



# Delay discounting mediates the association between posterior insular cortex volume and social media addiction symptoms

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## Abstract

Addiction-like symptoms in relation to excessive and compulsive social media use are common in the general population. Because they can lead to various adverse effects, there is a growing need to understand the brain systems and processes that are involved in potential social media addiction. We focus on the morphology of the posterior subdivision of the insular cortex (i.e., the insula), because it has been shown to be instrumental to supporting the maintenance of substance addictions and problematic behaviors. Assuming that social media addiction shares neural similarities with more established ones and consistent with evidence from the neuroeconomics domain, we further examine one possible reason for this association—namely that insular morphology influences one's delay discounting and that this delay discounting contributes to exaggerated preference for immediate social media rewards and consequent addiction-like symptoms. Based on voxel-based morphometry techniques applied to MRI scans of 32 social media users, we show that the gray matter volumes of the bilateral posterior insula are negatively associated with social media addiction symptoms. We further show that this association is mediated by delay discounting. This provides initial evidence that insular morphology can be associated with potential social media addiction, in part, through its contribution to poor foresight and impulsivity as captured by delay discounting.

**Keywords** Social media · Addiction · Morphology · Insular cortex · Delay discounting · Gray matter volume

Social networking site (SNS) addiction is an emergent potential disorder on the spectrum of technology addictions. It is a state of maladaptive dependency on the SNS that manifests in compulsively seeking and engaging in SNS use to such an extent that typical behavioral addiction symptoms emerge (e.g., withdrawal, salience, tolerance, mood modification, conflict, and repeated relapses) (Turel, He, Xue, Xiao, & Bechara, 2014). While there is still an ongoing debate whether such problems can or should be classified as a formal disorder of addiction (Carbonell & Panova, 2017), it is clear that regardless of their classification

and the terminology used, such maladaptive dependencies can adversely affect various facets of people's lives, including social functioning, wellbeing, academic, and professional performance, as well as mental health (He, Turel, Brevers, & Bechara, 2017a; Turel, Poppa, & Gil-Or, 2018; Serenko & Turel, 2015; Turel & Qahri-Saremi, 2016). Such addictions also can serve as a gateway for physically dangerous practices, such as using SNS while driving (Turel & Bechara, 2016a). Notable proportions of adolescents (4.5% in Banyai et al., 2017) and young adults (15.23% in Turel, Brevers, & Bechara, 2018) meet criteria for at-risk for social media addiction, and many others present symptoms similar to those observed in other behavioral addictions. Hence, there is a growing need to better understand the etiology and pathogenesis of such potential addictions (Turel, Romashkin, & Morrison, 2016).

Consistent with the American Medical Association's view of addictions as brain diseases, SNS addiction can be treated as an outcome of a deficit in people's brain systems involved in decision making. This deficit promotes uncontrolled problematic compulsive behaviors that can hurt normal functioning. Several studies adapting this perspective suggested that possible neurobiological roots of this problem include hypersensitization of the

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impulsive brain system to SNS cues (Turel et al., 2014), reduced gray matter volumes of the amygdala-striatal system (He, Turel, & Bechara, 2017a; He, Turel, Brevers, et al., 2017b; Montag et al., 2017), and increased anterior cingulate volumes (He, Turel, & Bechara, 2017a). These findings not only illuminate the complexity of the studied phenomenon but also suggest that it can be rooted, at least in part, in the decision-making faculties in people's brains.

In the current study, we extend this line of work to account for recent conceptualizations of tripartite models of addiction (Turel & Bechara, 2016b; Wood & Bechara, 2014). These models extend the dual system perspective, which portrays addictions as decision-making deficits in terms of strong uncontrollable motivation for immediate rewards coupled with weak inhibition abilities. The extension suggests that the insular cortex, or insula, is an additional central region for addiction development and maintenance. This region translates interoceptive states of homeostatic violation into what may subjectively become experienced as an urge or craving to engage in behaviors that resolve or reduce the homeostatic imbalance (Craig, 2009; Naqvi & Bechara, 2010). This craving can drive addictive behaviors by exciting the impulsive brain system, increasing the salience of the desired behavior, and by occupying the inhibition brain system, such that it cannot properly engage in reflection and behavior control (Noel, Brevers, & Bechara, 2013).

Indeed, when the insula is lesioned, a significant reduction in the drive to engage in addictive behaviors is observed (Naqvi, Rudrauf, Damasio, & Bechara, 2007). Consequently, functional and structural abnormalities of the insula have been observed across a wide range of substance use and other addictive behaviors (Droutman, Read, & Bechara, 2015). However, the involvement of the insula has been largely overlooked in the Internet addiction literature in general, and specifically in studies exploring the etiology of SNS addiction. In that sense, the insula is a “hidden island,” not just in terms of its neuroanatomical location, but also in terms of the paucity of research that addresses its possible involvement in SNS addiction or excessive use of SNS.

Structural imaging studies have largely shown that problematic use of substances, such as cocaine (Rando, Tuit, Hannestad, Guarnaccia, & Sinha, 2013), opioids (Wollman et al., 2017), alcohol (Heikkinen et al., 2017), and cigarettes (Peng et al., 2017), is associated with reduced gray matter volumes in the insular cortex, typically bilaterally. In many cases, these structural differences are especially pronounced in the posterior parts of the insula (Droutman et al., 2015). The importance of the posterior insula stems from its role; it processes incoming interoceptive signals and mobilizes insular activity in response to somatic inputs associated with hedonic experiences, which are then transferred to anterior dorsal and ventral parts of the insula (Markett, Heeren, Montag, Weber, &

Reuter, 2016). These regions are connected to and influence the functionality of the reward system (Noel et al., 2013), the abnormalities in which have been implicated in prior SNS addiction studies (He, Turel, & Bechara, 2017a; He, Turel, Brevers, et al., 2017b; Montag et al., 2017). Assuming neural similarities between SNS addiction and other addictions, we can hypothesize that: (H1) the gray matter volumes of the bilateral posterior insula are negatively associated with SNS addiction scores.

It is further suggested that modulation of the insula can contribute, at least in part, to addiction manifestations through its associations with deficiencies in assessing future consequences and specifically with delay discounting. Delay discounting is a behavioral measure of impaired foresight and consequent impulsive decision-making that captures general (non SNS-specific) preference for current small rather than for larger future rewards; it is operationalized with a delay discounting factor,  $k$ , that encapsulates the rate by which the subjective value of rewards is reduced as a function of time to receive the reward (Kirby & Santiesteban, 2003). This association is assumed, because the posterior insula has been shown to be activated in delayed reward decisions and to be a central brain region involved in delaying gratification (Wittmann, Leland, & Paulus, 2007). Consistent with functional studies that show changes in insular activation in people with deficient foresight (Wittmann, Leland, & Paulus, 2006), structural scans show that pathological gamblers (who have higher delay discounting compared with normal populations) have reduced bilateral insula volumes compared with healthy subjects (Mohammadi et al., 2016). Assuming similar neural underpinnings in the case of SNS addiction and the notion that structural changes in the insula are primarily in its posterior division (Droutman et al., 2015), we can hypothesize that: (H2) the gray matter volumes of the bilateral posterior insula are negatively associated with the delay discounting factor  $k$ .

Lastly, it has been shown that addictions often stem, in part, from problems in assessment of future consequences and misjudgment of future rewards and punishments (Bechara & Damasio, 2002; Bechara, Damasio, Damasio, & Lee, 1999) as often manifested in increased delay discounting factor  $k$  (Bickel, Jarmolowicz, Mueller, Koffarnus, & Gatchalian, 2012; Hamilton & Potenza, 2012; Robles, Huang, Simpson, & McMillan, 2011), including in the case of technology-related addictions (Saville, Gisbert, Kopp, & Telesco, 2010; Weinstein, Timor, Ben Abu, & Mama, 2016). We therefore can hypothesize that (H3) the delay discounting factor  $k$  is positively associated with SNS addiction. The proposed hypotheses suggest that (H4) delay discounting is at least a partial mediator of the association between posterior insula volumes and SNS addiction.

## Methods

### Participants

Participants were invited via university announcements to participate in a study on Facebook use. Upon arrival and after study introduction, they gave informed consent to the study procedures, which were approved by the Institutional Review Board of the University of Southern California. Inclusion criteria included Facebook use and age older than 18 years. Exclusion criteria included the existence of neurological disorders that may affect result interpretation (psychoses, current major depression episode, a history of major depression episodes or major depressive disorder, heavy drinking, substance abuse, schizophrenia, current and history of anxiety disorders, and bipolar disorder) and abnormal uncorrected vision. No exclusions based on these criteria were made.

The procedure yielded a sample of 33 participants (6 females, average age = 31.18, standard deviation [SD] = 9.32, range = 21–62 years) who reported using Facebook (average years of experience = 6.79, SD = 2.54, range = 1.0–12.0 years; average number of contacts on Facebook = 682.5, SD = 565.5, range = 20–2,000 contacts; average use frequency = 6.42, SD = 6.80, range = 1–30 times/day), and who met the inclusion criteria and did not meet any of the exclusion criteria. One participant (male) was later excluded from further analysis because of poor MRI image quality, leaving a final sample of 32 (6 females) on which we ran analyses. The sample size was tested with G\*Power (version 3.1.9.2) with large effect size estimation ( $r = 0.5$ ), and results suggested that a sample of 32 was sufficient for getting a statistical power larger than 0.95.

### Procedures

Measures were obtained from four sources. First, participants were asked to complete an online survey and interview that captured descriptive information and demographics, as well as their levels of SNS addiction. Second, participants completed online screening criteria forms for capturing exclusionary conditions, such as having a pacemaker or specific mental health conditions, particularly substance addictions. These data were used for exclusion-inclusion decisions but not for hypothesis testing. Third, computer-based delay discounting task (Wang et al., 2014) tests were administered to participants. Lastly, gray matter volumes of the whole brain as well as the regions of interest were captured by applying Voxel-Based Morphometry (VBM) techniques to high-resolution structural MRI scans.

### Online survey and screening

The online survey captured people's age, number of contacts on the SNS, the daily frequency of SNS use, and years of

experience with the SNS, using open ended and Likert-type questions. It also captured participants' sex. The survey also captured SNS addiction scores by using the 14-item instrument by Van Rooij et al. (2011) as applied to the Facebook use context (Turel et al., 2014). This scale captures typical behavioral addiction symptoms, such as loss of control, withdrawal, salience, relapse, and conflict. The score it produced, therefore, captured people's level of addiction as manifested by these symptoms.

Lastly, a short computerized version of the Structured Clinical Interview for DSM-IV (the SCID) was used to rule out certain mental health disorders, such as psychosis and substance use disorders (Bechara & Damasio, 2002). This interview was used to assign Axis I diagnoses, looked at both lifetime and current diagnoses, and was conducted by a trained research assistant who had several years of experience using this tool. It took approximately 20 minutes and focused on ruling out specific relevant issues, namely psychosis, depression, heavy drinking (abuse and dependence), substance abuse, pathological gambling, schizophrenia, anxiety disorders, and bipolar disorder.

### Delay discounting task

The Delay Discounting Task (DDT) was adapted from Wang et al. (2014). In this task, participants were asked to choose between an immediate monetary reward and a future (always 120 days later) reward. The sizes of the immediate and delayed reward were manipulated independently, with immediate reward ranging from 25 to 100 U.S. dollars (16 levels in \$5 increments) and delayed reward ranging from 38 to 150 U.S. dollars (16 levels in \$7–8 increments). There was a total of 256 trials with all possible combinations of immediate and delayed reward levels. Logistic regression was employed with the sizes of immediate and delayed rewards as the independent variables and choice of immediate or delayed option as the dependent variable. The temporal discounting factor ( $f$ ) was computed as follows:  $f = \beta_{\text{delayed}} / \beta_{\text{immediate}}$ , where  $\beta_{\text{immediate}}$  and  $\beta_{\text{delayed}}$  are the unstandardized regression coefficients for the immediate and delayed variables, respectively. The delay discounting factor  $k$  was calculated using the following equation,  $k = \left(\frac{1}{f} - 1\right) / 120$ , with larger  $k$  indicating steeper temporal discounting. This factor served as the index for one's delay discounting.

Note that while delay discounting is often measured in the context of monetary decisions, it has been identified as a strong predictor of the severity of addictive behaviors, including many that do not involve money (Bari & Robbins, 2013; Stevens et al., 2014). This happens, because steep delay discounting is often manifested across life domains in the presence of prefrontal and reward system abnormalities (Bechara & Damasio, 2002; Bechara et al., 1999; Bechara,

Dolan, & Hines, 2002). Hence, delayed discounting operationalized with monetary-choice tasks can be suitable for measuring general impulsivity problems, and it is consequently associated with various addictions (Amlung, Vedelago, Acker, Balodis, & MacKillop, 2017).

### MRI protocol and VBM analysis

High-resolution structural magnetic resonance imaging (MRI) scans were conducted to capture relevant brain morphology after the surveys and tasks were completed. The extracted voxel-wise gray matter volumes were partially correlated with the addiction score and other covariates, as captured in the surveys. Significant findings from the voxel-wise GMV correlation were extracted for plotting purposes and for mediation analysis. Regions of Interest (volumes of the bilateral insula and its subdivisions) were extracted using common maps. The scans were 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 MRI session lasted approximately 15 minutes, and one high-resolution structural scan was performed. The T1-weighted 3D-Magnetization Prepared Rapid Gradient Echo (MPRAGE) sequence was used to cover the whole brain (TR (repetition time)/TE (echo time) = 2,530/3.39 ms, flip angle = 7°, matrix = 256 × 256, 128 sagittal slices, 1.33-mm thickness). The scanned brains were divided into voxels (the three-dimensional equivalent of pixels) with volumes of 8 mm<sup>3</sup> (2-mm × 2-mm × 2-mm isotropic). These voxels served as the smallest data point for further analyses. Preprocessing of brain images was performed to bring all brains into a common three-dimensional space, and voxels' locations were mapped onto a common anatomical brain map, because people have brains different in size and shape.

Preprocessing and data analyses were performed with FSL (FMRIB Software Library)-VBM (voxel-based morphometry) (<http://fsl.fmrib.ox.ac.uk/fsl/fslwiki/FSLVBM/>), an optimized voxel-based morphometry analysis toolbox implemented in FSL, a statistical toolbox for neuroimaging data (<http://fsl.fmrib.ox.ac.uk/fsl/fslwiki/FSL>). This approach is operator-independent and efficient (Ashburner & Friston, 2000). First, structural images were extracted using the Brain Extraction Tool, BET. Next, tissue-type segmentation was performed using the FMRIB's Automated Segmentation Tool, FAST4. The resultant gray matter partial volume images were then aligned to the gray matter template in the Montreal Neurological Institute standard space (MNI152) using the affine registration tool FLIRT (FMRIB's Linear Image Registration Tool), followed by nonlinear registration using FNIRT (FMRIB's Non-Linear Image Registration Tool), which used a b-spline representation of the registration warp field (Rueckert et al., 1999). The spatially normalized images were then averaged to create a study-

specific template to which the native gray matter images were registered by using the abovementioned linear and nonlinear algorithms. 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, which represent the GMV, were then smoothed with an isotropic Gaussian kernel with a 3-mm standard deviation.

Several steps followed using the abovementioned FSL-VBM toolbox. First, hypothesis driven ROI-based analysis was performed. The GMV of anatomically defined regions of interest of eight brain regions, including the bilateral insula, and six subregions [bilateral dorsal anterior insula (DAI), bilateral ventral anterior insula (VAI), and posterior insula (PI)] were extracted. Harvard-Oxford cortical probability atlas (25 thresholds, 2-mm resolution) was used to extract the left and right insula. The three subregions of insula were obtained from NeuroSynth (<http://neurosynth.org/>) according to Droutman et al. (2015). The subregion masks extracted from NeuroSynth have overlaps. These were removed with the *fslmaths* tool. These masks were confirmed and restricted in the left and right insula ROIs (because the NeuroSynth results include extensions of regions to other parts of the frontal cortex). Table 1 and Fig. 1 display the number of voxels and MNI locations, after the above two preprocessing steps. Averaged GMV in each ROI was extracted for each participant using the *fslmeants* to sum voxel-by-voxel GMV within the ROI. These total GMV scores were then subjected to partial correlation analyses (accounting for the effects of control variables) using SPSS 24.

Next, mediation analysis was conducted using the Hayes' (2013) PROCESS macro implemented in SPSS 24 to explore whether increased delay discounting partially accounts for the association between ROI-based posterior insula GMV and SNS addiction. A bootstrapping procedure (with 5,000 bootstrap samples) to estimate 95% confidence intervals (CI) was used. A 95% CI for the product of indirect path coefficients that does not include zero provides evidence of a significant indirect effect (Preacher, Rucker, & Hayes, 2007).

Lastly, as a supplementary analysis, a voxel-wise (whole-brain) general linear model was built to examine the partial correlation between gray matter images and SNS addiction scores. Specifically, partial Pearson correlations were estimated in the FSL package to capture the addiction-GMV correlations after accounting for variation explained by the control variables. A total of six regressors (one variable for the SNS addiction score and five controls were included: age, gender, number of contacts on the SNS, SNS use frequency, and years of experience with the SNS). Given the positive correlation between SNS addiction score and (1) number of contacts on the SNS and (2) SNS use frequency, it was orthogonalized with these two variables in the model. We also checked the model with and without these controls. The main results

**Table 1** Summary of ROIs and correlation with SNS addiction and the delay discount factor

Brain region	Voxels	Cluster Geometric Center (MNI)			Partial-Correlation with SNS addiction	Partial-Correlation with delay discount factor k
		x	y	z		
L Insula	1181	-37.6	2.57	-0.325	-0.247	-0.202
R Insula	1181	37.6	2.57	-0.325	-0.179	-0.191
L DAI	234	-36.2	14.2	-0.701	-0.071	-0.113
R DAI	234	36.2	14.2	-0.701	-0.259	-0.218
L VAI	100	-34.3	9.72	-13.4	-0.142	-0.104
R VAI	100	34.3	9.72	-13.4	-0.214	-0.205
L PI	420	-37.9	-10.4	4.76	-0.610*	-0.604*
R PI	420	37.9	-10.4	4.76	-0.546*	-0.540*

MNI, Montreal Neurological Institute coordinates; DAI, dorsal anterior insula; VAI, ventral anterior insula; PI, posterior insula; L, left; R, right

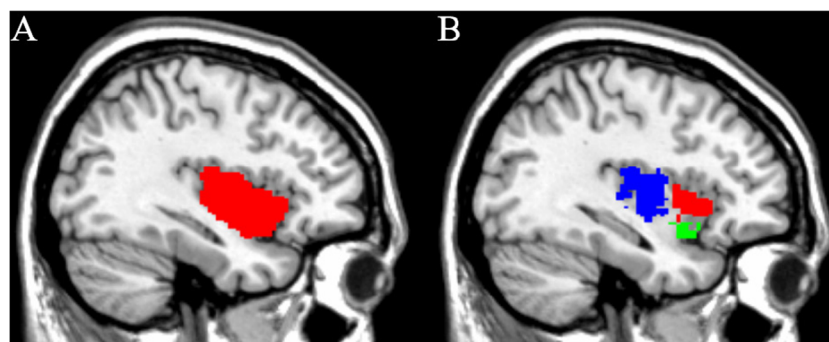
\* $p < 0.001$  Bonferroni corrected

remained unchanged (clusters remained the same, but the number of voxels has slightly changed; to 1443) when we did not include covariates. All continuous variables were mean centered as suggested by Mumford ([http://mumford.fmripower.org/mean\\_centering](http://mumford.fmripower.org/mean_centering)). Non-parametric permutation methods (Randomise v2.1 in FSL; see <http://fsl.fmrib.ox.ac.uk/fsl/fsl-4.1.9/randomise/index.html>) were used for inference on statistic maps. The null distribution at each voxel was constructed using 10,000 random permutations of the data to ensure that observed results are not due to chance. Because there are hundreds of voxels in the areas of interest, we corrected for multiple comparisons using Threshold-free cluster enhancement (TFCE) with  $p < 0.05$  across the whole brain. Then, to illustrate correlations, averaged GMV in the significant clusters were extracted.

## Results

The addiction scale was valid and reliable ( $\alpha = 0.96$ , Guttman Split-half coefficient = 0.91, total variance

explained by principal component = 0.67, composite reliability = 0.97, average variance extracted = 0.79). Hence, its average reasonably represented participants' self-reported SNS addiction levels or at least their levels of addiction-like symptoms in relation to SNS use. Addiction scores (average = 1.74, SD = 0.59, range = 1.00–3.00 on a 1–5 Likert type scale) as well as all extracted GMVs did not deviate from normality (Kolmogorov-Smirnov statistics with  $p > 0.20$ ). Hence, analyses executed in the FSL-VBM package, which assume reasonable normality, were deemed to be appropriate. Addiction scores were not significantly correlated with sex, age, and years of Facebook experience (all  $|r|s < 0.26$ ,  $p > 0.15$ ) but were somewhat correlated with number of contacts one has on the SNS ( $r = 0.35$ ,  $p < 0.06$ ) and the frequency of use ( $r = 0.37$ ,  $p < 0.04$ ). This is reasonable and consistent with the view that one's level of addiction to SNS should be associated with expanding activity (contacts and use frequency) on the SNS, but it is also different from mere use facets in that it manifests in addiction symptoms, such as withdrawal and conflict



**Fig. 1** The region of interest. (A) The insula was defined from Harvard-Oxford cortical probability atlas (25 thresholds, 2-mm resolution). (B) Three subregions of the insula were obtained from NeuroSynth ([http://](http://neurosynth.org/)

[neurosynth.org/](http://neurosynth.org/)) according to Drouman et al. (2015). The VAI is displayed in green, the DAI is displayed in red, and the PI is displayed in blue. This figure only illustrates the left hemisphere

(Turel & Serenko, 2012). The delay discounting factor  $k$  in this sample was similar to this obtained in Wang et al. (2014) with a mean of 0.00269 (SD = 0.00415, range = 0.000213–0.0223).

To examine the hypotheses, an ROI-based analysis was performed. The average GMVs in eight anatomically defined ROIs (bilateral insula, bilateral VAI, bilateral DAI, and bilateral PI) were extracted and their partial correlations with addiction scores and  $k$  were estimated. Results demonstrated negative correlations between left and right PI GMV and the delay discounting factor  $k$  ( $r = -0.604$ ,  $p < 0.001$  and  $r = -0.540$ ,  $p < 0.001$ , respectively, after correction; Table 1). They also showed that addiction scores were negatively correlated with the left and right PI volumes ( $r = -0.61$ ,  $p < 0.001$ , and  $r = -0.546$ ,  $p < 0.001$ , respectively; Table 1; Fig. 2). The GMV in all other ROIs did not significantly correlate with  $k$  or addiction. Two significant correlations between the GMV of left and right PI with number of contacts,  $r = -0.380$  and  $r = -0.400$  respectively, both  $p < 0.05$ , were observed.

After accounting for the control variables, the delay discounting factor  $k$  was positively correlated with SNS addiction ( $r = 0.634$ ,  $p < 0.001$ ) and negatively with the GMV of the left insula cluster ( $r = -0.563$ ,  $p < 0.01$ ). We next used the implicated ROIs (left and right PI) in mediation analysis. Results (Fig. 3) suggested significant indirect associations between the GMV of these regions and SNS addiction, through delay discounting (left PI: point estimate =  $-0.118$ , 95% CI =  $-0.012$  to  $-0.385$ ; right PI: point estimate =  $-0.125$ , 95% CI =  $-0.014$  to  $-0.327$ ).

To supplement the ROI-based analyses, we performed voxel-wise (whole-brain) correlation analysis (but not a full mediation test). Consistent with the ROI-based findings, it revealed that SNS addiction scores were negatively correlated with the GMV in the posterior parts of the left insula (local maxima in MNI coordinates  $x, y, z = -46, -22, 10$ , geometric center  $x, y, z = -47.8, -22.5, 14.8$ ), after accounting for control variable effects (Table 2). This cluster overlapped the left PI with 93 voxels, but no overlap was found between this cluster and either DAI or VAI regions. Right PI volume did not correlate with addiction after TFCE correction. Association between amygdala-striatal morphology and SNS addiction, as found in prior research (He, Turel, & Bechara, 2017a; He, Turel, Brevers, et al., 2017b; Montag et al., 2017), did not emerge as significant, after TFCE correction. No brain regions showed positive threshold-corrected correlation between their GMV and SNS addiction scores.

## Discussion

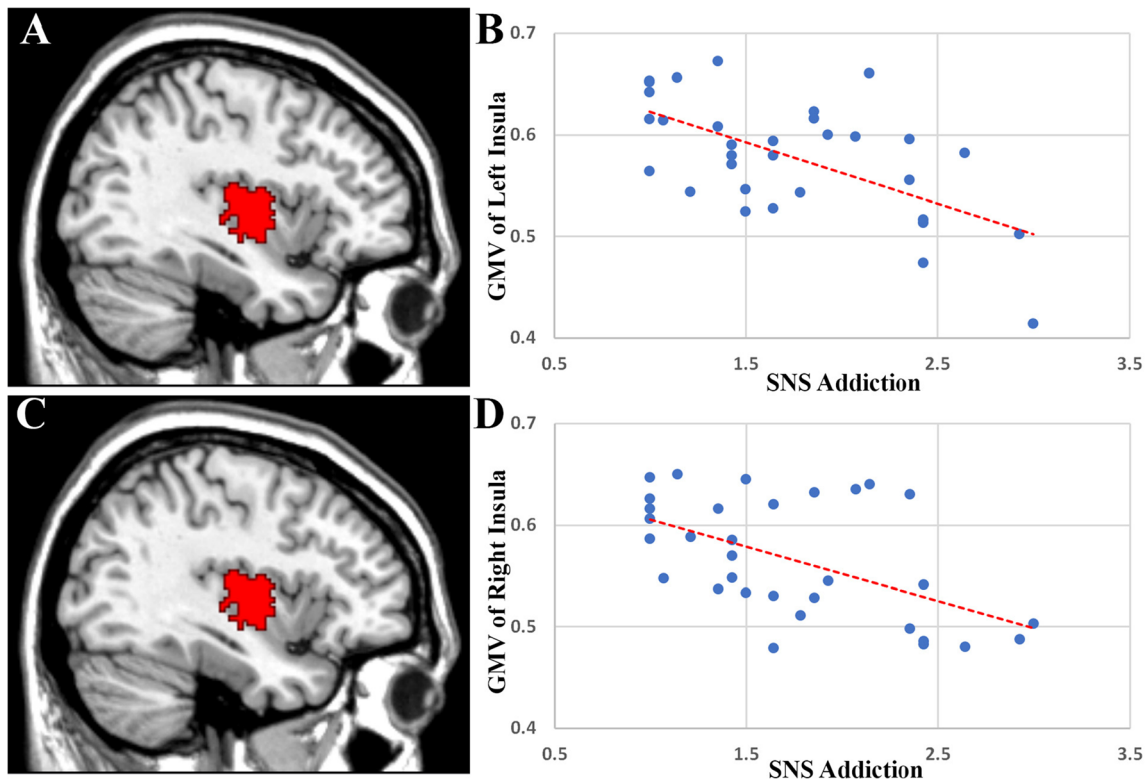
This study provides an integrated brain morphology-behavioral economics view of SNS addiction or excessive SNS use, with a specific emphasis on brain systems underlying interoceptive

awareness, the feeling of urge, and craving, which were implicated in the sustenance of other substance and behavioral addictions. It extends prior research in this area by focusing on "the hidden island of addiction," the insular cortex (Naqvi & Bechara, 2009), and specifically its posterior subdivision, the morphology of which has been shown to be associated with many substance addictions (Wood & Bechara, 2014).

In this respect, the findings add to prior functional and structural imaging studies that point to similarities between technology-related addictions and substance addictions (Turel et al., 2014; Zhou et al., 2011); they provide additional evidence regarding plausible similarities between SNS addiction and other substance and behavioral addictions in terms of the possible roles of the morphology of substrates involved in interoceptive awareness, as well as behavioral impulsivity as measured by delay discounting. The interoceptive awareness brain system is highly important for addiction maintenance, because it promotes subjective feelings of craving that can impair one's inhibition ability and excite his or her reward seeking (Naqvi & Bechara, 2009). Nevertheless, it has not been the focus of research on the family of Internet addictions in general, and specifically SNS addiction. This study partially bridges this gap and extends the understanding of technology-related addictions in several ways.

Relying on neuroanatomical knowledge from the substance and gambling addiction domains (Wood & Bechara, 2014), our first hypothesis (H1) was that the volumes of the bilateral posterior insula will be negatively associated with SNS addiction. The ROI analysis fully supported this assertion; the voxel-wise analysis partially and indirectly (through observed zero-order correlations) supported this hypothesis (only the left PI was significantly negatively correlated with SNS addiction). The observed possible laterality in the whole-brain analysis can be sex-related as our sample included mostly males (only six females, so more robust sex-based difference analyses were not feasible). This is because hemispheric damage to the insula can influence men and women differently; left-insula damage seems to disrupt addiction to smoking in males more than in females, while smoking in females was disrupted by either left or right insula damage (Gaznick, Bechara, & Tranel, 2014). Hence, sex-based differences in insular laterality should be examined in future technology-related addiction research.

Extending the pure neuroanatomical view regarding the role of posterior insula in SNS addiction, this study also sought to examine performance on a behavioral economics task used in addiction research, such as delay discounting, to assess the "shortsightedness" of affected individuals in terms of opting for smaller short-term gains over larger long-term gains. Specifically, we examined how posterior insula morphology is related to the behavioral mechanisms of delay discounting in the domain of SNS addiction. Many studies have revealed posterior insula activation and morphology

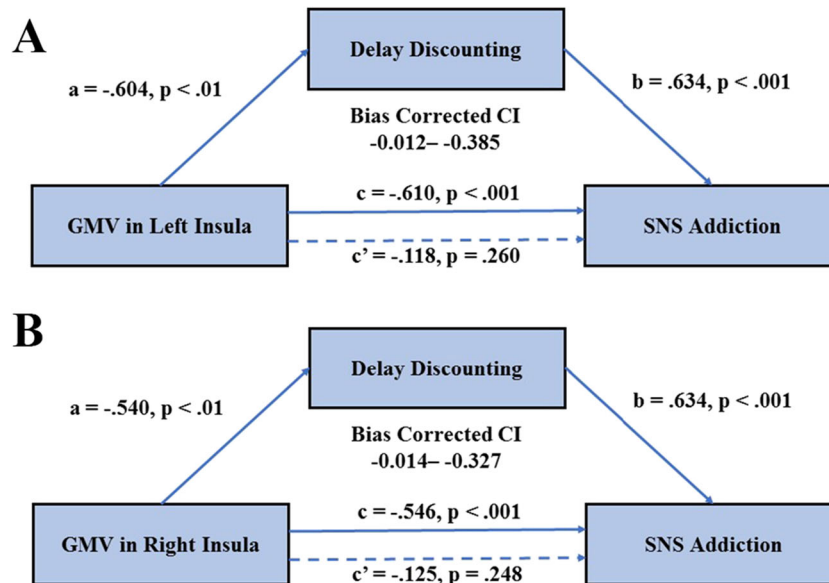


**Fig. 2** Visualized results of the ROI-based analyses. (A) location of ROI-posterior left insula. (B) The GMV of the posterior left insula is negatively correlated with SNS addiction. (C) Location of ROI- posterior right

insula. (D) The GMV of the posterior right insula is negatively correlated with SNS addiction

changes related to steep delay discounting in substance and gambling addictions (Bickel et al., 2012; Hamilton & Potenza, 2012; Mohammadi et al., 2016; Wittmann et al., 2006; Wittmann et al., 2007). We examined whether this would also apply to SNS addiction. H2 that focused on the bilateral

posterior insula volume's negative association with the delay discounting factor  $k$  was supported. H3 that focused on the positive association of the delay discounting factor  $k$  with SNS addiction was also supported. Lastly, H4 that suggested that the delay discounting factor  $k$  at least partially mediates



**Fig. 3** Moderation analyses using ROI-based GMV. (A) For posterior left insula, and (B) for posterior right insula. Given that in both cases  $c'$  is not statistically different from zero, they support full mediation

**Table 2** Summary of voxel-wise (whole brain) VBM results

Brain region	Voxels	Cluster Geometric Center (MNI)			Partial-Correlation with SNS addiction	TFCE corrected, <i>p</i> value (local maxima)
		x	y	z		
Negative correlation between GMV and SNS addiction score						
L Insula (mostly posterior parts)	1451	-47.8	-22.5	14.8	-0.632	<i>p</i> < 0.001
Positive correlation between GMV and SNS addiction score						
NONE						

MNI, Montreal Neurological Institute coordinates; VBM, Voxel-based morphometry; GMV, gray matter volume; TFCE, Threshold-Free Cluster Enhancement; SNS, social network sites; L, left; R, right

the association between the volume of the bilateral posterior insula and SNS addiction was supported as well (with ROI-based data). While we did not test a full mediation model with whole-brain data, the observed whole-brain data correlations with addiction and delay discounting provided preliminary potential support for possible mediation related to only the left posterior insula.

Overall, these findings point to an interesting perspective regarding the possible role of the posterior insula in SNS addiction development and maintenance. Many studies revealed associations between posterior insular cortex morphology and substance addictions, and suggested that abnormal volume of the insula among addicts reflects some deficit in decision making that arouses the impulsive brain system and prevents proper inhibition (see review in Wood & Bechara, 2014). We not only corroborate such views with our findings in the context of SNS, but also point to one specific mediational mechanism that links insular cortex volume and SNS addiction levels. It is possible that through morphological differences in the insula, people's preferences for immediate rewards are increased, and this impulsivity-prone disposition relates to their excessive and problematic use of SNS that can manifest in addiction-like symptoms. Because our findings are SNS specific, future research can extend the generalizability of this view of the insula as both a receiver of interoceptive signals and a center for processing the emotional value of future rewards to other addictive and problematic online and offline (e.g., substance use) behaviors.

Findings regarding the negative correlations between insular cortex volumes, SNS addiction and delay discounting are worthy of elaboration. Most studies thus far have clearly demonstrated a connection between reduced insular cortex volume (or reduced neural activity) and the presence of an addictive disorder (Mackey & Paulus, 2013). This suggests that addicts have a desensitized interoceptive insula system. Yet, brain lesions that include the insula disrupt certain addictions, such as smoking (Naqvi et al., 2014), and disrupt some of the cognitive distortions that draw gamblers to gambling behaviors (Clark et al., 2014). This suggests that the insula system is overactive in these cases of addiction and that insular damage

seems to correct the pathological state of addiction (see Droutman et al., 2015). One way to reconcile this apparent conflict is to consider the possibility that reduced gray matter of the insular cortex (or reduced BOLD activity within the insula in the case of functional neuroimaging studies) does not necessarily mean reduced function. In fact, this volume reduction could reflect a more sensitized (or an overactive) interoceptive insular cortex system. This possibility is especially relevant in light of some of the more recent perspectives arguing that while it is often assumed that a larger cortical volume or greater gray matter density is associated with better computational efficacy of that region (and many examples are consistent with this view), there are cases in which lower cortical volume is associated with better task performance (Kanai & Rees, 2011).

We also can draw a parallel with the neurodevelopmental literature, where age appropriate brain changes in adolescence result in reduction in gray matter (neuronal pruning), and increase in white matter (Durston et al., 2001). Similarly, developmentally appropriate decrease in neural recruitment between children, adolescents and adults, often is associated with increased performance in some domains and is consistently considered a sign of cognitive growth (Casey, Getz, & Galvan, 2008; Casey, Tottenham, Liston, & Durston, 2005). If in the case of the insula, lower recruitment indicates increased efficiency, then this could explain why lesions to the insula would wipe out this sensitized system and disrupt addictive behaviors. This is consistent with the view that as processes convert to be more automatic and habitual, lesser neural activation is needed; for example, there is reduced recruitment of cortical and subcortical systems when skill or performance learning becomes optimal (Babiloni et al., 2010; Gobel et al., 2011; Haslinger et al., 2004).

To elucidate the underlying mechanisms of the insula in normal versus addictive states, future studies should examine the white matter volume, structural connections to/from the insular cortex, and the ratio of white to gray matter. While many studies have shown decreased gray matter in the insula of individuals with substance use disorders, unfortunately there are no studies that have attempted to look at an increase



or decrease in the white matter associated with the insula itself. Also studies that address structural connectivity of the insula are currently lacking. Perhaps such studies should be conducted in the future to determine whether individuals with addictive behaviors develop increased insular white matter, which could reflect an enhancement in function and automaticity, despite an observed decrease in gray matter. Similarly, functional connectivity of the insula should be examined using diffusion tensor imaging (DTI) approaches. Understanding whether reduced gray matter volume of the insula means an overactive or an inactive insula is highly significant when considering strategies to correct addictive behaviors. It would be very important to know whether the correction of a behavioral addiction would require a boost or reduction of the neuronal function within the insula (Droutman et al., 2015).

This study is not without limitations. First, while our sample size seems to be sufficiently powered for the limited set of hypotheses we developed, it may still produce both Type I and Type II errors. Hence, our findings should be treated as preliminary and caution should be exercised when interpreting the results. Second, this study was conducted with a single SNS and a North American population. The model should be replicated with other SNS, technologies, and populations to increase its generalizability. Third, this study was correlational in nature and cannot support perfect causality arguments. Future research may employ longitudinal designs to be able to better establish causality. Fourth, even though participants with relevant psychiatric diagnosis were not found, it is possible that subclinical levels of psychiatric disorders could confound the results. Future research should include more potential confounds. Fifth, we used a monetary-choice delayed discounting task. Even though such tasks are good at predicting addictive behaviors (Amlung et al., 2017), future research may include tasks that involve social-media-specific choices.

Sixth, our whole-brain analysis did not find structural changes in regions that were implicated in prior social media addiction research, including the reward system (He, Turel, & Bechara, 2017a; He, Turel, Brevers, et al., 2017b; Montag et al., 2017), and anterior cingulate volumes (He, Turel, & Bechara, 2017a). These discrepancies may have several explanations but still point to a consistent picture regarding SNS addiction. Because the insular system can be considered a part of the reward system and it feeds into it (i.e., impulsivity and increased striatal (dopamine) output is exacerbated by inputs from the insula; see Noel et al., 2013), prior research and our work are consistent in showing that structural differences in components of the broader reward system are associated with SNS addiction. The difference in broader reward system components observed here versus in prior research can have several explanations. First, it is possible that different people (and samples) have different underlying brain morphology, as reflected in changes in different components of the reward

system and the systems that feed into it. Second, it also is possible that changes in specific regions depend on factors, such as length of addiction, sex and age, which may differ between samples. Hence, different samples may have varying degrees of amenability to changes in the morphology of the target brain regions.

Future research should therefore account for such factors, understand under what condition each one of the reward system region changes, what changes are more reliable in explaining addiction, and corroborate our findings as well as findings from prior research regarding brain structure abnormalities and SNS addiction. Lastly, while we associate gray matter volumes of bilateral posterior insula with one's level of SNS addiction, the techniques we use do not allow us to examine the different components of gray matter, such as neuronal and glial cells, axon terminals, and dendrites. Future research can use more microscopic techniques to examine how gray matter composition is associated with the examined variables and processes.

## Conclusions

Similar to its role in substance addictions, the insular cortex and specifically its posterior subdivision is also an important substrate in SNS addiction or excessive SNS use maintenance. Differences in its morphology, specifically reduced gray-matter volume, are associated with more frequent and disturbing SNS addiction symptoms. This association is partially mediated by a steep delay discounting performance, which manifests in SNS use behaviors that focus on immediate gains (e.g., dopamine release due to "likes"; Meshi, Morawetz, & Heekeren, 2013) and improperly weighing of future consequences (e.g., dying in a car accident due to the use of SNS while driving; Turel and Bechara, 2016).

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