

Adverse childhood experiences and cognitive function among adults with excess adiposity

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Summary

Background: Adverse childhood experiences (ACEs) and obesity are independently associated with brain/neurocognitive health. Despite a growing emphasis on the importance of early life adversity on health, the relationship between ACEs and neurocognition in adults with overweight/obesity is unclear. The objective was to examine associations between self-reported ACEs and measured neurocognitive domains in a sample of adults with overweight/obesity.

Methods: Participants were 95 predominantly white, highly educated adult women (76% female, 81% Caucasian, and 75% \geq bachelor's degree) with excess adiposity enrolled in the Cognitive and Self-regulatory Mechanisms of Obesity Study. ACEs and fluid/crystallized neurocognitive domains were measured at baseline using the Adverse Childhood Experiences Scale and the NIH Toolbox Cognition Battery and Automated Neuropsychological Assessment Metric, respectively.

Results: Higher ACEs scores were negatively correlated with fluid cognition ($r = -.34, P < .001$) but not crystallized cognition ($r = .01, ns$). Individuals with 3 and 4+ ACEs displayed significantly lower fluid cognition scores than those with fewer ACEs $F_{4,89} = 3.24, P < .05$. After accounting for body mass index (BMI), age, sex, race, and education, higher ACEs scores were still associated with poorer performance on overall fluid cognition ($\beta = -.36, P < .01$), along with the following subtests: Stroop Colour/Word test ($\beta = -.23, P < .05$), Go/No-Go omissions ($\beta = .29, P < .01$), and Picture Sequence Memory task ($\beta = -.30, P < .01$).

Conclusions: The role of ACEs in health may be related to their associations with executive function and episodic neurocognitive domains essential to cognitive processing and self-regulation. Obesity science should further examine the role of ACEs and neurocognition in obesity prevention, prognosis, and treatment using more rigorous, prospective designs and more diverse samples.

KEYWORDS

adverse childhood experiences, cognitive function, early life adversity, obesity

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1 | INTRODUCTION

Adverse childhood experiences (ACEs) and excess adiposity are two interrelated predictors of multiple negative biopsychosocial health outcomes, including neurocognitive deficits.^{1,2} These effects are alarming given the high prevalence of ACEs³ and overweight/obesity.⁴

The Kaiser Permanente ACE Study Survey³ of 17 337 adults reports that the majority of participants (64%) have experienced at least one ACE prior to the age of 18, while nearly a quarter (22%) endorse 3+ ACEs. ACEs include traumatic events or maltreatment such as abuse, household challenges, and deprivation/neglect. ACEs have deep theoretical ties to disrupted neurodevelopment, strong lines of empirical support from the animal literature, and emerging evidence in human samples—all indicating that ACEs are associated with detriments to neurocognitive/brain function.^{1,5-9} These adverse events are linked to impairment across brain structures and neurocognitive functions and are expected to yield difficulties in both fluid and crystallized cognitive domains.¹⁰⁻¹²

Fluid neurocognition is characterized by reasoning/problem-solving abilities and can be independent of past knowledge, whereas crystallized cognition is knowledge acquired over the lifetime and available in long-term storage (eg, language). Fluid cognition includes domains related to executive functioning (EF) (eg, cognitive flexibility and inhibitory control).¹³ Fluid cognition is more susceptible to age-related cognitive decline,¹⁴ adverse cardiometabolic effects,¹⁵ and daily stress reactivity¹⁶ and, thus, may be more impacted by the cumulative effect of ACEs than are crystallized tasks. However, early deprivation/neglect ACEs may be particularly harmful to crystallized abilities, like language development.¹⁷

ACEs have shown dose-response effects on health, such that the risk of multiple adverse outcomes increase as a person's ACE score increases, as well as threshold effects, such that ≥ 4 ACEs typically confer adverse health effects.¹⁸⁻²⁰ Such toxic stressors may result in neurocognitive injury via dysregulation of the body's stress systems (eg, hypothalamic-pituitary-adrenal axis [HPA]) or via cognitive deprivation.¹² Indeed, ACEs are linked to reduced cortical volume and differences in neural activation of brain regions associated with language, memory, socio-emotional processing, and EF.^{1,12}

Like ACEs, excess adiposity is also widespread among adults with 72% of adults aged 20 or older qualifying as overweight or obese.⁴ Obesity increases risk for 250+ comorbidities, including mild cognitive impairment and neurodegenerative disease.^{2,21} Excess adiposity is both caused and exacerbated by biologic changes in cardiometabolic and inflammatory processes that, like ACEs, may result in injury to neurocognitive functions, especially fluid cognitive abilities such as EF.^{2,22-25} These impairments range from impaired neuropsychological test performance to increased risk of neurodegenerative disease.² Associated changes in structural and functional brain integrity are also documented.^{25,26}

Clearly, both ACEs and obesity are independently associated with brain and neurocognitive health. They may also interact, as high rates of ACEs in communities are often paralleled by high rates of overweight/obesity. The states with the highest rates of adult obesity

are also among the states with the highest rates of ACEs (eg, Oklahoma).^{27,28} ACEs also predict future obesity, such that abuse in childhood predicts greater obesity up to 30 years later.²⁹ Meta-analyses of several hundred thousand participants indicated that persons with childhood maltreatment were 1.4 times more likely to develop obesity over the life course³⁰ and that the risk for obesity followed a dose-response relationship.³¹ All of the above findings highlight the origins of the ACE study,³ which began with trying to understand high drop-out rates in an obesity clinic.³² In sum, ACEs and obesity have been linked to one another since the original ACE study³ and, more recently, to adverse neurocognitive effects. Accordingly, understanding the relationships between ACEs, neurocognitive function, and indicators of brain health may be important in any effort to combat the onset or progression of neurocognitive dysfunction and obesity—especially given that poorer neurocognitive function is not only a consequence of ACEs⁸ and obesity² but may also adversely impact a person's weight loss treatment outcomes via mediating and/or moderating effects.³³⁻³⁷

Despite evidence linking ACEs to obesity and evidence linking these two factors to adverse neurobiological consequences, little empirical work has been done to examine whether ACEs uniquely relate to neurocognitive function in overweight/obese samples after adjusting for estimated excess adiposity and key demographics. The present study begins to address these gaps by examining the associations between self-reported ACEs and neurocognitive domains in a sample of adults with overweight/obesity enrolled in a behavioural weight loss trial and adjusting for measured body mass index (BMI) as well as age, sex, and education. Given the above evidence, we hypothesize that individuals with a higher ACEs score, especially ≥ 4 , will show poorer performance on neurocognitive testing. We expect this relationship to be more pronounced for fluid cognitive tasks and independent of BMI.

2 | METHODS

2.1 | Overview

The Cognitive and Self-regulatory Mechanisms of Obesity Study (COSMOS) trial (NCT02786238) is a larger, ongoing multi-year, multi-cohort trial with 108 enrolled participants (aged 21-65 years old). The parent project is a comparative effects pilot examining how two behavioural weight loss interventions impact physical, neurocognitive, and self-regulation factors. The data and analyses from the current project are limited to the baseline data from this trial and included 95 participants with ACEs scores and neurocognitive testing data (see the Supporting Information regarding those with incomplete ACEs data; $n = 13$). The 13 participants with missing data did not turn in the packet with the ACEs survey so were not included in the analyses. Full details about the parent study methodology can be found in the published trial protocol paper.³⁸

2.2 | Adverse childhood experiences

ACEs were measured using the Adverse Childhood Experiences Survey (ACEs Survey).³⁹ The ACEs Survey asks participants to indicate whether they have experienced 10 possible traumatic events occurring before age 18, including emotional, physical, and sexual abuse; emotional and physical neglect; domestic violence; parental separation/divorce; familial mental illness; substance use; and/or incarceration. Consistent with previous research, individuals were characterized into one of five groups: 0, 1, 2, 3, or 4+ ACEs.¹⁵⁻¹⁷

2.3 | Neurocognitive function domains

To assess their neurocognitive function across multiple domains, participants completed the NIH Toolbox Cognition Battery (NIHTB-CB)⁴⁰ and subtests of the Automated Neuropsychological Assessment Metrics-IV (ANAM-IV),⁴¹ both computerized neurocognitive test batteries. The NIHTB-CB tests for crystallized cognition are (a) Picture Vocabulary and (b) Oral Reading Recognition (language). The Fluid Cognition tests are (a) Flanker Inhibitory Control and Attention (inhibition/selective attention), (b) Dimensional Change Card Sort (cognitive flexibility), (c) List Sorting (working memory), (d) Picture Sequence Memory (episodic memory), and (e) Pattern Comparison Processing (processing speed). As supplemental measures of fluid executive function, participants also completed the Go/No-Go task and Stroop Colour/Word test (inhibitory control tasks) from the ANAM-IV.⁴¹ In an effort to standardize assessment, we computed standardized means (*z* scores) for each individual test variable and averaged these scores to create composite scores for crystallized cognition (using the two NIHTB-CB language tests) and for fluid cognition (using all other NIHTB-CB and ANAM-IV tests). Commission and omission errors on the Go/No-Go task were inverted, such that higher scores indicate superior performance, to be consistent with all other variables and ease interpretation.

2.4 | Adiposity variables and covariates

BMI (kg m⁻²) was obtained using measured height and weight from a standard medical scale to the nearest 0.1 of the kilogram. Participants were weighed wearing casual clothes and without shoes. Waist circumference (WC) was measured in centimetres according to the World Health Organization guidelines.⁴² Participants completed baseline self-report questionnaires assessing relevant covariates, including age (years), gender (0 = male, 1 = female), race-ethnicity (Caucasian, African American, American Indian/Native American/Alaskan Native, Asian/Pacific Islander, and Hispanic/Latino), and education level (middle school, high school, some college, associate's, bachelor's, graduate, or professional).

2.5 | Procedure

Participants were recruited from the local university/community and completed online/phone screenings. Eligible participants who wanted

to enroll provided written informed consent and were scheduled for their baseline assessments, which included a series of self-report questionnaires (including the ACEs Survey) and a laboratory testing session in which the NIHTB-CB and the ANAM-IV were administered by trained research staff. Participants received \$75 reimbursement for completing the visit.

2.6 | A priori statistical methods

All statistical tests were run using IBM SPSS Statistics software. Individuals were categorized into one of five groups based on their ACEs score (0, 1, 2, 3, or 4+ ACEs). ANOVA or χ^2 tests were used to examine group differences across these five groups across all study variables. Independent samples *t* test were also conducted to assess the differences in BMI and WC between high (≥ 4) and low (< 4) ACEs groups. Bivariate correlational analyses were run for ACEs group membership and fluid/crystallized cognition composite scores. One-way ANOVA tests were then run comparing cognition scores based on ACEs group. To determine if these bivariate relationships were robust, hierarchical multivariable regression analyses predicting cognitive scores were run with BMI, age, sex, race, and education covariates (step 1) and ACEs group (step 2).

3 | RESULTS

3.1 | Participant characteristics

In this sample of 95 adults with excess adiposity, 68% endorsed at least one ACE, 40% endorsed two or more, 23% endorsed three or more ACEs, and 17% endorsed four or more (see Table 1). The endorsement rates of ACEs type, from most to least frequent, were having a household member with depression, mental illness, or attempted suicide (28%); having biological parent lost through divorce, abandonment, or other reasons (27%); and having a parent or other adult in household enact verbal harm or fear of physical harm (27%), living with someone with problematic alcohol or drug use (26%), experiencing sexual abuse (23%), feeling unloved or unsupported (20%), experiencing physical abuse (9%), having a family member go to prison (5%), experiencing neglect (4%), or witnessing domestic violence (3%).

Individuals with ≥ 4 ACEs had significantly higher BMIs ($M = 38.9 \pm 5.9$) than those with less than four ACEs ($M = 35.1 \pm 5.9$); $t(92) = 2.4$, $P = .019$. This pattern also held for WC, such that those with ≥ 4 ACEs had greater abdominal adiposity ($M = 113.7 \pm 14.6$) than those with < 4 ACEs ($M = 106.1 \pm 12.4$); $t(92) = 2.2$, $P = .033$. See Table 1 for adiposity values across all ACEs groups.

3.2 | Preliminary analyses

As expected, ACEs group membership was found to have a significant negative association with the fluid cognition composite with a moderate effect size (see Table 2). The largest and statistically significant

TABLE 1 Characteristics of participants

	Total Sample	0 ACE (n = 30)	1 ACE (n = 27)	2 ACEs (n = 16)	3 ACEs (n = 6)	4+ ACEs (n = 16)	F or χ^2 Test	Partial η^2	P Value
		M (SD) or n (%)							
Demographic factors									
Age	45.6 (11.8)	47.2 (10.5)	46.2 (11.5)	46.9 (10.6)	35.3 (9.2)	43.9 (15.2)	1.5	.06	.220
Female Sex	72 (76%)	24 (80.0%)	18 (66.7%)	12 (75%)	5 (83.3%)	13 (81.3%)	2.0	--	.742
White race	76 (80%)	24 (82.8%)	21 (77.8%)	13 (81.3%)	5 (83.3%)	13 (81.3%)	15.7	--	.733
Highest level of education							25.5	--	.600
High school or less	6 (6.4%)	0 (0.0%)	2 (7.4%)	0 (0%)	1 (16.7%)	3 (18.8%)			
Some college/associates degree	17 (18.1%)	6 (20.7%)	3 (11.1%)	2 (12.6%)	1 (16.7%)	5 (31.3%)			
Bachelor's degree	29 (30.9%)	10 (34.5%)	9 (33.3%)	4 (25%)	2 (33.3%)	4 (25%)			
Graduate degree	42 (44.2%)	13 (44.8%)	13 (48.1%)	10 (62.6%)	2 (33.3%)	4 (25%)			
Adiposity variables									
Body mass index (BMI; kg m ⁻²)	35.7 (6.0)	35.7 (6.2) _{ab}	34.1 (5.9) _a	34.6 (5.5) _a	37.9 (5.4) _{ab}	38.9 (5.9) _b	2.1	.09	.093 ^a
Waist circumference, cm	107.4 (13.0)	106.6 (10.7)	105.7 (14.7)	105.5 (11.7)	106.7 (13.1)	113.7 (14.6)	1.2	.05	.331
NIHTB-CB cognitive t scores									
Oral Reading Recognition	56.2 (8.4)	57 (9.1)	56.6 (7.4)	53.7 (6.9)	57.3 (9.5)	55.7 (9.7)	0.4	.02	.780
Picture Vocabulary	55.5 (8.7)	54.7 (9.7)	56.5 (9.7)	52.7 (8.5)	55.7 (5.8)	57.6 (5.7)	0.8	.04	.537
Flanker Inhibitory Control/Attention	40.9 (7.3)	42 (7.9)	40.6 (6.7)	41.6 (6.3)	35.3 (6.7)	40.5 (7.9)	1.1	.05	.351
Dimensional Card Sorting/Flexibility	50.9 (10.5)	52.2 (9.7) _{ab}	53.5 (12.5) _a	49.5 (9.4) _{ab}	43.2 (8.1) _b	48.3 (8.6) _{ab}	1.7	.07	.159 ^a
Working Memory	53.6 (7.8)	54.7 (8.3)	53.3 (7.4)	55.3 (6.7)	49.7 (10.9)	51.7 (8.3)	0.9	.04	.478
Picture Sequence Episodic Memory	55.1 (12.4)	58.6 (14.1) _a	55.4 (12.1) _{ab}	56 (10.8) _{ab}	51.8 (7.8) _{ab}	48.6 (10.4) _b	1.9	.08	.119 ^a
Processing Speed	52.9 (13.5)	54.9 (11.7)	52.8 (13.8)	54.1 (15.7)	43.2 (12.3)	51.6 (14.2)	1.0	.04	.405
ANAM-IV executive function scores									
Stroop Colour/Word	36.2 (12.1)	37.9 (11)	36.7 (11.7)	36.1 (14.5)	36.8 (12.2)	31.7 (12.5)	0.7	.03	.596
Go/No-Go Omissions	0.7 (1.4)	0.6 (.9) _a	0.4 (.9) _a	0.8 (1.1) _{ab}	0.5 (.8) _{ab}	1.7 (2.3) _b	2.9	.12	.027 ^b
Go/No-Go Commissions	5.3 (3.1)	4.4 (2.5)	5.7 (3.6)	6.3 (3.5)	6.2 (4.1)	5.2 (2.2)	1.3	.05	.297
Composite cognition z scores									
Crystallized Cognition	0.0 (1.0)	0.1 (1.1)	0.1 (1.0)	-0.3 (0.9)	0.2 (0.9)	0.2 (0.9)	0.6	.03	.662
Fluid Cognition	0.0 (0.5)	0.2 (0.5) _a	0.1 (0.4) _{ab}	-0.0 (0.6) _{abc}	-0.3 (0.4) _{bc}	-0.2 (0.4) _c	3.2	.13	.016 ^b

Note. When the ANOVA omnibus *F* test comparing the five ACEs groups yielded a medium effect or larger ($\eta^2 > .06$), pairwise comparisons are indicated using subscripts, in which values with the same subscripts had statistically equivalent means or proportions. *t* scores: *M* = 50, *SD* = 10; *z* scores: *M* = 0, *SD* = 1.

Abbreviations: ACE, adverse childhood experience; ANAM-IV, Automated Neuropsychological Assessment Metric; NIHTB-CB, National Institutes of Health Toolbox-Cognition Battery.

^aMedium effect ($\eta^2 > .06$) but not statistically significant.

^bLarge effect ($\eta^2 > .14$) and/or *P* < .05.

effects were found for Picture Sequence Memory (episodic memory) and Go/No-Go omissions (executive skill of initiation) with medium effect sizes. However, all of the other fluid cognition tests (eg, cognitive flexibility, working memory, inhibitory control, and processing speed) were also related to ACEs in the expected negative direction and reached at least a small effect size magnitude, though they did

not reach statistical significance. In contrast, ACEs score was not significantly associated with the crystallized cognition composite score nor its subtests—with weak to no effect sizes. Consistent with the correlational results, one-way ANOVA results comparing ACEs group membership on fluid and crystallized cognition indicated there were no mean differences for crystallized cognition, $F_{4,88} = 0.60$, *ns*, but

TABLE 2 Correlations between adverse childhood experiences, cognitive function indices, and demographics

Variables	Correlations														
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. ACEs Groups	--														
2. Crystallized Cognition z score	.01	--													
3. Picture Vocabulary	.08	.87*	--												
4. Oral Reading	-.06	.86*	.51*	--											
5. Fluid Cognition z score	-.34*	.32*	.32*	.23*	--										
6. Flanker Inhibition/Attention	-.11	.15	.19	.09	.51*	--									
7. Working Memory	-.13	.26*	.25*	.21*	.49*	.05	--								
8. Card Sort Test/Flexibility	-.19	.12	.17	.04	.60*	.61*	.04	--							
9. Picture Sequence Episodic Memory	-.27*	.12	.16	.04	.44*	.01	.26*	-.01	--						
10. Processing Speed	-.12	.06	.05	.06	.63*	.27*	.16	.37*	.22*	--					
11. Stroop Colour/Word	-.16	.38*	.24*	.43*	.57*	.07	.31*	.12	.21*	.26*	--				
12. Go/No-Go Omissions ^a	-.28*	.20*	.23*	.12	.60*	.14	.12	.23*	.15	.26*	.38*	--			
13. Go/No-Go Commissions ^a	-.11	-.01	-.01	-.03	.31*	-.07	.06	.09	-.03	.03	-.06	.16	--		
14. Age	-.08	-.11	-.05	-.15	-.06	-.28*	.04	.21*	-.11	-.12	-.28*	-.25*	-.07	--	
15. Sex	.05	-.07	-.07	-.05	-.13	-.31*	-.08	-.07	.08	.01	-.04	.09	-.00	-.21*	--
16. Education	-.33*	-.15	-.22*	-.04	.09	.03	.20	-.10	-.00	-.00	-.05	.16	-.12	.13	-.13

^aScore inverted such that higher scores indicate better performance to be consistent with other cognitive measures.

*P < .05

significant mean differences were found for fluid cognition, $F_{4,89} = 3.24, P < .05$, with participants with 3 and ≥ 4 ACEs having lower fluid cognition scores than participants with zero ACEs.

3.3 | Regression analyses

With preliminary findings showing that participants with higher ACEs scores had deficits in the broad composite score of fluid cognition,

we ran hierarchical multivariable regression analyses to determine if bivariate effects were robust to the addition of key covariates: BMI, age, sex, race, and education. After adjusting for these covariates, higher ACEs scores still predicted the overall fluid cognition composite (see Table 3), such that higher ACEs scores were associated with lower scores on the fluid cognition composite variable. ACEs group membership explained approximately 12% of the variance in the fluid cognition composite score (Table 3). Higher ACEs scores were

TABLE 3 Regressions of adverse childhood experiences predicting fluid cognition tests

Variables	Fluid Cognition Composite				Stroop Colour/Word				Go/No-Go Omissions ^a				Picture Sequence Memory Test			
	β	R^2	ΔR^2	P	β	R^2	ΔR^2	P	β	R^2	ΔR^2	P	β	R^2	ΔR^2	P
Demographics (step 1)		.03	--	.550		.09	--	.071		.12	--	.018*		.02	--	.756
Sex	-.15			.176	-.11			.371	.14			.175	.05			.632
Age	-.11			.013*	-.30			.005*	.32			.003*	-.11			.340
Education	-.07			.551	-.02			.830	-.16			.129	.00			.969
BMI	-.06			.595	.00			.987	.09			.407	-.08			.461
ACEs (step 2)		.15	.12	.001*		.14	.05	.031*		.20	.08	.005*		.10	.08	.006*
Sex	-.15			.159	-.11			.303	.14			.164	.05			.614
Age	-.14			.177	-.33			.002*	.35			.001*	-.14			.196
Education	-.00			.973	-.07			.528	-.10			.313	-.06			.572
BMI	-.01			.935	.03			.749	.05			.459	-.04			.728
ACEs Group	-.36			.001*	-.23			.031*	-.29			.005*	-.30			.006*

Abbreviations: ACE, adverse childhood experience; BMI, body mass index.

^aScore inverted such that higher scores indicate better performance to be consistent with other cognitive measures.

*P < .05.

significantly associated with worse performance on the Stroop Colour/Word test, Go/No-Go task, and Picture Sequence Memory test, specifically accounting for 5% to 8% of the variance in these measures (Table 3). Standardized mean values for the fluid cognition composite and these individual tests are presented in Figure 1. ACEs were not found to be associated with the individual measures of working memory, cognitive flexibility, and inhibition/attention in these regression models.

3.4 | Post hoc analyses and results

To follow-up the standard ANOVAs run for Table 1, we also conducted linear contrast analyses for the primary variables (ie, adiposity and cognition) in order to better account for the ordered nature of the ACEs group categories. Linear contrasts for five ACEs groups (0, 1, 2, 3, and 4+ with coefficients: -2 -1 0 1 2) showed statistically significant contrasts for the following variables: BMI ($F_{1,89} = 5.20, P = .025$, partial $\eta^2 = .06$), fluid cognition composite ($F_{1,89} = 12.39, P = .001$, partial $\eta^2 = .12$), cognitive flexibility ($F_{1,88} = 5.18, P = .025$, partial $\eta^2 = .06$); episodic memory ($F_{1,88} = 6.40, P = .013$, partial $\eta^2 = .07$), and omissions ($F_{1,89} = 5.75, P = .019$, partial $\eta^2 = .06$). In each case, the linear trend was that groups with higher ACE scores had lower cognitive scores and higher BMIs. Statistically significant linear contrasts were not detected for the following variables: WC ($P = .134$, partial $\eta^2 = .03$), crystallized cognition composite ($P = .781$, partial $\eta^2 = .00$), vocabulary ($P = .459$, partial $\eta^2 = .01$), inhibitory control ($P = .138$, partial $\eta^2 = .03$), working memory ($P = .124$, partial $\eta^2 = .03$), processing speed ($P = .117$, partial $\eta^2 = .03$), Stroop Colour/Word ($P = .194$; partial $\eta^2 = .02$), or commissions ($P = .746$, partial $\eta^2 = .01$). Taking these together, these results are similar to the ANOVA results presented in Table 1 but suggest that linear contrast analyses may yield better powered contrasts of the ACEs groups, as several trending results became significant (ie, BMI, cognitive flexibility, and episodic memory).

Next, given recent data suggesting that ACEs-cognition relationships may be moderated by the type of ACE experienced: threat versus deprivation,^{43,44} we also categorized participants by their experience

of ACEs that were clearly deprivation type (ie, "absence of expected environmental inputs and complexity"⁴⁴ or Yes to items 4 or 5: emotional or physical neglect) versus clearly threat type (ie, "presence of experiences that represent a threat to physical integrity"⁴⁴ or Yes to items 1, 2, 3, or 7: emotional, physical, or sexual abuse, domestic violence). Given that items 6, 8, and 10 (ie, parental separation/divorce, familial mental illness, and incarceration) are less clear in their deprivation and/or threat outcomes, we did not use them to categorize participants into the deprivation vs threat groups but categorized them into a separate household dysfunction group. Using this categorization system, only 7.7% ($n = 5$) of our 65 participants with ≥ 1 ACE had deprivation-type ACEs only, whereas the majority endorsed both deprivation and threat-type ACEs ($n = 14, 21.5\%$), threat-only ($n = 22, 33.8\%$), or household dysfunction ACEs ($n = 23, 35.4\%$). In an ANCOVA comparing the crystallized cognition scores of these four groups with ACEs while adjusting for BMI, a significant omnibus test was observed ($F_{3,64} = 4.31, P = .008$, partial $\eta^2 = .18$). Pairwise comparisons showed that participants with deprivation-type ACEs-only exhibited significantly lower crystallized scores ($M = 47.87 \pm 9.95$) than individuals with threat-only ($M = 59.9 \pm 8.0; P = .002$) or household dysfunction ACEs ($M = 55.7 \pm 9.3; P = .035$). Fluid cognition did not differ among the four ACEs groups ($F_{3,65} = .59, P = .625$, partial $\eta^2 = .03$).

4 | DISCUSSION

The hypothesis that individuals with a higher total ACEs score would show poorer performance on neurocognitive testing, particularly in the fluid cognition domain, was supported. ACEs scores accounted for 12% in the composite score for fluid cognition, and 5% to 8% of the variance on specific tests measuring executive control and episodic memory. A higher number of ACEs was associated with lower scores on these domains, after adjusting for demographics, education, and obesity severity (ie, continuous BMI). In contrast, ACEs were not significantly related to the fluid domains of working memory, cognitive flexibility/set shifting, or inhibition of selective attention, or to the crystallized cognition domain.

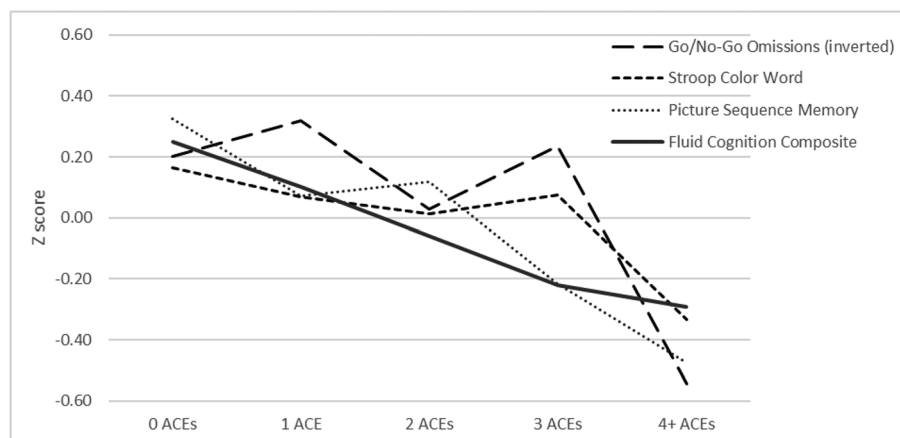


FIGURE 1 Fluid cognition performance across adverse childhood experiences (ACEs) groups

Theoretical and/or methodological explanations may drive this pattern of findings. First, fluid cognition may be more adversely impacted by ACEs given that it is not as resistant to age-related and/or stress-related decline as is language and other more crystallized knowledge.¹⁴ However, because this sample was highly educated (ie, high proportion with graduate training), range restriction may also limit the ability to detect findings between ACEs and the crystallized language domains—which are associated with educational achievement. Importantly, the issue of range restriction is also relevant for our obesity variables given that this sample is also comprised entirely of overweight/obese individuals, who are already at risk for relative neurocognitive impairment secondary to adverse metabolic, vascular, and inflammatory effects linked to excess adiposity.²² Indeed, the average score of these participants on certain tests (ie, Flanker Inhibitory Control/Attention) was low average with smaller standard deviations across the entire sample. Thus, although this sample was cognitively intact with cognitive performance within normal limits, the inclusion of age, sex, and education-matched peers without obesity may alter the strength of the associations between ACEs and cognitive test performance, a hypothesis in need of explicit testing. However, the patterns of results do suggest that ACEs remain a sensitive predictor of relative deficits in fluid cognitive abilities over and above the impact of obesity (as measured with BMI) even in a highly educated sample with excess adiposity.

Certain measures of fluid cognition were more related to ACEs than others in this sample. ACEs scores were most strongly related to Go/No-Go omissions, Stroop Colour/Word, and the Picture Sequence Memory test scores and accounted for 8%, 5%, and 8% of the variance in these tests, respectively. Two of these fluid cognition tests (Go/No-Go and Stroop) are measures of executive control. Specifically, the Go/No-Go test measures the ability to inhibit reactions to certain stimuli while initiating a response to others. The failure to inhibit a response yields greater commission errors, whereas the failure to enact a response results in greater omission errors. Yechiam et al⁴⁵ speculated that individuals who are prone to omissions on Go/No-Go-type tests are those who have higher attention to losses/punishment relative to gains, whereas people prone to commissions have higher attention to gains/rewards relative to losses. The finding that higher ACEs are linked to more omissions may then be consistent with the notion that high ACEs predict attentional biases that facilitate the ability to identify threats at the expense of the ability to attend to positive information.^{43,46} The lower scores on the Stroop may also support this idea, given that lower Stroop scores suggest a reduced ability to override a more automated or dominant reaction in favour of a less automated, non-dominant behaviour.⁴⁷ Other measures of EF (ie, set shifting, working memory, and attentional control) in this study also exhibited consistent negative relationships with ACEs with small effect sizes, although they did not reach significance.

In addition to decreased executive control, ACEs were also associated with poorer episodic memory in this sample, which is consistent with previous studies.^{8,9} Episodic memory is a type of declarative memory ability that allows a person to remember specific experiences and events in a temporal sequence and is typically linked to the

context and emotions surrounding the experience.⁴⁸ Given that episodic memory is largely encoded by the hippocampus,⁴⁸ this finding is consistent with literature linking childhood maltreatment with reduced hippocampal volume and activation as well as patterns of connectivity related to worse memory for contexts when threat is present.^{49,50} One proposed reason for this poor context encoding when under threat is that the individual narrows his/her attention to focus on the threatening stimuli, at the cost of his/her hippocampal processing of the larger context.⁵⁰ In sum, this pattern of results suggests that an individual with obesity and a history of ACEs may be primed to perceive threat and/or unpredictability, which may burden the cognitive systems associated with inhibitory control and response initiation as well as episodic memory encoding.

Although the ACEs-cognitive burden relationship has been consistently documented, ACEs-cognition relationships may be moderated by the type of ACE experienced: threat versus deprivation.⁴³ These categories may have distinct neurobiological correlates and consequent downstream effects on cognitive function. Deprivation-type ACEs are those in which normative cognitive and social input is limited or absent (eg, neglect), whereas threat-type ACEs occur when abnormal danger or risk to physical health is present (eg, sexual or physical abuse).⁴³ Other household dysfunction ACEs may be more ambiguous in their outcomes and depend on a child's support system or other factors (eg, parental incarceration leading to placement into a more stable environment). Post hoc probing of the sample revealed that only 8% of our sample with ACEs said that they experienced only deprivation type. The vast majority experienced either both types of ACEs, threat-only, or household dysfunction ACEs. Interestingly, participants with only deprivation-type ACEs exhibited significantly lower crystallized scores than individuals with threat-only or household dysfunction ACEs, an effect that was independent of BMI. Furthermore, given that the impact of ACEs on cognitive function involves neuroendocrine cascades (ie, disruptions of the HPA axis),¹ future studies should examine how these biologic factors intersect with excess adiposity, which is itself a disease of hormonal imbalance.⁵¹ Indeed, individuals with 4 or more ACEs had the most severe obesity levels and highest abdominal adiposity levels in this sample. Taking these together, the above findings suggest that the ACEs-cognition relationship is likely nuanced and may be neurocognitively toxic over and above excess adiposity.

Despite the fact that this study is the first to examine neuropsychological testing and ACEs in the context of an obesity treatment-seeking sample, certain limitations should be noted. First, this study is a cross-sectional examination of middle-aged adults with overweight/obesity. Future studies should seek to disentangle the relative and unique contributions of ACEs versus excess adiposity across time and in different age groups, particularly child and adolescent samples. ACEs overall and ACEs subtypes may promote obesity development differentially depending on when and which ACEs occurred relative to a child's maturation and/or pubertal milestones. Second, this study enrolled a highly educated predominantly white female sample and tested their cognitive function in a paradigm that was not overtly threatening and/or unpredictable. Accordingly, this pattern

of findings may not extend to other SES and/or marginalized groups or to contexts in which individuals were tested in situations of heightened stress or uncertainty. Lastly, while cognitive testing does provide some insight into neurocognitive health, future studies should incorporate more sophisticated neural interface technology (eg, functional magnetic resonance imaging or near-infrared spectroscopy) to elucidate the intersections between brain, cognition, and body as they relate to ACEs. Likewise, more precise measures of adiposity are needed to clearly ascertain the role of excess fat mass and its distribution in ACE-health relationships.

5 | CONCLUSION

Higher ACEs were associated with higher BMI and abdominal adiposity, even within a sample with overweight/obesity. Persons with more ACEs performed worse on cognitive tasks of executive control and episodic memory than did those with fewer ACEs, controlling for obesity severity. Given that these cognitive processes play important roles in self-regulation, ACEs may be important to obesity development, progression, and weight loss interventions. If replicated in more rigorous study designs, clinical implications of these results might include regular assessment of ACEs history and/or cognitive function to assess for obesity risk or heterogeneity of obesity treatment effects. Such information could ultimately be used for potential tailoring of obesity prevention or intervention efforts for at-risk individuals.

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DATA SHARING STATEMENT

Individual participant data will not be publicly available. Please contact the corresponding author to propose potential collaborative analyses of the trial data.

Clinical Trial Registry: Identifier NCT02786238; <https://clinicaltrials.gov/ct2/show/NCT02786238>

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CONFLICT OF INTEREST STATEMENT

The authors declared no conflict of interest.

AUTHOR CONTRIBUTIONS

M.H. is the PI for the COSMOS grant (K23DK103941) and lead author. L.C. conducted the initial analyses for this manuscript, drafted the results, and provided ACEs content expertise. J.C. was a postdoctoral fellow and provided detailed editing of manuscript content related to cognitive development. N.K., C.S., and M.S. were study interventionists and coordinators for the trial. S.A., M.A., and G.E. assisted with manuscript formatting and drafting for submission. All authors were involved in drafting the final paper and have final approval of the submitted version.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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