

Excess social media use in normal populations is associated with amygdala-striatal but not with prefrontal morphology



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ABSTRACT

This study aims to investigate the gray matter volume (GMV) of key neural systems possibly associated with Excess Social Media Use (ESMU) in the general user population. It employs a sex-balanced case (relatively high ESMU scores) - control (relatively low ESMU scores) design with 50 random university students who have reported varying levels of ESMU. The case and control groups included 25 subjects each. Brain volumes were calculated with Voxel-Based Morphometry techniques applied to structural MRI scans. Results based on voxel-wise and region-of-interest (ROI) analyses showed that the case group had reduced GMV in the bilateral amygdala and right ventral striatum. The GMV of the bilateral amygdala and right ventral striatum negatively correlated with ESMU scores in the voxel-wise analysis. No differences or correlations in relation to prefrontal regions were observed. Using the ROI analysis, the bilateral amygdala volumes correlated with ESMU scores, and insufficient evidence regarding the ventral striatum and ESMU was obtained. It is concluded that excess social media use in the general population is associated in part with GMV reduction in the bilateral amygdala, and possibly the striatum, but not in volumetric differences in prefrontal regions.

1. Introduction

Excess Social Media Use (ESMU) is a behavioral pattern on the spectrum of repeated compulsive online behaviors that can produce addiction-like symptoms, including salience, withdrawal, mood modification, relapse, conflict, and tolerance (Turel, 2015; Turel and Serenko, 2012). While there are no established clinical classification criteria for ESMU, and the appropriate terminology (e.g., addiction, disorder, problematic use, excess use) is not yet determined (Lortie and Guittin, 2013), studies show that most users present some degree of excess use and about 4.5% may be classified as at-risk for addiction (Bányai et al., 2017). Because ESMU can adversely impact large cross-sections of social media users (De Cock et al., 2014; Karaiskos et al., 2010; Kuss et al., 2013), through for instance diminished academic performance (Turel and Qahri-Saremi, 2016), wellbeing (Bright et al., 2015), and sleep (Turel et al., 2016), it is important to study its neural bases. Moreover, since ESMU does not involve neurotoxicity and begins at an early age (as opposed to, for instance, gambling), understanding its neural basis can provide a psycho-behavioral account of addiction, without confounding the finding with chemical effects.

To this end, we turn to the dual system theory (Bechara, 2005), according to which excess behaviors stem from an imbalance between hyperactivity of the reward system (mesolimbic dopamine amygdala-striatal) and hypo-activity of the inhibition system which includes prefrontal regions such as the orbitofrontal cortex (OFC), ventromedial prefrontal cortex (vmPFC), dorsolateral prefrontal cortex (dlPFC) and the Anterior Cingulate Cortex (ACC) (Brand et al., 2014). Using this theory we hypothesize regarding the GMV differences in these regions that may be associated with ESMU in a typical cross-section of social media users (i.e., people who present some degrees of ESMU).

First, the amygdala is important for excess behavior formation and maintenance because it governs learning (linking cues to emotive outcomes) and dopamine release in response to anticipated and received rewards (Bechara et al., 1999). Structural abnormalities in it (typically reduced GMV), are associated with excess behaviors, including substance use (Barros-Loscertales et al., 2011; Makris et al., 2008) and using Facebook (He et al., 2017). This reduced GMV can presumably reflect both hypo-activity in the reward anticipation stage, which promotes reward seeking, and hyper-activity in the reward outcome processing stage, that reflects fast and efficient reward production (Luijten

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et al., 2017). GMV reduction in this system may take place even at low levels of ESMU because the mesolimbic-dopamine system is highly morphologically flexible (Kanai and Rees, 2011). As such, assuming similarities between excess behaviors and ESMU, we hypothesize that (H1) the volumes of the bilateral amygdale will be reduced in high (top 50%) vs. low (bottom 50%) ESMU cases in a typical cross-section of users.

Second, the ventral striatum (Nucleus Accumbens, NAcc) is also important in reward processing (Cardinal et al., 2001); it is a central reward hub that has been shown to be involved in many excess behaviors (Gilman et al., 2014). Its activity has been shown to be associated with social media phenomena such as likes (Sherman et al., 2016) and reputation gains (Meshi et al., 2013). It has also been shown to be associated with social media addiction levels (Turel et al., 2014). From a structural standpoint, findings are less consistent. Some studies show that high frequency of checking Facebook on a smartphone is associated with reduced NAcc volumes (Montag et al., 2017) and others find no structural changes in NAcc associated with social media addiction (He et al., 2017). Given the theoretical role of NAcc in excess behaviors, the presumed morphological flexibility of this region (Kanai and Rees, 2011), and the notion that excess behaviors are often associated with reduced NAcc volumes (Seifert et al., 2015), we venture to hypothesize that (H2) the volumes of the bilateral NAcc will be reduced in high (top 50%) vs. low (bottom 50%) ESMU cases.

Third, excess Internet behaviors can also be associated with impairment of executive control abilities (Dong et al., 2011a, 2011b). The reason is that when prefrontal inhibition systems are weak, they cannot respond to and override impulsions mediated via the reward system. At low-medium levels of excess behavior, there are mixed findings. Some studies of relatively normal users showed that prefrontal systems are not impaired (Turel et al., 2014), and others showed volume reduction in regions such as the OFC in women (Altbäcker et al., 2016) and in right frontal pole in men (Kuhn and Gallinat, 2015), as a function of excess behavior. At higher levels of excess use (clinical cases of Internet addiction), reduced GMV in the OFC and dlPFC has been observed (Yuan et al., 2013, 2011). The reasons for such inconsistencies may be that prefrontal regions are much less morphologically flexible compared to mesolimbic-dopamine systems (Kanai and Rees, 2011) and that the subject of use in most prior studies (i.e., the Internet) includes many applications with varying use phenomenology (e.g., the use of videogames on the Internet is phenomenologically different from the use of social media). Moreover, many prefrontal functionalities can remain intact among Internet addicts (Nie et al., 2016) and even improve in response to hyper-activity in the amygdala-striatal system (He et al., 2017). Hence, we consider the possibility that especially at low-medium levels of ESMU there will be no observable prefrontal structural changes. We therefore cautiously hypothesize that (H3) the volumes of prefrontal regions will not significantly differ between high (top 50%) vs. low (bottom 50%) ESMU cases, in a typical cross-section of social media users.

Together, these hypotheses provide another look at past findings regarding GMV of key regions of the dual system that governs behavior and decision making, and their associations with a specific instance of the family of excess Internet use behaviors (He et al., 2017; Kuhn et al., 2011; Yuan et al., 2013, 2011; Zhou et al., 2011). They specifically replicate and extend such findings to the case a typical cross-section of SNS users (as opposed to comparing those who meet or do not meet criteria for addiction classification). Understanding the underlying brain differences associated with ESMU in typical users can help dealing with the aversive effects of excess use, even on those who do not meet addiction criteria.

2. Methods

Fifty participants who reported using Facebook were recruited for this study using a university bulletin board. ESMU was captured with

the 14-item Facebook-specific adaptation of the Compulsive Internet Use instrument, measured on a 1–5 Likert scale ($\alpha_{\text{All}} = 0.93$, $\alpha_{\text{Low ESMU}} = 0.90$, $\alpha_{\text{High ESMU}} = 0.87$) (Meerkerk et al., 2009; Turel et al., 2014) and median-split was employed for assigning people to the low- vs. high-ESMU groups. The study followed a sex-balanced case (relatively high ESMU, $n = 25$, 8 female, $M_{\text{age}} = 24.12$ [18–44], $SD_{\text{Age}} = 6.15$, $M_{\text{ESMU}} = 2.49$ [1.86–3.64], $SD_{\text{ESMU}} = 0.46$) - control (relatively low ESMU, $n = 25$, 8 female, $M_{\text{age}} = 29.80$ [19–55], $SD_{\text{Age}} = 10.90$, $M_{\text{ESMU}} = 1.34$ [1.00–1.78], $SD_{\text{ESMU}} = 0.26$) design. We were unable to assign people to addicted vs. non-addicted groups, given that such cutoffs are not formally defined. Nevertheless, based on prior research in the video-gaming domain (van Rooij et al., 2011), it was reasonable to assume that both groups largely do not meet addiction criteria, and hence represent normal users with respective low and high excess social media use (Low-ESMU range is aligned mostly with the low-use and no-addiction clusters in the abovementioned study, and High-ESMU here is mostly aligned with the high-use and no-addiction clusters in the abovementioned study). Participants were carefully screened for key neurological and psychiatric disorders using the Structured Clinical Interview for DSM-IV (SCID). They consented to participate in the study that was approved by the Institutional Review Boards of two research universities. No exclusions were made.

Brain GMV was calculated using Voxel-Based Morphometry techniques with high-resolution structural MRI scans performed in a 3T Siemens MAGNETOM Tim/Trio scanner at the Dana and David Dornsife Cognitive Neuroscience Imaging Center at the University of Southern California. The T1-weighted 3D-Magnetization Prepared Rapid Gradient Echo (MPRAGE) sequence was used to cover the whole brain [TR /TE = 2530/3.39 ms, flip angle = 7°, matrix = 256 × 256, 128 sagittal slices, 1.33 mm thickness]. The image quality was visually inspected for any distortion and noise before further analysis. The brain was extracted using BET, and then segmented into gray matter, white matter and CSF. The resulting gray-matter partial volume images were then aligned to the gray-matter template in the MNI152 standard space using the affine registration tool FLIRT, followed by nonlinear registration using FNIRT, which used a b-spline representation of the registration warp field. The spatially normalized images were then averaged to create a study-specific template, to which the native gray matter images were registered again using both linear and nonlinear algorithms as described above. The registered partial volume images were then modulated by dividing them with the Jacobian of the warp field to correct for local expansion or contraction. The modulated segmented images that represent the GMV, were then smoothed with an isotropic Gaussian kernel with a 3 mm standard deviation. The resulting images were re-sampled into 2 mm × 2 mm × 2 mm isotropic voxels (8 mm³). Voxel-wise general linear models were used for statistical inference. To increase robustness, both GMV differences between high and low ESMU participants as well as correlation between ESMU and GMV were analyzed using FSL-VBM toolbox. In both analyses we used age and sex as covariates (note that while sex composition was identical across groups, we used sex to account for possible variation within-groups). We corrected for multiple comparisons in the voxel-wise analysis. First, for the voxel-wise analysis, GMV was compared between the two groups. The null distribution at each voxel was constructed using 10,000 random permutations of the data. Threshold-free cluster enhancement (TFCE) with $p < 0.05$ was employed for correcting for multiple comparisons. This methods can have improved sensitivity and provide richer and more interpretable output compared to cluster-based thresholding methods (Smith and Nichols, 2009). It has been consequently widely used in VBM-based studies. To increase robustness, significant regions from the voxel-wise GMV group difference analysis were extracted for correlation analysis between GMV and ESMU. Second, the Regions of Interest (ROIs) as defined by the Harvard-Oxford cortical probability atlas (25 thresholds, 2 mm resolution) were extracted for correlation analysis between GMV and ESMU. We also compared the GMVs between groups. For all correlational analyses,

Table 1
Summary of voxel-based morphometry (VBM) results.

Voxel-wise						
Brain region	Voxels	Cluster Geometric Center (MNI)			Partial-Correlation with ESMU Score	TFCE corrected <i>p</i> value (local maxima)
		x	y	z		
Low (control) > High (case)						
L Amygdala	207	−24.3	−5.77	−17.8	−0.438**	<i>p</i> < 0.001
R Amygdala	190	25.3	−4.36	−17.6	−0.384**	<i>p</i> < 0.001
R ventral Striatum	132	20.4	7.42	−6.03	−0.366**	<i>p</i> < 0.005
High (case) > Low (control)						
NONE						
Region of Interest (ROI)-based						
Low (control) > High (case)						
L Amygdala	<i>t</i> (48) = 3.15, <i>p</i> = 0.003				−0.457**	Bonferroni corrected <i>p</i> < 0.001
R Amygdala	<i>t</i> (48) = 2.84, <i>p</i> = 0.007				−0.421**	<i>p</i> < 0.001
R ventral Striatum	<i>t</i> (48) = 2.21, <i>p</i> = 0.03				0.264	<i>p</i> < 0.069
L ventral Striatum	<i>t</i> (48) = 1.51, <i>p</i> = 0.14 (ns)				0.118	<i>p</i> = 0.414

MNI: Montreal Neurological Institute coordinates; TFCE: Threshold-Free Cluster Enhancement; EMSU: Excess Social Media Use; L: Left; R: Right.

** *p* < 0.01.

robust regression was used to minimize the impact of outliers in the behavioral data, using iteratively reweighted least squares implemented in the *robustfit* command in the MATLAB Statistics Toolbox. Reported correlations reflect non-robust Pearson product-moment correlation values, whereas the reported *p*-values are based on the robust regression results.

3. Results

Table 1 provides almost full support to the hypotheses by demonstrating reduced GMV in the case versus the control group in the bilateral amygdala and the right ventral striatum, and no prefrontal differences, across analyses. Results are illustrated in Fig. 1.

4. Discussion

The findings indicate that ESMU at presumably normal levels is similar to other excess problematic behaviors in that it is partially associated with individual differences in the morphology of the reward (amygdala-striatal) system (Barros-Loscertales et al., 2011; Makris et al., 2008). This lends support to H1, which focuses on the amygdala, and partial support to H2, which focuses on the NAcc. In contrast to clinical cases of very high ESMU, in a typical cross-section of users that includes low-medium levels of ESMU, people in the top 50% of ESMU scores did not present significant differences from those in the bottom 50% in prefrontal morphology. This lends support to H3 and highlights potential differences between morphology in presumably normal populations with reasonable levels of ESMU and those who present very high levels of ESMU and often meet at-risk for addiction criteria.

We conclude that ESMU in the general population primarily involves morphology of the amygdala-striatal system, but not prefrontal systems. This suggests that excess behaviors may have similar behavioral symptomatology, but the imbalance of the systems that drive these behaviors may stem from different structural differences. These differences in the case of typical levels of ESMU (reduced GMV of the amygdala-striatal system) can indicate one, or a combination, of three scenarios, which we cannot validate or refute in this study. The first possibility is that people with lower amygdala, and to some extent striatal volumes, are more prone than others to engage in excess behaviors, presumably because their amygdala-striatal system includes fewer neurons and can hence respond faster to learned stimuli, perhaps even before a person can recruit resources for engaging in inhibition (He et al., 2017). Since social media is an accessible and available platform for generating rewards (Meshi et al., 2013), to the point where people present addiction-like symptoms in relation to these rewards (Turel, 2015, 2016; Turel and Serenko, 2012; Turel et al., 2011), it is possible that social media is simply the platform of choice, the use of

which is difficult to inhibit due to the efficiency of the reduced-in-volume amygdala-striatal system. The second possibility is in line with the incentive-sensitization theory (Robinson and Berridge, 1993) and suggests that the use of social media sensitizes the amygdala-striatal system (Turel et al., 2014). Such sensitization can lead to volumetric adaptations through processes of neural pruning, with the objective to produce a more efficient amygdala-striatal system that can deal with the growing reward processing demands (Kanai and Rees, 2011). In both of these scenarios it is possible that the volume reductions reflect shortage of dopamine receptors, which have been implicated in the family of Internet addictions (Kim et al., 2011). The third possibility is that there is a common factor, such as genetics, that contribute to both a-priori lower amygdala-striatal volume and more flexible adaptations in response to changes in reward frequency and intensity (Brewer and Potenza, 2008; Clark, 2002). The reasons for and directionality of the observed inter-individual differences are difficult to pinpoint (Frank, 2013); we hence call for future research to examine volumetric progression of the amygdala-striatal system, from young age before social media use begins, as a means to better understand the direction of causation.

From a broader excess behavior research perspective, the findings further illustrate the idea that disruption of reward processing is at the heart of the development and maintenance of excess behaviors (Di Chiara et al., 1999). The observed morphological inter-individual differences can supplement prior activation-based findings and may serve as a basis for resolving inconsistencies in prior addiction research (Luijten et al., 2017). Specifically, the reduced volume of the amygdala-striatal system supports the reward deficiency theory (Cloninger, 1987) in that the reduced volume may reflect the occasionally observed hypo-activation of reward circuits in addicts; this lower activation reduces the rewards people feel, which in turn motivates them to engage more often in the target behavior. Our findings can also support impulsivity theory, according to which excess behaviors stem from hyper-activation of the amygdala-striatal system that produces strong incentive rewards (Noel et al., 2013). This idea is supported if we assume that lean amygdala-striatal system means faster and more efficient reward production, akin to pruning of other brain regions in an attempt to make them faster (Kanai and Rees, 2011). Such individual differences in amygdala-striatal volumes and the efficiency that is presumably associated with reduced GMV, are also in line with the incentive-sensitization theory (Robinson and Berridge, 2001), according to which people overtime develop a sense of “wanting” toward social media use in response to social media cues. Pruned amygdala-striatal system can produce a more obligatory and efficient response to such cues.

The support of these theories, which are not always compatible, may stem from the notion that volumetric differences in the amygdala-striatal system can reflect at the same time, both efficiency in some

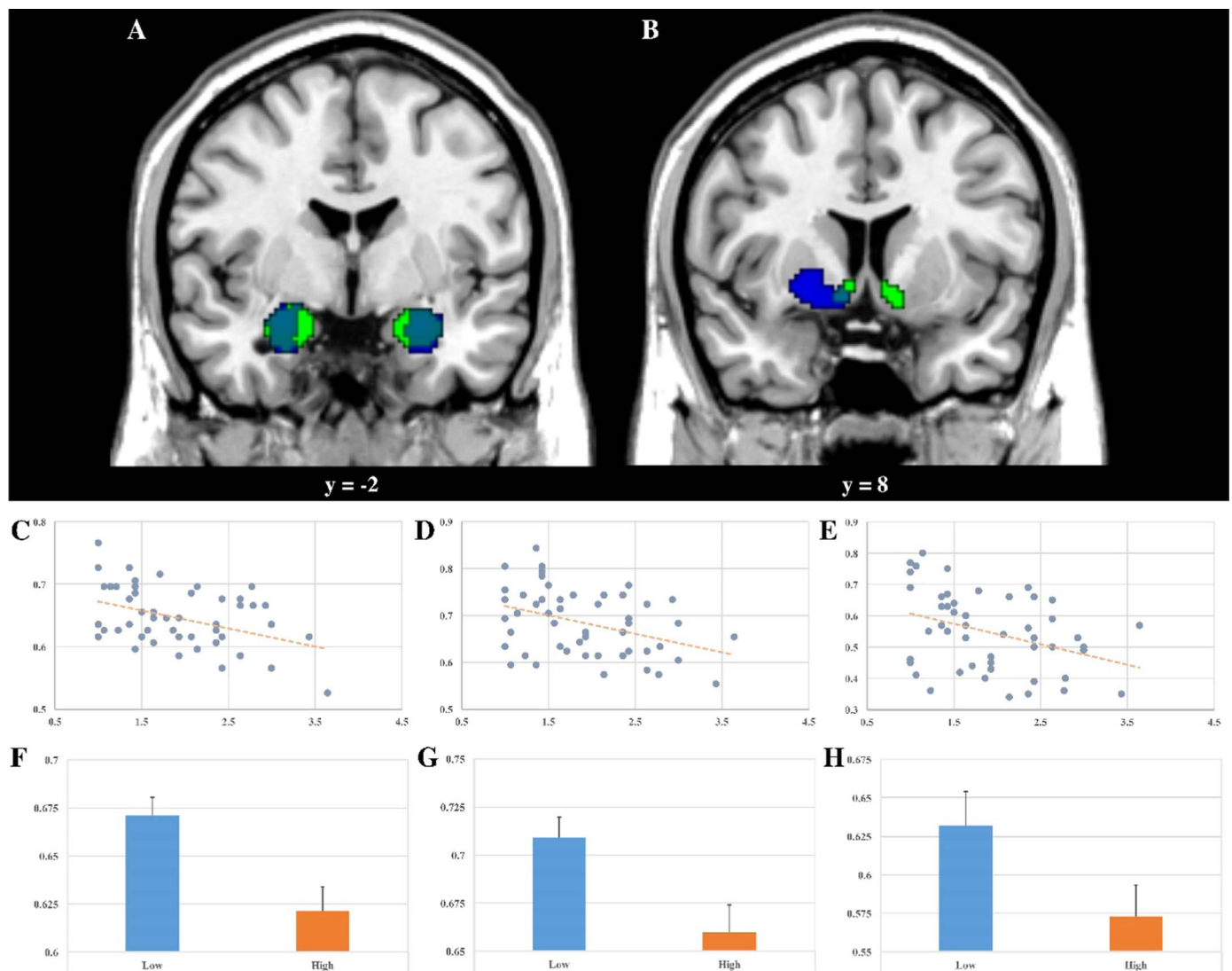


Fig. 1. (A & B) Voxel-wise VBM revealed that case (participants with higher ESMU) showed reduced GMV in bilateral amygdala and right striatum compared to controls (participants with lower ESMU score), after accounting for age and sex. Blue regions show the voxel-wise VBM results. Green regions show anatomical ROIs used to extract GMV. The overlapping regions are displayed in light blue. Figures are displayed in canonical view with left side of the paper represent the right hemisphere. These three regions (C = left amygdala, D = right amygdala, E = right striatum) also show significant negative correlation between GMV and ESMU score. The horizontal axis represents the ESMU score and vertical axis represents the GMV (arbitrary value) in each region. ROI analysis suggested that the case group (higher ESMU; in orange) have less GMV than the control group (lower ESMU; in blue) in left amygdala (F), right amygdala (G), and right striatum (H). The horizontal axis represents the ESMU group (case = relatively high versus control = relatively low) and vertical axis represents the GMV (arbitrary value) in each region. Error bar represents standard error. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article).

processes it supports (reward outcome processing) and deficiency in others (reward anticipation) (Luijten et al., 2017). That is, reduced-in-volume amygdala-striatal system may be associated with weaker ability to anticipate rewards as it lacks neurons needed for this task, hence promoting hypo-activation, at least at the reward anticipation stage. This is in line with reward deficiency theory. Simultaneously, the reduced volume can reflect efficiency (speed) in reward outcome processing. That is, it can produce faster and stronger rewards as the retained fewer neurons require less coordination and can respond faster to stimuli. This is line with the impulsivity and incentive-sensitization theories. Future research should further examine these issues, not just in the case of social media, but also with regards to substances.

Our findings regarding lack of prefrontal differences and associations replicate similar findings that showed no activation or structural differences in prefrontal regions in relation to excess social media use (He et al., 2017; Turel et al., 2014). They differ, however from other studies in which such prefrontal differences were observed (Brand et al., 2014; Yuan et al., 2013, 2011). They may therefore provide

initial support to proposed differences in morphological flexibility between subcortical and cortical regions. This can resolve inconsistencies and explain why in some cases (extreme and prolonged excess use) prefrontal morphological differences are present (Yuan et al., 2013, 2011) and in others, like this study, where people report not too-high levels of ESMU, such differences are not significant. Since some studies demonstrate prefrontal differences even in habitual users (Altbacker et al., 2016; Kuhn and Gallinat, 2015), the findings here may lend support to the idea that brain morphology can differ between excess Internet use and the more specific excess social media use. Future research should further examine such ideas.

Several limitations of this study should be mentioned. First, our sample included people with low-medium ESMU scores. Hence, it represents relatively normal variation in the population and excludes extreme cases of ESMU. Future research may focus on a broader range of ESMU scores. Second, our data were cross-sectional and cannot support causality arguments. Future research may employ longitudinal designs and more techniques for developing a deeper understanding of

the causal directions of the observed associations. Lastly, our techniques did not allow for a more nuanced analysis of the components of gray matter in the regions of interest (glial cells, neuronal cells, dendrites, axon terminals). Future research may look into gray matter composition and its association with ESMU.

Disclosure statement

The authors declare no conflict of interest.

Author contributions

Conception and design of the study: QH, OT, AB; acquisition and analysis of data: QH, OT, DB, AB; drafting the manuscript or figures: QH, OT, DB, AB.

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