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# Executive function differences as a function of parent-reported binge eating and weight: Results from the adolescent brain cognitive development study

Kaitlin N. Rozzell-Voss[1] | Patrycja Klimek-Johnson[2] | Dawn M. Eichen[3] | Tiffany A. Brown[4] | Aaron J. Blashill[5,6]

[1]University of Hawaiʻi at Mānoa, Honolulu, Hawaii, USA

[2]San Francisco VA Medical Center/University of California, San Francisco, California, USA

[3]University of California, San Diego, California, USA

[4]Auburn University, Auburn, Alabama, USA

[5]San Diego State University, San Diego, California, USA

[6]San Diego State University/University of California, San Diego Joint Doctoral Program in Clinical Psychology, San Diego, California, USA

**Correspondence**
Aaron J. Blashill, Department of Psychology, San Diego State University, 6363 Alvarado Court, Suite 101, San Diego, CA 92120, USA.
Email: ajblashill@sdsu.edu

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## Abstract

**Background:** Binge eating is a relatively common disordered eating behavior among children, and is associated with poor health outcomes. Executive function (EF)—higher order cognitive abilities related to planning and impulse control—may be implicated in both binge eating and pediatric obesity. Although EF deficits are evident among individuals with obesity and/or binge eating, findings are mixed across the lifespan.

**Methods:** The present study examined differences in EF among children with varying weight statuses and parent-reported binge eating. The sample included 10,017 children from the Adolescent Brain Cognitive Development study, aged 9–10 years.

**Results:** Children with parent-reported binge eating—either with overweight/obesity or normal weight—had significantly lower EF than those with no binge eating and a normal weight status but did not differ from those with no binge eating and overweight/obesity. Children with no binge eating and overweight/obesity also had statistically significantly lower EF than those with normal weight status. Although all significant differences between groups were negligible to very small, results may indicate similar neurocognitive profiles among children with binge eating and those with overweight/obesity.

**Conclusions:** Alterations in EF among children with binge eating may not be solely related to weight-specific factors, as significant differences also emerged among children with normal weight status, with versus without parent-reported binge eating. Future research is needed to understand temporal associations between

Data used in the preparation of this article were obtained from the Adolescent Brain Cognitive Development (ABCD) Study (https://abcdstudy.org), held in the NIMH Data Archive (NDA). This is a multisite, longitudinal study designed to recruit more than 10,000 children aged 9–10 and follow them over 10 years into early adulthood. The ABCD Study is supported by the National Institutes of Health and additional federal partners *under award numbers U01DA041022, U01DA041028, U01DA041048, U01DA041089, U01DA041106, U01DA041117, U01DA041120, U01DA041134, U01DA041148, U01DA041156, U01DA041174, U24DA041123, and U24DA041147*. A full list of supporters is available at https://abcdstudy.org/nih-collaborators. A listing of participating sites and a complete listing of the study investigators can be found at https://abcdstudy.org/principal-investigators.html. ABCD consortium investigators designed and implemented the study and/or provided data but did not necessarily participate in analysis or writing of this report. This manuscript reflects the views of the authors and may not reflect the opinions or views of the NIH or ABCD consortium investigators. The ABCD data repository grows and changes over time. The ABCD data used in this report came from 10.15154/1412097. DOIs can be found at https://ndar.nih.gov/study.html?id=500.

obesity, disordered eating, and neurocognition in children using multi-informant methods for assessing binge eating.

**KEYWORDS**
binge eating, executive function, loss of control eating, neurocognition, overweight/obesity

## 1 | INTRODUCTION

Overweight/obesity has been consistently associated with binge eating (BE) behavior—overeating accompanied by a sense of loss of control (LOC; e.g., refs [1,2]). Comorbidities of obesity, particularly childhood obesity, include metabolic and cardiovascular disorders and decreased quality of life.[3] LOC eating in children may lead to more severe eating pathology later in life.[4,5] Neurocognitive deficits, particularly executive function (EF) deficits (e.g., inhibitory control and set-shifting), are evident in individuals with overweight/obesity across the lifespan.[6–8] Moreover, recent longitudinal research has demonstrated that higher EF in children predicts lower body mass index (BMI) over time.[9] EF deficits are also evident among individuals with BE or LOC eating.[10,11] BE may explain the prospective association between EF and weight gain from middle childhood to adolescence.[12]

Among adults, those who engage in binge eating appear to perform worse on some specific EF tasks. One study found that adults with binge eating disorder (BED) and obesity had worse working memory and problem solving skills than adults with obesity, though performance on other EF tasks did not differ.[13] A systematic review of EF in adults with BED also found that compared to adults with obesity and no binge eating, adults with BED had significantly worse working memory, though results were mixed regarding differences in inhibitory control, cognitive flexibility, and decision making.[14] Across studies among adults with BED and/or obesity, the degree of EF impairment and in various EF tasks are inconsistent. Thus, the associations between overweight/obesity, BE, and neurocognition remain unclear. Investigations of these associations in pediatric samples may help better characterize the independent roles of weight status and BE on neurocognitive functioning and help to prevent and/or manage morbidity associated with pediatric obesity.

Investigations of associations between LOC eating, pediatric overweight/obesity, and EF are limited. Children with self-reported LOC eating and overweight demonstrated poorer working memory than children with overweight but no LOC eating; however, children with overweight but no LOC eating performed worse than children with normal weight on measures of planning and working memory.[15] Moreover, there were no significant group differences in measures of cognitive control/attention, cognitive flexibility, and decision-making. Thus, it remains unclear how children with BE and normal weight perform relative to those with overweight/obesity. Although overweight/obesity and BE are separate constructs with their own unique physical and mental health outcomes, they often co-occur.[16] It is necessary to examine differences among these distinct and overlapping groups on various outcomes, including EF. EF performance in childhood may help to highlight the underlying etiology of overweight/obesity and BE, and potentially inform targeted prevention and intervention efforts. Additional research on EF in pediatric samples is needed to better identify risk factors for obesity-related health outcomes and eating disorders.

Thus, the present study aimed to investigate differences in EF across four groups of 9–10-year-old children: those with no parent-reported BE + normal weight (NW); those with parent-reported BE + normal weight (NW + BE); those with no parent-reported BE + overweight/obesity (OW); and those with parent-reported BE + overweight/obesity (OW + BE). It was hypothesized that children in the three groups, NW + BE, OW, and OW + BE, would have lower EF than children in the NW group. However, given the limited and mixed literature on group differences in EF between OW and OW + BE, and NW + BE compared to OW or OW + BE, no directional hypotheses were proposed regarding differences between these three groups (OW, OW + BE, NW + BE).

## 2 | METHOD

### 2.1 | Participants

Participants were 11,875 children, aged 9–10 years and their caregivers from the Adolescent Brain and Cognitive Development (ABCD) study, a large longitudinal study in the United States (initial baseline data; Wave 1; Annual Release 2.0; ref.[17]), collected in 2016–2017. Details on the ABCD study sample are reported in Table 1. The main aims of the ABCD study are to investigate the interaction between various childhood experiences (e.g., physical exercise, substance use) and their influence on health outcomes.[18] Participants were recruited through selected schools located near the 21 data collection sites across the United States. To our knowledge, this is the first study to assess the associations among EF, overweight/obesity, and parent-reported BE among the baseline ABCD sample.

### 2.2 | Measures

#### 2.2.1 | Parent-reported BE

BE was evaluated using the computerized Kiddie Schedule for Affective Disorders and Schizophrenia based on DSM-5 criteria

TABLE 1 Characteristics of children in the sample.

| | Binge eating overweight/obesity (OW + BE) (n = 406) | | | Overweight/obesity (OW) (n = 3121) | | | Binge eating normal weight (NW + BE) (n = 125) | | | Normal weight (NW) (n = 6365) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Race and ethnicity[a] | *n* | % | $N_p$ | *n* | % | $N_p$ | *n* | % | $N_p$ | *n* | % | $N_p$ |
| White | 154 | 37.9 | 119,267 | 1169 | 37.5 | 894,844 | 59 | 47.6 | 41,318 | 3842 | 60.4 | 2,573,835 |
| Black | 73 | 18 | 45,862 | 690 | 22.2 | 426,876 | 22 | 17.7 | 14,446 | 701 | 11.0 | 427,908 |
| Asian/Pacific Islander | 7 | 1.7 | 10,562 | 43 | 1.4 | 54,129 | 2 | 1.6 | 3528 | 151 | 2.4 | 790,329 |
| Hispanic/Latino/a | 131 | 32.3 | 111,870 | 877 | 28.2 | 724,593 | 30 | 24.2 | 24,868 | 1005 | 15.8 | 172,161 |
| Multiracial | 41 | 10.1 | 17,695 | 336 | 10.8 | 166,479 | 11 | 8.9 | 3254 | 659 | 10.4 | 286,677 |
| Birth sex | | | | | | | | | | | | |
| Male | 238 | 58.6 | 177,287 | 1620 | 51.9 | 1,165,955 | 65 | 52.0 | 45,240 | 3295 | 51.8 | 2,153,980 |
| Female | 167 | 41.1 | 126,478 | 1501 | 48.1 | 1,103,771 | 60 | 48.0 | 42,966 | 3069 | 48.2 | 2,100,421 |
| Intersex | 1 | 0.2 | 1489 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | <0.01 | 369 |
| Gender | | | | | | | | | | | | |
| Male | 239 | 58.9 | 177,906 | 1618 | 51.8 | 1,164,919 | 65 | 52.0 | 45,240 | 3291 | 51.7 | 2,151,054 |
| Female | 166 | 41.1 | 126,550 | 1495 | 47.9 | 1,099,234 | 60 | 48.0 | 42,966 | 3067 | 48.2 | 2,098,786 |
| Transgender | 0 | 0 | 0 | 2 | 0.1 | 2075 | 0 | 0 | 0 | 2 | <0.01 | 498 |
| Gender queer | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | <0.01 | 232 |
| Other identity | 0 | 0 | 0 | 3 | 0.1 | 1103 | 0 | 0 | 0 | 1 | <0.01 | 944 |
| Unknown/refused to answer | 0 | 0 | 0 | 3 | 0.1 | 2396 | 0 | 0 | 0 | 3 | <0.01 | 3255 |
| | | *M*(*SD*) | *M*(*SE*) | | *M*(*SD*) | *M*(*SE*) | | *M*(*SD*) | *M*(*SE*) | | *M*(*SD*) | *M*(*SE*) |
| BMI percentile | | 95.99 (3.63) | 96.05 (0.20) | | 94.07 (4.21) | 94.10 (0.11) | | 57.32 (21.54) | 57.81 (2.36) | | 49.50 (2.27) | 49.89 (0.75) |

*Note*: BMI percentile is reported as the sample mean and standard deviation and population estimate mean and standard error.

Abbreviations: BMI, body mass index; $N_p$, estimated within-group population size.

[a]Missing values for *n* = 7 of those with normal weight, *n* = 1 of children with binge-eating and normal weight, and *n* = 6 groups with overweight/obesity.

(KSADS-5; ref.[19]) using the parent-reported item: "In the past 2 weeks, has your child had eating binges, when they lost control of their eating and ate way more than they needed because they were unable to stop themselves from eating?" Given that parents and/or guardians are typically consistent observers of their child's eating behaviors, there was a solid rationale for obtaining data on child BE from parents.[20] Although parent-reported BE demonstrated limited validity in one previous pediatric BE study,[21] another study demonstrated that child and parent reports on the Spanish K-SADS Lifetime Version 5 have high agreement on the BED module.[22] Furthermore, a different study using a similar single item assessing BE found that parent report of BE was significantly related to child BE.[23] This approach has been used across several research groups in previous studies both utilizing the ABCD dataset[24] and outside of the study.[25,26] Binary responses included in the ABCD dataset were the presence of any parent-reported BE (coded as 1) or absence of BE (coded as 0).

### 2.2.2 | Body mass index

Average standing height and weight were used to calculate BMI ($kg/m^2$). CDC 2000 Growth Chart SAS software was used to generate *z*-scores and percentiles based on sex, age, height, and weight.[27] Per the CDC guidelines, BMIz less than −4 or greater than 4 was excluded. BMI percentile was used to categorize individuals based on weight status: individuals at ≥85% were categorized as having overweight/obesity and individuals 5 to <85% were categorized as "normal" weight per CDC guidelines (Table 2)

### 2.2.3 | Executive function

EF was measured using the National Institutes of Health Toolbox List Sorting Working Memory Test (TLSWMT), Flanker Task (TFT), and

**TABLE 2** Means, standard deviations of executive function tasks by body mass index group status.

| | Binge eating overweight/obesity (OW + BE) (*n* = 406) | | Overweight/obesity (OW) (*n* = 3121) | | Binge eating normal weight (NW + BE) (*n* = 125) | | Normal weight (NW) (*n* = 6365) | |
|---|---|---|---|---|---|---|---|---|
| | *M* | *SD* | *M* | *SD* | *M* | *SD* | *M* | *SD* |
| Executive function | | | | | | | | |
| TDCCST | 94.13 | 20.00 | 94.50 | 23.21 | 91.37 | 16.25 | 97.38 | 37.57 |
| Flanker task | 93.51 | 20.19 | 94.05 | 23.51 | 92.15 | 14.30 | 95.94 | 31.35 |
| TLSWMT | 97.65 | 14.55 | 98.35 | 29.07 | 97.55 | 15.61 | 101.00 | 46.30 |

Abbreviations: TDCCST, toolbox dimensional change card sort task; TLSWMT, toolbox list sorting working memory test.

Dimensional Change Card Sort Task (TDCCST), described in further detail below.

*Working memory*

The Toolbox List Sorting Working Memory Test (TLSWMT) was used as a measure of working memory and information processing.[28] Participants viewed a series of visual stimuli (e.g., food, animals) simultaneously accompanied by auditory and text input of the given stimuli.[28] There were two types of trials among a total of 26 trials: (a) one-category trials with stimuli from a single category (e.g., food *or* animals); and (b) two-category trial with stimuli from two categories (e.g., food *and* animals). After viewing stimuli, participants were instructed to verbally recite the presented items in real-life size order from smallest to largest. During the two-category trials, participants were asked to order the stimuli within each category and recite them (e.g., food first and then animals). Raw accuracy scores were calculated based on the total sets correct across both one-category and two-category trials.

*Cognitive control/attention*

The Toolbox Flanker Task (TFT) is a measure of executive function, attention, and inhibition.[29] During the task, participants were instructed to pay attention to the target (arrow) located in the middle of the iPad screen. The four flanking stimuli (arrows) were located along the same horizontal axis as the main target (two on left and two on right) and were either facing the same (congruent trial) or a different (incongruent trial) direction as the target arrow. Participants were instructed to push a button to indicate whether the target arrow was facing left or right. The task yielded both accuracy and reaction time (RT) scores, which were used to yield a composite score.

*Set-shifting*

The Toolbox Dimensional Change Card Sort Task (TDCCST) is a measure of set-shifting and cognitive flexibility.[29] In this task, participants were presented with two visual stimuli at the bottom, and the target stimulus above them. Then, they were instructed to choose one of the two bottom stimuli that matched the target stimuli by a given dimension (i.e., shape or color). The task comprised 40 trials, began with a block of trials with one-category sorting (e.g., color-matching), and moved onto trials with two-category sorting (i.e., alternating between categories). The task yielded both accuracy and RT score, which were combined as a composite score.

## 2.3 | Statistical analysis

Children with missing data on either parent-reported BE or BMI ($n = 167$), BMIz under −4 ($n = 46$), children with underweight ($n = 421$), and children who met criteria for current psychotic, bipolar, or substance/alcohol use disorders ($n = 1225$) were excluded from further analyses. The final sample ($N = 10{,}017$) was used to calculate population estimates ($N_P$) using complex sample analyses for children with parent-reported (a) BE+ overweight/obesity ($n = 406$; $N_P = 305{,}254$), (b) BE + normal weight ($n = 125$; $N_P = 88{,}205$), (c) no BE + overweight/obesity ($n = 3121$; $N_P = 2{,}269{,}725$), (d) no BE + normal weight ($n = 6365$; $N_P = 4{,}254{,}769$).

All analyses were completed using SPSS version 25.0. Complex samples general linear regression models[30] were used to test mean score differences between groups on all measures of EF, using population-based estimates to mirror the 2010 census. Given parental education is positively associated with EF[31] and negatively associated with BMI,[32] parental education was controlled for in follow-up sensitivity analyses in addition to demographic variables sex, race, and ethnicity. Sensitivity analyses including parental education, sex, and race/ethnicity showed results consistent with analyses that did not include covariates. Results are further detailed using models without covariates for parsimony.

Effect sizes were calculated using Cohen's *d* ($(M_a – M_b) \div SD_{pooled}$) with 0.1, 0.2, 0.4 and 0.6 constituting very small, small, medium, and large effect sizes, respectively.[33] The Benjamini-Hochberg (B-H) correction was applied to correct for Type I error, using a false discovery rate of 0.05.[34]

## 3 | RESULTS

Birth sex of children was 52.1% male and 47.9% female. Greater than 99.8% identified as cisgender. Children were 52.3% White, 14.8% Black, 2.0% Asian/Pacific Islander, 20.4% Hispanic/Latino, and 10.5% multiracial. Parents of the children endorsed a median family income of $75,000–$99,999, and the median education level was completion of a Bachelor's degree. Table 1 provides descriptive statistics across weight status groups.

Parental education had minimal to small correlations with all 3 measures of EF ($r_s$ = .15, .29, .20, $p$ < .001) and both BMI ($r_s$ = −22, $p$ < .001) and presence/absence of parent-reported binge eating ($r_s$ = −.06, $p$ < .001). However, controlling for parental education did not significantly change the model fit or magnitude of effects; thus, unadjusted models were reported. The B-H alpha correction procedure indicated that the appropriate critical value for 18 individual tests was .022.

Children with parent-reported BE—either OW + BE or NW + BE—had significantly lower EF than those in the NW group, although these differences were minimal to very small (see Table 3). Children with OW had statistically significantly lower EF than those with NW, although effect sizes were negligible (Cohen's *d*s < 0.10). However, no differences emerged between groups OW + BE and NW + BE versus OW or among OW + BE versus NW + BE.

## 4 | DISCUSSION

This study examined EF differences across four groups of 9–10 year old children with and without parent-reported BE and with and without overweight/obesity (NW, NW + BE, OW, OW + BE). The largest differences, albeit very small in effect size, emerged in EF performance (i.e., set-shifting, cognitive control/attention, working memory) between OW + BE and NW + BE groups compared to the NW group. Specifically, both OW + BE and NW + BE groups demonstrated lower set-shifting and working memory than the NW group. Further, no significant differences emerged between the NW + BE group versus the OW + BE group. Children with OW + BE or NW + BE also appeared to perform similarly to the OW group.

These results suggest that overweight/obesity does not explain or exacerbate lower EF performance evidenced in children with BE. In contrast, Goldschmidt et al.[15] found worse working memory performance among children with both self-reported LOC eating and overweight than children with overweight but no LOC eating. Consistent with previous research,[7,35] children with overweight/obesity and no parent-reported BE had lower EF than those with normal weight. Thus, present findings may suggest shared clinical features between overweight/obesity and BE in children, but that weight status may not have an additive effect on EF in children.

Alternatively, the impact of weight status may emerge later in development. For example, Goldschmidt et al.[15] found large differences in working memory among children aged 9–12 years with overweight and LOC eating compared to those with normal weight or overweight and no LOC eating. They also found small-to-medium but statistically nonsignificant differences (likely due to power) in set-shifting and cognitive control/attention. Thus, a comparison between this prior study and the present study may demonstrate that larger neurocognitive differences may emerge later in eating behavior development, or alternatively, as the effects of pediatric overweight/obesity are prolonged. These results may have implications for early intervention for pediatric obesity, given that

**TABLE 3** Comparisons of executive function performance by group status.

| | OW + BE versus OW | | | | | OW + BE versus NW + BE | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **Executive function measure** | **B** | **95% CI** | ***t*** | ***p*** | ***d*** | **B** | **95% CI** | ***t*** | ***p*** | ***d*** |
| TDCCST | −0.37 | [−2.37, 1.63] | −0.39 | .70 | 0.02 | 2.76 | [−1.29, 6.81] | 1.42 | .17 | 0.14 |
| Flanker | −0.54 | [−2.67, 1.58] | −0.53 | .60 | 0.02 | 1.37 | [−1.92, 4.65] | 0.87 | .40 | 0.07 |
| TLSWMT | −0.7 | [−2.03, 0.62] | −1.1 | .28 | 0.03 | 0.10 | [−2.70, 2.89] | 0.07 | .94 | 0.01 |
| | **OW + BE versus NW** | | | | | **NW + BE versus NW** | | | | |
| **Executive function measure** | **B** | **95% CI** | ***t*** | ***p*** | ***d*** | **B** | **95% CI** | ***t*** | ***p*** | ***d*** |
| TDCCST | −3.25 | [−5.32, −1.17] | −3.26 | .004 | 0.09 | −6.01 | [−9.22, −2.79] | −3.88 | <.001 | 0.16 |
| Flanker | −2.43 | [−4.59, −0.27] | −2.34 | .03 | 0.08 | −3.8 | [−6.15, −1.44] | −3.35 | .003 | 0.12 |
| TLSWMT | −3.35 | [−4.94, −1.77] | −4.4 | <.001 | 0.07 | −3.45 | [−5.96, −0.94] | −2.86 | .01 | 0.08 |
| | **NW + BE versus OW** | | | | | **OW versus NW** | | | | |
| **Executive function measure** | **B** | **95% CI** | ***t*** | ***p*** | ***d*** | **B** | **95% CI** | ***t*** | ***p*** | ***d*** |
| TDCCST | −3.13 | [−6.29, 0.03] | −2.06 | .05 | 0.14 | −2.88 | [−3.46, −2.29] | −10.22 | <.001 | 0.09 |
| Flanker | −1.91 | [−4.38, 0.56] | −1.61 | .12 | 0.08 | −1.89 | [−2.67, −1.11] | −5.03 | <.001 | 0.07 |
| TLSWMT | −0.8 | [−3.60, 2.00] | −0.59 | .56 | 0.03 | −2.65 | [−3.64, −1.65] | −5.54 | <.001 | 0.06 |

Abbreviations: NW, normal weight; NW + BE, normal weight and parent-reported binge eating; OW, overweight/obesity; OW + BE, overweight/obesity and parent-reported binge eating; TDCCST, toolbox dimensional change card sort task; TLSWMT, toolbox list sorting working memory test.

among 9–10 year-olds in the present study, overweight/obesity does not appear to compound the negative effects of binge eating on EF. However, the present study may have detected smaller differences due to utilizing a parent-reported and binary measurement of BE. Future research is therefore needed to replicate EF differences in young children with LOC eating and varying weight statuses.

The present study is not without limitations. Notably, this study utilized a single item to assess parent-reported binge eating, which may not accurately capture binge eating behavior among children due to the use of a singular item or because parents may be unaware of their child's engagement in binge eating. Moreover, the item used in the present study assesses binge eating via the presence of LOC combined with an objectively large amount of food; however, researchers have proposed that LOC eating, independent of the amount of food consumed, may be more indicative of future eating pathology later in life.[36] Additionally, prior research suggested that EF tasks that incorporate disorder-specific stimuli show more pronounced EF deficits among binge eating samples.[37] However, the present study used general EF tasks; thus, the incorporation of disorder-specific stimuli may have led to different findings (e.g., larger effect sizes). Nevertheless, notable strengths of the present study are the utilization of a large and diverse sample that mirrors the U.S. population, and the comparisons between all possible groups with and without overweight/obesity and with and without BE.

The current study demonstrated that children with parent-reported BE and those with overweight/obesity but no BE performed similarly across measures of EF. Results may reflect shared phenotypes across children with overweight/obesity and those who engage in BE. Moreover, results suggest that associations between BE and EF may not be solely related to obesity-related factors and overweight/obesity may not have an additive effect on EF among children with BE. Larger differences among groups with BE and/or overweight/obesity could emerge in later developmental stages of eating pathology, or results may be impacted by methodological limitations such as parent-reported BE. Longitudinal research with multi-informant and rigorous methods is needed to corroborate evidence in the present study. A better understanding of the risk and maintenance factors of pediatric overweight/obesity and BE will be crucial in informing early intervention.

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PK and AJB developed the study concept. All authors contributed to the study design. PK and KRV performed the data analysis and contributed to the interpretation of findings under the supervision of AJB. PK and KRV drafted the paper, and AJB, DE, and TAB provided critical revisions. All authors approved the final version of the paper for submission. Ms. Da Yeoun Moon contributed general support for early revisions of this manuscript. Author time for Dr. Eichen was supported by a grant from the National Institute of Diabetes and Digestive and Kidney Diseases of the National Institutes of Health (K23DK114480)

## CONFLICT OF INTEREST STATEMENT

We have no conflicts of interest to disclose.

## ORCID

*Kaitlin N. Rozzell-Voss* https://orcid.org/0000-0002-7386-3689
*Patrycja Klimek-Johnson* https://orcid.org/0000-0002-3134-7328
*Dawn M. Eichen* https://orcid.org/0000-0001-5619-2177
*Tiffany A. Brown* https://orcid.org/0000-0002-7349-7228
*Aaron J. Blashill* https://orcid.org/0000-0002-4727-3888

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