ORIGINAL ARTICLE



Prevalence and risk factors of disordered eating behavior in youth with hypertension disorders

Ella C. Perrin¹ · Hanna L. Ravi² · Gagana S. Borra · Andrew M. South ^{1,3,4,5}

Received: 21 November 2022 / Revised: 8 February 2023 / Accepted: 13 February 2023 / Published online: 17 May 2023 © The Author(s), under exclusive licence to International Pediatric Nephrology Association 2023

Abstract

Background Adolescents with certain health conditions requiring lifestyle management, such as diabetes mellitus, have higher disordered eating behavior (DEB) risk than the general adolescent population, but DEB is underdiagnosed and can lead to adverse health consequences. In youth with other conditions requiring lifestyle counseling such as hypertension (HTN), DEB prevalence and associated risk factors are unknown. We hypothesized that youth with HTN disorders would have higher DEB prevalence than the general adolescent population, and that obesity, chronic kidney disease (CKD), and less specialized lifestyle counseling would be associated with higher DEB risk.

Methods Prospective cross-sectional study of youth aged 11-18 years with HTN disorders. We excluded patients with diabetes mellitus, kidney failure or transplantation, or gastrostomy tube dependence. We collected data via surveys and electronic health record abstraction. We administered the validated SCOFF DEB screening questionnaire. We compared DEB prevalence using a one-sample *z*-test of proportions (p_0 =0.1) and estimated DEB risk by obesity, CKD, and lifestyle counseling source using multivariable generalized linear models.

Results Of 74 participants, 59% identified as male, 22% as Black or African American, and 36% as Hispanic or Latino; 58% had obesity and 26% had CKD. DEB prevalence was 28% (95% CI 18–39%, p < 0.001). CKD was associated with higher DEB prevalence (adjusted RR 2.17, 95% CL 1.09 to 4.32), but obesity and lifestyle counseling source were not.

Conclusions DEB prevalence is higher in youth with HTN disorders and comparable to other conditions requiring lifestyle counseling. Youth with HTN disorders may benefit from DEB screening.

 $\textbf{Keywords} \ \ Blood \ pressure \cdot Feeding \ and \ eating \ disorders \ of \ childhood \cdot Chronic \ kidney \ disease \cdot Lifestyle \ counseling \cdot Pediatric \ obesity$

Previous presentations: Portions of this study were presented in abstract form at the American Heart Association Hypertension Scientific Sessions in San Diego, CA, on September 7, 2022.

- Andrew M. South asouth@wakehealth.edu
- Department of Pediatrics, Section of Nephrology, Brenner Children's, Wake Forest University School of Medicine, One Medical Center Boulevard, Winston Salem, NC 27157, USA
- Department of Pediatrics, University of Colorado Anschutz Medical Campus, Aurora, CO, USA
- Division of Public Health Sciences, Department of Epidemiology and Prevention, Wake Forest University School of Medicine, Winston Salem, NC, USA
- Cardiovascular Sciences Center, Wake Forest University School of Medicine, Winston Salem, NC, USA
- Center for Biomedical Informatics, Wake Forest University School of Medicine, Winston Salem, NC, USA

Introduction

The incidence and prevalence of eating disorders (ED) in adolescents have increased in recent decades, and ED prevalence is now 1–3% in adolescent females [1]. ED can cause many serious short- and long-term health complications, including electrolyte disturbances, cardiomyopathy, and death due to starvation or suicide [1]. The prevalence of disordered eating behavior (DEB)—a broader category of unhealthy eating behaviors—is approximately 10–13% in adolescent and young adult males and females [1–3]. These estimates have been derived from several heterogeneous generalized populations, with variability across age, sex, race, ethnicity, and presence of obesity. As DEB increases the risk of concurrent ED or ED development [1], it is critically important for healthcare providers to screen for DEB so they



can identify and treat DEB earlier and prevent progression to ED and the related health complications [4, 5].

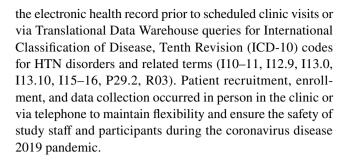
Several chronic health conditions are associated with increased risk of DEB. Type 1 diabetes mellitus has a strong relationship with developing ED or DEB in youth [6, 7]. Current or prior history of overweight or obesity has been associated with increased risk of ED, including both binge ED and restrictive ED [2, 8]. Increased emphasis in healthcare on identifying and addressing modifiable risk factors such as obesity may inadvertently contribute to the development of DEB or ED by placing an undue focus on weight and diet [5, 9]. There is currently limited data regarding which risk factors may make individuals more susceptible to developing DEB in response to counseling focused on weight loss [10]. Data are also emerging that adults with chronic conditions that place an emphasis on diet and physical activity—such as hypertension (HTN) and chronic kidney disease (CKD)—may have a higher DEB prevalence, although this relationship is incompletely described [10–12]. However, the relationship between HTN, a condition that is strongly associated with obesity but can be present in isolation, and DEB in pediatric populations has not been previously studied. In particular, it is unclear if HTN-related lifestyle counseling such as the DASH diet and avoiding sedentary behavior is associated with DEB [13]. Identifying risk factors associated with DEB can allow for more targeted screening, earlier diagnosis, and decreased adverse outcomes from ED [4].

The purpose of this novel study was to estimate DEB prevalence in youth with HTN disorders compared to what is reported in diverse samples of the general adolescent population and investigate whether obesity, CKD, or lifestyle counseling source are associated with higher DEB risk. We hypothesized that DEB prevalence would be higher than the general population with similar demographic characteristics to ours (estimated at 10%), that obesity and CKD would be associated with higher DEB risk, and that more specialized lifestyle counseling would be associated with lower DEB risk.

Methods

Study design and study population

This was a prospective cross-sectional study whose design was informed by power and sample size calculations a priori (see "Statistical analysis" below). Participants were recruited from the Hypertension and Nephrology Clinics at a tertiary care children's hospital from April 5, 2021, to February 9, 2022; these clinics provide care for all patients with HTN disorders and CKD in the referral region within our health-care system. Eligible patients were identified by screening



Ethical approval

The Institutional Review Board approved the study. We obtained written informed consent and assent from parents/ legal guardians and participants who were enrolled in person, and verbal consent and assent from those enrolled by telephone. Contact information for pediatric mental health services was provided to participants who screened positive for DEB.

Inclusion and exclusion criteria

Participants all had a diagnosis of a HTN disorder and had received subspecialty care in the clinics for at least 1 year. Exclusion criteria were age < 11 years or > 18 years, electronic health record-documented diagnosis of type 1 or type 2 diabetes mellitus, gastrostomy tube dependence, kidney failure with dialysis dependence, or kidney transplantation by ICD-10 codes, as well as inability to speak either English or Spanish.

Data collection

Demographic and clinical data were collected from a questionnaire administered to the participant and their parent or guardian, including self-identified sex, gender, race, ethnicity, insurance status, history of obesity or CKD, and history of lifestyle counseling received in the last year (any of nephrologist, nutritionist, and weight management clinic) (Fig. 1). We collected and reported race and ethnicity for descriptive purposes only. Participant DEB screening was conducted using the well-validated SCOFF questionnaire, a 5-question DEB screening survey that has 100% sensitivity and 88% specificity in adults, and has been validated in general pre-adolescent and adolescent populations, as well as in Spanish (Table 1) [14–17]. Additional participant clinical data were collected via electronic health record abstraction to validate self-reported responses, including age, height, weight, documentation of obesity or CKD by ICD-10 codes, blood pressure measurements, and serum creatinine most proximal to the most recent clinic visit, as well as source of lifestyle counseling received in the last year. We calculated blood pressure z-scores and classified blood pressure



Fig. 1 Demographic questionnaire

<u>Demographics</u>			
1. Participant sex (gender at birth): Male Female Other answer			
 Participant gender (gender the patient identifies with): Decline to answer 			
3. What race do you identify with?			
○ White ○ Black or African American ○ Asian ○ American Indian or Alaska Native			
○ Native Hawaiian or Other Pacific Islander ○ Multiracial ○ Other			
◯ Unknown ◯ Refused			
4. Are you of Hispanic, Latino/Latina, or Spanish Origin ethnicity?			
O NOT Hispanic, Latino/a, or Spanish Origin Hispanic, Latino/a, or Spanish Origin			
Other Ounknown Refused			
5. Insurance status:			
O Private Insurance O Public Insurance (Medicaid) Self Pay			
6. Do you have obesity (high weight)?: Y / N			
7. Do you have chronic kidney disease (kidney problems)?: Y / N			
8. From whom have you received lifestyle counseling in the past year (someone talking to			
you about diet and exercise modifications)? Check all that apply:			
○ Nephrologist (kidney doctor) ○ Nutritionist ○ Brenner FIT			

Table 1 Individual SCOFF scores by disordered eating behavior screen result

	Study population $N=74$	Positive DEB screen $N=21$ (28%)	Negative DEB screen N=53 (72%)
SCOFF score*	1 (0, 2)	2 (2, 2)	0 (0, 1)
Do you make yourself sick because you feel uncomfortably full?*	9 (12%)	7 (33%)	2 (4%)
Do you worry you have lost control over how much you eat?*	23 (31%)	15 (71%)	8 (15%)
Have you recently lost > 14 lb (6.3 kg) in a 3-month period?	11 (15%)	6 (29%)	5 (9%)
Do you believe yourself fat when others say you are too thin?*	15 (20%)	11 (52%)	4 (8%)
Would you say that food dominates your life?*	8 (11%)	7 (33%)	1 (2%)

N(%) or median (IOR)

categories per the American Academic of Pediatrics Clinical Practice Guideline [18]. Study data were collected and managed using a secure Research Electronic Data Capture tool hosted locally [19, 20].

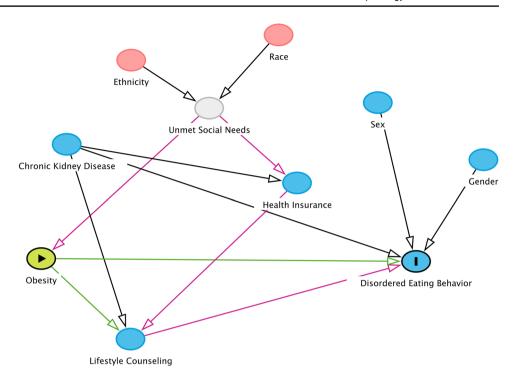
We defined a positive DEB screen as a SCOFF score of ≥ 2 (i.e., responded "yes" to at least two of five questions) per validated guidelines [14, 15]. We calculated body mass index and corresponding age- and sex-specific percentiles and z-scores per Centers for Disease Control and Prevention criteria. We estimated glomerular filtration rate by the

modified Schwartz equation [21]; the Clinical Laboratory uses a modified Jaffe assay traceable to isotope dilution mass spectrometry. For our primary analysis, we defined our exposures by several different criteria to mitigate misclassification bias and self-report bias. We defined obesity as follows: (1) self-report; (2) ICD-10 code documented diagnosis in the electronic health record; or (3) body mass index \geq 95th percentile for age and sex [22]. To account for discrepancies among definitions, we defined validated obesity as meeting at least two of the three criteria above in our primary



^{*}Between-group difference p < 0.05 by chi-square test, Fisher exact test, or Wilcoxon rank-sum test

Fig. 2 Example of the directed acyclic graph for the association between obesity and disordered eating behavior in youth with hypertension disorders. Green node with the arrow is the exposure, and blue node with the vertical bar is the outcome. Blue nodes are ancestors of the outcome, red nodes are potentially confounding factors, and the light gray node is an unmeasured variable. Green arrows are causal paths, red arrows are biasing non-causal paths, and black arrows are neither causal nor non-causal paths. The directed acyclic graph informs the minimally sufficient adjustment set containing insurance status and chronic kidney disease as variables to include in the multivariable model to close the biasing paths. Created using www.dagitty.net [27]



analysis. We defined CKD as follows: (1) self-report; (2) ICD-10 code documentation in the electronic health record; or (3) either estimated glomerular filtration rate < 60 ml/min/1.73 m² or \ge 60 ml/min/1.73 m² plus evidence of abnormal kidney structure or function per self-report or documentation according to Kidney Disease: Improving Global Outcomes pediatric criteria [23]. To account for discrepancies among definitions, we defined validated CKD as meeting the third criteria above in our primary analysis.

For our secondary analysis, we defined the source of lifestyle counseling that participants received on a three-value ordinal scale (nephrologist vs. nutritionist vs. weight management clinic) and as measured two ways: (1) self-report or (2) electronic health record documentation of clinic visits with the above providers in the last year; all participants received care from a nephrologist. We defined validated counseling from a nutritionist or the weight management clinic by presence of both self-report and documentation.

Statistical analysis

We reported descriptive statistics such as measures of central tendency, dispersion, and association including mean (SD), median (IQR), and frequencies with proportions. We compared groups with chi-square test, Fisher's exact test, two-sample *t*-test, ANOVA, Wilcoxon rank-sum test, or Kruskal–Wallis test, as appropriate. Power and sample size calculations determined a priori estimated that a sample size of 72 participants would provide over 80% power at a two-sided alpha of 0.05 for our primary analyses, based

on the one-sample z-test for binomial proportions (p_1 =0.3, p_0 =0.1) and the Pearson chi-square test for proportion of difference for the associations of obesity and CKD with DEB risk (p_0 =0.15, RR 3.0) [1–3, 24]. The reported prevalence estimates from these general adolescent populations were from diverse backgrounds with respect to age, sex, race, ethnicity, and obesity, including several with similar demographic characteristics to that seen in our clinics. Our secondary analysis estimating DEB risk by lifestyle counseling source was exploratory to inform the study design of future follow-up studies.

We compared DEB-positive screen prevalence in the study population to that reported in diverse samples of the general adolescent population using a one-sample z-test of proportions ($p_0 = 0.1$). For our primary and secondary analyses, we developed causal models to relate our variables of interest a priori, based on our clinical and epidemiological knowledge and the literature [25]. We then developed directed acyclic graphs for each exposure-outcome relationship to determine potential sources of bias and to close non-causal, biasing pathways by informing our minimally sufficient adjustment sets to include in each multivariable model (Fig. 2) [25, 26]. These included the following: insurance status and validated CKD for the obesity models; no adjustment set necessary for the CKD models; and validated obesity and validated CKD for the lifestyle counseling models. In our causal model, we believed that obesity was best defined as a potentially confounding factor as opposed to a modifying factor. We estimated the associations of obesity, CKD, and lifestyle counseling source with DEB risk using



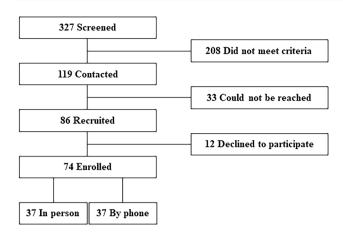


Fig. 3 Consort flow diagram demonstrating the number of patients approached during recruitment and the reasons why they were not enrolled

bivariate and multivariable generalized linear models with binomial or Poisson regression (binomial or Poisson distributions with log-link function) and reported RR with 95% CL.

We used our validated exposures in the primary and secondary analyses. In sensitivity analyses, we defined the obesity and CKD exposures as each individual measure of exposure separately, self-report or documentation, and, for lifestyle counseling, we defined the exposure as a composite of self-report or documentation, each individual measure separately, and then the highest value (i.e., most specific type) recorded (weight management clinic vs. nutritionist vs. nephrologist). We considered a two-sided alpha < 0.05 as statistically significant and used SAS Enterprise Guide Version 7.15 for Windows (Cary, NC) for all analyses.

Results

Among the 86 patients who met eligibility criteria and were contacted, 74 were enrolled (86%) (Fig. 3). Among the 74 enrolled participants, the mean age was 15.5 (*SD* 2.2) years, 59% identified as male gender, 22% identified as Black or African American, and 36% identified as Hispanic or Latino (Table 2). Validated obesity prevalence was 58% and validated CKD prevalence was 26% (Table 3). Validated nutritionist was present in 8% and validated weight management clinic was present in 5%.

DEB-positive screen prevalence was 28% and was statistically significantly higher than the general adolescent population (95% CI 18–39%, p < 0.001). Among the SCOFF questionnaire items, the questions "Do you worry you have lost control over how much you eat?" and "Do you believe yourself fat when others say you are too thin?" were most likely to be answered affirmatively in the screen-positive group

compared to the screen-negative group (71 vs. 15% and 52 vs. 8%, respectively, p < 0.05) (Table 1). Participants who screened positive for DEB were statistically significantly more likely to self-identify as females compared to those who screened negative (57 vs. 32%, p = 0.046) (Table 2). There was no difference in blood pressure between the two groups.

CKD was associated with a higher risk of DEB by our validated measure (RR 2.17, 95% CL 1.09 to 4.32) (Table 4, Fig. 4). However, validated obesity was not associated with a higher DEB risk (adjusted RR 1.67, 95% CL 0.64 to 4.36), nor was validated lifestyle counseling associated with a lower DEB risk (nutritionist adjusted RR 1.88, 95% CL 0.54 to 6.58; weight management adjusted RR 0.74, 95% CL 0.1 to 5.63).

Our sensitivity analysis confirmed that CKD was associated with a higher DEB risk by documentation (RR 2.89, 95% CL 1.51 to 5.51) but not self-report (RR 1.97, 95% CL 0.97 to 3.99) (Table 4). Our other exposure measures for obesity and lifestyle counseling were not associated with DEB risk.

Discussion

In this study of youth with HTN disorders who received care in a Hypertension Clinic or Nephrology Clinic for at least 1 year, we observed that DEB-positive screen prevalence was 28%, which was statistically significantly higher than that described in several samples of the general adolescent population (10–13%) [2, 3]. Our finding is comparable to the DEB prevalence observed in adolescents with type 1 diabetes mellitus (20–39%) [24, 28]. We also found that concurrent CKD was associated with more than double the risk of DEB compared to youth with HTN disorders who did not have CKD.

Several factors may contribute to the high prevalence of a positive screen for DEB in youth with HTN disorders. One factor may be a lack of appropriately specialized lifestyle counseling. Unsupervised weight loss and dieting in adolescents is associated with progression to unhealthy dieting practices and development of ED [2, 29]. Patients may receive harmful counseling and praise for weight loss from providers, family, or friends, reinforcing unhealthy behaviors [2, 6]. Adolescents with type 1 diabetes mellitus, another condition that requires intensive lifestyle counseling, are over twice as likely to develop DEB than their healthy peers [24, 28]. Although our study did not find a difference in DEB prevalence by source of lifestyle counseling, we note that this was an exploratory aim of our study given the low proportion of participants who received more-specialized counseling. Youth with HTN may also have an atypical ED presentation due to lack of low weight, which can lead to delayed diagnosis [5, 9, 29].



Table 2 Participant demographic and clinical characteristics by disordered eating behavior screen result

	Study population $N = 74$	Positive screen $N=21$ (28%)	Negative screen $N=53 (72\%)$
Age (years)	15.5 (2.2)	15.2 (2.0)	15.7 (2.3)
Female sex*	29 (39%)	12 (57%)	17 (32%)
Gender			
Female	29 (39%)	12 (57%)	17 (32%)
Male	44 (59%)	9 (43%)	35 (66%)
Decline to answer	1 (1%)	0 (0%)	1 (2%)
Race			
Asian	1 (1%)	0 (0%)	1 (2%)
Black or African American	16 (22%)	1 (5%)	15 (28%)
Multiracial	7 (9%)	2 (10%)	5 (9%)
Other	17 (23%)	6 (29%)	11 (21%)
Unknown	1 (1%)	0 (0%)	1 (2%)
White	32 (43%)	12 (57%)	20 (38%)
Ethnicity			
Hispanic or Latino	27 (36%)	9 (43%)	18 (34%)
Not Hispanic or Latino	46 (62%)	12 (57%)	34 (64%)
Unknown	1 (1%)	0 (0%)	1 (2%)
Insurance status			
Public insurance	47 (64%)	13 (62%)	34 (64%)
Private insurance	26 (35%)	7 (33%)	19 (36%)
Self-pay	1 (1%)	1 (5%)	0 (0%)
Height (cm)	165.0 (11.8)	162.3 (11.5)	166.0 (11.8)
Height z-score	0.0 (1.2)	-0.16 (1.22)	0.06 (1.2)
Weight (kg)	89.8 (34.8)	85.4 (29.0)	91.6 (36.9)
Weight z-score	1.7 (1.48)	1.65 (1.62)	1.72 (1.43)
Body mass index (kg/m ²)	32.6 (11.0)	31.8 (8.4)	32.9 (11.9)
Body mass index z-score	1.7 (1.15)	1.75 (1.15)	1.68 (1.16)
Body mass index classification*			
Class 3 severe obesity	19 (26%)	3 (14%)	16 (31%)
Class 2 severe obesity	15 (21%)	6 (29%)	9 (17%)
Class 1 obesity	12 (16%)	7 (33%)	5 (10%)
Overweight	11 (15%)	2 (10%)	9 (17%)
Normal body mass index	15 (21%)	2 (10%)	13 (25%)
Underweight	1 (1%)	1 (5%)	0 (0%)
Systolic BP (mmHg)	125.6 (15.5)	121.1 (13.8)	127.4 (15.9)
Diastolic BP (mmHg)	71.8 (9.1)	70.8 (9.3)	72.2 (9.0)
Systolic BP <i>z</i> -score, $n = 60$	1.15 (0.94)	0.82 (0.94)	1.32 (0.91)
Diastolic BP <i>z</i> -score, $n = 60$	0.61 (0.82)	0.63 (0.86)	0.6 (0.82)
BP severity			
Stage 2 hypertension	20 (27%)	5 (24%)	15 (28%)
Stage 1 hypertension	17 (23%)	2 (10%)	15 (28%)
Elevated BP	9 (12%)	3 (14%)	6 (11%)
Creatinine (mg/dl)	0.72 (0.23)	0.66 (0.2)	0.74 (0.24)
Estimated GFR (ml/min/1.73 m ²)	102.5 (29.9)	114.0 (47.1)	98.1 (18.9)

N (%) or mean (SD)



^{*}Between-group difference p < 0.05 by chi-square test or Fisher exact test. BP, blood pressure; GFR, glomerular filtration rate

Table 3 Obesity, chronic kidney disease, and lifestyle counseling status by disordered eating behavior screen result

	Study population $N = 74$	Positive screen $N=21 (28\%)$	Negative screen $N=53 (72\%)$
Obesity			
Validated	43 (58%)	14 (67%)	29 (55%)
Self-reported	35 (47%)	12 (57%)	23 (43%)
Documented	43 (58%)	13 (62%)	30 (57%)
Chronic kidney disease			
Validated*	19 (26%)	9 (43%)	10 (19%)
Stage III chronic kidney disease	3 (16%)	0 (0%)	3 (30%)
Stage II chronic kidney disease	5 (26%)	4 (44%)	1 (10%)
Stage I chronic kidney disease	11 (58%)	5 (56%)	6 (60%)
Self-reported	15 (20%)	7 (33%)	8 (15%)
Documented*	13 (18%)	8 (38%)	5 (9%)
Nutritionist lifestyle counseling			
Validated	6 (8%)	3 (14%)	3 (6%)
Self-reported or documented	28 (38%)	8 (38%)	20 (38%)
Self-reported	24 (32%)	7 (33%)	17 (32%)
Documented	10 (14%)	4 (19%)	6 (11%)
Weight management lifestyle counseli	ng		
Validated	4 (5%)	1 (5%)	3 (6%)
Self-reported or documented	13 (18%)	3 (14%)	10 (19%)
Self-reported	12 (16%)	3 (14%)	9 (17%)
Documented	5 (7%)	1 (5%)	4 (8%)
Highest level of self-reported counsels	ing		
Weight management clinic	12 (16%)	3 (14%)	9 (17%)
Nutritionist	19 (26%)	6 (29%)	13 (25%)
Nephrologist	32 (43%)	10 (48%)	22 (42%)
None	11 (15%)	2 (10%)	9 (17%)

N(%)

The high prevalence of concomitant obesity in youth with HTN may also contribute to the higher DEB prevalence compared to the general population, though we did not observe a difference in DEB prevalence based on obesity status by several measures despite 58% prevalence of obesity in our study population. It is possible that our cross-sectional design limited our ability to better investigate the potential role of obesity in DEB in our population. It is also possible that presence of obesity does not contribute to DEB risk in this population. Obesity has been consistently reported as a risk factor for ED, with one study of youth with obesity reporting a DEB prevalence of 9% in males and 20% in females [2, 30, 31]. Patients with ED and obesity often have a longer duration of illness before presentation, which can put them at higher risk of medical complications than those with normal or low weight [2, 9, 29]. ED and obesity also share many common risk factors, including body dissatisfaction, poor self-esteem, depression, and engaging in dieting behaviors [2, 28]. Some adolescents with obesity experience weight-shaming [2]. Youth with HTN may be experiencing

many of the same risk factors for ED development as other adolescents with obesity.

Another potential contributing factor is the negative effect of having a chronic health condition on self-esteem, especially during childhood. Youth with chronic health conditions including epilepsy and diabetes have reported lower self-esteem compared to healthy peers, which is an independent risk factor for ED [6, 32]. Our finding of a higher risk of DEB in youth with HTN disorders and CKD compared to HTN alone supports this theory. This is in line with recent evidence that there is a higher prevalence of DEB in adults with HTN and CKD [11, 12].

Lastly, it is possible that DEB may contribute to HTN development. In adults, mental health conditions including binge ED and bulimia nervosa have been associated with the subsequent development of HTN [11, 12, 33]. One study monitoring youth longitudinally at ages 6.5, 11.5, and 16 years found that those who screened positive for problematic eating attitudes at age 11.5 years were more likely to go on to develop HTN, although this relationship did not



^{*}Between-group difference p < 0.05 by chi-square test or Fisher exact test

Table 4 Unadjusted and adjusted associations of obesity, chronic kidney disease, and lifestyle counseling source with risk of positive screen for disordered eating behavior in youth with hypertension

	Unadjusted	Adjusted RR (95% CL)	
Model	RR (95% CL)		
Obesity ^a			
Validated	1.44 (0.66 to 3.15)	1.67 (0.64 to 4.36)	
Self-reported	1.49 (0.71 to 3.1)	1.88 (0.76 to 4.69)	
Documented	1.17 (0.55 to 2.48)	1.33 (0.53 to 3.34)	
Chronic kidney disease ^b			
Validated	2.17 (1.09 to 4.32)	2.17 (1.09 to 4.32)	
Self-reported	1.97 (0.97 to 3.99)	1.97 (0.97 to 3.99)	
Documented	2.89 (1.51 to 5.51)	2.89 (1.51 to 5.51)	
Nephrologist lifestyle counseling ^c			
Self-reported	1.35 (0.57 to 3.24)	1.19 (0.43 to 3.33)	
Nutritionist lifestyle counseling ^c			
Validated	1.89 (0.77 to 4.61)	1.88 (0.54 to 6.58)	
Self-reported or documented	1.01 (0.48 to 2.13)	0.93 (0.37 to 2.37)	
Self-reported	1.04 (0.48 to 2.24)	1.0 (0.39 to 2.56)	
Documented	1.51 (0.64 to 3.56)	1.41 (0.45 to 4.39)	
Weight management lifestyle counseling ^c			
Validated	0.88 (0.15 to 4.97)	0.74 (0.1 to 5.63)	
Self-reported or documented	0.78 (0.27 to 2.27)	0.71 (0.21 to 2.47)	
Self-reported	0.86 (0.3 to 2.47)	0.78 (0.22 to 2.68)	
Documented	0.69 (0.12 to 4.14)	0.61 (0.08 to 4.63)	
Highest level of self-reported counseling ^{c,d}			
Weight management clinic	0.9 (0.3 to 2.67)	0.8 (0.22 to 2.93)	
Nutritionist	1.13 (0.5 to 2.56)	1.1 (0.4 to 3.0)	

^aAdjusted for insurance status and chronic kidney disease

^dReferent group nephrologist or none

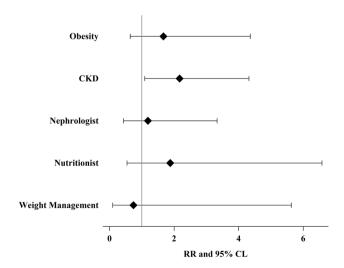


Fig. 4 Adjusted relative risk of positive disordered eating behavior screen by each validated exposure. Each validated exposure adjusted model is on the *Y*-axis with relative risks (black diamonds) and 95% confidence limits (black lines) on the *X*-axis. Vertical reference line of 1 indicates null risk

remain statistically significant when adjusted for body mass index at age 6.5 years [34].

Limitations of this study include its cross-sectional design, so causality and directionality cannot be inferred. It is possible that, despite our sample size calculations a priori, we underestimated the effect size estimates and thus would need a larger sample size to detect the true differences between exposure groups, or that there is no true difference in DEB-positive screen among patients with and without obesity or CKD. The small sample size and exploratory nature of the lifestyle counseling analysis likely contributed to insufficient power to detect an association of lifestyle counseling source with DEB. Given the three-level nature of this variable and the potential for measurement error, we did not power our study for this particular study aim. However, we specifically designed that aim to inform planned follow-up studies. We note that our other analyses were all adequately powered a priori, and we achieved our recruitment goals. We collected data via self-report, which can be subject to recall bias. We also collected data via chart review, which can be biased against care received outside



^bNo adjustment set necessary

^cAdjusted for obesity and chronic kidney disease

of the primary healthcare system. We attempted to account for these potential biases by using both self-report and electronic health record review to make the results more precise. There were also some discrepancies between self-reported diagnoses and lifestyle counseling sources and the electronic health record. However, our validated approach to defining our exposures helped mitigate misclassification bias. This study occurred in a single center with relatively homogeneous approaches to standards of care, so provider counseling style and precise content might not be generalizable. Furthermore, there likely exists variability across providers even within a particular subspecialty that could introduce bias. Our study population is not necessarily generalizable to the general adolescent population or individuals with other chronic medical conditions, but our research question did not attempt to investigate those direct comparisons. Planned follow-up studies will investigate these questions further, including with well-matched control populations. The patient populations that our clinics serve are very representative of our study population, and these participants would not have received subspecialty care elsewhere. The SCOFF survey is also brief and less sensitive for binge ED and ED not otherwise specified compared to anorexia nervosa and bulimia nervosa, and it has not been validated in populations with HTN.

Future studies should validate the SCOFF survey in youth with HTN to strengthen its applicability in research and the clinical setting. It would be important to further determine if DEB is associated with HTN disorder severity or use of anti-HTN medication. Future, larger, planned interventional studies with a more in-depth DEB screen at baseline and in follow-up could examine the role of provider counseling in DEB development. Randomization of participants to receive different types of lifestyle counseling with providers specifically trained about DEB could better define the role that lifestyle counseling plays in DEB development. Future studies should examine these relationships in youth derived from general populations, including to investigate whether these associations exist with DEB in youth with CKD without HTN disorders, and to what extent HTN disorders may mediate and/or moderate these associations.

35In conclusion, we observed that youth with HTN disorders had a high prevalence of screening positive for DEB and that CKD more than doubled this risk. Youth with HTN disorders may benefit from routine ED screening, though validation in this population is necessary. The American Academy of Pediatrics recommends that primary care providers conduct routine screening for EDs at annual wellness exams in this age group, and the American Academy of Child and Adolescent Psychiatric recommends mental health professionals conduct ED screening for all pre-adolescent and adolescent patients [35–37]. However, recent US Preventive Service Task Force guidelines reported insufficient evidence

to assess the benefits and harms of routine screening for EDs in adolescents [37]. Future recommendations should consider whether or not populations with specific risk factors such as HTN and CKD would benefit from routine ED screening.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00467-023-05921-1.

Acknowledgements We thank Ria Tilve, Beatrice Gar, and Abha Athawale for their assistance with data collection, all of whom have no conflicts of interest to disclose. We would like to acknowledge the biomedical informatics teams of the Wake Forest University School of Medicine Center for Biomedical Informatics and the Clinical and Translational Science Institute for their assistance with data extraction.

Funding The Wake Forest University School of Medicine Clinical and Translational Science Institute provided support (National Institutes of Health (NIH), National Center for Advancing Translational Sciences: UL1TR001420). AMS reports funding from the NIH National Heart, Lung, and Blood Institute: K23HL148394 and L40HL148910-2.

Data availability The datasets from this study are available from the corresponding author upon reasonable request.

Declarations

Conflict of interest The authors declare no competing interests.

References

- Rosen DS, American Academy of Pediatrics Committee on Adolescence (2010) Identification and management of eating disorders in children and adolescents. Pediatrics 126:1240–1253. https://doi.org/10.1542/peds.2010-2821
- Golden NH, Schneider M, Wood C, Committee On Nutrition, Committee On Adolescence, Section On Obesity (2016) Preventing obesity and eating disorders in adolescents. Pediatrics 138:e20161649. https://doi.org/10.1542/peds.2016-1649
- Irving LM, Neumark-Sztainer D (2002) Integrating the prevention of eating disorders and obesity: Feasible or futile? Prev Med 34:299–309. https://doi.org/10.1006/pmed.2001.0997
- Desocio JE, O'Toole JK, Nemirow SJ, Lukach ME, Magee MG (2007) Screening for childhood eating disorders in primary care. Prim Care Companion J Clin Psychiatry 9:16–20. https://doi.org/ 10.4088/pcc.v09n0103
- Taylor SA, Ditch S, Hansen S (2018) Identifying and preventing eating disorders in adolescent patients with obesity. Pediatr Ann 47:e232–e237. https://doi.org/10.3928/19382359-20180522-01
- Colton P, Olmsted M, Daneman D, Rydall A, Rodin G (2004) Disturbed eating behavior and eating disorders in preteen and early teenage girls with type 1 diabetes: a case-controlled study. Diabetes Care 27:1654–1659. https://doi.org/10.2337/diacare.27.7.1654
- Nielsen S (2002) Eating disorders in females with type 1 diabetes: an update of a meta-analysis. Eur Eat Disorders Rev 10:241–254. https://doi.org/10.1002/erv.474
- Sim LA, Lebow J, Billings M (2013) Eating disorders in adolescents with a history of obesity. Pediatrics 132:e1026-1030. https://doi.org/10.1542/peds.2012-3940
- Lebow J, Sim LA, Kransdorf LN (2015) Prevalence of a history of overweight and obesity in adolescents with restrictive eating disorders. J Adolesc Health 56:19–24. https://doi.org/10.1016/j. jadohealth.2014.06.005



- 10. Academy for Eating Disorders (2023) The Academy for Eating Disorders releases a statement on the recent American Academy of Pediatrics clinical practice guideline for weight-related care: first, do no harm. Newswise. https://www.newswise.com/articles/the-academy-for-eating-disorders-releases-a-statement-on-the-recent-american-academy-of-pediatrics-clinical-practice-guide line-for-weight-related-care-first-do-no-harm. Accessed 2 February 2023
- Gregg LP, Hedayati SS (2020) Treatment of psychiatric disorders in chronic kidney disease patients. In: Kimmel PL, Rosenberg ME (ed) Chronic Renal Disease, 2nd edn. Academic Press, pp 1123–1140. https://doi.org/10.1016/B978-0-12-815876-0.00068-1
- Stein DJ, Aguilar-Gaxiola S, Alonso J, Bruffaerts R, de Jonge P, Liu Z, Miguel Caldas-de-Almeida J, O'Neill S, Viana MC, Al-Hamzawi AO, Angermeyer MC, Benjet C, de Graaf R, Ferry F, Kovess-Masfety V, Levinson D, de Girolamo G, Florescu S, Hu C, Kawakami N, Maria Haro J, Piazza M, Posada-Villa J, Wojtyniak BJ, Xavier M, Lim CC, Kessler RC, Scott KM (2014) Associations between mental disorders and subsequent onset of hypertension. Gen Hosp Psychiatry 36:142–149. https://doi.org/10.1016/j. genhosppsych.2013.11.002
- Udo T, Grilo CM (2019) Psychiatric and medical correlates of DSM-5 eating disorders in a nationally representative sample of adults in the United States. Int J Eat Disord 52:42–50. https://doi. org/10.1002/eat.23004
- Luck AJ, Morgan JF, Reid F, O'Brien A, Brunton J, Price C, Perry L, Lacey JH (2002) The SCOFF questionnaire and clinical interview for eating disorders in general practice: comparitive study. BMJ 325:755–756. https://doi.org/10.1136/bmj.325.7367.755
- Morgan JF, Reid F, Lacey JH (1999) The SCOFF questionnaire: assessment of a new screening tool for eating disorders. BMJ 319:1467–1468. https://doi.org/10.1136/bmj.319.7223.1467
- Kutz AM, Marsh AG, Gunderson CG, Maguen S, Masheb RM (2020) Eating disorder screening: a systematic review and meta-analysis of diagnostic test characteristics of the SCOFF. J Gen Intern Med 35:885–893. https://doi.org/10.1007/ s11606-019-05478-6
- Garcia-Campayo J, Sanz-Carrillo C, Ibanez JA, Lou S, Solano V, Alda M (2005) Validation of the spanish version of the SCOFF questionnaire for the screening of eating disorders in primary care. J Psychosom Res 59:51–55. https://doi.org/10.1016/j.jpsychores. 2004.06.005
- Flynn JT, Kaelber DC, Baker-Smith CM, Blowey D, Carroll AE, Daniels SR, de Ferranti SD, Dionne JM, Falkner B, Flinn SK, Gidding SS, Goodwin C, Leu MG, Powers ME, Rea C, Samuels J, Simasek M, Thaker VV, Urbina EM; Subcommittee On Screening and Management of High Blood Pressure in Children (2017) Clinical practice guideline for screening and management of high blood pressure in children and adolescents. Pediatrics 140(3). https://doi.org/10.1542/peds.2017-1904
- Harris PA, Taylor R, Minor BL, Elliott V, Fernandez M, O'Neal L, McLeod L, Delacqua G, Delacqua F, Kirby J, Duda SN, RED-Cap Consortium (2019) The REDCap consortium: building an international community of software platform partners. J Biomed Inform 95:103208. https://doi.org/10.1016/j.jbi.2019.103208
- Harris PA, Taylor R, Thielke R, Payne J, Gonzalez N, Conde JG (2009) Research electronic data capture (REDCap)

 –a metadata-driven methodology and workflow process for providing

- translational research informatics support. J Biomed Inform 42:377–381. https://doi.org/10.1016/j.jbi.2008.08.010
- Schwartz GJ, Munoz A, Schneider MF, Mak RH, Kaskel F, Warady BA, Furth SL (2009) New equations to estimate GFR in children with CKD. J Am Soc Nephrol 20:629–637. https://doi. org/10.1681/ASN.2008030287
- Barlow SE, Expert Committee (2007) Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. Pediatrics 120 Suppl 4:S164–192. https://doi.org/10.1542/peds. 2007-2329C
- National Kidney Foundation (2002) K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis 39(2 Suppl 1):S1-266
- Bachle C, Lange K, Stahl-Pehe A, Castillo K, Scheuing N, Holl RW, Giani G, Rosenbauer J (2015) Symptoms of eating disorders and depression in emerging adults with early-onset, long-duration type 1 diabetes and their association with metabolic control. PLoS One 10:e0131027. https://doi.org/10.1371/journal.pone.0131027
- Cohen JB, D'Agostino McGowan L, Jensen ET, Rigdon J, South AM (2021) Evaluating sources of bias in observational studies of angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker use during COVID-19: beyond confounding. J Hypertens 39:795–805. https://doi.org/10.1097/HJH.0000000000002706
- Shrier I, Platt RW (2008) Reducing bias through directed acyclic graphs. BMC Med Res Methodol 8:70. https://doi.org/10.1186/ 1471-2288-8-70
- Textor J, Hardt J, Knuppel S (2011) Dagitty: a graphical tool for analyzing causal diagrams. Epidemiology 22:745. https://doi.org/ 10.1097/EDE.0b013e318225c2be
- Nilsson F, Madsen JOB, Jensen AK, Olsen BS, Johannesen J (2020) High prevalence of disordered eating behavior in danish children and adolescents with type 1 diabetes. Pediatr Diabetes 21:1043–1049. https://doi.org/10.1111/pedi.13043
- Jebeile H, Lister NB, Baur LA, Garnett SP, Paxton SJ (2021) Eating disorder risk in adolescents with obesity. Obes Rev 22:e13173. https://doi.org/10.1111/obr.13173
- Restrictive eating disorders in overweight or obese adolescents (2014) AAP Grand Rounds 32:68–68. https://doi.org/10.1542/gr. 32-6-68
- Flament MF, Henderson K, Buchholz A, Obeid N, Nguyen HN, Birmingham M, Goldfield G (2015) Weight status and DSM-5 diagnoses of eating disorders in adolescents from the community. J Am Acad Child Adolesc Psychiatry 54:403–411.e402. https://doi.org/10.1016/j.jaac.2015.01.020
- 32. Northam EA (1997) Psychosocial impact of chronic illness in children. J Paediatr Child Health 33:369–372. https://doi.org/10. 1111/j.1440-1754.1997.tb01622.x
- Grilo C (2016) [pp. 13.07] Hypertension and cardiovascular disease risk reduction in obese patients with binge eating disorder: controlled trial of stepped-care and behavioral weight loss. J Hypertens 34:e192–e193. https://doi.org/10.1097/01.hjh.00004 91879.94394.fc
- 34. Wade KH, Kramer MS, Oken E, Timpson NJ, Skugarevsky O, Patel R, Bogdanovich N, Vilchuck K, Davey Smith G, Thompson J, Martin RM (2017) Prospective associations between problematic eating attitudes in midchildhood and the future onset



- of adolescent obesity and high blood pressure. Am J Clin Nutr 105:306–312. https://doi.org/10.3945/ajcn.116.141697
- Hornberger LL, Lane MA, Committee On Adolescence (2021) Identification and management of eating disorders in children and adolescents. Pediatrics 147:e2020040279. https://doi.org/10.1542/ peds.2020-040279
- Lock J, La Via MC, American Academy of Child and Adolescent Psychiatry, Adolescent Psychiatry Committee on Quality Issues (2015) Practice parameter for the assessment and treatment of children and adolescents with eating disorders. J Am Acad Child Adolesc Psychiatry 54:412–425. https://doi.org/10.1016/j.jaac. 2015.01.018
- 37. Davidson KW, Barry MJ, Mangione CM, Cabana M, Chelmow D, Coker TR, Davis EM, Donahue KE, Jaen CR, Kubik M, Li

L, Ogedegbe G, Pbert L, Ruiz JM, Silverstein M, Stevermer J, Wong JB (2022) Screening for eating disorders in adolescents and adults: US preventive services task force recommendation statement. JAMA 327:1061–1067. https://doi.org/10.1001/jama. 2022.1806

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Springer Nature or its licensor (e.g. a society or other partner) holds exclusive rights to this article under a publishing agreement with the author(s) or other rightsholder(s); author self-archiving of the accepted manuscript version of this article is solely governed by the terms of such publishing agreement and applicable law.

