# Directing Technology Addiction Research in Information Systems: Part I. Understanding Behavioral Addictions

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## Abstract

Technology-related addictions have become common in many societies. Consequently, IS research has started examining such issues. In these embryonic stages of research, this line of work has already shown some promise in terms of understanding and tackling technology addiction problems. Nevertheless, there is a need to step back and understand the roots of technology-related addictions and how foundations evolved in reference disciplines in order to be able to conduct more scientifically informed research on such issues. This study, therefore, explains the concept of behavioral addictions (the family of addictions to which technology-related addictions presumably belong), reviews the field's history and evolution, explains the relevant brain circuitry, and discusses similarities and differences between behavioral and substance addictions. A synthesis of this information provides eight key observations and recommendations that should help the field move forward.

**Keywords:** Behavioral Addictions; Technology Addiction; Excessive Use of Information Systems; Information Systems.

## Introduction

The topic of behavioral addictions has traditionally been studied in the fields of psychology, psychiatry, and neuroscience and has been documented in the journals of these disciplines. One noticeable exception technology-related addictions "technology addiction"): this line of research has recently captured the attention of IS academics who have started documenting their discoveries in various IS outlets, ranging from conference proceedings to the prestigious "Basket of Eight Journals." Two arguments can be made to explain this unique positioning of the psychology/psychiatry research topic with the IS discipline. First, the concept of technology addiction revolves around the IT artifact and affects patterns of IT use and IT users. Having advanced knowledge of various technologies and technology use issues benefits the research process because it allows researchers to better understand all aspects, including abnormal, of human-computer interaction processes, explain their findings from the users' perspective, and make recommendations for system designers. Second, requiring IS scholars to publish their technology addiction research solely psychology/psychiatry journals would contribute to psychology/psychiatry research but do little for the advance of IS scholarship (Nunamaker & Briggs, 2011). For these reasons, IS scholars pursue technology addiction topics and document their discoveries in traditional IS outlets.

With this in mind, technology addiction has become an important IS research topic that represents the extreme, abnormal side of use, which has, unfortunately, become quite common. However, a key challenge faced by technology addiction researchers is that the theoretical foundations of this concept exist in a multi-disciplinary body of knowledge, including in journals in the fields of psychology, psychiatry, and neuroscience - i.e., reference disciplines (Wade, Biehl, & Kim, 2006). Acquiring such knowledge requires a substantial investment of effort and, most importantly, time, which is difficult to afford given the contemporary "publish or perish" culture and borderline-unrealistic promotion and tenure requirements. Nevertheless. attempting to study technology addiction without fully understanding the essence of behavioral addictions (the family of addictions to which technology addictions belong) as documented in reference disciplines may lead to erroneous conclusions and faulty recommendations. The authors of this paper have observed such inter-disciplinary frictions and inconsistencies when serving as the senior editors, associate editors, and reviewers, as well as being authors and readers, of papers on technology addiction.

To help IS researchers understand the theoretical and methodological aspects of technology addiction, we present a series of articles that rely on the neuroscience, psychiatry, and psychology literatures. Technology addiction is a type of behavioral addiction in which the addictive behavior is conducted by means of an IT artifact. Thus, before embarking on the investigation of technology addiction, IS researchers should understand the conceptualization, historical development, and underlying cognitive processes of behavioral addictions. This is the objective of the present, inaugural publication.

## What Are Behavioral Addictions?

The term addiction has traditionally been associated with a continued use of various substances such as alcohol, tobacco, and drugs. Gradually, the notion of behavioral addictions has attracted the attention of the research community and the general public with examples pertaining to gambling, sex, food, shopping, work, tanning, and exercise (Ascher & Levounis, 2015). More recent and widely publicized instances of behavioral addictions also include the use of information technologies (IT) such as video games, smartphones, and social networking sites (Griffiths, 2018) to such an extent that people ruin their marriages, destroy careers, fail school, and lose reallife friends. In one particular story, a couple's threemonth-old daughter died of starvation while her parents were raising a virtual child in a video game (Salmon, 2010). But what is this bizarre,

counterintuitive behavior in which individuals engage despite its potential harm, and why does it exist?

Behavioral addictions are a specific group of mental and behavioral disorders that do not include the ingestion of psychoactive substances (Demetrovics & Griffiths, 2012). They are defined as a psychological dependence on repetitive behaviors that feature the core components of addiction: salience, mood modification, tolerance, withdrawal, conflict, and relapse (Griffiths, 1996, 2005, & 2018) (see Table 1). These typically impair normal functioning and can easily be observed by most people. Behavioral addictions are fundamentally different from habit because the latter does not include the manifestation of the core addiction components, may be healthy, and may not adversely affect normal functioning.

The same definition and core symptoms apply to the concept of technology addiction, which is a psychological dependence on the use of a particular IT artifact resulting in the six symptoms discussed above (Turel, Serenko, & Giles, 2011). Whereas the magnitude of each core symptom may vary, typically all six of them must be present, at least to some extent, to conclude that a user experiences some degree of technology addiction. Ultimately, some impairment of normal functioning caused by the behavior pattern also needs to be apparent. Without such impairment, we may be looking at an extreme habit but cannot call the phenomenon an "addiction."

Because technology addiction is a form of behavioral addiction, it is important for researchers to understand the nature and development of this area of research as presented below.

# The History of Behavioral Addictions

Behavioral addictions attracted the attention of psychology and psychiatry researchers in the early 1980s. However, the very existence and conceptual foundations of behavioral addictions have been sporadically documented since the invention of writing. For example, the earliest dice and rolling boards ever discovered date to 3000 BC, and various forms of gambling have existed in virtually all human cultures throughout the entire history of our civilization (Schwartz, 2006). The first instance of behavioral addictions in the form of gambling addiction is mentioned in the Rig Veda, an ancient collection of Indian texts written between 1500 BC and 1200 BC. which clearly describes the financial, familial, and emotional consequences of an extreme gaming behavior when the gambler lost his wife, relatives, a house, money, and all personal belongings but was still unable to cease gambling (Griffith, 2017 (Mandala 10, Hymn 34)). Commodus, the Roman emperor who lived in the second century AD, bankrupted the regime

**Table 1. The Core Components of Behavioral Addictions** 

Component	Definition	Example
Salience	The behavior becomes an extremely important activity in one's life, dominates one's thinking, creates cognitive distortions, and causes cravings.	Someone addicted to shopping may devote as much time as possible to this activity as well as crave and ruminate on shopping when being unable to shop due to time or financial constraints.
Mood modification	The arousing or tranquilizing shift in one's mood as a result of an addictive behavior.	A morning gambling session may boost one's mood, but an evening session may have an opposite, calming effect on the same person. The predictable shift in one's mental state allows the person to manipulate his/her mood by means of addictive behavior.
Tolerance	Longer and/or higher intensity behavioral sessions are required to achieve the mood-modification effect.	To experience the same "high," a person addicted to tanning might need to spend increasing amounts of time tanning and/or increase the level of exposure in a tanning bed.
Withdrawal	Unpleasant psychological feelings (e.g., agitation, mood swings, emotional exhaustion) and/or physical discomfort (e.g., nausea, headache, insomnia, loss of appetite) are experienced when the addictive activity is reduced or suspended.	A person addicted to food may become very irritable even when not hungry if he/she is unable to eat.
Conflict	The addictive behavior is at odds with other activities – such as school, social life, hobbies, work, familial responsibilities, etc. – leading to various negative consequences for the individual and/or others.	Someone addicted to exercise may sacrifice family time and/or reduce work hours due to an inability to moderate the exercise activity.
Relapse	A person repeatedly tries to reduce or discontinue the addictive behavior but reverts to the previously established behavioral patterns. All attempts to moderate or terminate the behavior fail, and the previous levels of behavior are restored after abstinence periods.	Someone addicted to sex may make numerous attempts to cease the behavior but will revert to it even after long periods of abstinence.

because of his gambling addiction and turned the imperial palace into a casino (Schwartz, 2006). In the 16th century, Gerolamo Cardano, a prolific mathematician and avid gambler, publicly recognized himself as being "inordinately addicted to the chessboard and the dicing table" (David, 1962, pp. 56-57). In 1816, Andre Matthey, a Swiss doctor, invented the term "klopemanie" - currently referred to as kleptomania - which is the impulsive theft of objects having little or no value for the culprit (Whitlock, 1999). Cases describing individuals having unrestrained and excessive sexual appetites that require treatment were documented by Rush (1812) in the 18th century. In addition, pyromania, a disorder in which an affected individual deliberately sets things on fire, was welldocumented in the first half of the 19th century (Prichard, 1842).

In 1844, the Association of Medical Superintendents of American Institutions for the Insane was established – the organization which later changed its name to American Psychiatric Association (APA). In 1917, it

published the Statistical Manual for the Use of Institutions for the Insane (SMUII), which listed 22 mental diagnoses, but none of them pertained to behavioral addictions (APA, 1918) (see Figure 1). The first version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-I) published by the APA included a general description of obsessive compulsive reaction as the "persistence of unwanted ideas and of repetitive impulses to perform acts which may be considered morbid by the patient" (APA, 1952, p. 33), which somewhat corresponded to behavioral addictions. However, it did not offer definitions or examples of behavioral addictions. DSM-II included a similar description under the term of obsessive compulsive neurosis (APA, 1968). In 1968, both the World Health Organization (WHO) and the APA replaced the term drug addiction with drug dependence (Maddux & Desmon, 2000). As a result, dependence became an official term used in psychology and psychiatry literature and practice, and it even occasionally appeared as behavioral dependence.

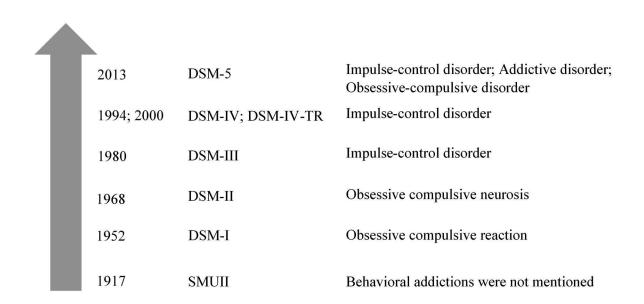


Figure 1: Classification of Behavioral Addictions by the APA

DSM-III included three types of behavioral addictions - pathological gambling, kleptomania, and pyromania - which were listed under impulse-control disorders and which had to have three essential elements: 1) inability to resist the impulse, drive, or temptation to act in a manner that is harmful for the individual or others; 2) some experienced tension before acting; and 3) a sense of pleasure, gratification, or release during the behavior. DSM-III also clearly excluded behavioral addictions from obsessive compulsive disorder/neurosis by arguing that "the individual derives pleasure from the particular activity and may wish to resist it only because of its secondary deleterious consequences" (APA, 1980, p. 235). DSM-IV (APA, 1994) and DSM-IV-TR (APA, 2000) slightly expanded the list of behaviors above by adding trichotillomania (pulling out of one's hair) and, again, differentiated such behaviors from obsessivecompulsive disorder. DSM-IV-TR also stated that an individual may or may not show regret, self-reproach, or guilt after the act. A distinctive feature of DSM-IV-TR is that it completely omitted the term "addiction" from its classification of mental disorders.

DSM-5 (the latest version of DSM when this paper was written) introduced several drastic changes to the classification of behavioral addictions. First, it reclassified trichotillomania as an obsessive-compulsive disorder and expanded this list by adding hoarding disorder (excessive acquisition of objects and the inability to discard them) and excoriation (skin-picking). Second, it retained kleptomania and pyromania as impulse-control disorders. The work group that developed DSM-5 also considered including other forms of behavioral addictions – such as sex, food, exercise, and shopping – but decided to omit them due

to insufficient evidence (O'Brien, 2014). Third, it brought back the term addiction and listed gambling disorder as a type of non-substance-related addictive disorder (APA, 2013). The rationale was that the term addiction more clearly describes a behavioral disorder than the term dependence does and that the former is less vague because dependence can be easily confused with other terms (e.g., interpersonal dependence). Fourth, it acknowledged a possibility of Internet gaming disorder by listing it under conditions for further study (section III of DSM-5).

The International Classification of Diseases (ICD), maintained by the World Health Organization (WHO) under the mandate of the United Nations, recently took this a step further. In its most recent version of ICD-11 released in 2018, it listed hoarding disorder and bodyfocused repetitive behavior disorders (hair-pulling, skin-picking, lip-biting) as obsessive-compulsive disorders, and pyromania, kleptomania, compulsive sexual behavior as impulse-control disorders (WHO, 2018). Most importantly, it introduced a separate category of disorders due to addictive behaviors, which includes gambling disorder and gaming disorder, where the latter is defined as a pattern of persistent or recurrent online and/or offline gaming behavior accompanied by impaired control. salience, and conflict in which the symptoms must be evident for a period of at least 12 months (see Reed et al. (2019) for a comprehensive review).

The latest DSM and ICD classifications (at the time of writing) are not the final word on these matters: they are expected to be gradually updated or even replaced. It is for this reason that Arabic numerals are now being used to indicate DSM versions: for example, future

updates may be referred to as 5.01, 5.1, 5.5, etc., depending on the magnitude and/or importance of the changes. Such indications would have been more difficult to achieve with the use of the Roman numerals (i.e., V). As evidenced by the need for such flexibility, the classifications of mental disorders are in constant flux as new empirical evidence is being accumulated at an exponentially growing pace.

## **Brain Circuitry of Addiction**

Behavioral addictions are considered a "primary, chronic disease of brain reward, motivation, memory and related circuitry" (ASAM, 2011, p. 1), where dopamine plays a critical role. Dopamine is a neurotransmitter (i.e., a chemical messenger) carrying signals between neurons (i.e., nerve cells). It is released under two conditions: 1) when a person is exposed to external (e.g., visual, olfactory, tactile, gustatory, auditory) or internal (e.g., mental) stimuli associated with a subject of behavioral addiction, and 2) when a person is engaged in a rewarding behavior, including those behaviors he/she is addicted to (Yacubian & Büchel, 2009). After being activated and released in the ventral tegmental area of the brain, dopaminergic neurons send projections to the brain regions responsible for the processing of rewards, such as the ventral striatum, amygdala, and ultimately the prefrontal cortex.

After synthesis, dopamine is stored in synaptic vesicles inside a neuron. Upon release, a single neuron does not travel throughout the person's entire mesolimbic pathway. Instead, the transmission process resembles a chain reaction in which discharged dopamine cells bind to dopamine receptors in other neurons (D2 receptors, in cases of addiction) and activate them, which, in turn, send activation signals to release their own dopamine: this results in a domino effect lasting until the signal reaches a terminal brain area (Beaulieu & Gainetdinov, 2011). Despite its complexity, the transmission process is almost instantaneous. When dopamine reaches its final destination, a person experiences the feeling of pleasure, excitement, or arousal associated with a reward - the strength of which (in healthy people) is proportional to the amount of dopamine that reached the corresponding brain regions (Volkow et al., 2002).

When individuals continuously engage in a behavior that is accompanied by a release of dopamine resulting in intrinsic rewards, their reward system becomes hyper-active in terms of the processing and integration of information among various circuits and functions of the brain, leading to two major changes (Reuter et al., 2005; Volkow et al., 2010). First, when people continuously engage in a rewarding behavior which is accompanied by sudden spikes in their

dopamine levels, the sensitivity and sometimes the number of D2 receptors decrease, and the amount of released dopamine declines, given the same behavioral pattern. As a result, they show a decreased sensitivity to the rewarding behavior and consequently need to engage in the behavior more frequently and more intensely to achieve the same level of pleasure as before. Second, the overstimulation of dopamine cells within the mesolimbic reward system usually occurs in the presence of stimuli associated with the behavior (e.g., the surrounding environment, preparation rituals, objects used to conduct the addictive behavior). Over time, people develop powerful learned associations between these stimuli and the subsequent reward (i.e., dopamine bursts entering the brain). Gradually, their brains develop new functional connections and start firing dopamine cells in the mere presence of (or even thoughts about) the related stimuli before the actual behavior takes place. Thus, the spikes of dopamine caused by the stimuli create an urge (strong, uncontrolled motivation) to engage in a behavior in order to secure a stronger subsequent reward. In other words, individuals become hyper-sensitive to behavior-associated stimuli. a phenomenon referred to as incentive salience (Robinson & Berridge, 1993, 2008).

There are two major conditions that strengthen incentive salience and produce a stronger anticipation of reward: reward uncertainty (Anselme, Robinson, & Berridge, 2013) and the experience of almost winning (i.e., near misses) (Griffiths, 1991). The feeling of uncertainty makes the reward more desirable because the person has to earn it, which causes stress. Stress, in turn, activates one's dopaminergic systems (Abercrombie et al., 1989; Robinson et al., 2015). In fact, previous research shows that the activity of dopaminoceptive regions in the human brain is correlated with reward uncertainty, and it achieves the highest level when the probability of reward is 50 percent (Preuschoff, Bossaerts, & Quartz, 2006). The presence of dopamine reinforces a learning process by motivating a person to attain the reward. Subsequently, an individual starts paying closer attention to the stimuli and subsequent actions, which produces a stronger stimuli-behavioral response link (Fiorillo, Tobler, & Schultz, 2003). This is consistent with the Pearce-Hall theory of learning (Pearce & Hall, 1980).

Near-misses produce biological and psychological outcomes similar to situations in which reward is obtained by implying a future acquisition of reward and heightening one's expectations (Griffiths, 1991). Evidence suggests that both reward acquisitions and near-misses activate identical brain regions (Habib & Dixon, 2010), and near-misses are accompanied by dopamine release, which signals the expectation of

future reward and encourages the behavior (Winstanley, Cocker, & Rogers, 2011). Whereas the effects of reward uncertainty and the near-misses have been mostly explored in the context of gambling, they apply equally to all potentially addictive behaviors in which an individual is uncertain about the likelihood of obtaining a reward and may experience near-misses. The observed effect of unexpected reward schedules on addictive behaviors dates back to Skinner's experiments with pigeons (Skinner, 1953); it also exists in the case of IT, which often provides rewards (e.g., game points, "likes") on an intermittent schedule (Meshi et al., 2016; Meshi, Morawetz, & Heekeren, 2013).

This presumably harmful (from the addiction perspective) brain functioning is, in fact, a result of evolution directed at the survival and perfection of all species, including humans. The entire development of the brain has been based on the recognition, anticipation, acquisition, and processing of rewards which enhance humans' chances of survival, improvement, and reproduction (Schultz, 1998). There are two types of rewards: primary and non-primary (Schultz, 2015). Primary rewards are necessary for survival and reproduction, such as substances (e.g., food, liquid) and behaviors (e.g., mating, caring for offspring). Non-primary rewards increase one's chance of attaining primary rewards and thereby

indirectly contribute to the survival, reproduction, and improvement of one's genes. For example, enjoying gourmet food and drink enhances one's ability to select a high-quality, nutritious diet. Romantic love, which, in contrast to straightforward sex, does not directly lead to reproduction, enhances one's attachment to a potential mating partner and their future offspring to increase their chances of survival as a family. Rewards from altruistic action, friendship, and social interaction have similar evolutionary advantages because they promote group cooperation, which improves one's chances for survival (Báez-Mendoza & Schultz, 2013). Novelty seeking, experimentation, exploration of the world, and curiosity - which are associated with reward uncertainty - help discover new food sources, shelters, and mating partners, thereby also contributing to survival and reproduction (Schultz, 2015). Thus, the brain mechanisms responsible for the development of behavioral addictions actually contribute to the survival and development of humans, and addictive behaviors may be considered an accidental byproduct of their workings.

A simplified depiction of the brain circuitry systems and the way they interact to produce addictive behaviors is given in Figure 2. It shows that behavioral addiction results from an imbalance between the reward and self-control/inhibition systems.

# Reward System (Amygdala-Striatum Dependent) Interoceptive Awareness/ Urge System (Insular Cortex Dependent)

Figure 2: The Brain Circuitry of Addictive Behavior

The incentive salience effect can often be exacerbated by deficit in self-control/inhibition circuitry (Bechara, 2005). That is, some people may take control over strong incentive motivation as mediated by the reward system by engaging their self-control or inhibition abilities, which are primarily prefrontal cortex dependent (Turel & Bechara, 2016b). However, when damage to prefrontal brain regions occurs, either through lesions (Bechara, 2004) or substance use (He et al., 2018b), it prevents people from integrating the needed information for making decisions and exercising judgment with long-term consequence considerations. That is, the interplay between the reward and self-control/inhibition systems can define people's propensity to develop addictions; an imbalance manifested hyper-active in reward/incentive systems and hypo-active selfcontrol/inhibition systems can produce addictive behaviors (Turel & Qahri-Saremi, 2016).

Another system that is part of this circuitry is the insular cortex, also known as the insula (Contreras, Ceric, & Torrealba, 2007). This system mediates the development of what may be subjectively felt as urges (or cravings and temptations). Importantly, it has trajectories to the reward and inhibition brain systems and hence can modulate their activity (Wood & Bechara, 2014). First, the insula sensitizes the reward system. Second, it impairs and preoccupies the inhibition system (Turel & Bechara, 2016a, 2016b; Turel et al., 2018b). It, therefore, has a dual effect that promotes addictive behaviors (i.e., strengthening the focus on rewards and, at the same time, weakening inhibition abilities). Indeed, in the context of addiction, it has been shown that damage to the insula helped people overcome smoking addiction (Nagvi et al., 2007). The temptation/urge the insula mediates also impairs people's abilities to reflect on the IT they use and makes them more prone to rely on automatic behaviors (Turel & Bechara, 2016b, 2017).

Thus, behavioral addictions are considered a disease of the brain's reward system (Holden, 2001) and involve the interplay and specifically an imbalance of the three aforementioned brain systems: reward (amygdala-striatum dependent), self-control/inhibition (prefrontal cortex dependent), and interoceptive awareness/urge (insular cortex dependent). Once an imbalance develops, these systems may be triggered by various external or internal stimuli beyond people's conscious awareness, and the enactment of the addictive behavior can dominate people's lives to the point when it adversely affects normal functioning. As such, behavioral addictions rely on the same neurological mechanisms as substance addictions. Nevertheless, behavioral addictions exhibit a number of unique characteristics that place them into an exclusive category of mental disorders.

# Behavioral Addictions vs. Substance Addictions

Behavioral and substance addictions have a shared symptomology: both types of addictions manifest in the aforementioned six core symptoms (Karim & Chaudhri, 2012). Both types are often accompanied by impulse-control disorders (Grant, 2008) and result in adverse consequences for the addicted individuals and their environments (Grant et al., 2010). Both types of addictions are also similar in terms of their neurobiological roots, including genetic factors (e.g., genes that affect D2 dopamine receptors), brain impairments of the reward and self-regulation systems (Grant, Brewer, & Potenza, 2006; Weinstein, 2013), risk factors that underlie their development (Kuss, Griffiths, & Binder, 2013; Lam et al., 2009), and their development processes (Grant & Chamberlain, 2014).

Despite many similarities, though, behavioral and substance addictions can differ along several dimensions. First, most addictive substances (excluding coffee and sugar) are largely regulated, and access to such substances by children and youth is typically restricted. For example, many countries forbid the selling of alcohol, tobacco, and marijuana to adolescents (Pacula et al., 2014). In contrast, many addictive behaviors (e.g., playing video games and even some forms of gambling) are relatively less restricted compared to substances (Singer & Singer, 2012). As such, two pronounced differences between behavioral and substance addictions are their prevalence in children and youth and the ease of access to the addictive object. For instance, while lifetime use of marijuana in the US in 2018 was 13.9 percent in 8th grade, 32.6 percent in 10th grade, and 43.6 percent in 12th grade (The National Institute on Drug Abuse Blog Team, 2018), a much higher proportion of children and youth in the US engages in video gaming and/or in using social networking sites (Twenge, Martin, & Campbell, 2018), which may lead to the development of IT-related behavioral addictions, because such uses of IT tend to be highly rewarding and provide rewards on a variable schedule.

Second, while the symptomology of behavioral and substance addictions is similar, its interpretation and severity can vary. For example, addiction requires "clinically significant" impairment of normal functioning (APA, 2013), which is part of the "conflict" symptom mentioned above. However, this can carry different meanings in behavioral and substance addictions. In behavioral addictions, clinically significant impairments can mean losing all of one's money (gambling), deteriorated school performance (video gaming), and, in some cases, loss of sleep, increased and cardio-metabolic deficits obesity, (Turel. Romashkin, & Morrison, 2016; Turel, Romashkin, &

Morrison, 2017). In contrast, in substance addictions, such impairments may manifest in much more lethal health risks, such as liver disease, cancer (Rehm et al., 2003), and/or overdose (Calcaterra, Glanz, & Binswanger, 2013). Differences in severity are also pronounced regarding withdrawal symptoms: in behavioral addictions, these symptoms are mostly in the form of psychological discomfort; in substance addictions, withdrawal symptoms can include severe physiological reactions, including tremors, nausea, and headaches (Winokur et al., 1980).

Third, we can reasonably speculate that relapse frequency (resuming the addictive behavior) and difficulty abstaining from the addictive behavior can vary between substance and behavioral addictions. First, while people can relatively easily avoid substances (e.g., they have to, in most cases, find a drug dealer or go to a liquor store), it may be more difficult for them to avoid common behavioral cues (e.g., technology cues are all around people in modern society). Combined with the notion that behavioral addictions are often perceived to be benign (Turel et al., 2014), we can expect higher relapse rates in the case of behavioral addictions. Second, once substance use stops, substance effects typically do not fully disappear (Zhao et al., 2017). There is often some retained neurotoxicity in the brain and changes to brain mechanisms that make abstinence difficult, and relapse more likely (He et al., 2018b). In contrast, while excessive behaviors are associated with brain changes (He et al., 2019; He, Turel, & Bechara, 2018a; He et al., 2017b), the brain may more easily recover after abstinence given that there is no neurotoxicity. The assertions above are preliminary; more evidence in support of such propositions is needed.

Ultimately, such differences can explain the relatively high prevalence rates of behavioral addictions. For example, using preliminary classification criteria, it has been suggested that there is about 4.5 percent prevalence rate of at-risk behavior for social media addiction in adolescents (Banyai et al., 2017) and between 11.6 percent (Turel & Cavagnaro, 2019) and 15.2 percent (Turel, Brevers, & Bechara, 2018a) in young adults. One explanation for such high numbers is that it is simply easier to meet addiction criteria in the case of behavioral addictions. While the symptoms of behavioral and substance addictions are identical. their interpretation is different, and, in the case of behavioral addictions, they are typically less severe or life-threatening (He et al., 2017b; Turel et al., 2018b; Turel et al., 2014).

Last, while the neural roots of behavioral and substance addictions are similar (Grant et al., 2006; Weinstein, 2013), there is growing evidence for some differences. Similarities stem from the ideas that all addictions are rooted in an imbalance between a

hyper-active reward system and a relatively weak (or hypo-active) self-control or inhibition brain faculty (Turel et al., 2014; Turel & Qahri-Saremi, 2016; Turel & Qahri-Saremi, 2018). This imbalance can be influenced by both nature and nurture (He, Turel, & Bechara, 2017a; He et al., 2017b). It is worth noting that children and youth are more vulnerable to addictions than older individuals, given that they have a built-in imbalance between the reward and selfcontrol systems. The reason for this imbalance is that these brain systems develop on different schedules (Casey, Getz, & Galvan, 2008; Giedd, 2004; Sowell et al., 1999). While the reward system is fully developed during adolescence, the self-control/inhibition brain system matures later on, and the connectivity between left and right hemispheres (corpus callosum), which is needed for proper decision making, matures even later (Casey et al., 2007; Casey, Giedd, & Thomas, 2000; Casey et al., 2005; Durston et al., 2001). It may be for this reason that there is a negative correlation between users' age and their technology addiction levels (e.g., see Serenko & Turel, 2015).

Focusing on specific similarities, the functional hyperactivity (Turel et al., 2014) and structural pruning (reduced grey matter volume, or fewer dopamine receptors) of the reward (amygdala-striatal) system exist in both substance and behavioral addictions (He et al., 2017a; He et al., 2017b). There are also similarities in structural changes in interoceptive-awareness brain systems (insular cortex dependent): reduced volumes of the posterior insula were observed in behavioral and substance addictions (Turel et al., 2018b). Moreover, some similarities in deficits in inter-hemispheric connectivity (white matter integrity), which prevents efficient communication between the hemispheres, were observed in substance and behavioral addictions (He et al., 2018a).

Nevertheless, some behavioral addictions do not present self-control impairments that are common in substance addictions: they simply involve a hyperactive reward system together with functional selfcontrol faculties and lack of motivation to engage the self-control system (Turel et al., 2014). Simply put, these findings show that the imbalance between reward and self-control systems in some behavioral addictions can be primarily a function of the hypersensitive (functionally and structurally) reward system. This is similar to less harming behaviors such as light smoking and moderate gambling. This is good news for people who try to treat or overcome technologyrelated behavioral addictions: most of them can do it if they have sufficient motivation, and techniques such as mediation and cognitive behavioral therapy may help (He et al., 2017a, 2018a; He et al., 2017b). That is, while recovery from substance addictions is difficult as it requires changes to damaged prefrontal brain

regions (He et al., 2018b), it may be easier to treat and overcome some behavioral addictions (at least for some users) given the right motivation and with the help of behavioral interventions (Turel et al., 2014).

Thus, despite some similarities, behavioral and substance addictions differ in terms of their prevalence, severity, core symptoms manifestation, self-control system impairment, and ease of treatment. While the observed similarities allow IS researchers to draw from the vast literature on addictions when conducting their studies, the differences suggest that IS researchers should examine technology addiction as a standalone phenomenon that may or may not function exactly like substance addictions and may or may not have the exact same biological-physiological and behavioral-psychological roots. This also means that more research on similarities and differences between substance and technology-related addictions is needed.

## Implications and Recommendations

The overview of the behavioral addiction literature presented above leads to several important observations that may be of interest to technology addiction researchers.

# Implication 1. IT users are not addicted to an IT artifact. Instead, they are addicted to a behavior conducted by means of an IT artifact (i.e., IT-mediated behavior).

Users can get addicted to a behavior that happens to be mediated via an IT artifact, rather than to an IT artifact per se. The IT artifact just provides easy, constant access to the addictive behavior. To illustrate this point, note that behavioral addictions have existed during the entire history of mankind. And, as the subjects of addiction and the aids facilitating addictive behaviors have changed, the underlying principles of behavioral addictions have remained the same. For example, while gambling technologies evolved, into online environments, gamblers' including behaviors did not change: over three millennia ago, some people were already addicted to the gambling process, and many still are. As various mechanical, electromechanical (e.g., pinball machines), and electronic gambling devices appeared, the overall addictive behavior has remained unaltered. Similarly, historically, a small percentage of people engaged in various forms of compulsive sexual behaviors; after the advent of the Internet, such activities simply evolved to cybersex addiction. This suggests that individuals get addicted to a behavior rather than the tool by means of which the behavior is conducted. In other words, users do not get addicted to an IT artifact - for example, a gambling website, a smartphone app, or a video game console. Instead, they are addicted to

the gambling, messaging, or gaming behavior conducted (i.e., mediated) through an electronic device. Thus, it is more precise to talk about addiction to the use of technology or to specific technologymediated behaviors rather than about addiction to broad technology objects (e.g., smartphones) that facilitate access to an addictive behavior. For example, a person who is addicted to the use of social media or to gaming may enact these addictive behaviors over smartphones, laptops, or desktop computers. It may, therefore, be inaccurate to call his/her addiction a "smartphone addiction": he/she is likely addicted instead to specific behaviors mediated via specific applications which can be enacted via any device. Note that this does not fully remove responsibility from technology developers and providers. In many cases (see implication #2), IT artifacts include features and functionalities that promote frequently repeated, automatic, and difficult-to-control behaviors, because their revenues depend on user traffic and time spent with the system (Turel, 2019).

# Implication 2. The features of many contemporary IT artifacts facilitate the development and reinforcement of technology addiction.

The propagation of technology addiction has likely never been an explicit goal of IT developers. However, to facilitate the adoption and continued use of their technologies, designers invented and incorporated in their products various features relying on behavioral reward mechanisms. For example, "loot boxes" (ingame purchased boxes with unknown/random sets of tools that are revealed only after the purchase) in video games are similar to gambling (Drummond & Sauer, 2018). As a result, rather than being intentional, technology addiction has become an accidental byproduct of the synergy between the workings of the brain's reward system and the capabilities of IT to allow users to easily engage in a rewarding (from the brain's perspective) behavior. To further illustrate this point, video games always incorporate elements of uncertainty (variable reward) and are full of near-miss effects which encourage and strengthen repetitive and possibly addictive play. As users advance through the game, the subsequent levels become more difficult, which addresses the issue of tolerance; as behavioral addiction strengthens, longer and more intense activity is required to obtain the same reward. Similarly, social media applications reward users through views. likes, and shares, and the number of such rewards is always somewhat unpredictable, which is a form of electronically delivered variable reward (Meshi et al., 2016; Meshi et al., 2013). Thus, the employment of many contemporary IT artifacts automatically engages the reward mechanism of their users. While the initial intention was to engage users through the exploitation of their reward systems rather than create "technology

addicts" (Eyal & Hoover, 2014), an unpredicted outcome is that some users develop addiction-like symptoms in relation to the use of such technologies. Thus, while technology is not the sole cause of behavioral addictions, its features and affordances can contribute to addiction formation through the provision of variable rewards and/or the elimination of features that promote self-control (e.g., use time alerts) (He et al., 2017a, 2018a; He et al., 2017b; Turel et al., 2018a).

# Implication 3. IT artifacts incorporating various socialization features are addiction-prone by their nature.

Social networking sites, multiplayer video games, and smartphones are perhaps the most frequently cited examples of potentially addictive technologies. This is not surprising because the human brain evolved in a way to encourage various forms of socialization, communication, and collaboration: engagement in such activities is accompanied by a release of dopamine as a form of reward (again, likely developed from an evolutionary perspective, to promote the survival and perfection of the species). The problem is that the technologies above continuously trigger socialization-based rewards (likes, views, shares, conversations, interactions) and, as a result, people use them for the sake of mere pleasure, which may lead to the development of the core symptoms of addiction. Again, users are not addicted to these IT systems; instead, they are addicted to a naturally rewarding electronic socialization process.

# Implication 4. Technology addiction researchers should rely on substance addiction literature with caution.

On the one hand, substance addictions and behavioral addictions share some similarities in terms of etiology and outcomes. On the other hand, there are some key differences - for example, age of onset, deficits in inhibition brain systems, and the magnitude of adverse effects. Hence, IS researchers should carefully build on literature on substance addiction from reference disciplines. In fact, one goal for the next decade is to better map similarities and differences between substance and behavioral addictions (He et al., 2017a. 2018a; He et al., 2017b) or to replicate common substance addiction models and phenomena in relation to behavioral technology addictions (see examples in He et al., 2018a; He et al., 2017b; Turel & Bechara, 2016b, 2017; Turel et al., 2018a; Turel et al., 2018b; Turel et al., 2014; Turel, Poppa, & Gil-Or, 2018c). Such attempts can allow a deeper and evidence-based, as opposed to blind, understanding of technology addiction.

# Implication 5. Literature obsolescence is an ongoing issue in behavioral addiction research.

In order to formally classify and include a mental disorder in the DSM and the ICD, the APA and WHO taskforce and workgroup members analyze thousands of peer-reviewed publications to reach a consensus. As the body of knowledge accumulates, behavioral mental disorders are re-classified, modified, added, and eliminated, and the classification system is in a constant state of flux, with new editions appearing every 10-15 years and superseding the previous ones. Hence, IS research has an opportunity and, some may say, a responsibility to inform this process. In the past, the process has relied on evidence published in psychology and psychiatry journals. Nevertheless, IS researchers should play a more active role in informing addiction classification and definition. While this has not been a common theme in IS journals as most treat addiction as a continuous construct (Turel & Serenko, 2012), a differentiation of abnormal from normal cases should be a future theme. Moreover, the unstable nature of this research stream should encourage IS researchers to stay current and follow recent work, including in reference disciplines. For example, one should follow the most recent DSM criteria and definitions as opposed to those provided in older versions.

# Implication 6. Contradictions in the technology addiction literature result from inconsistencies in the psychology, psychiatry, and medical literatures.

In addition to a fast pace of obsolescence, the behavioral addiction literature is rife contradictions. Currently, there are differences in the classification of mental disorders between the DSM and the IDC. Several countries developed their own adaptations of the ICD (e.g., ICD-10-CA in Canada), which differ from the one published by the WHO. This makes it challenging for IS researchers who lack extensive training in the psychology, psychiatry, or medical fields to select the most appropriate source to develop a conceptual foundation for an addictive behavior model in the context of IT. This means that papers on technology addiction should often use more cautious rather than deterministic language (e.g., "users exhibiting addiction symptoms," or "addictionlike symptoms" instead of "technology addicts").

# Implication 7. The "standing on the shoulders of giants" mindset may not fully apply to technology addiction research.

Traditionally, IS scholars have relied on the "standing on the shoulders of giants" mindset: researchers are expected to be aware of, utilize, and extend the body of knowledge published in IS journals while paying close attention to the classics. However, in addition to the literature obsolescence and contradiction issues discussed above, there is a substantial time gap between publications in the psychology and IS venues, which makes some IS works even more outdated. Thus, technology addiction researchers, reviewers, and editors should exercise extra caution when relying on or recommending the use of theories from previous IS publications. For example, a number of generally accepted and extensively cited technology addiction studies relied on the diagnostic features and criteria of pathological gambling, which was classified as an impulse-control disorder in DSM-IV-TR (APA, 2000) because that model was the closest one to mental disorders associated with IT at the date of these studies. Currently, DSM-5 (APA, 2013) classifies gambling disorder as an addictive, non-substancerelated disorder (i.e., not as an impulse-control disorder). As a result, referring to technology addiction as an impulse-control disorder is currently considered not only outdated but also incorrect. Given these realities, technology addiction researchers are urged to continuously update their knowledge by relying on the latest publications in the psychology literature.

# Implication 8. Many cases of technology addiction are curable, but it may not be easy.

Many recovery processes in the brain depend on one's ability to comprehend the situation and mobilize resources to deal with it. Such processes are prefrontal cortex dependent. For example, in cocaine addicts, there is damage to the prefrontal cortex which prevents people from exercising strong control over their desires and limits their ability to suppress urges and to recover (He et al., 2018b; Turel et al., 2011). Findings show that in many behavioral addictions (at least in low to moderately severe cases), there is limited damage or impairment (activation or structural) to the prefrontal cortex (He et al., 2017a, 2018a; He et al., 2017b). This means that people with these levels of behavioral addictions can have (or can be trained to have) the ability to overcome their addiction: they just need to have strong motivation to do so and overcome relapse challenges. Techniques, such as mindfulness training, and technological features, such as warnings about the time spent, can help train people to better control excessive behaviors (Black, 2014; Brewer, Elwafi, & Davis, 2013; Garland, 2016).

Note that this does not mean that all cases of behavioral addictions can be easily treated: it just means that a majority of IT users who present with low to moderate levels of IT addiction symptomology can take control over excessive use if they are sufficiently motivated or trained. Difficulties in recovery may relate to forces that influence relapse. Relapse is a common problem in overcoming addictive behaviors.

Consequently, it may take multiple attempts and interventions before one achieves full recovery (Bishop, 2018). For example, in the case of smoking. this behavior can take up to 24 years and require from 6 to 142 attempts (and subsequent relapses), while in the case of overeating, few actually achieve long-term weight loss (Bishop, 2018). As mentioned earlier in the text, there may be at least two forces that operate in opposite directions to influence relapse frequency in the case of technology use. The first force is through exposure to cues which promote addictive behaviors; such cues are thought to contribute to relapse (Bishop, 2018; Goltseker, Bolotin, & Barak, 2017; Rich & Torregrossa, 2019). Thus, even if one abstains from social networking sites (for example), the use of a smartphone (for other tasks or even by surrounding people) may act as a cue for relapse. The second, opposing force is control, which is easier to exercise in the case of technology-mediated addictions because there is no neurotoxicity like in the case of substance addiction, which makes brain structural and functional recovery more difficult in substance users (He et al., 2018b). The balance between such forces, combined with prefrontal brain capacity, should determine the ease of recovery and relapse likelihood. This is a fruitful area for future research.

## Conclusion

A first step in conducting academically sound and scientifically accurate research on technology addiction is to understand both its roots and the research on the general family of addictions to which it belongs – namely, behavioral addictions. We hope that IS scholars will rely on the provided summaries and implications to improve their understanding of this embryonic research area, resulting in more theoretically and methodologically sound studies. In the following (i.e., Part II) publication, we will focus exclusively on the topic of technology addiction by relying on the key issues discussed in the present work.

## References

Abercrombie, E. D., Keefe, K. A., DiFrischia, D. S., & Zigmond, M. J. (1989). Differential effect of stress on in vivo dopamine release in striatum, nucleus accumbens, and medial frontal cortex. *Journal of Neurochemistry*, *52*(5), 1655-1658.

Anselme, P., Robinson, M. J. F., & Berridge, K. C. (2013). Reward uncertainty enhances incentive salience attribution as sign-tracking. *Behavioural Brain Research*, 238(February), 53-61.

APA. (1918). Statistical Manual for the Use of Institutions for the Insane. New York: American Medico-Psychological Association.

- APA. (1952). Diagnostic and statistical manual of mental disorders: DSM-I. Washington: APA.
- APA. (1968). Diagnostic and statistical manual of mental disorders: DSM-II. Washington: APA.
- APA. (1980). Diagnostic and statistical manual of mental disorders: DSM-III. Washington: APA.
- APA. (1994). Diagnostic and statistical manual of mental disorders: DSM-IV. Washington: APA.
- APA. (2000). Diagnostic and statistical manual of mental disorders: DSM-IV-TR. Washington: APA.
- APA. (2013). Diagnostic and statistical manual of mental disorders: DSM-5. Washington: APA.
- ASAM. (2011). Public policy statement: Definition of addiction. Available online at https://www.asam.org/resources/definition-of-addiction. Chevy Chase, MD: American Society of Addiction Medicine.
- Ascher, M. S., & Levounis, P. (Eds.). (2015). *The behavioral addictions*. Washington, DC: American Psychiatric Publishing.
- Báez-Mendoza, R., & Schultz, W. (2013). The role of the striatum in social behavior. *Frontiers in Neuroscience*, 7(233), 1-14.
- Banyai, F., Zsila, A., Kiraly, O., Maraz, A., Elekes, Z., Griffiths, M. D., Andreassen, C. S., & Demetrovics, Z. (2017). Problematic social media use: Results from a large-scale nationally representative adolescent sample. *PLoS One*, *12*(1), 1-13.
- Beaulieu, J. M., & Gainetdinov, R. R. (2011). The physiology, signaling, and pharmacology of dopamine receptors. *Pharmacological Reviews*, 63(1), 182-217.
- Bechara, A. (2004). The role of emotion in decision-making: Evidence from neurological patients with orbitofrontal damage. *Brain and Cognition*, *55*(1), 30-40.
- Bechara, A. (2005). Decision-making, impulse control, and loss of willpower to resist drugs: A neurocognitive perspective. *Nature Neuroscience*, 8(11), 1458-1463.
- Bishop, F. M. (2018). Self-guided change: The most common form of long-term, maintained health behavior change. *Health Psychology Open*, *5*(1), 1-14.
- Black, D. S. (2014). Mindfulness-based interventions: An antidote to suffering in the context of substance use, misuse, and addiction. Substance Use & Misuse, 49(5), 487-491.
- Brewer, J. A., Elwafi, H. M., & Davis, J. H. (2013). Craving to quit: Psychological models and neurobiological mechanisms of mindfulness training as treatment for addictions. *Psychology of Addictive Behaviors*, 27(2), 366-379.

- Calcaterra, S., Glanz, J., & Binswanger, I. A. (2013). National trends in pharmaceutical opioid related overdose deaths compared to other substance related overdose deaths: 1999–2009. *Drug and Alcohol Dependence*, *131*(3), 263-270.
- Casey, B. J., Epstein, J. N., Buhle, J., Liston, C., Davidson, M. C., Tonev, S. T., Spicer, J., Niogi, S., Millner, A. J., Reiss, A., Garrett, A., Hinshaw, S. P., Greenhill, L. L., Shafritz, K. M., Vitolo, A., Kotler, L. A., Jarrett, M. A., & Glover, G. (2007). Frontostriatal connectivity and its role in cognitive control in parent-child dyads with ADHD. American Journal of Psychiatry, 164(11), 1729-1736.
- Casey, B. J., Getz, S., & Galvan, A. (2008). The adolescent brain. *Developmental Review*, 28(1), 62-77.
- Casey, B. J., Giedd, J. N., & Thomas, K. M. (2000). Structural and functional brain development and its relation to cognitive development. *Biological Psychology*, 54(1-3), 241-257.
- Casey, B. J., Tottenham, N., Liston, C., & Durston, S. (2005). Imaging the developing brain: What have we learned about cognitive development? *Trends in Cognitive Sciences*, *9*(3), 104-110.
- Contreras, M., Ceric, F., & Torrealba, F. (2007). Inactivation of the interoceptive insula disrupts drug craving and malaise induced by lithium. *Science*, *318*(5850), 655-658.
- David, F. N. (1962). Games, gods, and gambling: A history of probability and statistical ideas. Mineola, NY: Dover Publications.
- Demetrovics, Z., & Griffiths, M. D. (2012). Behavioral addictions: Past, present and future. *Journal of Behavioral Addictions*, 1(1), 1-2.
- Drummond, A., & Sauer, J. D. (2018). Video game loot boxes are psychologically akin to gambling. *Nature Human Behaviour*, 2(8), 530.
- Durston, S., Pol, H. E. H., Casey, B. J., Giedd, J. N., Buitelaar, J. K., & Van Engeland, H. (2001). Anatomical MRI of the developing human brain: What have we learned? *Journal of the American Academy of Child & Adolescent Psychiatry*, 40(9), 1012-1020.
- Eyal, N., & Hoover, R. (2014). *Hooked: How to build habit forming products*. New York, NY: Portfolio Hardcover.
- Fiorillo, C. D., Tobler, P. N., & Schultz, W. (2003). Discrete coding of reward probability and uncertainty by dopamine neurons. *Science*, 299(5614), 1898-1902.

- Garland, E. L. (2016). Restructuring reward processing with Mindfulness-Oriented Recovery Enhancement: Novel therapeutic mechanisms to remediate hedonic dysregulation in addiction, stress, and pain. In S. Sequeira (Ed.), Special issue: Advances in meditation research 1373, 25-37. New York, NY: New York Academy of Sciences.
- Giedd, J. N. (2004). Structural magnetic resonance imaging of the adolescent brain. In R. E. Dahl & L.
   P. Spear (Eds.), Adolescent brain development: Vulnerabilities and opportunities 1021, 77-85.
   New York, NY: New York Academy of Sciences.
- Goltseker, K., Bolotin, L., & Barak, S. (2017). Counterconditioning during reconsolidation prevents relapse of cocaine memories. *Neuropsychopharmacology*, *42*(3), 716-726.
- Grant, J. E. (2008). Impulse control disorders: A clinician's guide to understanding and treating behavioral addictions. New York, NY: W. W. Norton & Company.
- Grant, J. E., Brewer, J. A., & Potenza, M. N. (2006). The neurobiology of substance and behavioral addictions. *CNS Spectrums*, *11*(12), 924-930.
- Grant, J. E., & Chamberlain, S. R. (2014). Impulsive action and impulsive choice across substance and behavioral addictions: Cause or consequence? *Addictive Behaviors*, 39(11), 1632-1639.
- Grant, J. E., Potenza, M. N., Weinstein, A., & Gorelick, D. A. (2010). Introduction to behavioral addictions. *The American Journal of Drug and Alcohol Abuse*, 36(5), 233-241.
- Griffith, R. T. H. (2017). *The Rig Veda: Complete and illustrated*. Scotts Valley, CA: CreateSpace Independent Publishing Platform.
- Griffiths, M. (1991). Psychobiology of the near-miss in fruit machine gambling. *The Journal of Psychology*, *125*(3), 347-357.
- Griffiths, M. D. (1996). Behavioural addiction: An issue for everybody? *Employee Counselling Today: The Journal of Workplace Learning*, 8(3), 19-25.
- Griffiths, M. D. (2005). A 'components' model of addiction within a biopsychosocial framework. *Journal of Substance Use*, *10*(4), 191-197.
- Griffiths, M. D. (2018). Classifying behavioural addictions: The DSM, and over-pathologising everyday life. *Psychology Review*, 23(3), 18-21.
- Habib, R., & Dixon, M. R. (2010). Neurobehavioral evidence for the "near-miss" effect in pathological gamblers. *Journal of the Experimental Analysis of Behavior*, 93(3), 313-328.
- He, Q., Huang, X., Zhang, S., Turel, O., Ma, L., & Bechara, A. (2019). Dynamic causal modeling of insular, striatal, and prefrontal cortex activities during a food-specific Go/NoGo Task. Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, in-press.

- He, Q., Turel, O., & Bechara, A. (2017a). Brain anatomy alterations associated with Social Networking Site (SNS) addiction. *Scientific Reports*, 7(paper 45064), 1-8.
- He, Q., Turel, O., & Bechara, A. (2018a). Association of excessive social media use with abnormal white matter integrity of the corpus callosum. *Psychiatry Research: Neuroimaging*, 278, 42-47.
- He, Q., Turel, O., Brevers, D., & Bechara, A. (2017b). Excess social media use in normal populations is associated with amygdala-striatal but not with prefrontal morphology. *Psychiatry Research-Neuroimaging*, 269(1), 31-35.
- He, Q. H., Huang, X. L., Turel, O., Schulte, M., Huang, D., Thames, A., Bechara, A., & Hser, Y. I. (2018b). Presumed structural and functional neural recovery after long-term abstinence from cocaine in male military veterans. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 84, 18-29
- Holden, C. (2001). 'Behavioral' addictions: Do they exist? *Science*, 294(5544), 980-982.
- Karim, R., & Chaudhri, P. (2012). Behavioral addictions: An overview. *Journal of Psychoactive Drugs* 44(1), 5-17.
- Kuss, D. J., Griffiths, M. D., & Binder, J. F. (2013). Internet addiction in students: Prevalence and risk factors. *Computers in Human Behavior*, 29(3), 959-966.
- Lam, L. T., Peng, Z. W., Mai, J. C., & Jing, J. (2009). Factors associated with Internet addiction among adolescents. *Cyberpsychology & Behavior*, 12(5), 551-555.
- Maddux, J. F., & Desmon, D. P. (2000). Addiction or dependence? *Addiction*, *95*(5), 661-665.
- Meshi, D., Mamerow, L., Kirilina, E., Morawetz, C., Margulies, D. S., & Heekeren, H. R. (2016). Sharing self-related information is associated with intrinsic functional connectivity of cortical midline brain regions. *Scientific Reports*, 6(22491).
- Meshi, D., Morawetz, C., & Heekeren, H. R. (2013). Nucleus accumbens response to gains in reputation for the self relative to gains for others predicts social media use. *Frontiers in Human Neuroscience*, 7(439).
- Naqvi, N. H., Rudrauf, D., Damasio, H., & Bechara, A. (2007). Damage to the insula disrupts addiction to cigarette smoking. *Science*, *315*(5811), 531-534.
- Nunamaker, J. F., & Briggs, R. O. (2011). Toward a broader vision for information systems. *ACM Transactions on Management Information Systems*, 2(4), 1-12.
- O'Brien, C. P. (2014). Foreword. In K. P. Rosenberg & L. C. Feder (Eds.), *Behavioral addictions: Criteria, evidence, and treatment*. Waltham, MA: Elsevier.

- Pacula, R. L., Kilmer, B., Wagenaar, A. C., Chaloupka, F. J., & Caulkins, J. P. (2014). Developing public health regulations for marijuana: Lessons from alcohol and tobacco. *American Journal of Public Health*, 104(6), 1021-1028.
- Pearce, J. M., & Hall, G. (1980). A model for Pavlovian learning: Variations in the effectiveness of conditioned but not of unconditioned stimuli. *Psychological Review*, 87(6), 532-552.
- Preuschoff, K., Bossaerts, P., & Quartz, S. R. (2006). Neural differentiation of expected reward and risk in human subcortical structures. *Neuron*, *51*(3), 381-390.
- Prichard, J. C. (1842). On the different forms of insanity in relation to jurisprudence. London: Hippolyte Bailliere.
- Reed, G. M., First, M. B., Kogan, C. S., Hyman, S. E., Gureje, O., Gaebel, W., Maj, M., Stein, D. J., Maercker, A., Tyrer, P., Claudino, A., Garralda, E., Salvador-Carulla, L., Ray, R., Saunders, J. B., Dua, T., Poznyak, V., Medina-Mora, M. E., Pike, K. M., Ayuso-Mateos, J. L., Kanba, S., Keeley, J. W., Khoury, B., Krasnov, V. N., Kulygina, M., Lovell, A. M., de Jesus Mari, J., Maruta, T., Matsumoto, C., Rebello, T. J., Roberts, M. C., Robles, R., Sharan, P., Zhao, M., Jablensky, A., Udomratn, P., Rahimi-Movaghar, A., Rydelius, P.-A., Bährer-Kohler, S., Watts, A. D., & Saxena, S. (2019). Innovations and changes in the ICD-11 classification of mental, behavioural and neurodevelopmental disorders. World Psychiatry, 18(1), 3-19.
- Rehm, J., Room, R., Monteiro, M., Gmel, G., Graham, K., Rehn, N., Sempos, C. T., & Jernigan, D. (2003). Alcohol as a risk factor for global burden of disease. *European Addiction Research*, *9*(4), 157-164.
- Reuter, J., Raedler, T., Rose, M., Hand, I., Gläscher, J., & Büchel, C. (2005). Pathological gambling is linked to reduced activation of the mesolimbic reward system. *Nature Neuroscience*, 8(2), 147-148.
- Rich, M. T., & Torregrossa, M. M. (2019). Chapter 8 maladaptive memory mechanisms in addiction and relapse. In M. Torregrossa (Ed.), *Neural mechanisms of addiction* (pp. 103-122). London: Academic Press.
- Robinson, M. J., Anselme, P., Suchomel, K., & Berridge, K. C. (2015). Amphetamine-induced sensitization and reward uncertainty similarly enhance incentive salience for conditioned cues. *Behavioral Neuroscience*, *129*(4), 502-511.
- Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving. An incentive-sensitization theory of addiction. *Brain Research Reviews*, 18(3), 247-291.

- Robinson, T. E., & Berridge, K. C. (2008). The incentive sensitization theory of addiction: Some current issues. *Philosophical Transactions of the Royal Society B*, 363(1507), 3137-3146.
- Rush, B. (1812). *Medical inquiries and observations upon the diseases of the mind*. Philadelphia, PA: Kimber & Richardson.
- Salmon, A. (2010). Couple: Internet gaming addiction led to baby's death. *Cable News Network (CNN)*. Retrieved from http://www.cnn.com/2010/WORLD/asiapcf/04/01/korea.parents.starved.baby/index.html
- Schultz, W. (1998). Predictive reward signal of dopamine neurons. *Journal of Neurophysiology*, 80(1), 1-27.
- Schultz, W. (2015). Neuronal reward and decision signals: From theories to data. *Physiological Reviews*, 95(3), 853-951.
- Schwartz, D. G. (2006). Roll the bones: The history of gambling. New York, NY: Gotham Books.
- Serenko, A., & Turel, O. (2015). Integrating technology addiction and use: An empirical investigation of Facebook users. *AIS Transactions on Replication Research*, 1(1), 1-18.
- Singer, D. G., & Singer, J. L. (2012). *Handbook of children and the media*. London: Sage.
- Skinner, B. F. (1953). *Science and human behavior*. New York, NY: Free Press.
- Sowell, E. R., Thompson, P. M., Holmes, C. J., Jernigan, T. L., & Toga, A. W. (1999). In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. *Nature Neuroscience*, 2(10), 859-861.
- The National Institute on Drug Abuse Blog Team. (2018). *Marijuana 2018*. Washington, DC: NIDA.
- Turel, O. (2019). Potential 'dark sides' of leisure technology use in youth. *Communications of the ACM*, 62(3), 24-27.
- Turel, O., & Bechara, A. (2016a). Social Networking Site use while driving: ADHD and the mediating roles of stress, self-esteem and craving. Frontiers in Psychology, 7(455).
- Turel, O., & Bechara, A. (2016b). A triadic reflectiveimpulsive-interoceptive awareness model of general and impulsive information system use: Behavioral tests of neuro-cognitive theory. Frontiers in Psychology, 7(Article 601), 1-11.
- Turel, O., & Bechara, A. (2017). Effects of motor impulsivity and sleep quality on swearing, interpersonally deviant and disadvantageous behaviors on online social networking sites. *Personality and Individual Differences*, 108(1), 91-97.
- Turel, O., Brevers, D., & Bechara, A. (2018a). Time distortion when users at-risk for social media addiction engage in non-social media tasks. *Journal of Psychiatric Research*, 97, 84-88.

- Turel, O., He, Q., Brevers, D., & Bechara, A. (2018b). Delay discounting mediates the association between posterior insular cortex volume and social media addiction symptoms. *Cognitive, Affective, & Behavioral Neuroscience, 18*(4), 694-704
- Turel, O., He, Q., Xue, G., Xiao, L., & Bechara, A. (2014). Examination of neural systems subserving Facebook "addiction." *Psychological Reports*, *115*(3), 675-695.
- Turel, O., Poppa, N. T., & Gil-Or, O. (2018c). Neuroticism magnifies the detrimental association between social media addiction symptoms and wellbeing in women, but not in men: A three-way moderation model. *Psychiatric Quarterly*, 89(3), 605-619.
- Turel, O., & Qahri-Saremi, H. (2016). Problematic use of social networking sites: Antecedents and consequence from a dual system theory perspective. *Journal of Management Information Systems*, 33(4), 1087-1116.
- Turel, O., & Qahri-Saremi, H. (2018). Explaining unplanned online media behaviors: Dual system theory models of impulsive use and swearing on social networking sites. *New Media & Society*, 20(8), 3050-3067.
- Turel, O., & R. Cavagnaro, D. (2019). Effect of abstinence from social media on time perception: Differences between low- and at-risk for social media "addiction" groups. *Psychiatric Quarterly*, 90(1), 217-227.
- Turel, O., Romashkin, A., & Morrison, K. M. (2016). Health outcomes of information system use lifestyles among adolescents: Videogame addiction, sleep curtailment and cardio-metabolic deficiencies. *PLoS One*, *11*(5), 1-14.
- Turel, O., Romashkin, A., & Morrison, K. M. (2017). A model linking video gaming, sleep quality, sweet drinks consumption and obesity among children and youth. *Clinical Obesity*, 7(4), 191-198.
- Turel, O., & Serenko, A. (2012). The benefits and dangers of enjoyment with social networking websites. *European Journal of Information Systems*, 21(5), 512-528.
- Turel, O., Serenko, A., & Giles, P. (2011). Integrating technology addiction and use: An empirical investigation of online auction users. *MIS Quarterly*, 35(4), 1043-1061.
- Twenge, J. M., Martin, G. N., & Campbell, W. K. (2018). Decreases in psychological well-being among American adolescents after 2012 and links to screen time during the rise of smartphone technology. *Emotion*, 18(6), 765-780.

- Volkow, N. D., Fowler, J. S., Wang, G. J., & Goldstein, R. Z. (2002). Role of dopamine, the frontal cortex and memory circuits in drug addiction: Insight from imaging studies. *Neurobiology of Learning and Memory*, 78(3), 610-624.
- Volkow, N. D., Wang, G. J., Fowler, J. S., Tomasi, D., Telang, F., & Baler, R. (2010). Addiction: Decreased reward sensitivity and increased expectation sensitivity conspire to overwhelm the brain's control circuit. *Bioessays*, 32(9), 748-755.
- Wade, M., Biehl, M., & Kim, H. (2006). Information Systems is not a reference discipline (and what we can do about it). *Journal of the Association for Information Systems*, 7(1), 247-269.
- Weinstein, A. (2013). Internet and videogame addiction and the neurobiological basis of behavioral addictions. *Journal of Behavioral Addictions*, 2, 5-6.
- Whitlock, T. (1999). Gender, medicine, and consumer culture in Victorian England: Creating the kleptomaniac. *Albion*, *31*(3), 413-437.
- WHO. (2018). The International Classification of Diseases ICD-11 for Mortality and Morbidity Statistics. Geneva, Switzerland: World Health Organization.
- Winokur, A., Rickels, K., Greenblatt, D. J., Snyder, P. J., & Schatz, N. J. (1980). Withdrawal reaction from long-term, low-dosage administration of diazepam: A double-blind, placebo-controlled case study. Archives of General Psychiatry, 37(1), 101-105.
- Winstanley, C. A., Cocker, P. J., & Rogers, R. D. (2011). Dopamine modulates reward expectancy during performance of a slot machine task in rats: Evidence for a 'near-miss' effect. *Neuropsychopharmacology*, 36(5), 913-925.
- Wood, S. M. W., & Bechara, A. (2014). The neuroscience of dual (and triple) systems in decision making. In V. F. Reyna & V. Zayas (Eds.), Bronfenbrenner series on the ecology of human development. *The neuroscience of risky decision making* (pp. 177-202). Washington, DC: American Psychological Association.
- Yacubian, J., & Büchel, C. (2009). The genetic basis of individual differences in reward processing and the link to addictive behavior and social cognition. *Neuroscience*, *164*(1), 55-71.
- Zhao, H. C., Qiao, L., Fan, D. Q., Zhang, S. Y., Turel, O., Li, Y. H., Li, J., Xue, G., Chen, A. T., & He, Q. H. (2017). Modulation of brain activity with noninvasive transcranial Direct Current Stimulation (tDCS): Clinical applications and safety concerns. *Frontiers in Psychology*, *8*(685).

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