

# From Food to Function: Dietary Scores Predict Cognitive Outcomes and Neural Signatures in Youth \*

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## Abstract

**Background:** Adolescence represents a critical period of neuroplasticity when nutritional inputs may significantly influence cognitive development, yet the neural mechanisms mediating these relationships remain poorly understood.

**Methods:** Using data from the Adolescent Brain Cognitive Development Study (N=3,308; mean age 13.7 years, 47% female), we constructed nutrition-based cognitive potential scores (PolyIQ) via principal component analysis of dietary patterns and elastic net regression. We examined structural brain mediation of nutrition-cognition relationships using T1-weighted and diffusion-weighted MRI data, focusing on regions supporting memory and language function.

**Results:** PolyIQ scores significantly predicted both crystallized ( $\beta=0.55$ ,  $p < 0.0001$ ) and fluid intelligence ( $\beta=0.59$ ,  $p < 0.0001$ ) after controlling for socioeconomic factors. White matter integrity in the bilateral fornix emerged as the most consistent neural mediator, explaining 4.1-5.9% of nutrition-cognition relationships across both cognitive domains. Gray matter volumes in the left parahippocampal gyrus, left pars opercularis, and right middle temporal gyrus additionally mediated effects on crystallized intelligence, accounting for 3.5-8.1% of the total relationship.

**Conclusions:** Nutrition influences adolescent cognitive development through specific neurobiological pathways, particularly temporal-limbic circuits involving the fornix and parahippocampal region. These findings identify brain-based mechanisms linking dietary quality to cognitive outcomes and highlight the importance of nutritional interventions during this critical developmental period.

**Keywords:** Adolescent development, nutrition, neuroimaging, mediation analysis, cognitive function, ABCD Study.

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# 1 Introduction

Adolescence is a period of heightened neuroplasticity and cognitive refinement, making it a sensitive window during which nutritional inputs can have significant and lasting impacts on brain development. The adolescent brain undergoes substantial structural and functional maturation, characterized by synaptic pruning, increased myelination, and refinement of neural circuits that support higher-order cognition (Giedd, 2008; Blakemore, 2012). These neurodevelopmental processes are highly susceptible to environmental influences, with nutrition emerging as a key modifiable factor that may shape cognitive trajectories (Nyaradi et al., 2013b). Extensive literature has linked dietary quality with cognitive performance, physical development, and brain integrity, while a growing body of evidence points to how socioeconomic and psychological factors may modulate these nutritional effects, creating complex pathways from food consumption to cognitive outcomes.

Research consistently demonstrates that healthier dietary patterns are linked to improved cognitive outcomes across the lifespan. In adults, plant-based diets such as the Mediterranean, DASH, and MIND diets are associated with slower cognitive decline and enhanced mental performance, particularly among individuals at elevated cardiovascular risk (Van den Brink et al., 2019). Among youth, higher overall diet quality has been tied to better executive functioning and cognitive development (Cohen et al., 2016). Using data from more than 3,000 participants in the ABCD Study, Green (2022) reported that diet quality was positively associated with crystallized cognition, though no significant relationship was found with fluid cognition. These findings align with prior research indicating that "prudent" diets—characterized by high intake of vegetables and low-fat dairy—are linked to stronger baseline cognitive scores and reduced cognitive decline (Parrott et al., 2013). Additionally, distinct childhood dietary patterns, such as "healthy" versus "full-fat," have been associated with divergent trajectories of weight development, underscoring the long-term physiological and cognitive implications of early nutritional habits (Rashid et al., 2020b).

However, the relationship between nutrition and development is significantly shaped by socioeconomic, cultural, and behavioral factors. Data from the Amsterdam Born Children and their Development (ABCD) cohort revealed that children's dietary patterns varied significantly by ethnicity and socioeconomic status (Rashid et al., 2018, 2020a). Notably, diets high in vegetables and low-fat dairy were found to be especially beneficial for cognitive function among individuals from lower socioeconomic backgrounds (Parrott et al., 2013). In the U.S.-based ABCD Study, lower-income and racial or ethnic minority adolescents were more likely to consume fewer healthy foods and greater amounts of added sugars

(Nagata et al., 2025). Several behavioral and demographic variables—such as excessive screen time, high reward sensitivity, and poor planning—have been linked to both elevated BMI risk (Gray et al., 2020; Allen, 2023; Nagata et al., 2023) and increased consumption of fat and sugar (Adise et al., 2024), suggesting that nutritional pathways to cognitive outcomes are embedded within broader socioeconomic and behavioral contexts.

From a neurobiological perspective, nutrition serves as a fundamental driver of brain development through multiple mechanisms. Nutrients provide essential building blocks for brain tissue, regulate neurotransmitter synthesis, support myelination, and influence neuroinflammatory processes (Gómez-Pinilla, 2008). Macronutrients such as omega-3 fatty acids support membrane fluidity and synaptic function, while micronutrients like iron, zinc, and B vitamins are critical cofactors in neurodevelopmental processes (Georgieff, 2007). Longitudinal studies show that Western-style dietary patterns—high in processed foods and low in micronutrients—are linked to reduced hippocampal volume and poorer cognitive outcomes in adolescents (Jacka et al., 2015). Structural brain measures also predict BMI and future weight gain (Adise et al., 2021), suggesting bidirectional relationships between nutrition, brain structure, and cognitive function. Key circuits implicated in these relationships include the prefrontal-hippocampal network supporting executive function and memory, and frontotemporal networks supporting language acquisition and semantic knowledge (Beilharz et al., 2015).

The consequences of poor nutrition during youth can be particularly severe and long-lasting. Li et al. (2024) found that higher BMI in childhood predicted increased depressive symptoms in adolescence and that lower baseline cognitive performance was associated with greater weight gain over time. Similarly, Agarwal et al. (2023) identified high fat and sugar intake, excessive screen time, and exposure to family violence as key risk factors for maladaptive behaviors in adolescence, including obesity and substance use. Moreover, eating disorders are already present in 1.4% of children as young as 9 to 10 years old, emphasizing the importance of early dietary interventions to prevent long-term adverse outcomes (Rozzell et al., 2019).

Despite this growing evidence linking nutrition to cognitive outcomes, several important gaps remain in our understanding. Most studies have focused on single nutrients or broad dietary patterns without examining how these nutritional factors predict cognitive potential specifically. While associations between diet and cognition have been established, the neural mechanisms mediating these relationships remain poorly characterized, particularly during adolescence. Additionally, the potential moderating roles of socioeconomic status and physical health markers like adiposity are not well integrated into existing neurobiological models.

The present study addresses these gaps by investigating how dietary patterns predict

cognitive outcomes in youth, specifically fluid and crystallized intelligence, after accounting for socioeconomic and developmental factors. We also examine which structural brain features mediate the relationship between nutrition and cognitive performance in adolescents, and whether socioeconomic status and adiposity moderate or mediate the relationships between diet, brain structure, and cognition. By addressing these questions, we aim to elucidate the neurobiological pathways through which nutrition shapes cognitive development during this critical period, with potential implications for targeted interventions to promote optimal brain development and reduce cognitive disparities.

## 2 Methods

*Study Population.* We used data from the Adolescent Brain Cognitive Development (ABCD) Study, a longitudinal and nationally representative study of child and adolescent development. The ABCD cohort consists of over 11,000 youth recruited at ages 9–10 and followed across adolescence with repeated measures of neurocognition, mental health, physical health, and environmental exposures. For the present analysis, we focused on participants with complete dietary intake data from the Block Kids Food Screener (BKFS), macronutrient profiles, adiposity measures, multiple structural brain metrics (cortical morphology and white matter microstructure) and valid cognitive scores. After applying quality controls and excluding cases with missing or unreliable responses, we retained a final sample of 3308 participants. The average age was 13.7 old years ( $SD = 2.54$ ), with 47% identifying as female.

*Nutrition data.* Dietary intake was assessed using the 41-item BKFS, which evaluates frequency and quantity of foods consumed over the previous week. The BKFS captures intake across key categories including fruits, vegetables, whole grains, dairy, meat/fish, legumes, added sugars, and saturated fat, as well as intake in terms of macronutrients and metrics such as glycemic index and glycemic load. Descriptive statistics of consumption are reported in Appendix 1.

*Adiposity.* Body Mass Index (BMI) was calculated using participants’ measured height and weight. Specifically, weight in pounds was converted to kilograms ( $1 \text{ lb} = 0.453592 \text{ kg}$ ) and height in inches was converted to meters ( $1 \text{ inch} = 0.0254 \text{ m}$ ). BMI was then computed as weight in kilograms divided by height in meters squared ( $\text{kg}/\text{m}^2$ ). To account for developmental variation, BMI values were standardized using age- and sex-specific norms from the CDC LMS (Lambda–Mu–Sigma) growth charts. Each child’s age in months was matched to the appropriate LMS parameters (L, M, S), and a BMI z-score was computed. These z-scores were then converted to percentiles using the standard normal distribution. We also retained waist circumference as a second measure of adiposity. The

two capture different physiological aspects of body composition that may differentially affect brain development. BMI reflects overall body fatness. Waist circumference, by contrast, specifically captures central (abdominal) adiposity. For our data, higher sugary beverage consumption was robustly linked to higher BMI and waist circumference, and whole grain intake was associated with lower BMI. The relationship between adiposity and food intake. Details about the relationship between nutrition and adiposity can be found in Appendix 2.

*Cognitive Composite Scores.* To assess cognitive performance, we focused on fluid and crystallized intelligence using tasks from the NIH Toolbox included in the ABCD Year 4 dataset. Crystallized IQ was derived as the average of two standardized scores, Picture Vocabulary and Oral Reading Recognition. One test from the NIH Toolbox—the Card Sorting task—was excluded from the analysis due to a high rate of missing data. Fluid IQ was computed as the average of three standardized task scores, Flanker Inhibitory Control and Attention, List Sorting Working Memory and Episodic Memory. Prior to computation, all cognitive scores were z-scored, and missing values were estimated using multiple imputation with predictive mean matching (PMM) via the mice package in R (5 imputations, 5 iterations). The imputed values from the first completed dataset were retained for analysis. A detailed study of the composite score is presented in Appendix 3.

*PolyIQ Score Construction Based on Nutrition.* We constructed poly-nutritional IQ scores (PolyIQ) using an elastic net regularization framework. First, we applied principal component analysis (PCA) to food frequency variables. From these PCAs, we retained the first five food components (Food-PC1 to Food-PC5) as predictors of cognitive scores. To model the predictive relationship between nutrition and IQ, we used the crystallized IQ and fluid IQ composite scores as outcomes and the five food PCA components as input features. We then fit elastic net regression models using 10-fold cross-validation (`cv.glmnet` in R,  $\alpha = 0.5$ ) to optimize the penalty parameter. The coefficients obtained at the minimum cross-validated error were used to generate PolyIQ scores, *PolyFluid* and *PolyCryst*, that represent the predicted fluid and crystallized IQ based on dietary PCs. In other words, these scores are linear combinations of food intake components weighted to best predict each cognitive dimension, offering a nutrition-based latent cognitive index. They represent the portion of cognitive potential attributable to diet.

*Structural brain metrics.* We analyzed multiple structural brain metrics—cortical morphometry (thickness, area, volume) and white matter microstructure (FA)—to capture both gray and white matter properties. These data were extracted from T1- and diffusion-weighted MRI. To examine how nutrition influences cognitive development, we selected structural brain features based on their established neuroanatomical roles in memory,

language, and executive function, as well as their known sensitivity to both developmental and environmental influences. Given the extensive phenotypic variability within the ABCD Study cohort, we grounded our selection of regions and tracts in prior research to focus on theoretically relevant metrics and enhance our ability to detect meaningful associations (see Table 1). A full list of the specific measures can be found in Appendix 4.

For white matter, we focused on fractional anisotropy (FA) in tracts known to support distinct cognitive domains. The fornix and hippocampal cingulum were selected for their roles in memory consolidation and hippocampal connectivity. The left inferior longitudinal fasciculus, part of the ventral visual stream, was included for its role in semantic processing and reading, linking it to crystallized intelligence. The forceps major, a posterior callosal tract, supports visual-spatial integration and processing speed, and was included for its potential link to fluid reasoning performance. These tracts represent plausible neurodevelopmental pathways through which nutrition could influence cognition by promoting white matter integrity and myelination. Out of the 3308 participants, only 1520 had complete data.

For gray matter, we examined volume, surface area, and cortical thickness across a set of regions selected for their relevance to cognitive function. This included subcortical structures such as the hippocampus and parahippocampal gyrus, which are central to memory, cognitive integration, and reward-based planning. These areas are highly plastic during development and are particularly sensitive to nutritional influences in childhood. We also included cortical regions implicated in language processing, such as the inferior frontal and superior temporal gyri, which support language production and comprehension. In addition, we examined the dorsolateral prefrontal cortex (DLPFC), a key hub within the executive control network. Out of the 3308 participants, only 1864 had complete data for volumes, 2053 for surface area and 1647 for cortical thickness.

<b>Functional Domain</b>	<b>Brain Region</b>	<b>Structural Feature</b>
Memory & Learning	Hippocampus	Volume
Memory & Learning	Parahippocampal gyrus	Thickness / Area
Memory & Learning	Cingulum	Fractional Anisotropy (FA)
Memory & Learning	Fornix	Fractional Anisotropy (FA)
Language & Crystallized IQ	Inferior frontal gyrus (Broca)	Thickness
Language & Crystallized IQ	Inferior longitudinal fasciculus	Fractional Anisotropy (FA)
Executive Function & Fluid IQ	DLPFC (Middle frontal gyrus)	Thickness
Executive Function & Fluid IQ	Caudate nucleus	Volume
Executive Function & Fluid IQ	Forceps major	Fractional Anisotropy (FA)

**Table 1:** Structural features by region and cognitive domain

*Covariates.* To account for individual differences in biological and environmental conditions that may influence cognitive outcomes, we retained a set of covariates that represent both developmental and contextual factors. Age and pubertal stage serve as proxies for biological maturation, capturing chronological and hormonal changes that impact brain and behavior during adolescence. Income-to-needs ratio (INR) and socioeconomic status (SES) were included as indicators of environmental context and access to resources, both of which are known to shape neurocognitive development. INR was calculated based on reported income and 2021 federal poverty thresholds adjusted for household size. We used the SES latent measure provided by ABCD. While INR and SES are both factors of socioeconomic conditions, INR is a relative measure of economic hardship while SES is a composite measure incorporating income, parental education, and occupational status. In our sample, the two were significantly correlated but not identical (Pearson correlation  $r=0.53$ ,  $p < 0.0001$ ).

We also included sleep measures because sleep has established links to cognitive performance and brain health that need to be differentiated from nutrition. Sleep duration (SleepD) and sleep efficiency (SleepE) were retained to control for variability in sleep quantity and quality. Social jetlag (SJL) was included to account for misalignment between biological rhythms and social schedules, particularly relevant in adolescent populations. These measures were extracted from the Munich Chronotype Questionnaire (MCTQ) that assess individuals’ habitual sleep-wake patterns. SleepD was calculated as the average sleep duration during a 7 day period (combining weekdays and weekend), SleepE was defined as the percentage of time asleep over time in bed, and SJL was defined as the absolute difference between the midsleep point on free days and the midsleep point on workdays.

Variable	Mean	SD
age	13.699	2.544
PubertalStage	3.593	0.736
INR	3.871	2.621
SES	0.085	0.870
SleepD	6.688	1.197
SleepE	73.232	16.390
SJL	2.050	1.590

**Table 2:** Means and standard deviations of key variables.

As shown in Table 2, the sample was, on average, in mid-pubertal stages. INR and SES values reflect a diverse socioeconomic distribution, with some degree of economic disadvantage. Participants reported an average of 6.7 hours of sleep per night across the week, with an average sleep efficiency of 73.2%, indicating that a substantial portion of time in

bed was not spent asleep. Social jetlag averaged 2.05 hours, suggesting noticeable shifts in sleep timing between school and free days. These descriptive statistics highlight meaningful individual differences across biological and environmental dimensions, justifying their inclusion as control variables in subsequent analyses.

*Empirical strategy.* We conducted multivariate linear regressions to examine the predictors of cognitive outcomes. These models allowed us to estimate independent and interactive effects of covariates and PolyIQ scores on cognition while controlling for demographic and contextual covariates. These models also included interaction terms to examine moderation effects (e.g., how the influence of diet varies by SES or sleep patterns). To explore mechanisms, we employed causal mediation analysis to determine whether brain structure metrics—both gray and white matter—mediated the effect of PolyIQ on IQ. We first examined the associations between these brain measures and cognitive performance, applying false discovery rate (FDR) correction to account for multiple comparisons. We then assessed whether nutritional factors may serve as potential mediators in these brain–cognition relationships. Mediation analyses were performed using nonparametric bootstrapping to estimate average causal mediation effects (ACME) and assess the proportion of the total effect explained by structural brain features. Additionally, our strategy included adiposity measures (BMI and waist circumference) as potential mediators or moderators, testing whether central or general adiposity linked dietary patterns to changes in neural structure and, consequently, to cognitive performance. This multi-layered approach allowed us to quantify both direct and indirect pathways from food intake to cognitive outcomes, via physical health and neural structure, while adjusting for confounding variables.

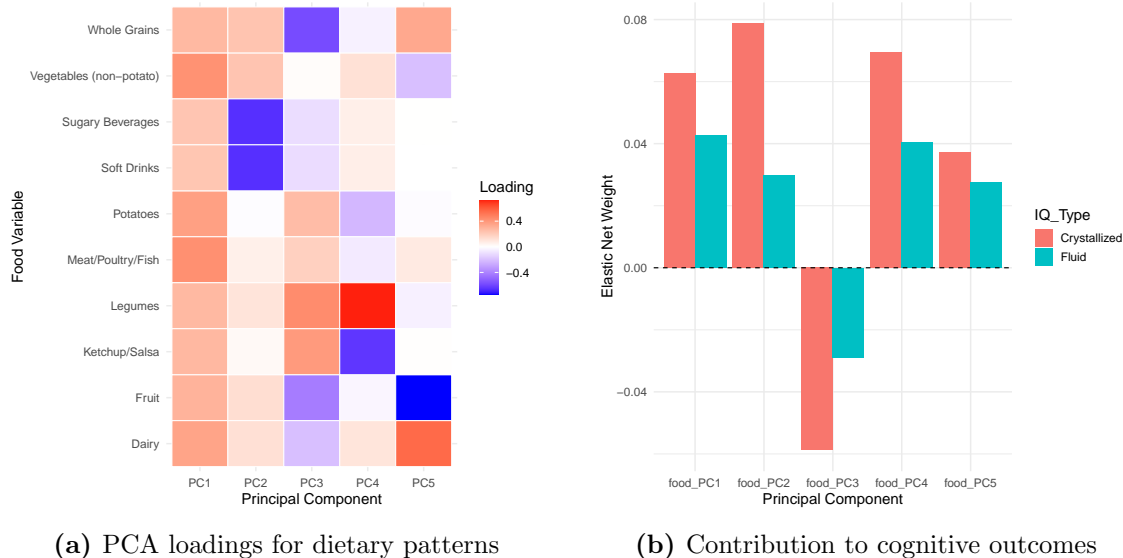
## 3 Results

### 3.1 PolyIQ scores

The first five principal components explained approximately 77% of the total variance in the data. Loadings are depicted in Figure 1(left). Food-PC1 - **Whole-Food Dietary Pattern**- captured strong positive loadings on fruits (0.42), vegetables (0.39), legumes (0.35), dairy (0.38), and meat/fish (0.37). This component represents adherence to dietary guidelines recommending consumption of a variety of minimally processed foods, and higher scores indicate broadly nutritious and balanced diets rich in essential nutrients, fiber, and high-quality protein. Food-PC2 - **Sugar-Avoidance Pattern**- was defined by strong negative loadings on sugary beverages (-0.48) and soft drinks (-0.45), contrasting individuals with low versus high consumption of sugar-sweetened drinks. This compo-

ment primarily reflects adherence to recommendations to limit added sugars, particularly from liquid sources that provide calories without nutritional value. Food-PC3 -**Non-Traditional Protein Pattern**- reflected a contrast between traditional health-associated foods—such as whole grains (-0.37) and fruits (-0.32) with negative loadings—and higher intake of legumes (0.41), potatoes (0.38), and condiments (0.36) with positive loadings. This component may capture a culturally or socioeconomically patterned dietary style that prioritizes accessible, energy-dense protein and starch sources over more expensive produce and grains. Food-PC4 - **Balanced Staples Pattern**- captured variation in consumption of balanced dietary staples, with moderate positive loadings on lean proteins (0.28), vegetables (0.24), and dairy (0.27), along with modest negative loadings on condiments (-0.22). This pattern represents a diet featuring nutritionally complete meals with limited use of added fats and sodium from condiments. Last, Food-PC5 -**Dairy-Fruit Contrast** - was characterized by positive loadings on dairy products (0.39) and negative loadings on fruits (-0.35), potentially reflecting contrasting sources of dietary calcium and carbohydrates. Food-PC4 and Food-PC5 accounted for smaller proportions of variance and reflected more nuanced dietary contrasts.

Elastic net models revealed that multiple dietary patterns predicted cognitive outcomes. Crystallized IQ was most strongly associated with food-PC2 ( $\beta = 0.079$ ) and food-PC4 ( $\beta = 0.070$ ), reflecting reduced sugary drink consumption and a balanced intake of staple foods. Food-PC1, a general healthy eating pattern, also contributed positively ( $\beta = 0.063$ ). For fluid IQ, predictive weights were smaller but followed a similar pattern, with food-PC4 ( $\beta = 0.040$ ) and food-PC1 ( $\beta = 0.043$ ) showing the strongest associations. These results suggest that broader dietary quality and specific patterns of food consumption may shape cognitive development in distinct ways. The results are described in Figure 1.



**Figure 1: Construction of nutrition-based cognitive potential scores (PolyIQ).** (A) Principal component loadings showing dietary patterns: PC1 (whole foods), PC2 (sugar avoidance), PC3 (processed foods), PC4 (balanced staples), PC5 (dairy-fruit contrast). (B) Elastic net regression coefficients indicating each dietary pattern’s contribution to fluid and crystallized intelligence predictions. Higher values indicate stronger predictive relationships.

### 3.2 Nutrition and IQ

In multivariable linear regression models (Table 3), nutrition-based PolyIQ scores remained significant predictors of cognitive outcomes after controlling for socioeconomic status (SES), income-to-needs ratio (INR), sleep duration and efficiency, age, pubertal stage, and sex. For crystallized IQ, the effect of diet was robust ( $\beta = 0.55$ ,  $p < 0.0001$ ), comparable in magnitude to that of SES ( $\beta = 0.17$ ) and INR ( $\beta = 0.16$ ), explaining 16% of the variance. For fluid IQ, although overall variance explained was smaller (9%), diet remained a significant predictor ( $\beta = 0.59$ ,  $p < 0.0001$ ), stronger than either SES or INR. We also found social jetlag showed consistent associations with cognitive performance. In the model predicting crystallized IQ, greater social jetlag was significantly associated with lower IQ scores ( $\beta = -0.115$ ,  $p < 0.0001$ ). The same was true, although less striking, in the case of fluid intelligence ( $\beta = -0.065$ ,  $p < 0.0001$ )

	Cryst. IQ	Fluid IQ
Intercept	0.028 (0.023)	-0.003 (0.019)
PolyIQ_cryst	0.554*** (0.086)	
SJL	-0.115*** (0.020)	-0.065*** (0.017)
SleepD	-0.008 (0.029)	0.031 (0.024)
SleepE	0.022 (0.029)	-0.001 (0.025)
Age	-0.205*** (0.047)	-0.043 (0.040)
Pubertal Stage	0.073*** (0.019)	-0.011 (0.016)
Female	-0.044 (0.038)	0.027 (0.032)
SES	0.170*** (0.019)	0.124*** (0.016)
INR	0.155*** (0.017)	0.080*** (0.015)
PolyIQ_fluid		0.590*** (0.131)
R <sup>2</sup>	0.165	0.091
Adj. R <sup>2</sup>	0.163	0.088
Num. obs.	2905	2905

\*\*\*  $p < 0.001$ ; \*\*  $p < 0.01$ ; \*  $p < 0.05$

**Table 3:** Linear regression models predicting IQ from nutrition PolyIQ and covariates.

Interaction models further showed that the effect of crystallized PolyIQ on IQ was moderated by SES and sleep factors. Also, we found that nutrition also partially mediated the effects of socioeconomic factors on cognitive outcomes, accounting for 6-8.5% of SES and INR effects on IQ (see Appendix 5).

### 3.3 Neural correlates

The main associations and the detailed mediation results are reported in Appendix 6.

*Volumes.* Of the 30 regions we retained, we found that 25 regional brain volumes were associated with either crystallized or fluid IQ after FDR corrections. Mediation analyses revealed that volumes in specific brain regions partially explained the effect of nutrition-based cognitive potential on IQ. Notably, volumes in the left parahippocampal gyrus, left pars opercularis, and right middle temporal gyrus significantly mediated the relationship

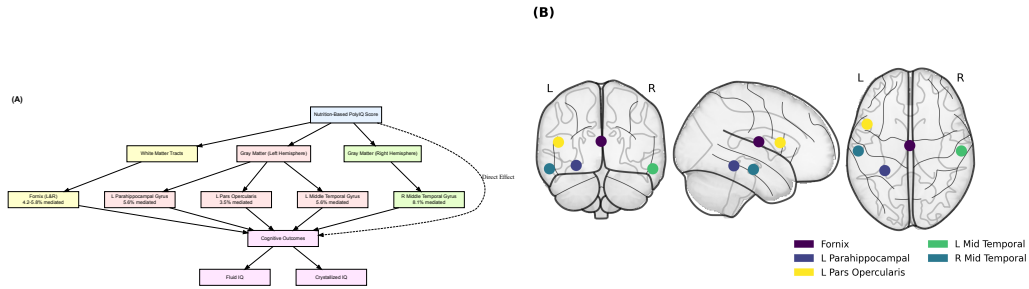
between crystallized PolyIQ scores and crystallized IQ. The left parahippocampal gyrus also mediated the effect of fluid PolyIQ on fluid IQ, suggesting its broader relevance across cognitive domains. While the proportion of variance mediated was modest (ranging from 3.6% to 8.1%), these effects were statistically significant.

*Surface Area.* From the 12 regions we retained, we did not find any significant surface area-IQ associations after FDR corrections in the regions of interest.

*Cortical Thickness.* Of the 12 regions we retained, only the left and right ParaHippocampal gyri were associated with both crystallized and fluid IQ. Cortical thickness in the left parahippocampal gyrus may partially mediate the effect of fluid PolyIQ scores on fluid IQ, although this mediation was only marginally significant ( $p = 0.050$ ). Approximately 3.6% of the total effect of nutrition-based fluid cognitive potential on IQ performance may be explained by structural differences in this brain region, which is known to support memory and contextual reasoning.

*FA.* Six of the seven tracts showed significant associations with IQ. The left and right fornix and left and right hippocampal cingulum were positively correlated with both crystallized and fluid IQ, consistent with their known roles in memory and learning. The left inferior longitudinal fasciculus was more strongly associated with crystallized IQ, reflecting its role in semantic processing, while the forceps major was associated with fluid IQ, suggesting a role in cross-hemispheric visual integration. Mediation analyses revealed that fractional anisotropy (FA) in the fornix significantly mediated the relationship between nutrition-based PolyIQ scores and IQ. For both crystallized and fluid intelligence, white matter microstructure in the left and right fornix accounted for a small but statistically significant proportion of the total effect (4.1–6.9%). The indirect effects were stronger for fluid IQ, aligning with the role of the fornix in memory and flexible reasoning.

Table 4 summarizes the main mediation effects and Figure 2 illustrates the key brain regions that mediate the relationship between nutrition-based cognitive potential (PolyIQ) and cognitive outcomes in youth. Together, the findings suggest that nutrition may influence cognitive development through specific neuroanatomical pathways connecting medial temporal structures to frontal and lateral temporal regions, with the fornix serving as a critical white matter tract linking these areas. These brain regions support memory formation, language processing, and semantic knowledge—functions essential for both fluid reasoning and crystallized intelligence.



**Figure 2:** Mediation effects: (A) Path diagram of the analysis (left). Simplified mediation model showing the proportion of variance in the nutrition-cognition relationship explained by each neural mediator. (B) Brain regions mediating the effects (right): (i) Cortical regions that significantly mediated the relationship between nutrition-based cognitive potential (PolyIQ) and crystallized intelligence, including the left parahippocampal gyrus (dark blue), left pars opercularis (yellow), and middle temporal gyrus (green tones). (ii) White matter tract visualization of the fornix (purple), which consistently mediated both fluid and crystallized intelligence outcomes. L = left hemisphere; R = right hemisphere.

Brain Region	Cognitive Domain	% Mediated
<i>White Matter</i>		
Fornix (bilateral)	Fluid IQ	5.8-5.9%
Fornix (bilateral)	Crystallized IQ	3.5-4.2%
<i>Gray Matter</i>		
L Parahippocampal Gyrus	Crystallized IQ	5.6%
L Parahippocampal Gyrus	Fluid IQ	3.6%
L Pars Opercularis	Crystallized IQ	3.5%
L Middle Temporal Gyrus	Crystallized IQ	5.6%
R Middle Temporal Gyrus	Crystallized IQ	8.1%

**Table 4: Summary of neural mediation effects.** Percentage of total nutrition-cognition relationship explained by each brain region. All effects significant at  $p < 0.05$ . Complete statistics in Appendix 6.

### 3.4 Nutrition and Neural Correlates

We examined the relationship between neural correlates and food PCs after controlling for age, sex, SES, and INR (see Appendix 7). Most notably, we found that the sugar-avoidance pattern (PC2) demonstrated beneficial associations with gray matter volumes in language-processing regions, including the left pars opercularis and bilateral middle temporal gyri ( $p < 0.05$ ), indicating that limiting sugar-sweetened beverage consumption may preserve cortical structures essential for semantic processing. The processed food

pattern (PC3) showed detrimental associations with bilateral fornix white matter integrity ( $p < 0.05$ ) and left parahippocampal volume ( $p < 0.05$ ), consistent with evidence that ultra-processed foods may impair brain structure development.

### 3.5 Adiposity as a Behavioral Pathway Linking Nutrition and Brain Structure

We extended our analysis to examine whether dietary patterns predictive of PolyIQ scores were also associated with physical health markers known to impact neurodevelopment. Multivariate regression models revealed that higher sugary beverage consumption was significantly associated with both higher BMI and larger waist circumference, while whole grain intake was associated with lower BMI (see Appendix 3). Notably, waist circumference—but not BMI—remained a significant negative predictor of fluid IQ after adjusting for SES, INR, and age (see Appendix 8). Building on these findings, we examined the potential link between adiposity and the brain regions that mediated the relationship between PolyIQ scores and cognitive outcomes.

The fornix (bilaterally) and left parahippocampal gyrus (volume and cortical thickness) emerged as mediators in the case of fluid intelligence. We found that central adiposity was associated with hippocampal cortical thickness after controlling for age and sex, raising the possibility that waist circumference may reflect not only metabolic burden but also a downstream effect of dietary habits with consequences for memory-related neurocircuitry supporting fluid intelligence. This effect vanished however after controlling for SES and INR.

For crystallized intelligence, we found a significant negative association between volumes in the middle temporal gyrus (both left and right hemispheres) and adiposity (BMI and waist circumference) even after controlling for SES and INR.

These findings suggest that adiposity may be part of a behavioral pathway through which nutrition affects brain structure and, in turn, cognitive performance. Specifically, diets high in added sugars and low in whole grains may elevate waist circumference and BMI, which are associated with alterations in temporal lobe structures (lateral and medial) that support both crystallized and fluid intelligence.

## 4 Discussion

Our findings provide new evidence for the neurobiological pathways linking dietary patterns to cognitive outcomes in children, emphasizing the mediating role of specific brain structures and physical health markers. We show that brain morphology partially mediates the effect of a nutrition-based cognitive potential score (PolyIQ) on both fluid and

crystallized IQ. Among all brain features examined, white matter integrity in the fornix emerged as the most consistent mediator across both IQ domains, while the left parahippocampal gyrus stood out for its relevance in both cortical thickness and volume-based models.

### **The Fornix as a Core Mediator**

The fornix, a major white matter tract connecting the hippocampus to other cortical and subcortical memory systems, was the only region to significantly mediate the effect of nutrition on both crystallized and fluid IQ. This finding supports its central role in episodic and semantic memory and suggests that nutritional input may influence cognitive development through its effects on hippocampal–fornical circuitry. Prior literature has linked fornix development to SES, early adversity, and dietary quality, further validating its role as a sensitive biomarker of cognitive potential.

### **Cortical and Subcortical Gray Matter Contributions**

While the hippocampus and caudate nucleus are known for their involvement in learning and executive function, our data indicate that their volumes are only modestly predictive of fluid intelligence and do not significantly mediate the effects of nutrition. Instead, cortical structures in the medial and lateral temporal lobe—including the left parahippocampal gyrus, left pars opercularis, and right middle temporal gyrus—were identified as key mediators, particularly for crystallized IQ. These regions are involved in memory encoding, language processing, and contextual learning, which are foundational to knowledge accumulation and flexible reasoning.

The repeated emergence of the left parahippocampal gyrus across multiple models—both in volume and cortical thickness—highlights it as a convergent neural substrate of nutritional effects on cognition. This region’s role in memory, navigation, and contextual integration may make it particularly responsive to early-life dietary exposures.

### **Integrated Temporal-Limbic Circuitry**

The joint involvement of the fornix and left parahippocampal gyrus points to a coordinated medial temporal lobe network through which nutrition may support cognitive development. The fornix facilitates communication between the hippocampus and frontal/subcortical areas, while the parahippocampal region contributes to higher-order memory integration. Their joint mediation suggests that nutrition supports both the integrity of individual structures and the efficiency of information flow across the memory system. Such enhancements may enable children to better encode, consolidate, and retrieve information—critical for both fluid reasoning and crystallized knowledge.

### **Role of Adiposity and Metabolic Pathways**

Beyond direct effects of diet on brain and cognition, our findings implicate central adiposity—measured via waist circumference—as a parallel pathway. Waist circumference negatively predicted fluid IQ even after adjusting for BMI, SES, and income-to-needs ratio, and was linked to structural features such as fornix FA and parahippocampal morphology. This supports the view that adiposity may reflect metabolic dysregulation (e.g., inflammation, insulin resistance) with downstream effects on brain development, particularly in memory-related regions. Given the high plasticity and metabolic sensitivity of the developing brain, these findings raise concerns about dietary patterns that promote central adiposity in youth.

### **Sugar Avoidance and Cognitive-Neural Benefits**

Consistent with the known detrimental effects of added sugars on neurodevelopment, dietary patterns characterized by lower consumption of sugar-sweetened beverages (PC2) were associated with higher crystallized IQ and increased volumes in language and memory-related brain regions, particularly the middle temporal gyrus and pars opercularis. This finding aligns with experimental evidence demonstrating that high sugar intake can impair synaptic plasticity, promote neuroinflammation, and reduce brain-derived neurotrophic factor expression in regions critical for learning and semantic processing. The positive association between sugar avoidance and temporal lobe structure provides neuroanatomical evidence supporting public health recommendations to limit sugar-sweetened beverage consumption during adolescence, a period of heightened neuroplasticity when dietary influences on brain development may have lasting consequences. These results complement our other dietary findings, demonstrating that multiple dimensions of healthy eating—including whole food consumption and sugar avoidance—converge to support optimal cognitive and neural development.

### **4.1 Limitations and Future Directions**

Several limitations of the present study warrant consideration when interpreting the results. First and foremost, the cross-sectional nature of our analysis precludes causal inferences about the directionality of the observed relationships. Although we identify significant associations between dietary patterns, brain structure, and cognitive outcomes, we cannot determine whether nutritional factors actively shape neural development and subsequent cognitive abilities, or whether pre-existing cognitive and neural traits influence dietary choices. Bidirectional relationships are likely, as children with stronger executive function may make healthier food choices, while nutritionally dense diets simultaneously support optimal brain development ([Diamond, 2013](#)).

The dietary assessment methods also present inherent limitations. The Block Kids

Food Screener, while validated for population-level research, captures only a snapshot of dietary intake and may be subject to recall bias and social desirability effects. Furthermore, parental reporting of children’s dietary intake could introduce additional biases, particularly for older adolescents who consume more meals outside the home (Burrows et al., 2010). Future research would benefit from more comprehensive dietary assessment methods, including multiple 24-hour recalls or biomarker validation, to strengthen the reliability of nutritional data.

Another limitation is our treatment of SES and income-to-needs ratio as static variables. Socioeconomic circumstances may fluctuate throughout childhood, and the timing, duration, and developmental period of economic hardship or advantage may differentially impact nutritional access and cognitive development (Evans and Fuller-Rowell, 2013). More granular measures of socioeconomic trajectories and food security would provide greater insight into these complex relationships.

While our mediation analyses identified significant brain-based pathways linking diet to cognition, these pathways accounted for a relatively modest proportion of the total effect (typically 4-8%). This suggests that numerous other mechanisms—including unmeasured neurobiological pathways, metabolic factors, gut microbiome composition, and gene-environment interactions—likely contribute to the diet-cognition relationship (Sarkar et al., 2018). Additionally, our focus on specific brain regions, while theoretically grounded, precluded examination of broader network-level properties that might mediate nutritional effects on cognition.

## 4.2 Developmental Trajectories and Longitudinal Implications

Our cross-sectional findings raise important questions about developmental trajectories that can only be addressed through longitudinal research. The ABCD Study’s ongoing data collection provides an unprecedented opportunity to examine how early dietary patterns predict changes in brain structure and cognitive performance across adolescence. Several developmental hypotheses warrant investigation in future longitudinal analyses.

First, nutritional effects on brain structure may accumulate over time, with dietary patterns during sensitive periods having disproportionate impacts on specific neural circuits. The fornix and parahippocampal regions identified in our study undergo protracted development throughout adolescence, potentially creating extended windows of nutritional sensitivity (Lebel and Deoni, 2018). Tracking how nutrition-brain-cognition relationships evolve with age could identify optimal periods for dietary intervention.

Second, the relative contributions of fluid and crystallized intelligence to overall cognitive functioning change across development, with fluid abilities typically peaking in early adulthood while crystallized abilities continue to develop across the lifespan (Li et al.,

2004). The differential associations between dietary patterns and these cognitive domains observed in our study may have important implications for academic and socio-emotional development as adolescents transition to adulthood.

Third, the interaction effects between nutrition and socioeconomic factors observed in our data suggest possible divergent developmental trajectories based on environmental context. Children from lower-SES backgrounds showed stronger associations between nutrition and fluid intelligence, suggesting that dietary quality might serve as a protective factor against environmental adversity. Longitudinal research could determine whether nutritional interventions might help narrow socioeconomic achievement gaps over time.

### 4.3 Implications for Interventions and Public Health Policy

Our findings have several implications for designing effective nutritional interventions and informing public health policies aimed at supporting optimal cognitive development in youth. First, the specificity of the brain-based pathways identified—particularly the fornix and parahippocampal structures—suggests that nutritional interventions should target macronutrients and micronutrients known to support white matter integrity and hippocampal development. These include omega-3 fatty acids, choline, zinc, and B vitamins (Nyaradi et al., 2013a).

Second, the observed interaction between nutrition and socioeconomic factors underscores the importance of contextually sensitive interventions. Rather than one-size-fits-all approaches, nutritional programs should be tailored to family resources, cultural preferences, and community contexts. School-based interventions that combine nutritional education with improved access to healthy foods show particular promise for reaching children across socioeconomic strata (Wang and Stewart, 2013).

Third, our finding that central adiposity independently predicts lower fluid intelligence, even after controlling for BMI and socioeconomic factors, points to the potential cognitive benefits of targeted obesity prevention efforts. Public health policies that focus on reducing consumption of ultra-processed foods and sugar-sweetened beverages—which showed the strongest associations with waist circumference in our sample—may yield both metabolic and cognitive benefits.

Fourth, the observed relationships between social jetlag, nutrition, and cognitive outcomes highlight the importance of integrative approaches that address multiple health behaviors. Policies supporting later school start times to align with adolescent chronobiology may improve both sleep patterns and dietary choices, with cascading benefits for brain development and academic performance (Minges and Redeker, 2016).

Finally, our results suggest that nutrition should be considered a core component of educational policy. The cognitive impacts of dietary quality observed in our study—comparable

in magnitude to socioeconomic effects—indicate that nutritional support may be as important as traditional educational interventions for promoting academic success. Integrating evidence-based nutrition programs into educational settings could help maximize cognitive potential during this critical developmental period.

Future intervention studies should build on these findings by testing whether dietary improvements lead to measurable changes in the neural structures identified in our study, and whether these changes mediate subsequent improvements in cognitive performance. Such research would provide stronger causal evidence and help refine the timing, content, and delivery of nutritional interventions aimed at supporting optimal brain development during adolescence.

## 5 Conclusion

This study provides novel evidence linking dietary patterns to brain structure and cognitive functioning in children, revealing both direct and indirect pathways through which nutrition may shape intellectual development. We demonstrate that healthy dietary habits are consistently associated with higher IQ, and that these associations are partly mediated by structural features of the brain—most notably, white matter integrity in the fornix and gray matter morphology in the parahippocampal and temporal regions. These brain-based mediators align with known memory and language networks, underscoring the neurobiological plausibility of a nutrition–cognition link.

We also show that central adiposity, as indexed by waist circumference, is a meaningful predictor of fluid intelligence and interacts with neural pathways in ways that merit further investigation. While some associations with sugary dietary patterns and higher IQ were observed, they likely reflect residual sociodemographic confounding rather than nutritional benefit per se.

Taken together, our findings highlight the importance of early-life dietary quality not only for physical health but also for neurocognitive development. They suggest that investment in healthy eating habits during childhood could yield long-term cognitive dividends by supporting brain systems foundational to learning and memory. Future longitudinal and intervention studies will be crucial to establishing causality and identifying specific mechanisms through which nutritional inputs shape the developing brain.

## References

- S. Adise et al. Sex-specific impulsivity predicts fat and sugar intake among adolescents: Findings from the abcd study. *Appetite*, 192:107081, 2024.
- Shana Adise, Nicholas Allgaier, Jennifer Laurent, Sage Hahn, Bader Chaarani, Max Owens, DeKang Yuan, Philip Nyugen, Scott Mackey, Alexandra Potter, et al. Multimodal brain predictors of current weight and weight gain in children enrolled in the abcd study®. *Developmental Cognitive Neuroscience*, 49:100948, 2021.
- K. Agarwal et al. Risk assessment of maladaptive behaviors in adolescents: Nutrition, screen time, prenatal exposure, childhood adversities—abcd study. *Journal of Adolescent Health*, 2023.
- N. Allen. A compositional analysis of leisure screen time and body mass index in the abcd study. *Obesity*, 31:146–147, 2023.
- Jessica E Beilharz, Jayanthi Maniam, and Margaret J Morris. Diet-induced cognitive deficits: the role of fat and sugar, potential mechanisms and nutritional interventions. *Nutrients*, 7(8):6719–6738, 2015.
- Sarah-Jayne Blakemore. Imaging brain development: the adolescent brain. *Neuroimage*, 61(2):397–406, 2012.
- Tracy L Burrows, Rebecca J Martin, and Clare E Collins. A systematic review of the validity of dietary assessment methods in children when compared with the method of doubly labeled water. *Journal of the American Dietetic Association*, 110(10):1501–1510, 2010.
- Juliana FW Cohen, Mary T Gorski, Staci A Gruber, LBF Kurdziel, and Eric B Rimm. The effect of healthy dietary consumption on executive cognitive functioning in children and adolescents: a systematic review. *British Journal of Nutrition*, 116(6):989–1000, 2016.
- Adele Diamond. Executive functions. *Annual review of psychology*, 64(1):135–168, 2013.
- Gary W Evans and Thomas E Fuller-Rowell. Childhood poverty, chronic stress, and young adult working memory: The protective role of self-regulatory capacity. *Developmental science*, 16(5):688–696, 2013.
- Michael K Georgieff. Nutrition and the developing brain: nutrient priorities and measurement. *The American journal of clinical nutrition*, 85(2):614S–620S, 2007.

- Jay N Giedd. The teen brain: insights from neuroimaging. *Journal of adolescent health*, 42(4):335–343, 2008.
- Fernando Gómez-Pinilla. Brain foods: the effects of nutrients on brain function. *Nature reviews neuroscience*, 9(7):568–578, 2008.
- J.C. Gray et al. Demographic, psychological, behavioral, and cognitive correlates of bmi in youth: Findings from the abcd study. *Psychological Medicine*, 50(9):1539–1547, 2020.
- R.J.F. Green. Health behaviors and the adolescent brain: Predicting cognition from physical activity and diet in children enrolled in the abcd study. *Unpublished manuscript*, 2022.
- Felice N Jacka, Nicolas Cherbuin, Kaarin J Anstey, Perminder Sachdev, and Peter But-terworth. Western diet is associated with a smaller hippocampus: a longitudinal inves-tigation. *BMC medicine*, 13:1–8, 2015.
- Catherine Lebel and Sean Deoni. The development of brain white matter microstructure. *Neuroimage*, 182:207–218, 2018.
- Shu-Chen Li, Ulman Lindenberger, Bernhard Hommel, Gisa Aschersleben, Wolfgang Prinz, and Paul B Baltes. Transformations in the couplings among intellectual abil-ities and constituent cognitive processes across the life span. *Psychological science*, 15(3):155–163, 2004.
- Z.A. Li et al. Weight indices, cognition, and mental health from childhood to early ado-lescence. *JAMA Pediatrics*, 178(8):830–833, 2024.
- Karl E Mingos and Nancy S Redeker. Delayed school start times and adolescent sleep: a systematic review of the experimental evidence. *Sleep medicine reviews*, 28:86–95, 2016.
- J.M. Nagata et al. Physical activity, screen time, and bmi among adolescents: A cross-sectional analysis. *JAMA Network Open*, 2023.
- J.M. Nagata et al. Social epidemiology of early adolescent nutrition. *Pediatric Research*, 2025. In press.
- Anett Nyaradi, Jianghong Li, Samuel Hickling, Janine Foster, and Wendy H Oddy. Prospective associations between dietary patterns and cognitive performance during adolescence. *Journal of Child Psychology and Psychiatry*, 54(9):1017–1024, 2013a.

- Anett Nyaradi, Jianghong Li, Siobhan Hickling, Jonathan Foster, and Wendy H Oddy. The role of nutrition in children’s neurocognitive development, from pregnancy through childhood. *Frontiers in human neuroscience*, 7:97, 2013b.
- M.D. Parrott et al. Dietary patterns and cognitive function in older adults. *The Journals of Gerontology: Series A*, 68(6):682–689, 2013.
- V. Rashid et al. Ethnicity and socioeconomic status are related to dietary patterns at age 5 in the abcd cohort. *BMC Public Health*, 18(1):1–10, 2018.
- V. Rashid et al. Beyond maternal education: Socio-economic inequalities in children’s diet in the abcd cohort. *PLoS One*, 15(10):e0240423, 2020a.
- V. Rashid et al. Weight development between age 5 and 10 years and its associations with dietary patterns at age 5 in the abcd cohort. *BMC Public Health*, 20:1–11, 2020b.
- K. Rozzell et al. Prevalence of eating disorders among us children aged 9 to 10 years: Data from the abcd study. *JAMA Pediatrics*, 173(1):100–101, 2019.
- Amar Sarkar, Siobhán Harty, Soili M Lehto, Andrew H Moeller, Timothy G Dinan, Robin IM Dunbar, John F Cryan, and Philip WJ Burnet. The microbiome in psychology and cognitive neuroscience. *Trends in cognitive sciences*, 22(7):611–636, 2018.
- Annelien C Van den Brink, Elske M Brouwer-Brolsma, Agnes AM Berendsen, and Ondine van de Rest. The mediterranean, dietary approaches to stop hypertension (dash), and mediterranean-dash intervention for neurodegenerative delay (mind) diets are associated with less cognitive decline and a lower risk of alzheimer’s disease—a review. *Advances in nutrition*, 10(6):1040–1065, 2019.
- Dongxu Wang and Donald Stewart. The implementation and effectiveness of school-based nutrition promotion programmes using a health-promoting schools approach: a systematic review. *Public health nutrition*, 16(6):1082–1100, 2013.

## Supplementary material

### Appendix 1: Descriptive analysis of nutrition

We analyzed dietary intake data from 3,308 participants using the BKFS questionnaire. Average daily consumption was highest for meat/poultry/fish (mean = 0.95 cup/oz equivalents), dairy (0.64), and fruit (0.63), with lower intake for vegetables excluding potatoes (0.42), whole grains (0.29), and potatoes (0.17). Legume consumption was minimal (mean = 0.04, highly right-skewed).

Intake of added sugars averaged 1.69 teaspoons per day, while saturated fat intake was 2.52 grams per day. Total energy intake averaged 6.69 (standardized units), with carbohydrates, proteins, and total fats contributing approximately 4.57, 3.53, and 3.49 units, respectively. Fiber intake averaged 2.09 units.

The glycemic index (GI) and glycemic load (GL) were stable across participants (means = 3.93 and 3.81), showing limited variation. In contrast, consumption of sugary beverages (mean = 2.30 kcal/day), soft drinks (2.82 g/day), and condiments like ketchup and salsa (0.60 g/day) exhibited high skewness and kurtosis, indicating that a subset of participants consumed disproportionately large amounts.

### Appendix 2: Adiposity and nutrition

We examined anthropometric data and found that the average body weight was 4.88 (SD = 0.25), and average height was 4.19 (SD = 0.05), both in standardized units. Mean waist circumference was 3.48 (SD = 0.16), with a range from 3.18 to 4.03, showing modest variability. The average BMI was 3.14 (SD = 0.22), with values ranging from 2.74 to 3.78 and moderate right skew (skew = 0.78, kurtosis = 0.30), indicating some children had notably higher BMI values. All anthropometric variables were approximately normally distributed, except for a slight positive skew in BMI and waist circumference, which may reflect early signs of overweight or obesity in a subset of participants.

Higher sugary beverage consumption was robustly linked to higher BMI and waist circumference, supporting its role as a key nutritional risk factor. Whole grain intake was associated with lower BMI, consistent with healthy eating guidelines. Interestingly, fruit intake showed a positive association with adiposity, which could reflect confounding factors such as fruit juice consumption or self-report bias. Total energy, saturated fat, and fiber intake were not significant predictors in this sample. Demographic variables (age, sex, SES, INR) explained a substantial share of variance, emphasizing their importance in childhood obesity risk.

	BMI	Waist circumference
(Intercept)	2.926*** (0.163)	3.316*** (0.125)
bkfs_dt_sug_t_y	-0.028 (0.017)	-0.039** (0.013)
bkfs_satfat_g_y	-0.022 (0.028)	-0.001 (0.021)
bkfs_dt_fibe_y	0.021 (0.020)	-0.014 (0.015)
bkfs_fruit_ce_y	0.043* (0.021)	0.047** (0.016)
bkfs_whlgrain_oze_y	-0.067** (0.023)	-0.025 (0.018)
bkfs_group_sugarybevg_total_kcal_y	0.006* (0.003)	0.005* (0.002)
bkfs_dt_kcal_y	-0.004 (0.039)	0.019 (0.030)
age	0.025*** (0.005)	0.015*** (0.004)
Sex2	0.020* (0.009)	-0.029*** (0.007)
SES	-0.059*** (0.006)	-0.038*** (0.004)
INR	-0.005** (0.002)	-0.003* (0.001)
R <sup>2</sup>	0.107	0.081
Adj. R <sup>2</sup>	0.103	0.077
Num. obs.	2454	2440

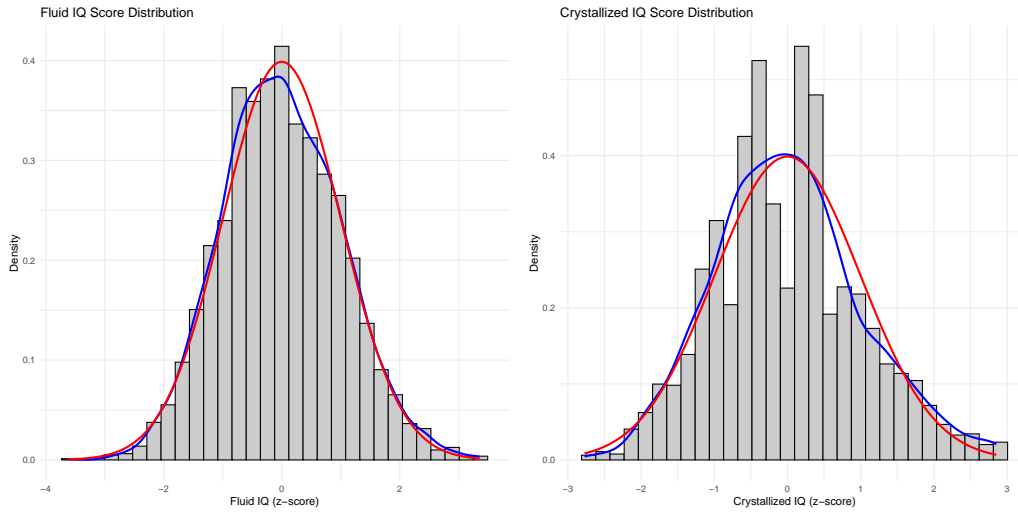
\*\*\*  $p < 0.001$ ; \*\*  $p < 0.01$ ; \*  $p < 0.05$

**Table 1:** Relationship between Nutrition, BMI and waist circumference

### Appendix 3: Analysis of IQ

The composite scores are illustrated in Figure 1. Both fluid and crystallized IQ scores are approximately normally distributed, though the crystallized IQ distribution shows slight skewness and kurtosis relative to the ideal Gaussian curve.

Crystallized and fluid intelligence scores are significantly correlated (Pearson's  $r = 0.44$ ,  $p < 0.001$ ), yet they show distinct patterns of association with demographic variables. SES and INR are the primary correlates of both components. , but the relationship is stronger for crystallized IQ. Regression analyses reveal that children from higher SES and INR backgrounds tend to have significantly higher crystallized IQ scores relative to fluid IQ scores. Pubertal stage is also positively associated with crystallized IQ but unrelated to



**Figure 1:** Distribution of composite scores. The blue line represents the kernel density estimate, while the red line shows the closest fitting normal distribution.

fluid IQ, suggesting that biological maturation contributes more to the accumulation of knowledge than to abstract reasoning. Interaction analyses indicate that the relationship between fluid and crystallized IQ does not vary by sex or SES. Overall, these results support the idea that crystallized intelligence is more environmentally shaped, while fluid intelligence may reflect more stable, intrinsic cognitive abilities.

To explore their relative influence, we computed the difference between fluid and crystallized IQ scores and compared SES and INR levels between children with relatively higher fluid intelligence and those with relatively higher crystallized intelligence. Children with higher crystallized IQ scores than fluid IQ scores were, on average, from households with significantly higher SES and INR (two-sample t-tests,  $p < 0.0001$  for both). Regression analysis confirm this result.

	Cryst. IQ	Fluid IQ	Difference
(Intercept)	0.032 (0.025)	0.001 (0.026)	-0.027 (0.023)
Sex2	-0.042 (0.042)	0.014 (0.043)	0.047 (0.038)
age <sub>z</sub>	-0.018 (0.016)	-0.001 (0.017)	0.015 (0.015)
PubertalStage <sub>z</sub>	0.055** (0.021)	-0.017 (0.021)	-0.060** (0.019)
INR <sub>z</sub>	0.199*** (0.020)	0.127*** (0.020)	-0.085*** (0.018)
SES <sub>z</sub>	0.227*** (0.020)	0.206*** (0.020)	-0.053** (0.018)
R <sup>2</sup>	0.143	0.086	0.025
Adj. R <sup>2</sup>	0.141	0.085	0.023
Num. obs.	3075	3075	3075

\*\*\*  $p < 0.001$ ; \*\*  $p < 0.01$ ; \*  $p < 0.05$

**Table 2:** Linear regression models predicting IQ from demographic variables

	Cryst IQ	Cryst IQ	Cryst IQ	Cryst IQ
(Intercept)	0.028 (0.021)	0.028 (0.021)	0.027 (0.021)	0.033 (0.021)
fluidIQ	0.448*** (0.020)	0.454*** (0.026)	0.448*** (0.020)	0.448*** (0.020)
Sex2	-0.041 (0.034)	-0.041 (0.034)	-0.041 (0.034)	-0.040 (0.034)
age_z	-0.016 (0.013)	-0.016 (0.013)	-0.016 (0.013)	-0.016 (0.013)
PubertalStage_z	0.054** (0.017)	0.054** (0.017)	0.054** (0.017)	0.053** (0.017)
INR_z	0.135*** (0.016)	0.135*** (0.016)	0.135*** (0.016)	0.138*** (0.016)
SES_z	0.134*** (0.016)	0.134*** (0.016)	0.135*** (0.017)	0.130*** (0.017)
fluidIQ:Sex2		-0.015 (0.039)		
fluidIQ:SES_z			0.003 (0.019)	
fluidIQ:INR_z				-0.033 (0.019)
R <sup>2</sup>	0.262	0.262	0.262	0.263
Adj. R <sup>2</sup>	0.261	0.261	0.261	0.261
Num. obs.	3075	3075	3075	3075

\*\*\*  $p < 0.001$ ; \*\*  $p < 0.01$ ; \*  $p < 0.05$

**Table 3:** Relationship between Crystallized and Fluid IQ

#### Appendix 4: Full list of gray and white matter measures

Variable Code	Region	Structure Type
fxrh	Right Fornix	FA
fxlh	Left Fornix	FA
cghrh	Right Hippocampal Cingulum	FA
cghlh	Left Hippocampal Cingulum	FA
ilfrh	Right Inferior Longitudinal Fasciculus	FA
ilflh	Left Inferior Longitudinal Fasciculus	FA
fmaaj	Forceps Major	FA
LHippo	Left Hippocampus	Volume
RHippo	Right Hippocampus	Volume
LCaudate	Left Caudate	Volume
RCaudate	Right Caudate	Volume
LParaHippo	Left Parahippocampal Gyrus	Volume
RParaHippo	Right Parahippocampal Gyrus	Volume
LParsOper	Left Pars Opercularis	Volume
RParsOper	Right Pars Opercularis	Volume
LParsOrbi	Left Pars Orbitalis	Volume
RParsOrbi	Right Pars Orbitalis	Volume
LParsTriang	Left Pars Triangularis	Volume
RParsTriang	Right Pars Triangularis	Volume
LFusi	Left Fusiform Gyrus	Volume
RFusi	Right Fusiform Gyrus	Volume
LMidTemp	Left Middle Temporal Gyrus	Volume
RMidTemp	Right Middle Temporal Gyrus	Volume
LRostMid	Left Rostral Middle Frontal Gyrus	Volume
RRostMid	Right Rostral Middle Frontal Gyrus	Volume
LCaudMid	Left Caudal Middle Frontal Gyrus	Volume
RCaudMid	Right Caudal Middle Frontal Gyrus	Volume
LFrontPole	Left Frontal Pole	Volume
RFrontPole	Right Frontal Pole	Volume
LInsula	Left Insula	Volume
RInsula	Right Insula	Volume
LPrecun	Left Precuneus	Volume
RPrecun	Right Precuneus	Volume
LPostGyrus	Left Postcentral Gyrus	Volume
RPostGyrus	Right Postcentral Gyrus	Volume
LParaCentLobule	Left Paracentral Lobule	Volume
RParaCentLobule	Right Paracentral Lobule	Volume
LParaHippo	Left Parahippocampal Gyrus	Thickness
RParaHippo	Right Parahippocampal Gyrus	Thickness

**Table 4:** Fractional anisotropy and cortical volume variables retained in the analyses

Variable Code	Region	Structure Type
LParsOper	Left Pars Opercularis	Thickness
RParsOper	Right Pars Opercularis	Thickness
LParsOrbit	Left Pars Orbitalis	Thickness
RParsOrbit	Right Pars Orbitalis	Thickness
LParsTriang	Left Pars Triangularis	Thickness
RParsTriang	Right Pars Triangularis	Thickness
LRostralMF	Left Rostral Middle Frontal Gyrus	Thickness
RRostralMF	Right Rostral Middle Frontal Gyrus	Thickness
LCaudalMF	Left Caudal Middle Frontal Gyrus	Thickness
RCaudalMF	Right Caudal Middle Frontal Gyrus	Thickness
LDLPFC	Left Dorsolateral Prefrontal Cortex	Area
RDLPFC	Right Dorsolateral Prefrontal Cortex	Area
LParaHippo	Left Parahippocampal Gyrus	Area
RParaHippo	Right Parahippocampal Gyrus	Area
LEntor	Left Entorhinal Cortex	Area
REntor	Right Entorhinal Cortex	Area
LParsOper	Left Pars Opercularis	Area
RParsOper	Right Pars Opercularis	Area
LTempPole	Left Temporal Pole	Area
RTempPole	Right Temporal Pole	Area
LRomidF	Left Rostral Middle Frontal	Area
RRomidF	Right Rostral Middle Frontal	Area

**Table 5:** Surface area and cortical thickness variables retained in the analyses

### Appendix 5: Nutrition and IQ

Interaction models showed that the effect of crystallized PolyIQ on IQ was moderated by contextual factors: it was weaker in individuals with greater social jetlag, and stronger in those with higher SES. Interestingly, while the association between fluid PolyIQ and fluid IQ was robust, it was significantly stronger in lower-income individuals, suggesting that nutritional influences may be particularly important in settings of socioeconomic disadvantage. Last, we found that nutrition also partially mediated the effects of socioeconomic factors on cognitive outcomes, accounting for 6-8.5% of SES and INR effects on IQ.

	Cryst. IQ	Fluid IQ	Cryst. IQ	Cryst. IQ	Fluid IQ
Intercept	0.028 (0.023)	-0.003 (0.019)	0.024 (0.023)	0.028 (0.023)	0.002 (0.020)
PolyIQ_cryst	0.554*** (0.086)		0.539*** (0.087)	0.551*** (0.087)	
SJL	-0.115*** (0.020)	-0.065*** (0.017)	-0.118*** (0.020)	-0.115*** (0.020)	-0.067*** (0.017)
SleepD	-0.008 (0.029)	0.031 (0.024)	-0.007 (0.029)	-0.009 (0.029)	0.032 (0.024)
SleepE	0.022 (0.029)	-0.001 (0.025)	0.022 (0.029)	0.024 (0.029)	-0.001 (0.025)
Age	-0.205*** (0.047)	-0.043 (0.040)	-0.204*** (0.047)	-0.205*** (0.047)	-0.043 (0.040)
Pubertal Stage	0.073*** (0.019)	-0.011 (0.016)	0.074*** (0.019)	0.074*** (0.019)	-0.010 (0.016)
Female	-0.044 (0.038)	0.027 (0.032)	-0.044 (0.038)	-0.043 (0.038)	0.027 (0.032)
SES	0.170*** (0.019)	0.124*** (0.016)	0.170*** (0.019)	0.172*** (0.019)	0.122*** (0.016)
INR	0.155*** (0.017)	0.080*** (0.015)	0.153*** (0.017)	0.159*** (0.018)	0.084*** (0.015)
PolyIQ_fluid		0.590*** (0.131)			0.606*** (0.132)
PolyIQ_cryst x SJL			-0.198* (0.101)		
PolyIQ_cryst x SES				0.216* (0.103)	
PolyIQ_cryst x INR				-0.276** (0.099)	
PolyIQ_fluid x SES					0.173 (0.154)
PolyIQ_fluid x INR					-0.446** (0.149)
R <sup>2</sup>	0.165	0.091	0.167	0.168	0.094
Adj. R <sup>2</sup>	0.163	0.088	0.164	0.165	0.090
Num. obs.	2905	2905	2905	2905	2905

\*\*\* $p < 0.001$ ; \*\* $p < 0.01$ ; \* $p < 0.05$

**Table 6:** Linear regression models predicting IQ from nutrition PolyIQ and covariates.

We tested whether dietary quality mediates the relationship between socioeconomic context and cognitive outcomes. Using nonparametric causal mediation analysis, we found that nutrition-based PolyIQ scores significantly mediated the effect of both SES and INR on crystallized and fluid IQ. Specifically, between 6% and 8.5% of the total effect of SES

and INR on crystallized and fluid IQ scores was explained by indirect pathways through nutrition. Although the majority of the SES and INR effects remained direct—likely reflecting broader environmental influences such as educational access and parental engagement—these results highlight nutrition as a statistically significant and biologically plausible pathway through which socioeconomic disparities shape cognitive outcomes.

Path	ACME	CLACME	ADE	Total	PropMediated
SES → Crystallized IQ	0.0162	[0.0102, 0.0200]	0.1735	0.1897	0.0853
INR → Crystallized IQ	0.0102	[0.0055, 0.0200]	0.1623	0.1725	0.0591
SES → Fluid IQ	0.0097	[0.0053, 0.0200]	0.1233	0.1329	0.0727
INR → Fluid IQ	0.0063	[0.0030, 0.0100]	0.0911	0.0973	0.0644

**Table 7:** Causal mediation results: SES and INR effects on IQ via nutrition

### Appendix 6: Associations between brain correlates and IQ composites

*Volumes.* Of the 30 regions we retained, we found that 25 regional brain volumes were associated with either crystallized or fluid IQ after FDR corrections and we found 5 mediations.

Volume	r_cryst	p_cryst	r_fluid	p_fluid	p_cryst_fdr	p_fluid_fdr
LParaHippo	0.162	0.00000	0.117	0.00000	0.00000	0.00000
RParaHippo	0.157	0.00000	0.077	0.00086	0.00000	0.00123
LParsOper	0.078	0.00080	0.063	0.00622	0.00100	0.00811
RParsOper	0.101	0.00001	0.077	0.00085	0.00002	0.00123
LParsOrbi	0.147	0.00000	0.086	0.00020	0.00000	0.00036
RParsOrbi	0.121	0.00000	0.076	0.00101	0.00000	0.00138
RParsTriang	0.081	0.00049	0.056	0.01515	0.00067	0.01894
LFusi	0.182	0.00000	0.093	0.00006	0.00000	0.00012
RFusi	0.158	0.00000	0.106	0.00000	0.00000	0.00002
LMidTemp	0.149	0.00000	0.099	0.00002	0.00000	0.00006
RMidTemp	0.184	0.00000	0.093	0.00005	0.00000	0.00012
LRostMid	0.131	0.00000	0.099	0.00002	0.00000	0.00006
RRostMid	0.115	0.00000	0.079	0.00068	0.00000	0.00107
LCaudMid	0.104	0.00001	0.086	0.00020	0.00001	0.00036
RCaudMid	0.074	0.00142	0.086	0.00020	0.00170	0.00036
LFrontPole	0.078	0.00080	0.043	0.06318	0.00100	0.07290
RFrontPole	0.097	0.00003	0.080	0.00059	0.00004	0.00099
LInsula	0.125	0.00000	0.118	0.00000	0.00000	0.00000
RInsula	0.123	0.00000	0.130	0.00000	0.00000	0.00000
LPrecun	0.156	0.00000	0.122	0.00000	0.00000	0.00000
RPrecun	0.165	0.00000	0.123	0.00000	0.00000	0.00000
LPostGyrus	0.112	0.00000	0.096	0.00003	0.00000	0.00008
RPostGyrus	0.111	0.00000	0.096	0.00003	0.00000	0.00008
LParaCentLobule	0.106	0.00000	0.106	0.00000	0.00001	0.00002
RParaCentLobule	0.120	0.00000	0.103	0.00001	0.00000	0.00003
LParsTriang	0.046	0.04642	0.049	0.03469	0.05356	0.04163

**Table 8:** Regions with significant Volume–IQ associations after FDR correction.

PolyIQ	Volume	ACME	Total_Effect	Prop_Mediated	p_ACME
Crystallized	LParaHippo	0.0304	0.5453	0.056	0.014
Crystallized	LParsOper	0.0193	0.5453	0.035	0.022
Crystallized	LMidTemp	0.0305	0.5453	0.056	0.022
Crystallized	RMidTemp	0.0442	0.5453	0.081	0.006
Fluid	LParaHippo	0.0241	0.6650	0.036	0.042

**Table 9:** Mediation of the effect of nutrition-based PolyIQ scores on IQ via gray matter volumes.

*Cortical Thickness.* Of the 12 regions we retained, only the left and right ParaHippocampal gyri were associated with both crystallized and fluid IQ (see Table 11).

thick2	r_cryst	p_cryst	r_fluid	p_fluid	p_cryst_fdr	p_fluid_fdr
LParaHippo	0.122	0.00000	0.064	0.00374	0.00000	0.02943
RParaHippo	0.077	0.00057	0.063	0.00490	0.00343	0.02943

**Table 10:** Regions with significant Cortical Thickness–IQ associations after FDR correction.

PolyIQ	Thick	ACME	Total_Effect	Prop_Mediated	p_ACME
Fluid	LParaHippo	0.0241	0.6650	0.036	0.050

**Table 11:** Mediation of the effect of nutrition-based PolyIQ scores on IQ via gray matter cortical thickness

*FA.* Six of the seven tracts showed significant associations with IQ and there were 2 mediations

Tract	r_cryst	p_cryst	r_fluid	p_fluid	p_cryst_fdr	p_fluid_fdr
fxrh	0.056	0.02985	0.072	0.00513	0.04179	0.00898
fxlh	0.093	0.00028	0.103	0.00006	0.00065	0.00039
cghrh	0.096	0.00017	0.083	0.00113	0.00059	0.00397
cghlh	0.117	0.00000	0.079	0.00195	0.00003	0.00456
ilflh	0.077	0.00269	0.055	0.03150	0.00471	0.03675
fmaj	0.038	0.13549	0.068	0.00799	0.15315	0.01119

**Table 12:** White matter tracts with significant FA–IQ associations after FDR correction.

PolyIQ	Tract	ACME	Total_Effect	Prop_Mediated	p_ACME
Crystallized	fxlh	0.0267	0.6357	0.042	0.008
Crystallized	fxrh	0.0224	0.6357	0.035	0.030
Fluid	fxlh	0.0387	0.6675	0.058	0.008
Fluid	fxrh	0.0392	0.6675	0.059	0.010

**Table 13:** Mediation of the effect of nutrition-based PolyIQ scores on IQ via white matter FA in the fornix.

## Appendix 7: Diets and neural correlates

Our analyses revealed specific associations between dietary patterns and neural structure, even after adjusting for age, sex, SES, and INR. Most notably, the sugar-avoidance pattern (PC2) demonstrated beneficial associations with gray matter volumes in language-processing regions, including the left pars opercularis and bilateral middle temporal gyri ( $p < 0.05$ ), indicating that limiting sugar-sweetened beverage consumption may preserve cortical structures essential for semantic processing. The processed food pattern (PC3) showed detrimental associations with bilateral fornix white matter integrity ( $p < 0.05$ ) and left parahippocampal volume ( $p < 0.05$ ), consistent with evidence that ultra-processed foods may impair brain structure development. Notably, socioeconomic factors (SES and INR) showed robust associations across multiple brain regions, reinforcing the importance of controlling for environmental context when examining nutrition-brain relationships.

	fxlh	fxrh	LParaHippo. (Vol)	LParsOper	LMidTemp	RMidTemp	LParaHippo. (Thick)
(Intercept)	0.397*** (0.010)	0.397*** (0.010)	0.235 (0.355)	1.535*** (0.355)	1.916*** (0.336)	1.893*** (0.330)	0.578 (0.348)
food_PC1	0.001 (0.000)	0.001 (0.000)	0.006 (0.013)	0.016 (0.013)	0.020 (0.013)	0.020 (0.012)	-0.006 (0.013)
food_PC2	0.000 (0.000)	0.000 (0.000)	0.014 (0.017)	0.030 (0.017)	0.031* (0.016)	0.036* (0.015)	0.001 (0.016)
food_PC3	-0.001* (0.001)	-0.001* (0.001)	-0.061* (0.024)	-0.011 (0.024)	-0.016 (0.023)	-0.003 (0.022)	-0.032 (0.023)
food_PC4	0.001 (0.001)	0.001 (0.001)	0.039 (0.024)	0.047* (0.024)	0.030 (0.023)	0.052* (0.022)	0.021 (0.024)
age	0.000 (0.001)	0.000 (0.001)	-0.015 (0.026)	-0.101*** (0.026)	-0.123*** (0.024)	-0.122*** (0.024)	-0.056* (0.025)
Sex2	-0.001 (0.001)	-0.001 (0.001)	-0.295*** (0.047)	-0.371*** (0.047)	-0.689*** (0.044)	-0.746*** (0.043)	0.157*** (0.046)
SES	0.001 (0.001)	0.001 (0.001)	0.123*** (0.031)	0.042 (0.031)	0.102*** (0.029)	0.102*** (0.029)	0.091** (0.031)
INR	-0.000 (0.000)	-0.000 (0.000)	0.027** (0.010)	0.005 (0.010)	0.020* (0.010)	0.030** (0.010)	0.026** (0.010)
R <sup>2</sup>	0.010	0.010	0.054	0.050	0.152	0.181	0.027
Adj. R <sup>2</sup>	0.005	0.005	0.050	0.046	0.148	0.177	0.024
Num. obs.	1519	1519	1862	1862	1862	1862	2018

\*\*\* $p < 0.001$ ; \*\* $p < 0.01$ ; \* $p < 0.05$

**Table 14:** Relationship between Nutrition and Neural mediators

## Appendix 8: Adiposity, IQ and neural correlates

We examined the correlation between markers of adiposity and cognitive scores. Both BMI and waist circumference were significantly and negatively associated with IQ. Crystallized IQ was modestly inversely correlated with BMI ( $r = -0.17$ ,  $p < 0.0001$ ), suggesting that higher body mass may be linked to lower accumulated knowledge. Fluid IQ showed a slightly weaker but still significant negative correlation with BMI ( $r = -0.14$ ,  $p < 0.0001$ ), and similarly with waist circumference ( $r = -0.13$ ,  $p < 0.0001$ ), indicating that higher adiposity is associated with reduced cognitive flexibility and reasoning capacity. All correlations were statistically significant and negative, though effect sizes were modest. These findings suggest that greater adiposity is linked to lower cognitive performance,

particularly in tasks requiring reasoning and accumulated knowledge, even in a pediatric population.

Regression analysis confirmed these results. Furthermore, when both BMI and waist circumference were included in the model, waist circumference—but not BMI—remained a significant predictor of lower fluid IQ, suggesting that central adiposity may be more cognitively relevant than overall body mass. Further, the negative association between BMI and fluid IQ was significantly stronger in higher-SES children, pointing to potential differential vulnerability or expectations across socioeconomic strata.

	Cryst IQ	Fluid IQ	Fluid IQ	Fluid IQ
(Intercept)	1.377*** (0.349)	0.586* (0.285)	1.089** (0.364)	0.631* (0.286)
BMI	-0.288*** (0.080)	-0.155* (0.065)	0.035 (0.107)	-0.170** (0.066)
age	-0.054** (0.019)	-0.017 (0.016)	-0.018 (0.016)	-0.017 (0.016)
Sex2	0.003 (0.033)	-0.037 (0.027)	-0.052 (0.028)	-0.038 (0.027)
SES	0.215*** (0.023)	0.173*** (0.019)	0.171*** (0.019)	0.627** (0.214)
INR	0.065*** (0.008)	0.034*** (0.006)	0.034*** (0.006)	0.033*** (0.006)
anthro_waist_cm			-0.309* (0.141)	
BMI:SES				-0.142* (0.067)
R <sup>2</sup>	0.145	0.101	0.103	0.102
Adj. R <sup>2</sup>	0.143	0.099	0.101	0.100
Num. obs.	2454	2454	2437	2454

\*\*\* $p < 0.001$ ; \*\* $p < 0.01$ ; \* $p < 0.05$

**Table 15:** Relationship between IQ, BMI and waist circumference

Given waist circumference—but not BMI—is a significant negative predictor of fluid IQ after adjusting for SES, INR, and age, we examined the potential link between adiposity and the brain regions that mediated the relationship between PolyIQ scores and cognitive outcomes (Table 16).

	LParaHippo (thick)	LParaHippo (thick)	LMid.Temp. (Vol)	RMid.Temp. (Vol)	LMid.Temp. (Vol)	RMid.Temp. (Vol)
BMI	1.723** (0.574)	1.172* (0.579)	2.964*** (0.568)	3.357*** (0.556)	2.530*** (0.446)	2.469*** (0.439)
Waist	-0.333* (0.139)	-0.163 (0.142)	-0.303* (0.139)	-0.435** (0.135)		
Age	-0.046 (0.025)	-0.057* (0.025)	-0.121*** (0.024)	-0.109*** (0.024)	-0.117*** (0.024)	-0.116*** (0.024)
Female	0.154*** (0.045)	0.157*** (0.044)	-0.713*** (0.043)	-0.766*** (0.043)	-0.695*** (0.043)	-0.758*** (0.042)
SES		0.025* (0.010)	0.019 (0.010)		0.020* (0.010)	0.031** (0.010)
INR		0.085** (0.031)	0.106*** (0.029)		0.105*** (0.029)	0.104*** (0.029)
Intercept					-0.221* (0.104)	-0.208* (0.103)
R <sup>2</sup>	0.011	0.026	0.151	0.156	0.149	0.177
Adj. R <sup>2</sup>	0.010	0.023	0.149	0.154	0.147	0.175
Num. obs.	1997	1997	1843	1843	1852	1852

\*\*\* $p < 0.001$ ; \*\* $p < 0.01$ ; \* $p < 0.05$

**Table 16:** Relationship between adiposity and neural mediators