

Screen media activity and brain structure in youth: Evidence for diverse structural correlation networks from the ABCD study



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ABSTRACT

The adolescent brain undergoes profound structural changes which is influenced by many factors. Screen media activity (SMA; e.g., watching television or videos, playing video games, or using social media) is a common recreational activity in children and adolescents; however, its effect on brain structure is not well understood. A multivariate approach with the first cross-sectional data release from the Adolescent Brain Cognitive Development (ABCD) study was used to test the maturational coupling hypothesis, i.e. the notion that coordinated patterns of structural change related to specific behaviors. Moreover, the utility of this approach was tested by determining the association between these structural correlation networks and psychopathology or cognition. ABCD participants with usable structural imaging and SMA data ($N = 4277$ of 4524) were subjected to a Group Factor Analysis (GFA) to identify latent variables that relate SMA to cortical thickness, sulcal depth, and gray matter volume. Subject scores from these latent variables were used in generalized linear mixed-effect models to investigate associations between SMA and internalizing and externalizing psychopathology, as well as fluid and crystallized intelligence. Four SMA-related GFAs explained 37% of the variance between SMA and structural brain indices. SMA-related GFAs correlated with brain areas that support homologous functions. Some but not all SMA-related factors corresponded with higher externalizing (Cohen's d effect size (ES) 0.06–0.1) but not internalizing psychopathology and lower crystallized (ES: 0.08–0.1) and fluid intelligence (ES: 0.04–0.09). Taken together, these findings support the notion of SMA related maturational coupling or structural correlation networks in the brain and provides evidence that individual differences of these networks have mixed consequences for psychopathology and cognitive performance.

1. Introduction

Brain structure undergoes remarkable changes in the second decade of life (Pfefferbaum et al., 2016), characterized by a reduction of gray matter and an increase in white matter (Giedd et al., 2015), with enduring impacts on cognition (Walhovd et al., 2016). Specifically, coordinated cortical thinning (Ducharme et al., 2016) is governed by evolutionary novelty and functional specialization (Sotiras et al., 2017), showing regional and temporal specificity with development (Houston et al., 2014). Evidence from several recent studies is consistent with the hypothesis that changes of brain structure are correlated across areas with similar function that recapitulate functional networks (Geng et al., 2017), which has been termed maturational coupling or structural correlation networks (SCNs), and has been proposed as a putative

region-specific biomarker for developmental psychopathology (Saggar et al., 2015). Thus, brain regions that change together, i.e. increase or decrease in volume at the same rate over the course of years in the same individual, show structural covariance (Vandekar et al., 2015) or anatomical connectivity across individuals, reflecting synchronized developmental change in distributed cortical regions (Alexander-Bloch et al., 2013). For example, developmental changes in maturational coupling within the default-mode network (DMN) align with developmental changes in structural and functional DMN connectivity (Khundrakpam et al., 2017). These structural changes can also be affected by environmental characteristics, such as childhood abuse (Gold et al., 2016) or urban upbringing (Besteher et al., 2017), and have direct implications for brain functions such as general cognitive ability (Vuoksimaa et al., 2016), behavioral inhibition (Sylvester et al., 2016), and

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subjective ratings of empathy (Bernhardt et al., 2014). Finally, these maturational differences seem to be triggered by regional variation of gene expression having a direct impact on cortical thickness (Fjell et al., 2015). Together, structural brain changes are a consequence of a coordinated process that reflects an interaction between environment and genes that impact specific neural functions.

Screen media activity (SMA) (e.g., watching television or videos, playing video games, or using social media) is among the most common recreational activity in children and adolescents (Kenney and Gortmaker, 2017; Loprinzi and Davis, 2016). As many as 99% of adolescents use the internet, approximately 85% engage in electronic gaming (Rikkers et al., 2016), and nearly 97% of US youth have at least one electronic item in their bedroom (Hale and Guan, 2015). Relatively few studies have examined the relationship between SMA and brain structure or function. In one study with 18 year-old college students, individuals with internet gaming addiction showed less gray matter volume in bilateral anterior cingulate cortex, precuneus, supplementary motor area, superior parietal cortex, left dorsal lateral prefrontal cortex, left insula, and bilateral cerebellum (Wang et al., 2015) than matched controls. Among young adult female habitual internet users, more gray matter volume of bilateral putamen and right nucleus accumbens and lower gray matter volume of orbitofrontal cortex were associated with more frequent use (Altbacker et al., 2016). Functional neuroimaging studies have provided some evidence that those with internet addiction fail to recruit frontal-basal pathways that are important in inhibiting unwanted actions (Li et al., 2014). However, there are no large studies in youth in general, and prepubescent adolescents in particular, focused on SMA and structural or functional brain characteristics.

There is some controversy about whether excessive SMA is associated with problematic outcomes among youth and adolescents. Whereas some have reported that frequent SMA is associated with internalizing psychopathology including depression (Goldfield et al., 2016) and anxiety (Holfeld and Sukhawathanakul, 2017), externalizing psychopathology (Cerniglia et al., 2016), greater risk behaviors (Fischer et al., 2011), and even suicide (Twenge et al., 2017), others have not found evidence for an association between SMA and problematic outcomes (Ferguson, 2015, (Ferguson, 2017)). Even fewer studies have examined the relationship between different types of SMA and brain structure in healthy youth. In a recent cross-sectional study of healthy children ages 8–12, time spent reading was positively correlated with higher functional connectivity between the Brodmann Area 37 and left-sided language, visual, and cognitive control regions, but screen time was related to lower connectivity between the left visual word form area and regions related to language and cognitive control (Horowitz-Kraus and Hutton, 2017). However, this study had a small number of participants. The goal of this investigation was to determine whether SMA is related to specific SCN, i.e. whether exposure to SMA correlates to specific brain areas across cortical thickness, volume, and sulcal depth. Moreover, the second goal was to determine whether such SCN can be related to individual differences in psychopathology and cognition. Based on the maturational coupling hypothesis (Alexander-Bloch et al., 2013; Raznahan et al., 2011), i.e. coordinated patterns of structural change related to specific behaviors, we hypothesized that those individuals engaged in significant SMA relative to those with less SMA exposure would show greater maturity in sensorimotor areas, i.e. lower cortical thickness associated with SMA in primary sensory and motor areas. Moreover, based on the emerging findings of disorganized SCNs in psychiatric populations (Wang et al., 2016; Xia et al., 2018), we hypothesized that SMA-related SCNs related to mismatch between sensorimotor and executive and value-based processing areas are associated with psychopathology or cognitive performance.

2. Materials and methods

The Adolescent Brain and Cognitive Development Study (ABCD) is a multi-site, longitudinal neuroimaging study following 9–10 year-old

youth through adolescence. The ABCD study team employed a rigorous epidemiologically informed school-based recruitment strategy, designed with consideration of the demographic composition of the 21 ABCD sites and the US as a whole (Volkow et al., 2017). The total sample size for the ABCD Study is projected to be 11,500; the first data release (February 2018) included 4524 youth who completed the baseline protocol before September 2017, and is the basis for these analyses (<https://ndar.nih.gov/study.html?id=500>, <https://doi.org/10.15154/1412097>, accessed 3/22/2018).

2.1. Screen media activity (youth-report)

Youth were asked to indicate how long they were engaged in the following SMA activities during the weekday and weekend: (1) Watch TV shows or movies? (2) Watch videos (such as YouTube)? (3) Play video games on a computer, console, phone or other device (Xbox, PlayStation, iPad)? (4) Text on a cell phone, tablet, or computer (e.g. GChat, WhatsApp, etc.)? (5) Visit social networking sites like Facebook, Twitter, Instagram, etc.? (6) Video chat (Skype, FaceTime, etc.)? Seven potential answer choices included: none, < 30 min, 30 min, 1 h, 2 h, 3 h, and 4 + hours. In addition, two questions that were included in the analyses focused on specific types of videos and games: (1) How often do you play mature-rated video games (e.g., Call of Duty, Grand Theft Auto, Assassin's Creed, etc.)? (2) How often do you watch R-rated movies? Four potential answer choices included: (1) never, (2) once in a while, (3) regularly, and (4) all the time. The youth report was used in the multivariate analyses to characterize and quantify SMA.

2.2. Mental health symptoms (parent report)

Youth's behavior was assessed using the *Child Behavior Checklist* (CBCL) (Achenbach, 2009). The CBCL's syndrome scales t-scores of externalizing, and internalizing psychopathologies were used for the analyses.

2.3. Cognition (youth performance)

The neurocognitive assessment included the NIH Toolbox (Luciana et al., 2018). For this report, measures of fluid intelligence (Li et al., 2004) (i.e., those abilities that rely on solving problems, thinking, acting quickly, and encoding new episodic memories) comprised of the Toolbox Pattern Comparison Processing Speed Test, List Sorting Working Memory Test, Picture Sequence Memory Test, Flanker Task, and Dimensional Change Card Sort Task. Crystallized intelligence (i.e., those abilities that are more dependent on experience, representing accumulated store of verbal knowledge or skills and rely more heavily on education and cultural exposure) was measured with the Toolbox Picture Vocabulary Task and Oral Reading Recognition Task (Akshoomoff et al., 2013).

2.4. Structural image processing

All structural neuroimaging processing was completed using FreeSurfer version 5.3.0 according to standardized processing pipelines (Casey et al., 2018). Subjects that did not pass FreeSurfer Quality Control measure were excluded from further analyses ($n = 247$; 5% of total sample), which left $n = 4277$ participants. Cortical reconstruction and volumetric segmentation was performed by the ABCD Data Acquisition and Integration Core using the FreeSurfer image analysis suite (<http://surfer.nmr.mgh.harvard.edu/>, accessed 3/22/2018). Details of these procedures are described in prior publications (Dale et al., 1999; Fischl et al., 1999). Briefly, this process includes removal of non-brain tissue using a hybrid watershed/surface deformation procedure (Segonne et al., 2004), automated Talairach transformation, segmentation of the subcortical white matter and deep gray matter volumetric structures (including hippocampus, amygdala, caudate, putamen, ventricles) (Fischl et al., 2002), intensity normalization, tessellation of the

gray/white matter boundary, automated topology correction (Fischl et al., 2001), and surface deformation following intensity gradients to optimally place the gray/white and gray/cerebrospinal fluid borders at the location where the greatest shift in intensity defines the transition to the other tissue class (Fischl and Dale, 2000). Images were registered to a spherical atlas, which is based on individual cortical folding patterns to match cortical geometry across subjects and the cerebral cortex was parcellated into 34 regions per hemisphere based on the gyral and sulcal structure (Desikan et al., 2006). Cortical thickness was measured as the shortest distance between the pial and the white matter tessellated surfaces (Dale et al., 1999). For sulcal depth, regions that moved outward during inflation were positive and represent the depths of sulci, and regions that moved inward were negative and represent the height of gyri (Fischl et al., 1999). FreeSurfer morphometric measures are related to histological measurements (Cardinale et al., 2014) and demonstrate good test-retest reliability (Han et al., 2006; Iscan et al., 2015).

2.5. Statistical analysis

All statistical analyses were conducted in R 3.4.0 (2017-04-21) ((2010)) using RStudio (Team, 2016b) and RMarkdown (Team, 2016a). Descriptive statistics were obtained using the tableone package in R (Yoshida and Bohn, 2017). Group Factor Analyses (GFA) were conducted using the R package GFA (Leppäaho et al., 2017). Mixed-effects model analyses were conducted using the R package gamm4 (Wood, 2017). Missing data were imputed during the GFA; however, all mixed model analyses were conducted with complete cases only. All R and R Markdown scripts are available as html documents in the supplemental materials.

2.6. Group Factor Analysis

Group factor analysis (Klami et al., 2015) identified latent variables (LVs) related to SMA, cortical thickness, sulcal depth, and gray matter volume. The goal of GFA is to find factors that separate relationships within groups of variables from those between groups. Thus, given a collection X_1, \dots, X_M of M groups of variables of dimension D_1, \dots, D_M , the task is to find $K < D_1 + \dots + D_M$ factors that describe dependencies between multiple groups X_m , while allowing for within-group factors that account for covariance unique to each group. The GFA solution differs from canonical correlation analysis or standard exploratory factor analysis (EFA) by utilizing a Bayesian inferential framework to place an Automatic Relevance Determination (ARD) prior on the factor solution (Tipping, 2001). The ARD prior assumes a low-rank representation of the factor loadings. The main advantages of GFA are that (i) it is conceptually simple, essentially a Bayesian EFA model which differentiates within-group from between-group associations, and (ii) it enables factor analysis in scenarios with two or more groups of data, giving factor solutions not driven by method variance particular to one variable grouping. The solution comprises as a set of factors that contain a projection vector for each of the variable groups having non-zero weights for that factor. The number of factors presented here were chosen based on SMA loadings with brain structure variables and accounting for at least 1% of the variance across all variable groupings.

2.7. Generalized Linear Mixed Models

Subsequent association analyses were conducted within a Generalized Linear Mixed Models (GLMM) framework. Specifically, GFAs that accounted for at least 1% of the variance across SMA and structural brain imaging estimates were used as independent variables to predict the dependent variables (CBCL-Internalizing, CBCL-Externalizing, Cognition-Fluid Intelligence, Cognition-Crystallized Intelligence). All variables were standardized to zero mean and unit variance to be able to calculate coefficients that correspond to Cohen's d effect sizes. We used the R gamm4 library (Wood, 2017), estimating the parameters of the mixed

Table 1

Quartile table showing the relationship between youth-reported total screen activity and socio-demographic variables.

Total Week Screen Media	1st Quartile	2nd Quartile	3rd Quartile	4th Quartile	p
Activity (hours) [youth report]	0–11	11–19.5	19.5–33.8	33.8–163	
n	1076	1085	1034	1062	
Average Screen Time (hours/week)	6.94 (2.76)	15.25 (2.48)	26.08 (4.05)	54.01 (19.13)	<0.001
Age in Months (mean (sd))	119.38 (7.23)	120.32 (7.26)	120.38 (7.57)	120.04 (7.26)	0.006
Height (in) (mean (sd))	55.26 (2.92)	55.43 (3.12)	55.49 (3.15)	55.56 (3.41)	0.147
Weight (lbs) (mean (sd))	77.71 (19.23)	81.09 (21.32)	82.58 (23.02)	87.65 (25.89)	<0.001
Body Mass Index (mean (sd))	17.75 (3.45)	18.40 (3.75)	18.67 (4.04)	19.72 (4.39)	<0.001
Gender (female) (%)	625 (58.1)	520 (47.9)	453 (43.8)	421 (39.6)	<0.001
Race/Ethnicity (%)					<0.001
White	745 (69.3)	700 (64.5)	587 (56.8)	487 (45.9)	
Black	43 (4.0)	55 (5.1)	99 (9.6)	202 (19.0)	
Hispanic	149 (13.9)	210 (19.4)	222 (21.5)	246 (23.2)	
Asian	36 (3.3)	30 (2.8)	21 (2.0)	10 (0.9)	
Other	102 (9.5)	90 (8.3)	105 (10.2)	116 (10.9)	
Parental Education (%)					<0.001
<= 12 grades	87 (8.1)	117 (10.8)	152 (14.7)	172 (16.2)	
HS Degree	25 (2.3)	38 (3.5)	46 (4.4)	50 (4.7)	
Some College	160 (14.9)	221 (20.4)	300 (29.0)	406 (38.2)	
Bachelor	363 (33.7)	400 (36.9)	290 (28.0)	263 (24.8)	
Higher	441 (41.0)	308 (28.4)	246 (23.8)	168 (15.8)	
Not known	0 (0.0)	1 (0.1)	0 (0.0)	3 (0.3)	
Parents married (yes) (%)	872 (81.0)	829 (76.4)	719 (69.5)	615 (57.9)	<0.001
Parental Income (%)					<0.001
[<50K]	144 (13.4)	171 (15.8)	252 (24.4)	399 (37.6)	
[>=50K & <100K]	243 (22.6)	313 (28.8)	327 (31.6)	310 (29.2)	
[>=100K]	610 (56.7)	517 (47.6)	378 (36.6)	262 (24.7)	
Refused to state	79 (7.3)	84 (7.7)	77 (7.4)	91 (8.6)	
Parental Age (mean (sd))	41.55 (6.09)	41.03 (6.31)	40.16 (7.16)	39.16 (7.19)	<0.001

model using Maximum Likelihood Estimation (MLE). Site and family nested within site were used as random effects and age, sex, race/ethnicity, parental education, marital status, parental income, parental age, and body mass index were used as covariates. Model comparisons were conducted using the anova function in R and the Akaike's Information Criterion (AIC).

3. Results

3.1. Demographics and sample characteristics

Table 1 shows general demographics of the sample by quartiles of youth-reported total SMA. First, there was no difference in average age across the quartiles of screen time. Second, males were more frequently in the higher quartiles. Third, youth in the higher quartile had a higher

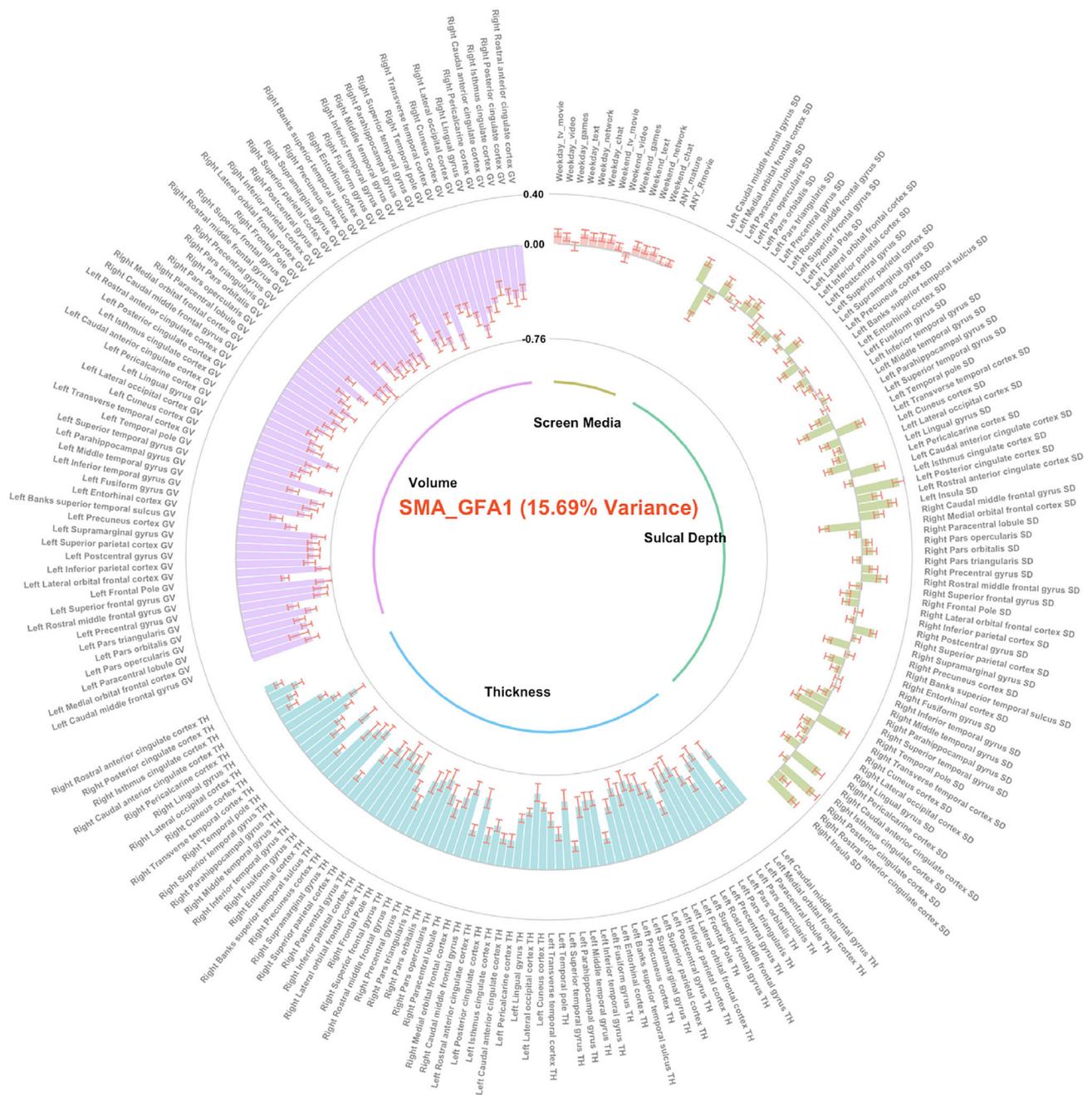


Fig. 1. General factor analysis of screen media activity, cortical thickness, sulcal depth and gray matter volume related loadings for GFA 1 with 95% credible interval. The median factor loadings and the 95% credible interval drawn from the posterior distribution of the GFA W matrix are shown for cortical thickness, sulcal depth, and gray matter volume.

BMI. Fourth, Black and Hispanic youth reported significantly more screen time use than White and Asian youth. Fifth, parents of youth in the higher quartiles were slightly younger, less well educated, were less likely to be married, and had lower household income.

3.2. Group factor analysis (GFA) relating SMA to structural estimates

Cortical thickness (Supp. Fig. 1), sulcal depth (Supp. Fig. 2), and gray matter volume (Supp. Fig. 3) distributions did not show excessive extreme values and were similar to those previously reported (Tamnes et al., 2017; Vandekar et al., 2015). Examining the correlational structure between SMA variables, cortical thickness, sulcal depth, and gray matter volume revealed that variables are relatively strongly correlated within variable groupings, i.e. within a particular

measurement domain, but weakly correlated across groups with sulcal depth being the exception showing relatively weak correlation within the group (Supp. Fig. 4). The GFA extracted 132 factors accounting for 58% of the variance (Supp. Fig. 5a) of which 10 factors (each of which accounted for > 1% of the variance) accounted for a total of 37% of the variance between SMA and structural brain indices (Supp. Fig. 5b) that were strongly orthogonal (Supp. Fig. 6). Only the four SMA-related GFAs that accounted for >1% of the variance across all groups were used in subsequent analyses (i.e., GFAs 1–3, 5, which will now be referred to as the 4th GFA).

The first SMA-related factor, which accounted for about 16% percent of the variance (Fig. 1), loaded positively on SMA and negatively on both cortical thickness (most negative for occipital areas and least for anterior cingulate) and GMV (most negative for orbitofrontal areas and least for

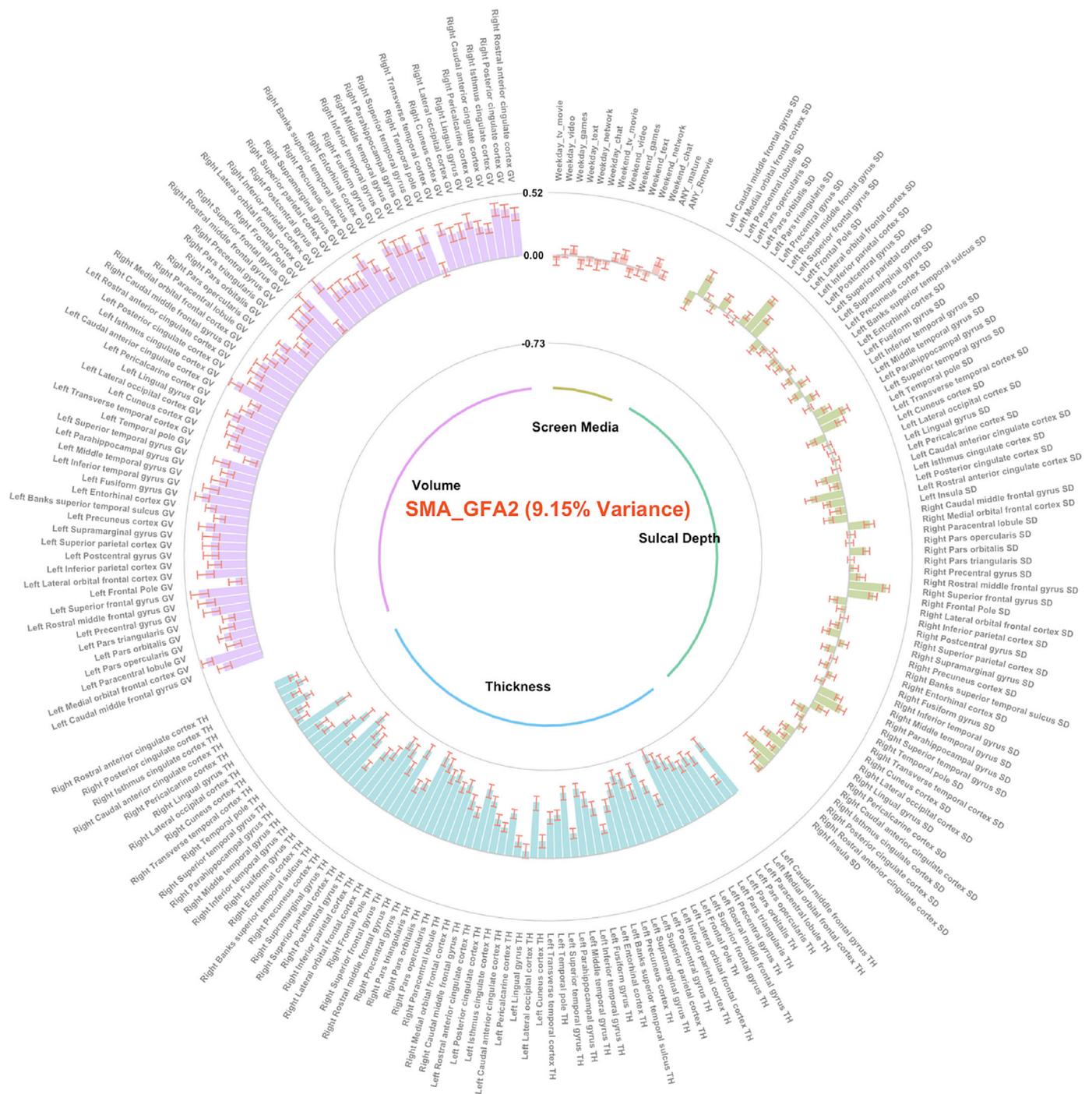


Fig. 2. General factor analysis of screen media activity, cortical thickness, sulcal depth and gray matter volume related loadings for GFA 2 with 95% credible interval. Panels analogous to Fig. 1.

entorhinal and anterior cingulate) with a mixed loading on sulcal depth (most positive on posterior cingulate, most negative for medial orbitofrontal and temporal pole). The second SMA-related factor, which accounted for 9% of the variance loaded most strongly on gaming with a negative loading on thickness (most negative for superior frontal areas and least for visual cortical areas) but a positive loading on GMV (most positive for orbitofrontal areas and least temporal pole) and a mixed loading on sulcal depth (most positive on superior frontal cortex, most negative for posterior cingulate) (Fig. 2). The third SMA-related factor, which accounted for 2% of the variance, loaded strongly on all SMA with mixed and selective loadings on cortical thickness (most negative for

visual areas and most positive for temporal and orbitofrontal areas), sulcal depth (most posterior parietal areas, most negative for primary visual cortex), and volumes (most positive for posterior cingulate and temporal cortex, most negative for primary visual cortices) (Fig. 3). The fourth SMA-related factor, which accounted for 2% of the variance, also loaded positively on SMA (relatively more on social media activity) and selectively on some areas for cortical thickness (most negative for hippocampus and most positive for visual cortices), sulcal depth (most negative for fusiform area and most positive for visual areas) and volumes (most negative for inferior temporal cortex, most positive for primary visual cortices) (Fig. 4).

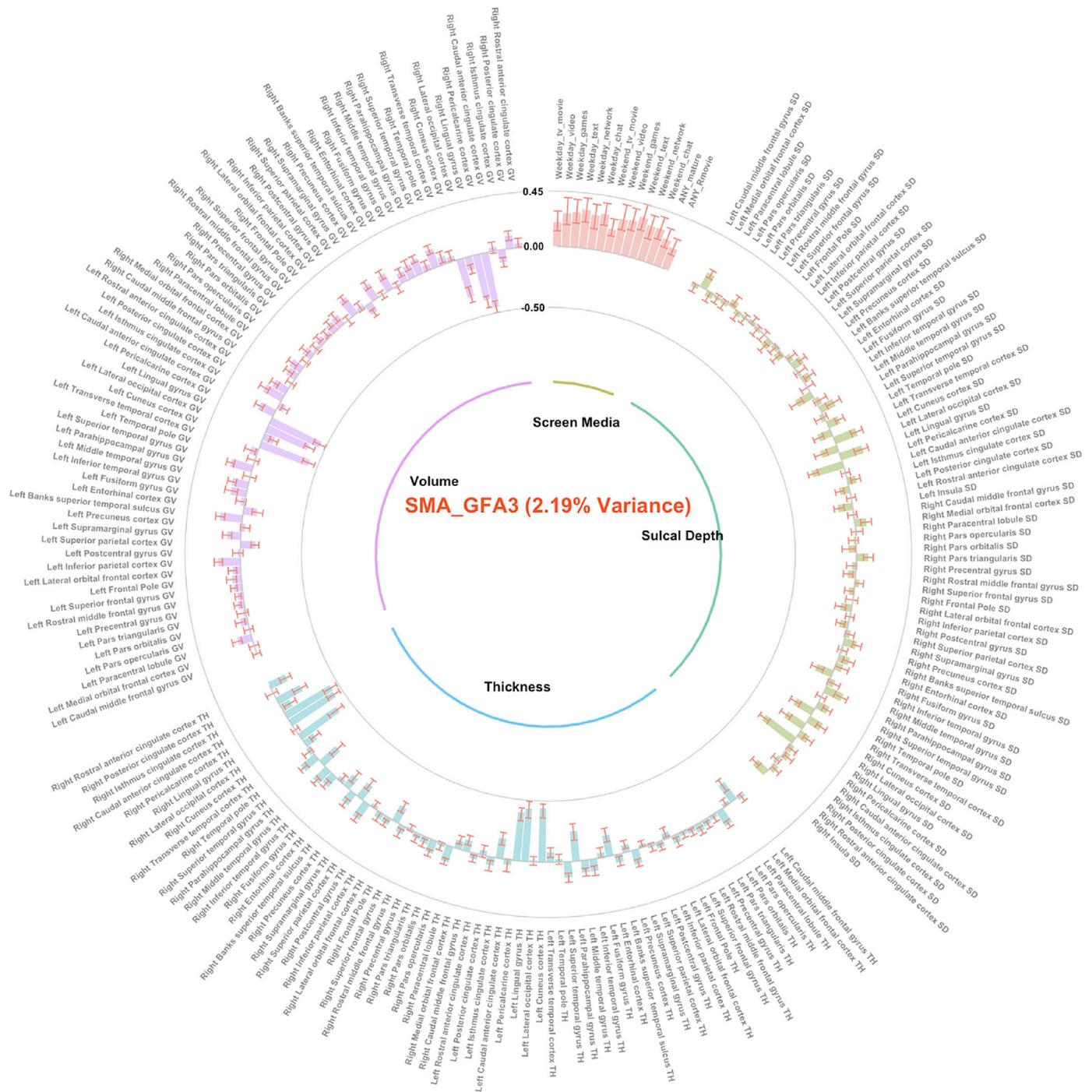


Fig. 3. General factor analysis of screen media activity, cortical thickness, sulcal depth and gray matter volume related loadings for GFA 3 with 95% credible interval. Panels analogous to Fig. 1.

3.3. Generalized mixed models predicting psychopathology and cognition

Mixed model analysis showed that the SMA-related GFA model for predicting overall internalizing behaviors ($df = 25$, AIC: 11726, 2.53% accounted covariance (AC)) did not significantly improve over the base model ($df = 21$, AIC = 11727, 2.35% AC) ($\text{Chi}^2 = 9.2492$, $p = 0.05516$), that comprised age, sex, BMI, race/ethnicity, parental education, parental marital status, parental age, and parental income (Fig. 5). In comparison, the SMA-related GFA model (AIC = 11705, $df = 25$, 4.36% AC) for predicting overall externalizing behaviors significantly improved ($\text{Chi}^2 = 48.081$, $p = 9.076e-10$) the base model ($df = 21$, AIC = 11746,

3.29% AC). Specifically, those individuals with higher SMA-related GFA 1 ($\beta = 0.059$) and GFA 4 ($\beta = 0.095$) scores had significantly higher externalization scores (see Fig. 6). The SMA-related GFA model for predicting fluid intelligence ($df = 25$, AIC = 10333, 10% AC) significantly ($\text{Chi}^2 = 34.181$, $p = 6.842e-07$) improved over the base model ($df = 21$, AIC = 10359, 9.2% AC). Specifically, those individuals with higher SMA-related GFA 2 ($\beta = 0.043$) had higher fluid abilities whereas those with higher SMA-related GFA 4 ($\beta = -0.086$) scored lower on fluid abilities (Fig. 7). Lastly, the SMA-related GFA model for predicting crystallized intelligence ($df = 25$, AIC: 9878.6, 19.1% AC) significantly ($\text{Chi}^2 = 87.655$, $p = 2.2e-16$) improved the base model ($df = 21$, AIC:

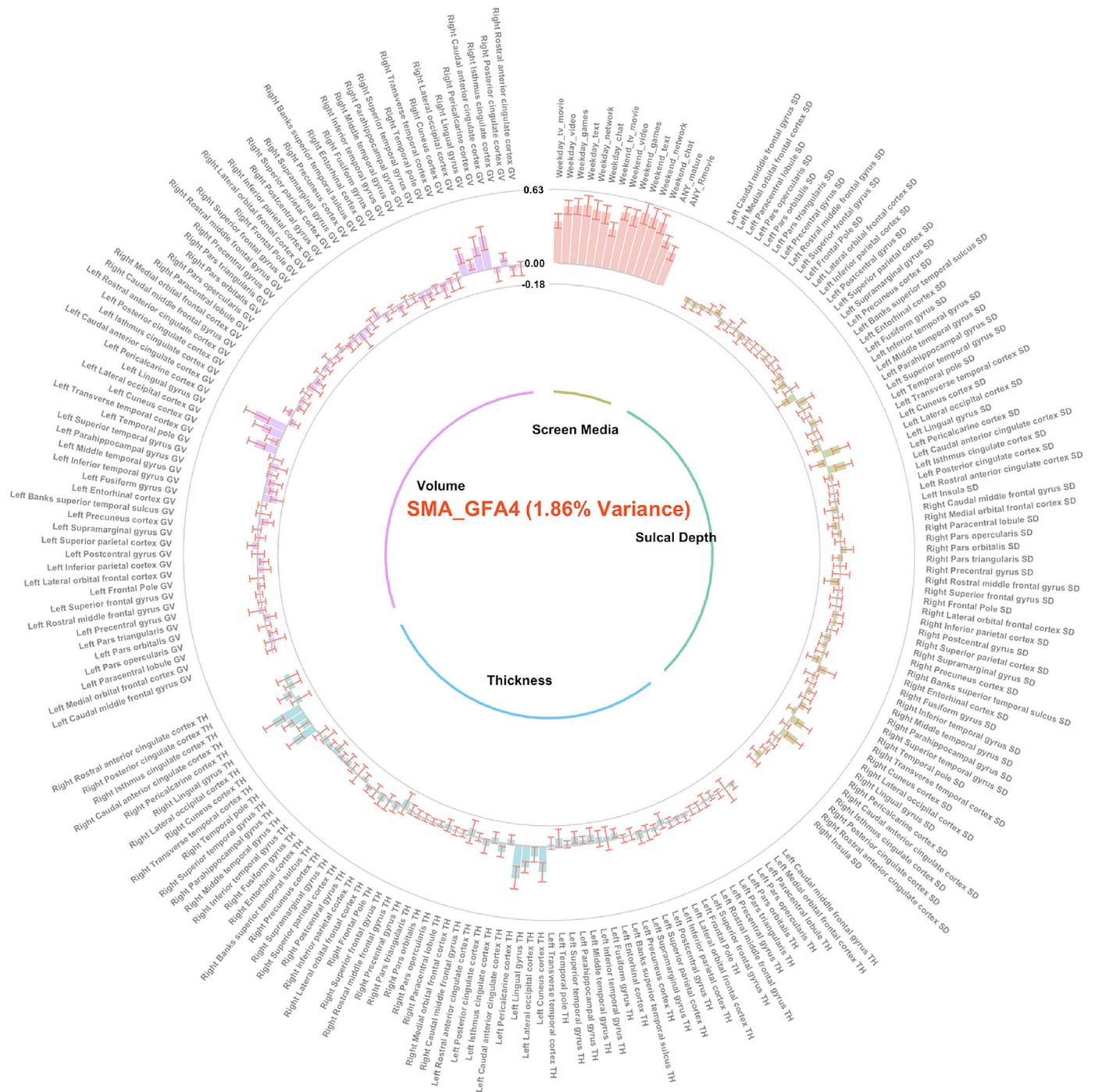


Fig. 4. General factor analysis of screen media activity, cortical thickness, sulcal depth and gray matter volume related loadings for GFA 5 with 95% credible interval. Panels analogous to Fig. 1.

9958.3, 17.2% AC). Specifically, those individuals with higher SMA-related GFA 1 ($\beta = -0.109$) and higher SMA-related GFA 4 ($\beta = -0.080$) had lower crystallized abilities whereas those with higher SMA-related GFA 2 ($\beta = 0.0871$) showed better performance on crystallized abilities (Fig. 8).

4. Discussion

This investigation applied a multivariate exploratory approach to the first data release of the ABCD study (1) to parse the relationship between SMA and structural brain indices (i.e., cortical thickness, sulcal depth, and

gray matter volume) and (2) to evaluate its impact on psychopathology and overall cognitive functioning. First, the Group Factor Analysis extracted four SMA-related factors that integrated across cortical thickness, sulcal depth, and gray matter volume. Second, these factors revealed that different brain regions supporting similar functions were correlated with SMA. Third, some but not all of the factors related to SMA were related to greater externalizing as opposed to internalizing psychopathology and were predominately related to crystallized intelligence and, to a lesser extent, fluid intelligence. Taken together, this brain behavior investigation shows that SMA is significantly related to brain structure with mixed consequences for psychopathology and cognitive performance.

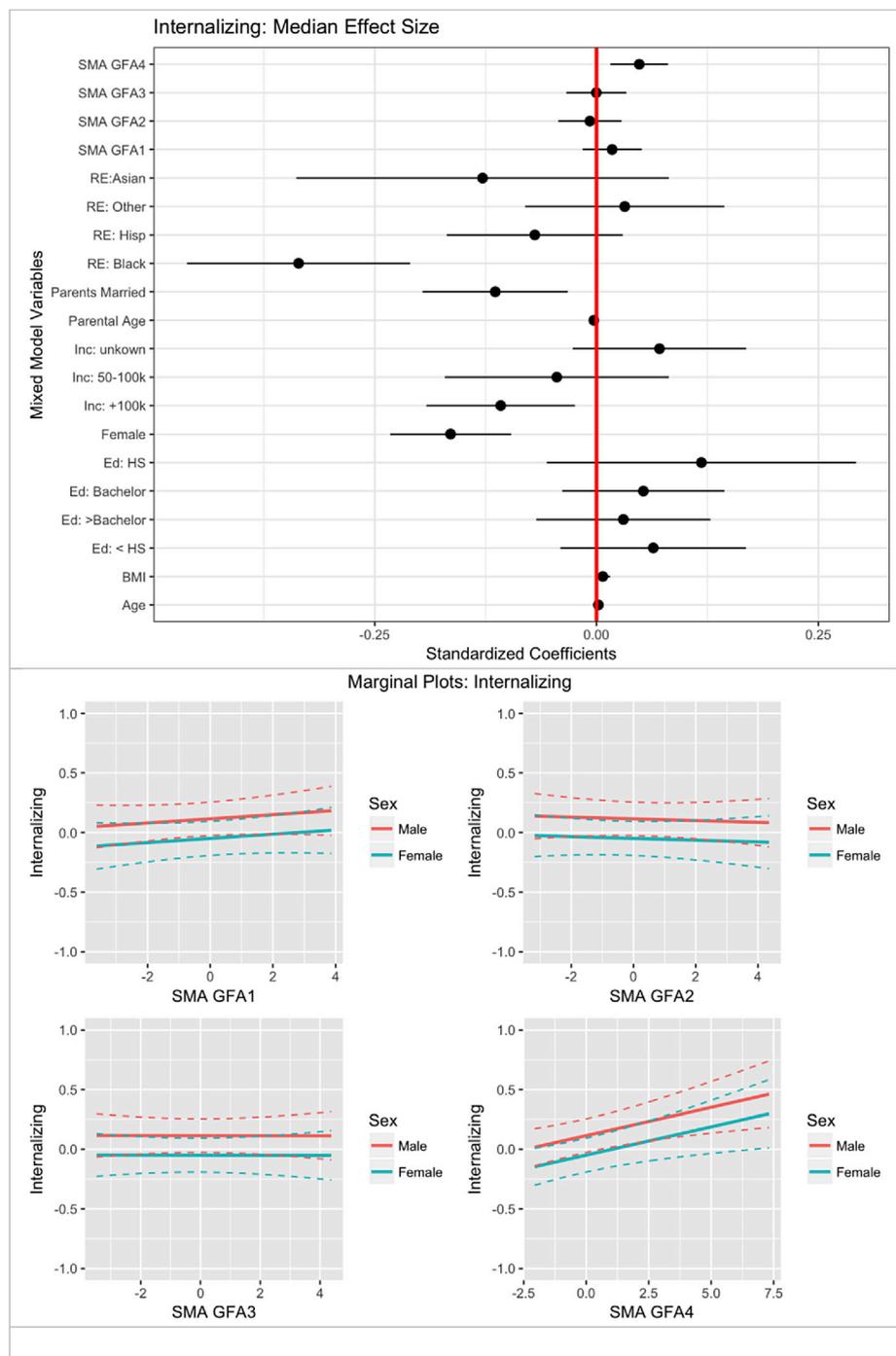


Fig. 5. Mixed model results for internalizing pathology. (a) median coefficients and 95% confidence interval, (b) marginal effect plots separated by sex.

4.1. Screen media activity and brain structure

The SMA-related GFAs linking to brain structure showed the greatest effect size and the most consistent loadings across factors for visual areas including occipital cortex, calcarine areas, and other primary visual cortices. Thus, those individuals with significant exposure to activities that engage the visual system (TV or video watching, gaming, and social network activities) show structural patterns suggestive of greater maturation in the visual system (i.e., thinner cortex, reduced volume, and a more complex pattern of changes in sulcal depth). Several studies have shown that maturational patterns show a strong covariance across brain areas of similar function (Alexander-Bloch et al., 2013; Geng et al., 2017;

Sotiras et al., 2017). Specifically, several investigations have suggested that homologous brain areas (Khundrakpam et al., 2017) undergo coordinated cortical thinning guided by evolutionary novelty and functional specialization (Sotiras et al., 2017). Moreover, these structural brain-related changes have been related to individual differences for specific brain areas, e.g. the anterior cingulate to behavioral inhibition (Sylvester et al., 2016), insula to empathy (Bernhardt et al., 2014), ventromedial prefrontal cortex to anxiety (Newman et al., 2016), prefrontal cortex to levels of depression (Vijayakumar et al., 2017), and general cortical thinning to personality characteristics (Ferschmann et al., 2018). Other investigators reported altered resting state connectivity patterns between sensorimotor and cognitive control regions as

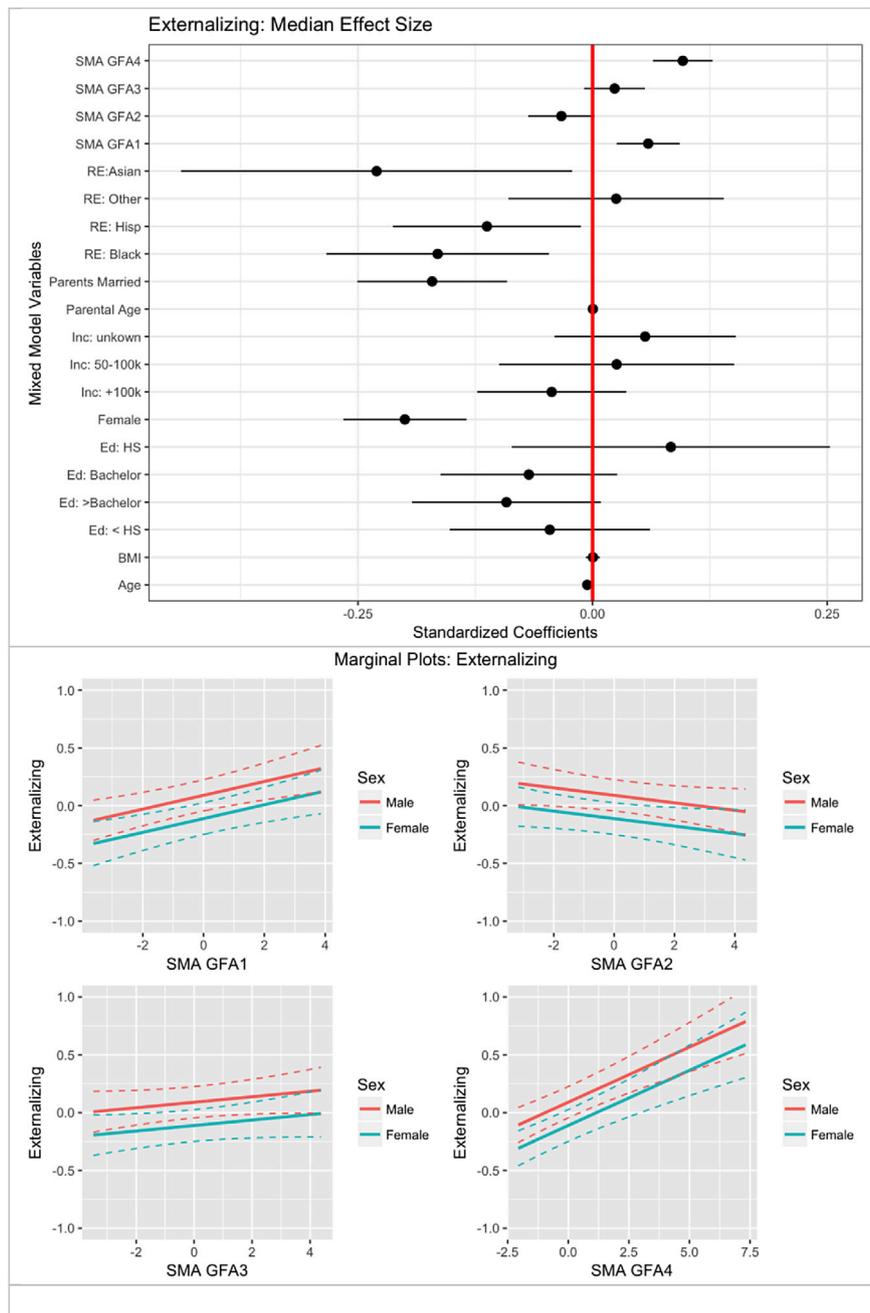


Fig. 6. Mixed model results for externalizing pathology, panels analogous to Fig. 5.

function of screen-based media use and suggested that excessive screen time may adversely affect cognitive control (Horowitz-Kraus and Hutton, 2017). The current investigation adds an important new element to these prior studies. Specifically, SMA is related across subjects to areas that are important for sensory processing but also for higher order cortical functions, i.e. the prefrontal cortex and posterior cingulate. However, it is important to point out that SMA is related to several correlated brain structural patterns that are orthogonal, i.e. the latent variables describing the relationship between brain structure and SMA are not correlated. Thus, screen media activity cannot be reduced to a unidimensional impact on brain structure. Taken together, although there is some evidence that SMA-related latent variables are associated with more psychopathology and poorer performance on cognitive tests, there are other latent variables that show no such relationship. Thus, it is difficult to conclude that SMA related brain structural characteristics have uniformly negative consequences.

4.2. Psychopathology, cognition, and SMA related brain structures

The complex relationship between SMA and brain structure is further supported by examining its psychopathological and cognitive correlates. There was little evidence that SMA related brain structure differences were associated with internalizing pathology, i.e. increase in anxiety, depression, or other avoidance or withdrawal behaviors. Some have suggested that reduced volume and cortical thickness in frontolimbic regions may serve as a neurobiological predictors of emergent internalizing psychopathology in adolescence (Jones et al., 2017). However, others have observed delayed thinning in the ventromedial prefrontal cortex in high anxious children (Newman et al., 2016). Thus, internalizing psychopathology may play a role in the development of cortical structures during adolescence; however, the current ABCD sample provides little evidence that SMA contributes to brain structure characteristics that can be related to internalizing behaviors. In comparison, we

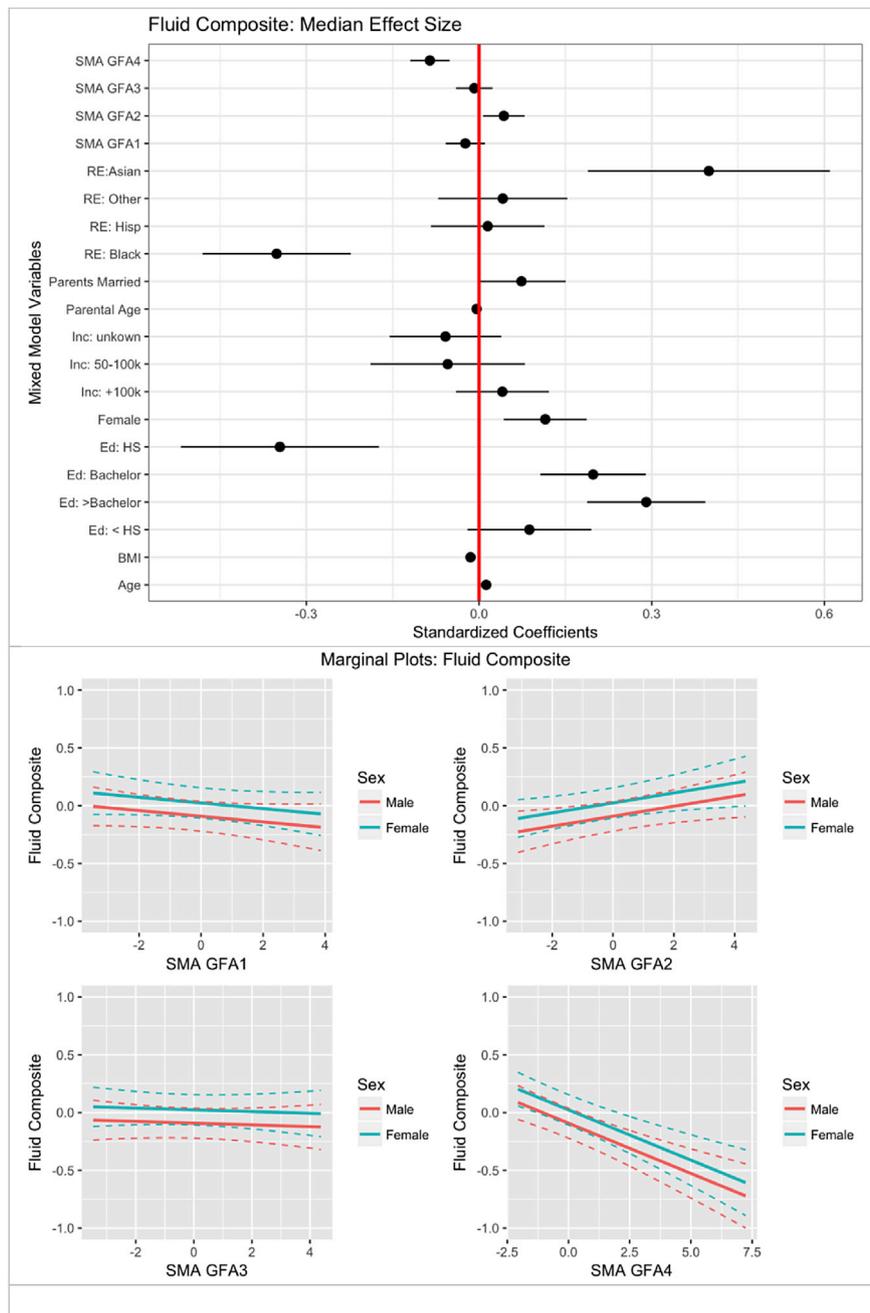


Fig. 7. Mixed model results for fluid intelligence, panels analogous to Fig. 5.

did find a strong relationship to externalizing psychopathology. Specifically, those youth who had higher scores on GFA 1 and 4, i.e. had thinner occipital cortices and smaller volume in orbitofrontal areas as well as thinner hippocampi and smaller inferior-temporal cortical volumes, showed greater levels of externalizing psychopathology. Some investigators have reported, among other areas, increased externalizing in individuals with thinner parahippocampal gyrus (Gold et al., 2016), whereas others showed that youths with conduct disorder had reduced cortical thickness in the superior temporal gyrus and sulcal pathology in orbitofrontal cortex, as well as increased cortical folding in the insula (Fairchild et al., 2015). The current study adds to these findings that some of these effects may also be related to SMA.

The complexity of SMA associated structural brain characteristics extends to the relationship with cognitive performance. In particular, for both fluid and crystallized intelligence, some GFAs were related to better performance whereas others related to poorer performance on these

tests. For fluid intelligence, GFA2, which loaded on gaming activities and greater cortical thinning in prefrontal areas and increased orbitofrontal volume, showed a positive relationship, whereas GFA4 which loaded on social media and reduced hippocampal thickness and lower inferior-temporal cortical gray matter volume, showed a strong negative relationship. Others have shown a relationship between cognitive abilities and structural variability in medial frontal lobes and paracingulate (Fornito et al., 2008). Moreover, a series of investigations have examined the relationship between brain structures and cognitive control in the context of internet gaming disorder. Some of the investigators found widespread reduction of cortical volume (Lin et al., 2015; Wang et al., 2015; Yuan et al., 2013), whereas others have found more circumscribed cortical thickness and gray matter volume reduction most notably within the orbitofrontal cortex (Altbacker et al., 2016; Park et al., 2017; Zhou et al., 2017), but also in frontal pole volume (Kuhn and Gallinat, 2015). The diversity of these findings underscores that SMA does not have a

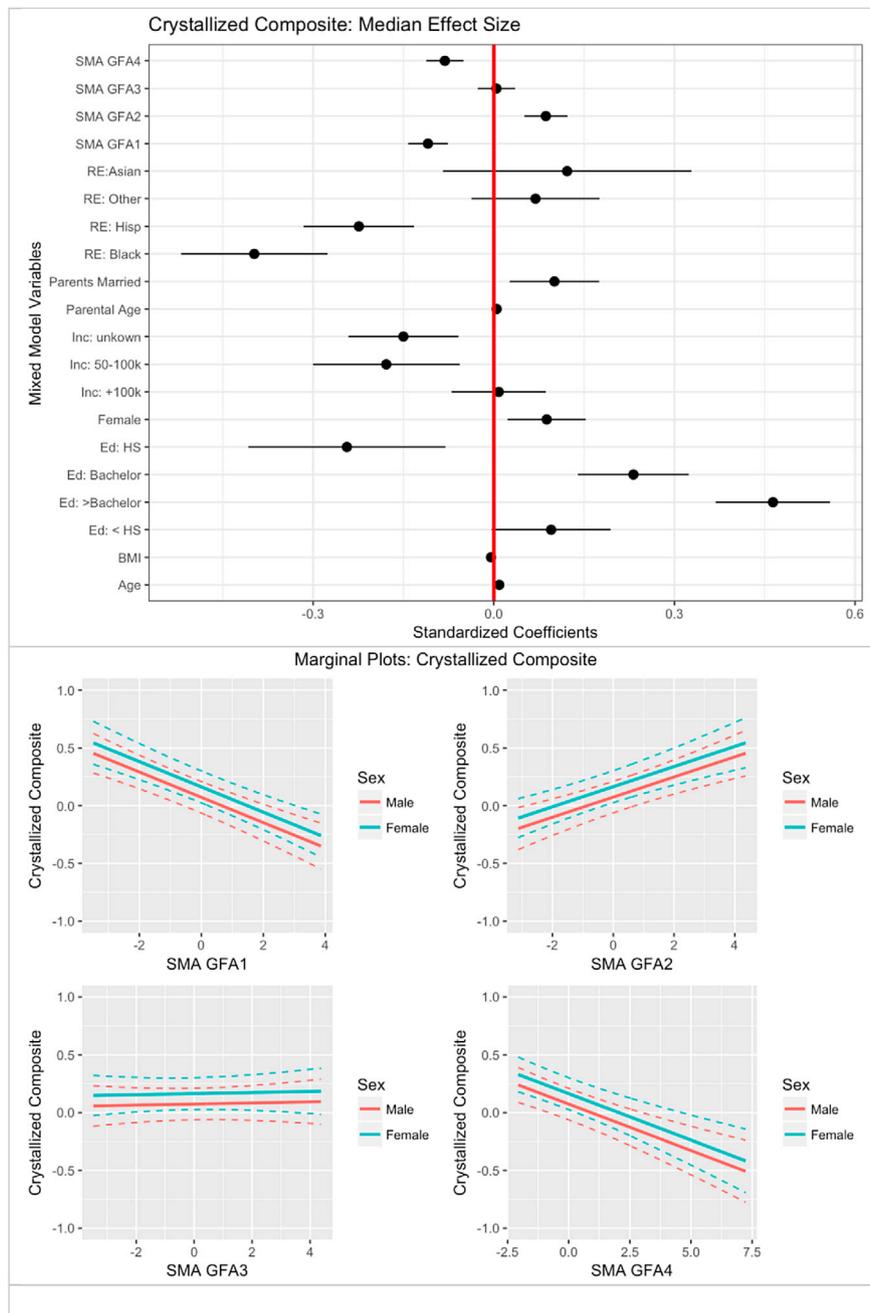


Fig. 8. Mixed model results for crystallized intelligence, panels analogous to Fig. 5.

simple effect on fluid intelligence but rather exerts differential effects via different brain networks. The SMA associated structural brain characteristics were most strongly related to crystallized intelligence, which are more dependent on experience, represent accumulated store of verbal knowledge and skills, and are influenced by education and cultural exposure (Akshoomoff et al., 2013). Here, GFA 1 and 4, which were general and social media related SMA that loaded strongly on occipital and orbitofrontal areas, showed the strongest negative relationship to crystallized intelligence. In contrast, GFA2, which loaded strongly on gaming and was associated with thinner prefrontal cortices but greater orbitofrontal cortical volume had a positive relationship to this ability. Since crystallized cognitive abilities have a strong cultural and educational component and it should not be surprising that these GFAs are also related to parental education, race/ethnicity, and parental income (Suppl. Table 1). Thus, future investigations will need to further delineate the complex relationship between SMA, structural brain

characteristics, and other moderating influences. Nevertheless, these results support the general conclusion that SMA associated brain characteristics are not uniformly related to better or poorer cognitive performance. Instead, specific components that affect particular brain areas contribute differentially to cognition.

4.3. Limitations

First, this is a cross-sectional assessment, which enables establishment of associations but does not allow drawing causal inferences. Although multivariate methods such as GFA enable differential examination of the impact of SMA and structural brain characteristics on outcome variables, they cannot address the “chicken and egg” question. Therefore, the longitudinal component of ABCD is essential to begin to delineate causal longitudinal pathways. Second, the current analysis pathway used the standard FreeSurfer regions (Desikan et al., 2006);

however, others have utilized a higher resolution approach (Vandekar et al., 2015) or have even suggested a connection-based approach, which may be more anatomically meaningful than the traditional lobar structure (Krugger, 2018). Third, although we have focused on the relationship between SMA and brain structure, others have pointed out that developmental trajectories of structural brain changes are influenced in complex ways by multiple factors (Foulkes and Blakemore, 2018) and that better control for these variables may yield more precise results (LeWinn et al., 2017). Fourth, subcortical structures, which also undergo significant development, were not available from the official ABCD data release, thus it was not possible to relate SMA to striatal characteristics, which could be highly informative. Therefore, future investigations may need to employ complex machine learning approaches aimed at identifying individual-level patterns that meaningfully relate variables across levels of analyses, i.e. examine cognitively relevant brain structures to specific SMA in socio-demographically select individuals exposed to particular environmental characteristics. Fifth, the SMA-related factors provide a latent variable approach to examine media related structural brain correlation networks with respect to other youth activities. Currently, we did not take into account the engagement of youth in other recreational activities. Moreover, school-related screen media activity was no considered in the screen media assessment, which may ultimately under-estimate the time spent on SMA. However, it will be important to determine whether these factors can be used to predict other recreational activities.

4.4. Conclusions

This investigation of the ABCD cohort aimed at relating an important youth behavior, i.e. screen media activity, to structural characteristics of the brain and revealed that there are significant associations but that they are complex. Whereas some SMA associated brain structures have relevance for externalizing psychopathology, fluid and crystallized intelligence, others do not. Moreover, whereas some SMA associated brain structures are related to poorer cognitive performance, others are related to better cognitive performance. This diversity of findings provides an important public health message, i.e. screen media activity is not simply “bad for the brain” or “bad for brain related functioning”. Instead, future investigations will need to examine how various forms of screen media activity influence specific psychopathology and cognitive functions, and how this influences changes throughout development.

Conflicts of interest

The authors have no conflicts of interest to declare.

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may not reflect the opinions or views of the NIH or ABCD consortium investigators. The ABCD data repository grows and changes over time. The ABCD data used in this report came from <https://dx.doi.org/10.15154/1412097>.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.neuroimage.2018.10.040>.

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