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EXAMINATION OF NEURAL SYSTEMS SUB-SERVING FACEBOOK "ADDICTION"[1, 2]

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Summary.—Because addictive behaviors typically result from violated homeostasis of the impulsive (amygdala-striatal) and inhibitory (prefrontal cortex) brain systems, this study examined whether these systems sub-serve a specific case of technology-related addiction, namely Facebook "addiction." Using a go/no-go paradigm in functional MRI settings, the study examined how these brain systems in 20 Facebook users (M age = 20.3 yr., SD = 1.3, range = 18–23) who completed a Facebook addiction questionnaire, responded to Facebook and less potent (traffic sign) stimuli. The findings indicated that at least at the examined levels of addiction-like symptoms, technology-related "addictions" share some neural features with substance and gambling addictions, but more importantly they also differ from such addictions in their brain etiology and possibly pathogenesis, as related to abnormal functioning of the inhibitory-control brain system.

While the Internet is largely beneficial to society, it can also bring about negative consequences (D'Arcy, Gupta, Tarafdar, & Turel, 2014), including behavioral and psychological signs that have been labeled by some researchers as "addiction"[3] to the use of specific applications on the Internet (Griffiths, 1998; Young, 1998a; Griffiths, 1999; Young, 2004; Turel,

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[3]The authors use quotation marks to indicate that the appropriateness of the term "addiction" to the described cases is still being examined.

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Serenko, & Giles, 2011). Such "addictions" can result in academic failure, sleep deprivation, social isolation, health issues, and many other impairments for adolescents and young adults; they also result in reduced work performance and marital discord and separation for adults (cf. Griffiths, 1995; Young, 1998b; Pratarelli, Browne, & Johnson, 1999; Chou, Condron, & Belland, 2005; Block, 2008; Byun, Ruffini, Mills, Douglas, Niang, Stepchenkova, *et al*., 2009; Young, 2010; Kuss, Griffiths, & Binder, 2013). It is therefore worthwhile to examine the possible neural basis of such "addictions."

Research across multiple countries, including the United States, estimates the prevalence of such "addictions" to be between 0.7% and 11% (Greenfield, 1999; Johansson & Götestam, 2004; Kim, Ryu, Chon, Yeun, Choi, Seo, *et al*., 2006; Cao & Su, 2007; Rendi, Szabo, & Szabó, 2007; Ghassemzadeh, Shahraray, & Moradi, 2008; Park, Kim, & Cho, 2008; Shaw & Black, 2008; Siomos, Dafouli, Braimiotis, Mouzas, & Angelopoulos, 2008; Bakken, Wenzel, Götestam, Johansson, & Oeren, 2009),[4] and that it is more prevalent among youth and young adults (Kuss, *et al*., 2013), presumably because the inhibitory system of such individuals develops more slowly than their impulsive system (Casey, Giedd, & Thomas, 2000; Casey, Tottenham, Liston, & Durston, 2005; Steinberg, 2005; Casey, Getz, & Galvan, 2008; Steinberg, 2008; Steinberg, Graham, O'Brien, Woolard, Cauffman, & Banich, 2009). Given the symptoms and prevalence of this phenomenon, calls have been issued to study its possible neurological roots (Block, 2008) and to focus on "addiction" to specific, intrinsically rewarding applications on the Internet (e.g., Facebook, videogames) (Yellowlees & Marks, 2007). Consequently, the concept of "Internet Gaming Disorder" was included in the Appendix (section 3, potential disorders requiring further research) of the DSM–V, and it is possible that more application-specific "addictions" will be considered for inclusion in future versions of the DSM. Moreover, several scales for measuring such "addictions" have been developed (van Rooij, Schoenmakers, Vermulst, van den Eijnden, & van de Mheen, 2011; Andreassen, Torsheim, Brunborg, & Pallesen, 2012). Nevertheless, the DSM is not conclusive on the existence of this possible disorder, and many researchers also still question whether the observed phenomenon reflects a pathological "addictive" state or merely a "bad habit," especially when applied to the vast general population of users who show addiction-like symptoms in relation to Internet application use (Griffiths, 1998, 1999; LaRose, 2010; Bergmark, Bergmark, & Findahl, 2011).

[4]This is a wide range and it can be assumed that it is a consequence of multiple factors, including the type of Internet application examined (e.g., Facebook, videogames), demographics and socio-economic differences between the samples, and national differences in accessibility to technologies and the availability of alternative activities.

This study attempts to address one aspect of this issue by focusing on a possibly "addictive" Internet technology, namely Facebook. Several studies have demonstrated that Facebook "addiction" is a plausible phenomenon and that addiction-like symptoms in relation to Facebook use may be prevalent in the general population (Echeburua & de Corral, 2010; Karaiskos, Tzavellas, Balta, & Paparrigopoulos, 2010; Kuss & Griffiths, 2011; Griffiths, 2012). These behaviors are usually labeled as "addictive" based on DSM criteria for dependence on substances, including tolerance, withdrawal, and loss of control to the point that the behavior causes a significant impairment to the individual (World Health Organization, 1992; American Psychiatric Association, 2000). Perhaps many Facebook users may be labeled as "addicts" simply because they easily meet several of these criteria, especially when the definition of "significant impairment" is subjective and variable. The following research question is therefore posed:

Research question 1. Does Facebook "addiction" constitute a pathological problem similar to those observed in the case of other substance and behavioral addictions, in the general user population?

One objective way to identify similarities or fundamental differences between Facebook (Internet) and other addictions is to look at the neural systems sub-serving these possible disorders. Thus, one goal of this study was to examine neural activities in two key brain systems implicated in substance addiction, the impulsive, amygdala-striatal system and the reflective-inhibitory prefrontal brain system (e.g., Jentsch & Taylor, 1999; Volkow & Fowler, 2000; Arnsten & Li, 2005; Bickel, Miller, Yi, Kowal, Lindquist, & Pitcock, 2007) when Facebook users are exposed to Facebook cues. The amygdala-striatal (mesolimbic dopamine-dependent) neural system is critical for the incentive motivational effects of a variety of rewards (Stewart, Dewit, & Eikelboom, 1984; Robbins, Cador, Taylor, & Everitt, 1989; Wise & Rompre, 1989; Robinson & Berridge, 1993; Di Chiara, Tanda, Bassareo, Pontieri, Acquas, Fenu, *et al*., 1999; Everitt, Parkinson, Olmstead, Arroyo, Robledo, & Robbins, 1999; Balleine & Dickinson, 2000; Koob & Le Moal, 2001). It becomes hyperactive and begins to intensify the incentive value of rewards in individuals with substance abuse problems (Bechara, 2005). As cue-behavior-reward associations are strengthened, they begin to drive behavior without the necessary involvement of conscious processes (Everitt, *et al*., 1999; Robinson & Berridge, 2003; Everitt & Robbins, 2005). Because Facebook use can provide strong rewards (Turel & Serenko, 2012; Meshi, Morawetz, & Heekeren, 2013),[5] it is expected that

[5]The terms "rewards" and "incentives" are used interchangeably. However, please note that rewards are one form of incentives, and other incentives can include avoiding negative consequences.

similar learning mechanisms take place with Facebook use, which can lead to "addiction"-like symptoms.

> *Hypothesis 1*. If Facebook "addiction" is sub-served by similar pathological issues underlying other addictions, the impulsive amygdala-striatal system activity in response to Facebook stimuli will be positively correlated with "addiction"-like symptoms.

While the amygdala-striatal system provides the drive for impulsive behaviors, diagnosis of addictions typically also requires poor control abilities that fail to inhibit impulsive behaviors and the consideration of long-term goals (Noel, Brevers, & Bechara, 2013). This inhibitory system depends primarily on the functions of the prefrontal cortex (Fellows, 2004; Wheeler & Fellows, 2008). A critical neural region in this system is the ventromedial prefrontal cortex (which is considered inclusive of the medial orbitofrontal cortex; Bechara, Tranel, & Damasio, 2000). Other important regions in the inhibitory system include the lateral orbitofrontal and inferior frontal gyrus regions, as well as the anterior cingulate cortex, which are involved in a variety of simple inhibitory processes (Glascher, Adolphs, Damasio, Bechara, Rudrauf, Calamia, *et al*., 2012). Good inhibitory functioning reflects the ability to actively stop a pre-potent behavioral response after it has been triggered (Logan, Schachar, & Tannock, 1997; Braver & Ruge, 2006). Inhibitory processes are activated primarily by antecedent cues (e.g., Wood & Neal, 2007), and inhibition is therefore especially relevant in the face of these cues. Individuals with hypoactivity of these systems have a tendency to act more impulsively (Bechara, 2005).

> *Hypothesis 2*. If Facebook "addiction" is sub-served by similar pathological issues underlying other addictions, the reflective-inhibitory prefrontal system activity in response to Facebook stimuli will be negatively correlated with "addiction"-like symptoms.

Method

Participants

The sample was recruited in two phases. The first involved an online questionnaire which captured demographic, exclusion criteria (self-reported neurological or psychiatric history as well as uncorrected vision), and "addiction" variables. It was administered to 45 Facebook users who were recruited using an announcement on a bulletin board at a North American university, and who were given a small gift card in exchange for their time. None of the users who completed this questionnaire met any of the exclusion criteria. In the second phase, twenty participants who

completed the screening survey were invited and agreed to participate in the fMRI scan. The selection was made such that the sexes are balanced, and that there is sufficient variability in addiction scores. The sample was equally distributed between men and women, and the average age of the participants was 20.3 yr. ($SD = 1.30$, range = 18–23). The participants had normal or corrected-to-normal vision, and were free of neurological or psychiatric history (self-reported). All participants gave informed consent to the experimental procedure, which was approved by the University of Southern California Institutional Review Board.

Measures

Participants were asked to complete an online version of the Facebook "addiction" scale (adapted from van Rooij, *et al*., 2011). This scale asked them to report the frequency (1: Never, 5: Very Often) of typical Facebook "addiction" symptoms such as withdrawal, salience, relapse, loss of control, and conflict. It therefore presumably captures the "level of 'addiction'" and was valid and reliable ($\alpha = .92$, Spearman-Brown Coefficient for split-half reliability = 0.91, Guttman split-half coefficient = 0.91, Average Variance Extracted (AVE) = 0.53, and composite reliability = 0.91). Given its validity and reliability, the mean of all items was calculated, which represents the average severity of the addiction symptoms per individual. The mean score was 2.20 ($SD = 0.72$, range = 1.07–3.64), and the scores seemed to be reasonably normally distributed (skewness = 0.32, $SE = 0.51$; kurtosis = –0.59, $SE = 0.99$); the Kolmogorov-Smirnov test (statistic = 0.097, $df = 20$, $p = .20$) and the Shapiro-Wilks test (statistic = 0.97, $df = 20$, $p = .81$) were non-significant. Hence, no transformations to normality were applied. The behavioral questionnaire also captured age, sex, mental history, and exclusion criteria (e.g., non-corrected bad vision and any peripheral neuropathies). No mental issues, including drug and alcohol abuse were reported by the sample; no participant met any of the exclusion criteria.

fMRI Procedures and Tasks

fMRI scans were performed one week after the completion of the behavioral questionnaire. In these scans, participants rested in the supine position on the fMRI scanner bed to view the task back-projected onto a screen through a mirror attached to the head coil. Foam pads were used to minimize head motion. Stimulus presentation and timing of all stimuli and response events were achieved using Matlab (Mathworks) and Psychtoolbox (www.psychtoolbox.org) on an IBM-compatible PC. The participants' responses were collected online using an MRI-compatible button box.

The participants performed two Facebook-specific go/no-go tasks while in the scanner: (1) a sign go/Facebook no-go task (SGo task) in which they

FIG. 1. The illustration of the event-related Facebook-specific go/no-go tasks: (1) sign go/Facebook no-go task (SGo task), and (2) Facebook go/sign no-go task (FGo task). Participants were asked to press a button as soon as possible in the go trials (traffic sign pictures in SGo task and Facebook-related pictures in FGo task) and withhold the response in the no-go trials (Facebook-related pictures in SGo task and traffic sign pictures in FGo task). The order of tasks was counterbalanced across subjects and across sessions.

were asked to press a button when they saw a traffic sign image, and refrain from pressing the button when they saw a Facebook-related image; and (2) a Facebook go/sign no-go task (FGo task) in which they were asked to press a button when they saw a Facebook-related image, and refrain from pressing the button when they saw a traffic sign image. This go/no-go paradigm allows examination of both the brain responses to Facebook stimuli and the inhibition of pre-potent responses to Facebook stimuli. Examples of stimuli are shown in Fig. 1. Traffic signs included common (excluding red) signs.

Each task consisted of 120 go trials (75%) and 40 no-go trials (25%). No-go trials were presented in pseudo-randomized order, designed so that no-go trials appeared with equal probability after 1–5 consecutive go trials, and no two no-go trials appeared consecutively. Each stimulus was presented for 500 msec., followed by a fixation cross for 1.5–4 sec. with a mean of 2.5 sec. The sequence was optimized for design efficiency using an in-house program. Each task ran for 8 min. The order of the two versions of go/no-go tasks was counterbalanced across participants.

Following signal detection theory, the hit rate, false alarm rate, sensitivity index

$$d' = Z_{\text{Hitsrate}} - Z_{\text{false alaram rate'}},$$

and decision bias

$$C = -0.5 \times \left(Z_{\text{Hits rate}} + Z_{\text{false alaram rate}} \right)$$

were calculated for each task (Macmillan & Creelman, 1996). The mean reaction time for go trials and no-go trials (false alarm trials only) for each task were also calculated. The reaction time for go trials served as an index for habitual-impulsive responding to the stimuli, with longer reaction times indicating less habitual response, while decision bias *C* served as an index of response inhibition, with higher values indicating better inhibitory control.

fMRI Protocol

Functional MRI (fMRI) imaging was conducted in a 3T Siemens MAGNETOM Tim/Trio scanner. Functional scanning used a z-shim gradient echo EPI sequence with PACE. This sequence was aimed at reducing signal loss in the prefrontal and orbitofrontal areas. The parameters were: TR/TE = 2000/25 msec.; flip angle = 90°; 64 × 64 matrix size with resolution $3 \times 3\,mm^2$. Thirty-one 3.5-mm axial slices were used to cover the whole cerebral cortex and most of the cerebellum with no gap. The slices were tilted about 30° clockwise along the AC-PC plane to obtain better signals in the orbitofrontal cortex. An anatomical T1-weighted structural scan was also done (TR/TE = 1950/2.26 msec.; flip angle = 7°; 176 sagittal slices; spatial resolution = 1 × 1 × 1.95 mm) for registration purposes.

fMRI Analysis

Image preprocessing and statistical analyses were carried out using FSL (www.fmrib.ox.ac.uk/fsl). Images were realigned to compensate for small residual head movements (Jenkinson & Smith, 2001). Translational movement parameters never exceeded one voxel in any direction for any participant. Data were spatially smoothed using a 5-mm full-width-half-maximum (FWHM) Gaussian kernel and were filtered using a nonlinear high pass filter with a 100-second cutoff.

A two-step registration procedure was used whereby EPI images were first registered to the MPRAGE structural image, and then into standard MNI space, using affine transformations (Jenkinson & Smith, 2001). Registration from MPRAGE structural image to standard space was further refined using FNIRT nonlinear registration (Andersson, Jenkinson, & Smith, 2007a, 2007b). Statistical analyses were performed in the native image space, with the statistical maps normalized to the standard space prior to higher-level analyses. The data were modeled at the first-level using a general linear model within FSL's FILM module. Brain activation in every trial was modeled for go and no-go trials, respectively, at the single-participant level. Error-related trials (misses and false alarms) were modeled together as a nuisance variable. The event onsets were convolved with canonical hemodynamic response function (HRF, double-gamma) to generate regressors. Temporal derivatives were included as covariates of

no interest to improve statistical sensitivity. Null events were not explicitly modeled, and therefore constituted an implicit baseline. The six movement parameters were also included as covariates in the model.

A higher-level analysis created cross-run contrasts for each participant, using a fixed-effect model. A 2 task (go vs no-go) × 2 stimuli (traffic sign vs Facebook pictures) within-subjects factor design was used. The main effects and interaction were modeled as well as 4 single-condition effects (Facebook go; Facebook no-go; traffic sign go; traffic sign no-go). Higher-level random-effects models were tested for group analyses using FMRIB's Local Analysis of Mixed Effect stage 1 only (Beckmann, Jenkinson, & Smith, 2003; Woolrich, Behrens, Beckmann, Jenkinson, & Smith, 2004) with automatic outlier detection (Woolrich, 2008). The brain activation associated with each contrast was first tested in all participants using one-sample *t* tests. Then, the brain activation was correlated with the addiction score. Group images were thresholded with a height threshold of $Z > 2.3$ and a cluster probability of $p < .05$, corrected for whole-brain multiple comparisons based on a Gaussian random field theory. The sex of participants was included as a covariate for all fMRI analyses.

Regions of interest (ROI) were used to show the direction of the activation and in correlation analyses and scatterplots. ROIs were created from clusters of voxels with significant activation in the voxel-wise analysis. Analyses were performed by extracting parameter estimates (betas) for each event type from the fitted model, and averaging them across all voxels in the cluster for each participant and session. Percent signal changes were calculated using a method suggested by Mumford.[6]

Results

Behavioral Results

There was no significant correlation between "addiction" score and age ($r = -.20$, $p = .40$), but the "addiction" score was significantly correlated with sex ($r = .45$, $p = .05$). This sex difference was also supported by a *t* test ($t_{18} = -2.12$, $p < .05$, Cohen's $d = 0.96$), and variance was reasonably equal (Levene's test $F = 1.03$, $p < .32$). This implies that at least in the sample, women (coded as 1) presented stronger addiction-like symptoms with regards to Facebook use (female $M = 2.52$) than did men (male $M = 1.89$). There was no significant correlation between "addiction" score and other behavioral measures of the go/no-go tasks (all with $p > .05$).

Table 1 summarizes the major behavioral measures for both fMRI tasks, including hit rates, false alarm rates, sensitivity index *d'*, decision bias *C*, and reaction times for go trials and no-go trials (false alarm rates

[6]http://mumford.fmripower.org/perchange_guide.pdf

TABLE 1
BEHAVIORAL MEASURES FROM THE FACEBOOK-SPECIFIC GO/NO-GO TASK

Variable	SGo Task		FGo Task		t	p	Cohen's d
	M	*SD*	*M*	*SD*			
Hits rate	0.90	0.11	0.90	0.12	−0.02	.98	−.009
False alarm rate	0.13	0.13	0.06	0.05	2.42	.03	1.08
Go trial response time (msec.)	522.3	86.7	487.3	78.2	44.2	.002	1.66
No-go trial response time (msec.)	433.2	50.7	389.0	44.2	2.53	.03	1.13
d'	2.85	0.54	3.04	0.61	−1.10	.29	−0.49
C	−0.31	0.46	−0.07	0.27	−1.70	.12	−0.76

Note.—SGo = a sign go/Facebook no-go task; FGo = a Facebook go/sign no-go task. *$p < .05$ corrected for multiple comparison with a Bonferroni correction.

for inhibitory failures only). For every behavioral measure, a paired *t* test was performed to test the difference between tasks (SGo vs FGo task). In all cases Levene's test statistics were non-significant (all $p > .10$), indicating that equal variance can be assumed. Analysis revealed that the average reaction time for go trials was significantly longer in the SGo task than in the FGo task ($p < .05$ corrected). Analysis also revealed differences in the false alarm rate and reaction time for no-go trials between the two tasks, but they were no longer significant after multiple comparison correction (Table 1).

fMRI Results

The fMRI analyses were used in a confirmatory manner. First, it was examined whether the amygdala-striatal system was engaged in the Facebook-go trials. As showed in Table 2 and Fig. 2, this pattern was supported because the Facebook-go trials activated a large sector of the amygdala-striatum system along with other brain regions, including the occipital cortex, parietal cortex, and precentral gyrus/insula. Next, in order to test for possible hyperactivity of the amygdala-striatal system as a function of one's "addiction" score (Hypothesis 1), the "addiction" scores were correlated with the brain activity in Facebook-go trials. The results revealed that bilateral ventral striatum activity in Facebook-go trials was correlated positively with the "addiction" score (Fig. 3). Hypothesis 1 was further supported by the results in Table 1, which demonstrate that on average there are significantly more false alarms in the case of sign-go (i.e., Facebook-no-go) tasks, and that response times were shorter in Facebook-go trials.

To test Hypothesis 2, it was first examined whether the inhibitory control system (especially the ACC and dorsolateral prefrontal cortex) was engaged in Facebook-no-go trials. Table 2 and Fig. 2 lend support to such an effect, with activation of the ACC, right DLPFC/insula, and bilateral

TABLE 2
SUMMARY OF FMRI RESULTS

Brain Region	Voxels	MNI Coordinates			*Z*
		x	y	z	
Brain activity of Facebook-go trials					
B Occipital cortex/Amygdala/Striatum	31,860	30	−62	−18	6.41
L Insula	1,596	−50	4	2	3.60
L Postcentral cortex	968	−62	−16	46	4.28
Brain activity of Facebook-no-go trials					
B Occipital/ Parietal/ Temporal cortices	31,386	−40	−80	−14	5.94
R DLPFC	3,239	50	10	42	4.03
Cingulate cortex	781	4	12	48	3.16
Whole brain correlation between brain activity in Facebook-go trials and addiction score (Positive)					
Bilateral ventral striatum	1,021	−20	14	−8	3.67
Whole brain correlation between brain activity in Facebook-no-go trials and addiction score					
No significant correlations were observed					

occipital/parietal cortex. Furthermore, the prefrontal activation in Facebook-no-go trials was compared with this of traffic sign-no-go trials. No significant differences were found. This suggests that in both conditions participants engaged in similar levels of inhibition.

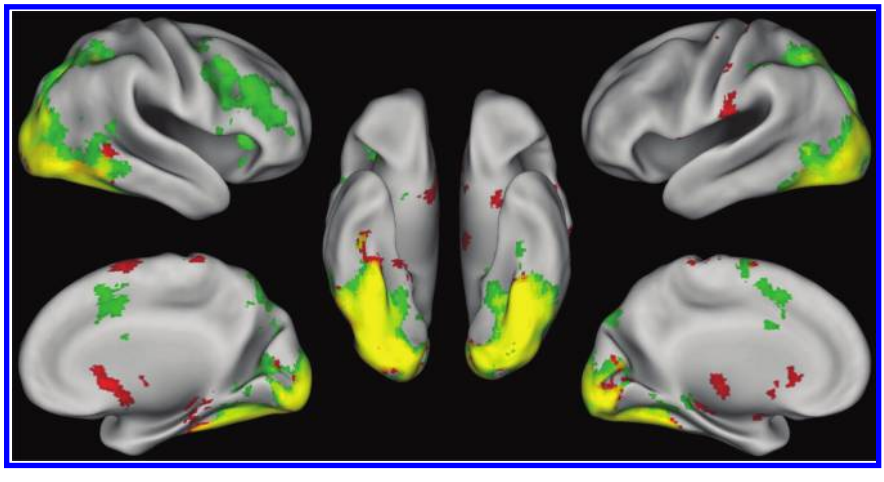

FIG. 2. Activation of Facebook-go (in red) and Facebook-no-go (in green) trials. Yellow areas indicate common activation for go and no-go trials.

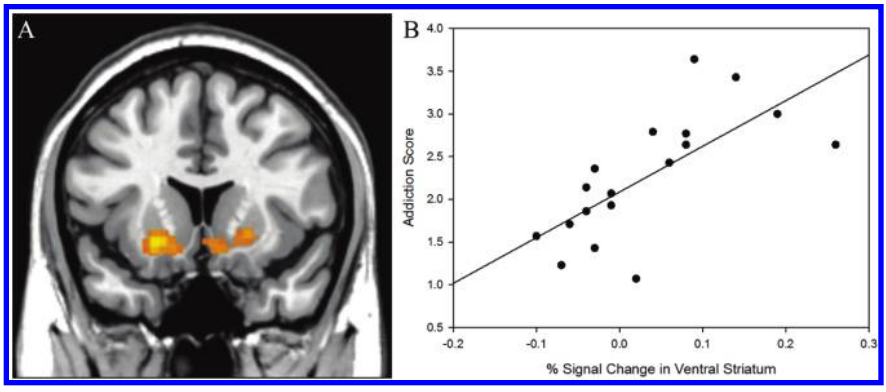

FIG. 3. The ventral striatum signal showed positive correlation with the addiction score in Facebook go trials. (A) Coronal image shows the ventral striatum signal. (B) Scatter plot shows the correlation pattern.

Next, the authors tested whether the activity of the inhibitory control neural systems, as a manifestation of inhibition attempts in response to Facebook cues, was associated negatively with "addiction" scores. That is, the "addiction" scores of the participants were correlated with the brain activity in Facebook-no-go trials. The results indicated no significant association between any component of the inhibition system (ventromedial prefrontal cortex, lateral orbitofrontal, and inferior frontal gyrus regions, and anterior cingulate cortex) and "addiction" scores (all with $p > .05$).

DISCUSSION

While the activation of the amygdala-striatal (impulsive) brain system was positively associated with one's Facebook "addiction" score (i.e., the level of addiction-like symptoms presented), there was no association between this score and activation of the prefrontal cortex (inhibition) brain system. The findings, therefore, suggested that at least individuals with low to medium levels of addiction-like symptoms have a hyperactive amygdala-striatal system, which makes this "addiction" similar to many other addictions, but they do not have a hypoactive prefrontal lobe inhibition system, which makes it different from many other addictions, such as to illicit substances. Hence, technology "addictions" may not present the exact same brain etiology and possibly pathogenesis that drives substance and gambling addictions. The detected hyperactivity of the impulsive brain system supplements and confirms findings of other studies which discovered similarities between brain systems sub-serving technology-related addictions and other addictions (Ko, Liu, Hsiao, Yen, Yang, Lin, *et al*., 2009; Han, Hwang, & Renshaw, 2010; Han, Kim, Lee, Min, & Renshaw, 2010; Han, Bolo, Daniels, Arenella, Lyoo, & Renshaw, 2011; Han, Kim, Lee, & Renshaw, 2012; Han, Lyoo, & Renshaw, 2012;

Ko, Liu, Yen, Yen, Chen, & Lin, 2013; Ko, Liu, Yen, Chen, Yen, & Chen, 2013). However, the findings regarding the frontal lobe inhibition system also point to possible dissimilarities between substance and gambling addictions, and Facebook "addiction," which exist at least at the examined levels of addiction symptoms.

Several implications of these findings should be noted. First, studies on technology-related "addictions" indicate that many individuals present at least some (and in some cases many) addiction-like symptoms with low-medium frequency and intensity (and in some cases high) in relation to the use of presumably addictive technologies (La Barbera, La Paglia, & Valsavoia, 2009; Turel & Serenko, 2012). This has raised public and scientific awareness of this potential problem (Block, 2008; Byun, *et al*., 2009), and has resulted in the inclusion of Internet Gaming Disorder as a "condition for further study" in DSM–V (American Psychiatric Association, 2013).

Without discounting the existence and importance of this problem and its possible adverse consequences, one may question (1) whether the term "addiction" is the most appropriate one for this problem (LaRose, Lin, & Eastin, 2003; Yellowlees & Marks, 2007; Turel, *et al*., 2011; Kuss & Griffiths, 2011); and (2) whether, at least when applied to the general population of users, commonly used addiction scales, which include relatively easy-to-meet criteria and benign symptoms (LaRose, *et al*., 2003; LaRose, 2010), actually capture "addiction" or merely capture symptoms emerging from a strong bad habit of implicit and automatic high engagement with a technology (Charlton & Danforth, 2007; Yellowlees & Marks, 2007; Turel & Serenko, 2012). The findings of this study partially addressed these questions and implied that at least at low-medium levels of addiction-like symptoms, the observed symptoms are associated with some brain changes (sensitization of the amygdala-striatal system), but not with changes in all key brain systems associated with substance addictions (especially the prefrontal cortex). In this sense, it adheres to calls by researchers (Block, 2008) and the DSM–V (American Psychiatric Association, 2013) to further examine the pathology of technology-related addictions.

Second, the findings lend support to past research pointing to the importance of the amygdala-striatal system in addiction pathology (Everitt, *et al*., 1999; Jentsch & Taylor, 1999; Volkow & Fowler, 2000; Everitt & Robbins, 2005; Koob & Volkow, 2010). In this study, this system responded to Facebook cues, produced more false alarms in sign-go (Facebook inhibition) tasks, and resulted in shorter response times in Facebook-go trials (compared to the response times with regards to neutral signs). Furthermore, the activation of this system was positively and significantly correlated with the "addiction" scores. Thus, these symptoms, at least in part, manifest from automatically and easily retrieved implicit associations and

the consequent hyperactivity in the bilateral ventral striatum (Everitt, *et al*., 1999). In this respect, Facebook "addiction" is similar to substance and gambling addictions.

It was also hypothesized that the level of addiction-like symptoms in relation to Facebook use would be negatively associated with activation of prefrontal inhibition brain structures, including the ventromedial prefrontal cortex, lateral orbitofrontal, and inferior frontal gyrus regions, and the anterior cingulate cortex (Di Chiara, 2000; Goldstein & Volkow, 2002; Volkow, Fowler, Wang, & Swanson, 2004). These results were consistent with previous reports that no-go trials with various stimuli activate the inhibitory control system (Menon, Adleman, White, Glover, & Reiss, 2001; Garavan, Ross, Murphy, Roche, & Stein, 2002), but also suggested that participants, regardless of their level of addiction-like symptoms, presented normal functioning of the inhibition system (i.e., no significant hypo-activity was detected). That is, no impairment of the inhibition system was observed.

These findings imply that technology-related "addictions," at least at lower-to-medium levels of addiction-like symptoms, differ from other addictions, e.g., to illicit substances, on at least one dimension. While gambling and substance addictions often involve the impairment of both the impulse and inhibition brain systems (Noel, *et al*., 2013), technology-related "addictions," at least at the examined levels of addiction-like symptoms, involve only changes to the amygdala-striatal system. Perhaps this is a result of differences between the adverse consequences in the case of technology-related "addictions" (e.g., missing school) and those in the case of other addictions (e.g., major health risks and troubles with the law), the latter of which are likely to be more severe. While substance addicts often respond to substance-related cues without reflection, and have weak abilities to inhibit or exert cognitive control over their behaviors, it seems that technology-related "addicts" respond to Facebook cues in a similar way, but have the capacity to inhibit such behaviors. Given the abovementioned possible differences between the adverse consequences of substance and technology use, users of applications such as Facebook perhaps lack the motivation to engage the prefrontal brain system (Shuster & Toplak, 2009). This proposition, however, warrants further research.

Lastly, the findings point to potential practical implications. They imply that individuals who present low-medium levels of addiction-like symptoms in relation to Facebook have an imbalance between their amygdala-striatal and prefrontal cortex systems. Thus, their problematic use of Facebook (i.e., use that results in at least some addiction-like symptoms) can be overcome by restoring the homeostasis between these two systems. This could be achieved by cognitive behavioral therapy (CBT). Indeed, several attempts to apply CBT in technology-related addiction cases have been reported to be success-

ful (Young, 2007; van Rooij, Zinn, Schoenmakers, & van de Mheen, 2012), and can perhaps also help with Facebook "addiction."

Limitations and Future Research

Several limitations of this study that point to future research should be acknowledged. First, the sample included educated young adults residing in one country. Given possible age-, socio-economic-, and nation-based differences in technology "addictions," future research may extend the generalizability of the findings to other populations, and in line with the ecological model include a range of ecological risk factors, beyond the individual, in the model (Catala-Minana, Lila, & Oliver, 2013; Raynor, 2013; Sterk, Elifson, & DePadilla, 2014). It can perhaps also focus on additional possible consequences of such "addictions," e.g., obesity. Second, the sample was limited in the range of addiction-like symptoms it presented. Because the sample included participants with low-medium levels of addiction-like symptoms, future research could examine those few extreme users with very high addiction scores to find if there is a point of inflection after which prefrontal cortex impairments might be observable. This study was correlational in nature, and hence caution regarding causality arguments should be exercised. Addiction co-morbidity did not exist in the current sample. Nevertheless, longitudinal studies can be executed to see if prefrontal cortex weaknesses in these few extreme users progress into not only Internet "addiction," but also into other addictive behaviors. Since Internet use starts at a very early age, typically before any exposure to addictive substances, this would help address an important theoretical question in addiction research on whether brain abnormalities precede substance abuse, or whether these abnormalities are actually the consequences of substance abuse (Ersche, Jones, Williams, Turton, Robbins, & Bullmore, 2012).

While the authors have acknowledged the yet-unknown appropriateness of the term "addiction" when applied to Facebook by using quotation marks, more research on similarities and differences between Facebook "addiction" and substance and gambling addictions should be conducted. Noteworthy is the fact that this study focused on "addiction" to an activity that is legal and has socially acceptable symptoms, or at least symptoms not judged harshly by society. In contrast, addictions to substances or gambling can have much more severe consequences. Perhaps these differences in societal view of such addictions and possible differences in the severity of the consequences act as inhibition (de)motivators, and consequently people may lack the motivation to inhibit their Facebook use, rather than having impaired inhibition systems. This proposition, however, merits further research. Similarly, Facebook "addiction" may not be a cause but a mediator or moderator of some other facet of experience. It could also serve

as a gateway for developing other addictions (i.e., when prefrontal cortex changes are observed), at which point early interventions could help avert more serious addictions. This idea, too, merits further research. Lastly, this study points to the possibility that some interventions, such as CBT or trans-cranial magnetic stimulation (Hallett, 2000), may be efficacious in treating Facebook "addiction." However, more research on the efficacy of these therapeutic strategies to deal with such "addictions" is needed.

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