parental self-efficacy (Byrne et al., 2015; Robinson et al., 2013) or strong internal motivation to recover (Vall & Wade, 2015). Additionally, food exposure may challenge beliefs about weight gain, even as individuals are gaining weight, by violating expectancies about the *degree* or *rate* of weight gain (Schaumberg et al., 2021). Moreover, weight gain itself may serve as a form of exposure that violates beliefs about the consequences of weight gain (Levinson et al., 2019), such as “my life will be worse off if I gain weight.”

This open trial has a number of limitations that should be considered when interpreting these findings. Similar to prior research (e.g., Farrell et al., 2019a; Hildebrandt et al., 2013), food exposure was one ingredient of a larger treatment program, and we did not have a control group. Thus, we cannot conclude whether our findings are specifically related to food exposure, other aspects of treatment, and/or other non-treatment variables (e.g., passage of time). Although larger than prior studies investigating the specific effects of food exposure in adolescents with EDs, our sample size of 54 participants was insufficient to detect very small effects. While exposure was a prominent feature of our PHP, the program included other treatments provided by an interdisciplinary team of therapists, physicians, psychiatrists, and dietitians. Without control or comparison groups, we cannot conclude that improvements are specifically related to the effects of food exposure. Our sample was predominantly White, female, and received treatment at a specialized clinic in an academic medical center. Results may not generalize to more diverse populations, older patients, or different treatment settings. With the exception of body weight, we relied on subjective self-report questionnaires to assess distress levels and ED symptomatology. Future research should consider using multimodal methods to evaluate the efficacy of exposure therapy at targeting fear and anxiety, including behavioral, physiological, and neurobiological measures. Participants provided self-report distress ratings immediately before and after eating feared foods, and we did not track SUDS ratings within the session or during longer time intervals following food exposure. Thus, our definition of within-session habituation is limited to this pre-eating to post-eating interval, and we did not capture potential habituation that occurred in the hours following food exposure. Future research should consider tracking SUDS ratings at multiple intervals throughout, and for longer periods following, exposure trials.

Despite these limitations, our findings provide support for integrating food exposure into PHPs for adolescents with EDs undergoing weight restoration. This study also adds to the limited existing knowledge about the relationship between habituation and outcomes of exposure-based treatment. We hope these findings inform larger, controlled studies with multimodal measures that aim to better understand the effectiveness of food exposure, and the mechanisms underlying exposure therapy’s efficacy, for individuals with EDs.

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Data Availability Statement:

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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Public Significance Statement:

This study provides some evidence that partial hospitalization programs that include food exposure may be useful for adolescents with eating disorders (EDs) who are experiencing weight restoration. Between-session habituation, but not within-session habituation, predicted favorable treatment outcomes. Further research is needed to determine whether clinicians can disregard within-session habituation when conducting food exposure for EDs, and understand the importance of between-session habituation as a potential mechanism of food exposure.

Table 1.

Random intercepts model results predicting change in pre-exposure SUDS over time (i.e., between-session habituation) and change in SUDS difference over time (i.e., within-session habituation)

	B **(95% CI)**	***SE***	***t***	***p***
Pre-exposure SUDS				
Intercept	1.63 (1.87, 2.80)	2.88	0.57	.57
Day	−0.01 (−0.03, −0.001)	0.01	−2.18	.03
%MBMI group-mean centered	−0.09 (−0.15, −0.03)	0.03	−2.88	.004
%MBMI group mean	0.05 (−0.01 0.11)	0.03	1.72	.09
SUDS difference				
Intercept	0.82 (−0.84, 2.48)	0.85	0.97	.34
Day	−0.005 (−0.01, 0.002)	0.004	−1.44	.15
%MBMI group-mean centered	0.02 (−0.01, 0.06)	0.02	1.33	.19
%MBMI group mean	−0.01 (−0.02, 0.01)	0.01	−0.79	.43

Day = centered at 0, indicating day of intake; %MBMI = percent median body mass index; %MBMI group-mean centered = group-mean (i.e., person-mean) centered %MBMI (level 1 variable); %MBMI group mean = group (i.e., person) %MBMI mean used to compute group-mean centered %MBMI (level 2 variable)

Table 2.

Random intercepts model results for pre-exposure SUDS (i.e., between-session habituation) and SUDS difference (i.e., within-session habituation) predicting change in %MBMI, eating disorder symptoms, and fear of food

	***B* (95% CI)**	***SE***	***t***	***p***
%MBMI				
Intercept	85.99 (77.29, 94.69)	4.44	19.17	< .001
Day	0.17 (0.16, 0.18)	0.004	40.67	< .001
SUDS pre (L1)	−0.15 (−0.24, −0.06)	0.05	−3.32	< .001
SUDS pre (L2)	1.30 (−0.02, 2.61)	0.68	1.92	.06
SUDS difference (L1)	0.06 (−0.09, 0.21)	0.08	0.82	.41
SUDS difference (L2)	−0.35 (−4.41, 3.70)	2.09	−0.17	.87
ChEAT Total				
Intercept	0.18 (−10.04, 10.44)	5.28	0.03	.97
Day	−0.22 (−0.29, −0.15)	0.03	−6.42	< .001
SUDS pre (L1)	2.14 (1.10, 3.17)	0.53	4.03	< .001
SUDS pre (L2)	5.68 (4.15, 7.20)	0.79	7.23	< .001
SUDS difference (L1)	−0.92 (−2.66, 0.82)	0.89	−1.03	.31
SUDS difference (L2)	−0.22 (−4.50, 4.08)	2.21	−0.10	.92
FOFM Anxiety about Eating				
Intercept	0.82 (−0.22, 1.86)	0.54	1.53	.13
Day	−0.02 (−0.03, −0.01)	0.004	−4.06	< .001
SUDS pre (L1)	0.40 (0.28, 0.51)	0.06	6.73	< .001
SUDS pre (L2)	0.63 (0.47, 0.78)	0.08	7.84	< .001
SUDS difference (L1)	−0.07 (−0.26, 0.12)	0.10	−0.71	.48
SUDS difference (L2)	−0.11 (−0.54, 0.33)	0.22	−0.48	.64
FOFM Food Avoidance Behaviors				
Intercept	0.74 (−0.21, 1.70)	0.49	1.51	.14
Day	−0.01 (−0.02, −0.007)	0.003	−4.20	< .001
SUDS pre (L1)	0.33 (0.23, 0.43)	0.05	6.61	< .001
SUDS pre (L2)	0.49 (0.34, 0.63)	0.07	6.59	< .001
SUDS difference (L1)	0.00 (−0.16, 0.17)	0.08	0.03	.97
SUDS difference (L2)	−0.05 (−0.45, 0.35)	0.21	−0.25	.80
FOFM Feared Concerns				
Intercept	0.93 (0.01, 1.86)	0.48	1.96	.05
Day	−0.01 (−0.02, −0.008)	0.003	−4.36	< .001
SUDS pre (L1)	0.24 (0.13, 0.34)	0.05	4.37	< .001
SUDS pre (L2)	0.60 (0.46, 0.73)	0.07	8.40	< .001
SUDS difference (L1)	−0.08 (−0.26, 0.11)	0.09	−0.81	.42
SUDS difference (L2)	−0.20 (−0.60, 0.19)	0.20	−0.99	.33

Day = centered at 0, indicating day of intake; %MBMI = percent median body mass index; SUDS = subjective units of distress; SUDS pre L1 = participant-mean centered pre-exposure SUDS (level 1 variable); SUDS pre L2 = participant mean pre-exposure SUDS (level 2 variable); SUDS

difference L1 = participant-mean centered SUDS difference (level 1 variable); SUDS difference L2 = participant mean SUDS difference (level 2 variable); ChEAT = Children's Eating Attitudes Test; FOFM = Fear of Food Measure