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Genetic and environmental influences on problematic Internet use: A twin study

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ABSTRACT

Despite many studies on the prevalence and correlates of problematic Internet use (PIU), we know little about its etiological components. Our main aim is to find out to what extent PIU is influenced by genetic and environmental factors using the classic twin design. A total of 237 Turkish twin-pairs aged 10–25 participated in the study. PIU was measured using the 'Problematic Internet Use Scale' (PIUS) developed by Ceyhan, Ceyhan and Gürcan. For male twin-pairs, the monozygotic (MZ) twin correlations were larger than the dizygotic (DZ) twin correlations, indicating that genetic factors influenced scores on the PIUS. However, for female twin-pairs, the MZ correlations were smaller than the DZ correlations for the PIUS, showing that genetic factors did not play a role in female twin-pairs. The influence of both genetic and environmental factors was explored with model-fitting analysis. Results showed that both for the "*social comfort/benefit*" and "*negative consequences associated with the Internet use*" sub-dimensions, the best-fitting models were the ADE models whereas both for the "*excessive use*" sub-dimension and "*PIUS-Total*", the best-fittings models were the ACE models. The key result of this study is that the genetic and non-shared environmental effects are equally influential on the overall PIU in male twin-pairs.

1. Introduction

Computers, mobile-phones, and the Internet are inevitable information and communication vehicles in our daily lives. However, some individuals demonstrate problematic or addictive behaviors related to use of such technologies. Problematic Internet use (PIU) has been described as a multidimensional syndrome consisting of cognitive and behavioral symptoms that result in negative social, academic, professional, or health consequences (Caplan, 2002, 2005; Caplan & High, 2006; Davis, 2001; Davis, Flett, & Besser, 2002). This phenomenon is also labeled by researchers with various terms like *Internet addiction* (Young, 1998), *pathological Internet use* (Davis, 2001; Young & Rogers, 1998), *compulsive Internet use* (Greenfield, 1999), *excessive Internet use* (Hansen, 2002), *Internet abuse* (Morahan-Martin, 2005, 2008) and other similar terms.

PIU or Internet addiction (IA) is characterized by an individual's inability to control his/her use of the Internet, leading to feelings of distress and functional impairment of daily activities (Shapira, Goldsmith, Keck, Khosla, & McElroy, 2000). However, we now know well that the Internet itself (as a medium) is not addictive, but some content and/or interactive applications such as online shopping, gaming, chatting, gambling, social networking or cybersex appeared to play a significant role in the development of PIU/IA. Furthermore, because the classical concept of addiction used to describe a physical dependence on a substance, this type of addiction has been conceptualized as a form of behavioral (non-chemical) addiction. Some other examples of the behavioral addiction include pathological gambling, video-game playing, television viewing, and physical exercise. Shaffer et al. (2004) have expressed that their analysis of extant literature reveals that the specific objects of addiction play a less central role in the development of addiction than previously thought, and this situation identifies the need for a more comprehensive philosophy of addiction. LaRose, Lin, and Eastin (2003) highlight that genetics and family history may predispose experimentation and initial reactions to addictive behaviors, while Shaffer et al. (2004) consider genetic risk and psychosocial elements as distal antecedents of the addiction syndrome.

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Davis (2001) introduces a cognitive-behavioral model of PIU that defines two distinct types of PIU as specific and generalized PIU. According to this distinction, specific PIU includes those people that are dependent on a specific function of the Internet whereas generalized PIU involves a multidimensional overuse of the Internet (Davis, 2001). This model also hypothesizes that maladaptive cognitions and psychosocial pathology predisposes some Internet users to develop PIU/IA. Indeed, PIU/IA is associated with various psychosocial correlates or psychiatric comorbidities. Some of the variables that have been linked to PIU/IA include depression (Caplan, 2002; Ceyhan & Ceyhan, 2008; Davis et al., 2002; Yen, Ko, Yen, Wu, & Yang, 2007; Young & Rogers, 1998), anxiety disorder (Bernardi & Pallanti, 2009; Shapira et al., 2000), social phobia (Yen et al., 2007), attention deficit hyperactivity disorder (Bernardi & Pallanti, 2009; Yen et al., 2007), obsessive–compulsive personality disorder (Bernardi & Pallanti, 2009), impulsivity (Meerkerk, van den Eijden, Franken, & Garretsen, 2010), loneliness (Caplan, 2002; Ceyhan & Ceyhan, 2008; Davis et al., 2002; Morahan-Martin & Schumacher, 2000), low self-esteem (Caplan, 2002; Kim & Davis, 2009; Niemz, Griffiths, & Banyard, 2005), shyness (Odacı & Çelik, 2013), external locus of control (Chak & Leung, 2004), higher novelty seeking and lower reward dependence (Ko et al., 2010), aggressive behaviors (Ko, Yen, Liu, Huang, & Yen, 2009; Odacı & Çelik, 2013), hostility (Yen, Yen, Wu, Huang, & Ko, 2011; Yen et al., 2007), substance use (Ko et al., 2006), and problematic alcohol use (Ko et al., 2008). Besides, some researchers have been argued that comorbidity of two disorders may indicate the casual relationship and/or common etiology shared by them (e.g., Mueser, Drake, & Wallach, 1998).

Although PIU/IA has been identified as a potential mental health issue, until now it is not included as a psychiatric disorder or a mental illness in the revised fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders-IV Text Revision* (DSM-IV-TR) (American Psychiatric Association [APA], 2000) or in the tenth edition of the *International Classification of Diseases* (ICD-10). Besides, PIU/IA was considered for the DSM-V 'behavioral addiction' category but was rejected due to a lack of sufficient research evidence, however, APA decided to include it in the appendix of the DSM-V in order to stimulate future research (APA, 2010).

Some researchers, on the other hand, have suggested that PIU can be defined as an impulse-control disorder (Shapira et al., 2003; Young, 1998) or another form of an obsessive–compulsive spectrum disorder (Shapira et al., 2000). In fact, studies have shown that problematic Internet users' use of the Internet met the DSM-IV criteria for an impulse-control disorder not otherwise specified (Shapira et al., 2000), and the diminished impulse-control is an indicator of more severe PIU (Davis et al., 2002). According to Yellowlees and Marks (2007), the existence of such a relationship has caused some researchers to postulate a role of brain chemistry in addictive Internet use because many other impulse-control disorders and behavioral addictions are thought to operate dopaminergically. Indeed, more recent evidences showed that PIU/IA is associated with dysfunctions in the dopaminergic brain systems (e.g., Hou et al., 2012; Kim et al., 2011).

Beard (2005) also proposes a model to conceptualize PIU integrating biochemical, genetic, psychological, familial, environmental, and cultural dynamics. The genetic component of Beard's model recognizes that perhaps a combination of genes makes a person prone to developing addictive behaviors whereas the biochemical component of this model acknowledges that the neurochemical changes (e.g., insufficient amount of dopamine) may force a person engaging in an addictive behavior to maintain the homeostasis of his/her body. The psychological component, on the other hand, recognizes the use of learning processes to initiate, maintain, or change addictive behaviors, whereas the social component highlights familial, social, or cultural dynamics that may incite addictive behaviors.

However, we still do not have a valid answer regarding whether PIU/IA is a separate disorder, a manifestation of an underlying psychiatric illness, or both (Mitchell, 2000; Morahan-Martin, 2005, 2008; Pies, 2009; Shapira et al., 2000). Besides, Pies (2009) has summarized that a syndrome and its symptoms may be understood as a specific disease entity when at least one of the following criteria are met: (1) a pattern of genetic transmission is discovered, sometimes leading to the identification of a specific genetic locus; (2) the syndrome's etiology, pathophysiology and/or pathologic anatomy become reasonably well understood; and (3) the syndrome's course, prognosis, stability, and response to treatment are seen to be relatively predictable and consistent across many different populations (p. 35).

In the literature, however, there are a small number of molecular genetics studies (DNA researches) associated with PIU/IA. According to the findings of these studies, the genetic polymorphisms of the dopamine D2 receptor gene (DRD2 Taq1A1) and G allele of the norepinephrine transporter gene (Net-8) were found more frequently in a group of male adolescents with Internet addiction tendency (Kim, Lee, Han, Suh, & Kee, 2006); the genetic polymorphisms of the serotonin transporter gene (SS-5HTTLPR), which was associated with depression, was also found more frequently in a group of male adolescents with excessive Internet use (Lee et al., 2008); similarly, the genetic polymorphisms of the dopamine D2 receptor gene (DRD2 Taq1A1) and low activity alleles (i.e., COMTL) in the Catecholamine-O-Methyltransferase (COMT) gene were found more prevalent in a group of male adolescents with excessive Internet video game play (Han et al., 2007); and more recently, the genetic polymorphisms of the nicotinic acetylcholine receptor gene (CHRNA4) was found more frequently in the problematic Internet users (Montag, Kirsch, Sauer, Markett, & Reuter, 2012). Taken together, these findings suggest that a biological or genetic predisposition to PIU/IA may exist. However, this predisposition seems more powerful especially in males. While above mentioned molecular genetic studies have shown that relationships do exist between some genetic polymorphisms and PIU/IA, the joint contribution of both genetic and environmental influences is still vague. Thus, it is necessary to expand research on PIU/IA behaviors to consider genetic influences as well as environmental ones. Besides, as emphasized by some researchers, no twin studies investigated the heritability of PIU/IA to date (Montag et al., 2012; Pezoa-Jares, Espinoza-Luna, & Vasquez-Medina, 2012). Therefore, the main purpose of this study is to quantify the relative importance of genetic and environmental influences on PIU, using the classical twin design.

2. Methods

2.1. Design: Rationale of behavioral genetics and the classical twin design

In the present study, we adopted a quantitative behavioral genetics approach, and used the classical twin design. Quantitative behavioral genetic approaches are designed to separate out genetic influences and environmental influences by apportioning the observed differences between people (phenotypic variance) into three subcomponents: *heritability* (h^2), *shared* (*common*) *environment* (c^2 or C), and *non-shared* (*unique*) *environment* (e^2 or E) (Plomin, DeFries, McClearn, & Rutter, 2001). Heritability is a statistic that describes the proportion of observed variance for a behavior that can be ascribed to genetic differences among individuals in a particular population (Plomin, 1989). It should be noted however that the effects of some genes may be independent of other genes

(additive), or alternatively, the effects of some genes may be 'non-additive' and depend on other genes, either at the same locus (dominance) or at other loci (epistasis) (McGue & Bouchard, 1998; Neale & Maes, 2004; Sherman et al., 1997). The heritability of any human behavior can be estimated by comparing the correlation levels of reared-together monozygotic (MZ) and reared-together dizygotic (DZ) twins. This method is known as the classical twin design. Because MZ twins are genetically identical – whereas DZ twins share only half their variable genes, on average – it is assumed that differences in intra-pair correlations can be used to estimate the amount of the total variation that is genetic in origin (Richardson & Norgate, 2005). As mentioned earlier, this genetic contribution (heritability) may include additive genetic influences (a^2 or A) and/or non-additive (dominance or epistasis) genetic influences (d^2 or D). Environmental influences can also be calculated in the classical twin design. Shared environmental influences (e.g., parenting style, socio-economic status, etc.) increase similarities between twins, whereas non-shared environmental influences (e.g., differential prenatal exposure, differential parental treatment, or different educational/school experiences, etc.) make twins different from each other.

The classical twin design adopts the equal environment assumption. This assumption suggests that the shared environment has the same influence on MZ and DZ twins. Although the validity of the equal environment assumption has been debated, its validity was confirmed in recent studies (e.g., Derks, Dolan, & Boomsma, 2006; Hettema, Neale, & Kendler, 1995; Xian et al., 2000). Table 1 displays the genetic hypothesis testing for a single continuous trait in the classical twin design (Duffy, 1994).

Model-fitting in twin studies is a sophisticated method of estimating genetic and environmental effects (Rijsdijk & Sham, 2002). In the classical twin design, however, it is not possible to test an ACDE model including effects of additive genetic (A or a^2), shared environment (C or c^2), non-additive genetic (D or d^2), and non-shared environment (E or e^2). Therefore, researchers must limit their comparisons to sub-models including three of the four sources of individual differences: ACE model (including additive genetic, shared environment and non-shared environment), or ADE model (including additive genetic, non-additive genetic and non-shared environment) (Franić, Dolan, Borsboom, & Boomsma, 2012). If the DZ correlation is greater than the half of the MZ correlation, the ACE model is the correct model and the estimate of D in the ADE model is always zero. However, if the DZ correlation is less than half of the MZ correlation, the ADE model is the correct model and the estimate of C in the ACE model is zero (Rushton, Bons, & Hur, 2008).

2.2. Participants and procedure

The participants consisted of 237 Turkish twin-pairs ($n = 474$). There were 270 females (57%) and 204 males (43%) ranging in age from 10 to 25 ($M = 15.39$, $SD = 3.73$). Of the twins, 80 pairs (34%) were MZ (identical) twins (49 female and 31 male), and 157 pairs (66%) were DZ (fraternal) twins. Of the 157 DZ twins, 98 pairs (62%) were same-sex twins (57 female and 41 male) and 59 pairs (38%) were opposite-sex twins. Zygosity was determined by the twins' self-report and physical similarity. 206 Twin-pairs (87%) reported that they had access to a computer and Internet connection at home. However, majority has either mobile-phone with Internet connection or has access to a computer with Internet connection at their schools. All of the twins were reared together. Before the survey questionnaire was administered, participants were informed about the purpose of this study and told that they had right to withdraw from the study at any time during or after the study. All the participants volunteered for the study and no time limit was imposed for the completion of the questionnaire. Instructions for accomplishing the task were presented in both written and verbal forms. In addition, all of the participants responded to the items independent of communication with their twins.

Table 1
Genetic hypothesis testing for a single continuous trait in the classical twin design.[a]

Relationship	Interpretation
$r_{MZ} > 4r_{DZ}$	Epistasis
$r_{MZ} > 2r_{DZ}$	Genetic dominance (or epistasis; shared environment small)
$2r_{DZ} > r_{MZ} > r_{DZ}$	Additive genes and shared environment (genetic dominance small)
$r_{MZ} = 2r_{DZ}$	Additive genetic effect – either monogenic or polygenic
$r_{MZ} = r_{DZ} > 0$	No genetic contribution – effects of family environment
$r_{MZ} = r_{DZ} = 0$	No familial aggregation

[a] *Source:* Duffy (1994). r_{MZ} = MZ intra-pair correlation; r_{DZ} = DZ intra-pair correlation.

2.3. Measure

2.3.1. Problematic Internet use scale

The Problematic Internet Use Scale (PIUS) is a 33-item self-report scale yielding a total possible score ranging from 33 to 165. This scale is for measuring the generalized PIU, and developed by Ceyhan, Ceyhan, and Gürcan (2007) in the Turkish language. The PIUS is well validated, and factor analyses show its unidimensional nature. However, the PIUS has also shown adequate psychometric properties: it consists of three factors: factor 1 – negative consequences associated with Internet use (17 items), factor 2 – social comfort/benefit (10 items), and factor 3 – excessive use (6 items). The definitions of the PIUS factors (subscales) are as follows: (1) *Negative consequences associated with Internet use*: the severity of personal, social and academic (school-related) problems resulting from one's Internet use. A sample item from this factor includes "*I can't stop thinking about the Internet when I don't have access to the Internet*". (2) *Social comfort/benefit*: the extent to which an individual perceives Internet use as entailing greater social benefits than face to face communication. A sample item from this factor includes "*I feel very free when I am on the Internet*". (3) *Excessive use*: the degree to which an individual feels that she or he spends an excessive amount of time online. A sample item from this factor includes "*I do not spend more time online than I intend to*". The items of PIUS are scored on a 5-point Likert-type scale (1 = not at all true of me and 5 = very true of me). Higher scores are associated with unhealthy use of the Internet, therefore can be considered to be a predisposition to risk of Internet addiction. In this sample, internal consistency coefficients (Cronbach's alphas) were 0.91, 0.81, 0.71, and 0.93 for negative consequences associated with Internet use, social comfort benefit, excessive use, and the entire scale respectively.

2.4. Statistical analyses

First of all, means and standard deviations for sex and zygosity groups were calculated for the PIUS and its three subscales, and to investigate whether levels of PIU significantly differ from in MZ and DZ twin-pairs, paired *t*-test comparisons were performed. Then, twin resemblance in PIUS and its sub-dimensions was assessed by maximum likelihood estimates of correlation coefficients. Comparing the MZ twin correlations with the DZ twin correlations provided a first estimate of the sources of variation in individual differences in PIU. When comparing the results of correlations, the interpretation scheme given in Table 1 was used regarding the genetic hypothesis testing for a single continuous trait in the classical twin design. In the present study, the influence of both genetic and environmental factors was explored with model-fitting analysis. Models were fitted using the structural

equation modeling software package OpenMx (Boker et al., 2011) to dissect genetic and environmental variance components. Based on the twin correlations, two types of model were examined only for the male twin-pairs: ACE (additive genetic (a^2), shared environmental (c^2), and non-shared environmental (combined with measurement error) (e^2) influences) and ADE (additive genetic (a^2), non-additive (dominance or epistasis) genetic (d^2), and non-shared environmental (combined with measurement error) (e^2) influences). In the beginning, a saturated model was fitted to estimate the correlations between twin pairs. In the model-fitting phase, the saturated model is used as a starting-point for the comparison of different nested models. The fit of the various models are evaluated using likelihood ratio tests in which the negative log-likelihood (−2LL) of the nested model is compared with −2LL of the saturated model. Goodness-of-fit was assessed by the Akaike Information Criterion (AIC = $\chi^2 - 2[df]$) than hypothesis based approaches. AIC provides a simple and effective means for the selection of best approximating model for inference. The model with the smallest AIC values is the best model among all models specified (Burnham & Anderson, 2002).

3. Results

3.1. Descriptive statistics

For the entire sample, the mean PIUS total score was 67.62 (*SD* = 23.37). Table 2 shows three categories for the PIUS scores and the percentage of participants within each category, indicating that 33.8% of the participants exhibited low-to-moderate levels of PIU whereas 10.5% of the participants exhibited moderate-to-high levels of PIU.

The means and standard deviations for each of the PIUS subscales by sex and zygosity are listed in Table 3. No significant mean differences were found between MZ co-twins or between same-sex DZ co-twins. However, significant mean differences were found between opposite-sex DZ co-twins. According to the paired *t*-test results, males appeared to be more problematic Internet users than their female co-twins (excessive use: $t_{(58)} = -3.631$, $p < 0.05$; social comfort/benefit: $t_{(58)} = -3.842$, $p < 0.05$; negative consequences: $t_{(58)} = -4.016$, $p < 0.05$; PIU-total: $t_{(58)} = -4.473$, $p < 0.05$).

3.2. Twin correlations

Table 4 shows the maximum likelihood estimates of correlations with pairs of twins and 95% confidence intervals (CI) for the PIUS and its subscales. For male twin-pairs, the MZ-$_{MM}$ correlations were at least twice larger than the DZ$_{SS}$-$_{MM}$ correlations, indicating that genetic factors influenced scores on the PIUS. This was not the case for female twin-pairs, where MZ-$_{FF}$ correlations were no larger than DZ$_{SS}$-$_{FF}$ correlations. In fact, for female twin-pairs, the MZ correlations were smaller than the DZ$_{SS}$ correlations for the PIUS and its three subscales, showing that genetic factors did not play a role in female twin-pairs. Besides, another sex-specific effect was detected, namely the opposite-sex DZ correlations tended to be much smaller than the same-sex DZ correlations, indicating that the intra-pair similarity in DZ twins varied as a function of sex.

Table 2
Three categories for PIU scores and the percentage of participants within each.[a]

PIU levels	Range	%
None to low	33–66	55.7
Low to moderate	67–99	33.8
Moderate to high	100–165	10.5

[a] (n = 237 twin-pairs).

3.3. Univariate model-fitting analyses

It is important to keep in mind that the heritability estimates are zero for any trait in which the DZ correlation is equal to or larger than the MZ correlation. Therefore, in the present study, genetic models for female twin-pairs could not be fitted. However, for male twin-pairs, the correlation ratio between MZ and DZ twins was appropriate for the genetic model-fitting. For this reason, we first examined the PIUS scores and the percentage of MZ-$_{MM}$ and (same-sex) DZ$_{SS}$-$_{MM}$ participants within each category. Table 5 shows these data, indicating that 39.95% of the MZ-$_{MM}$ twins and 45.15% of the DZ$_{SS}$-$_{MM}$ twins exhibited low-to-moderate levels of PIU, whereas 16.25% of the MZ-$_{MM}$ twins and 12.20% of the DZ$_{SS}$-$_{MM}$ twins exhibited moderate-to-high levels of PIU.

Finally, Table 6 displays the results of model-fitting and parameter estimates of the univariate models for PIU in male twins.

For the "*excessive use*" sub-dimension, the best-fitting model was the ACE model where the additive genetic component accounted for 19% of the variance, the shared environment accounted for 18.9% of the variance, and the non-shared environment accounted for 61.9% of the variance. For the "*social comfort/benefit*" sub-dimension, the best-fitting model was the ADE model where the additive genetic component accounted for 21.2% of the variance, the non-shared environment accounted for 44.5% of the variance, and the dominance genetic component accounted for 34.2% of the variance. For the "*negative consequences associated with the Internet use*" sub-dimension, the best-fitting model was the ADE model where the additive genetic component accounted for 01% of the variance, the non-shared environment accounted for 13.5% of the variance, and the dominance genetic component accounted for 86.4% of the variance. Lastly, for the "*problematic Internet use*" in general, the best-fitting model was the ACE model where the additive genetic component accounted for 41.7% of the variance, the shared environment accounted for 16.7% of the variance, and the non-shared environment accounted for 41.5% of the variance.

4. Discussion

To the best of our knowledge this is the first study to examine the genetic and environmental etiology of PIU, using the classical

Table 3
Means and standard deviations of the PIUS by sex and zygosity.

Zygosity	MZ				DZ$_{SS}$				DZ$_{OS}$			
Sex	Females		Males		Females		Males		Females		Males	
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Excessive use	16.15	5.14	17.56	5.69	16.99	5.85	17.61	5.09	15.13	5.06	18.23	5.30
Social comfort	18.62	6.88	22.32	9.34	20.03	8.23	21.00	7.74	18.03	6.20	22.30	8.10
Negative consequences	9.39	3.91	11.85	5.85	10.85	5.63	13.09	9.48	9.30	4.21	12.35	5.39
PIU-total	61.54	17.57	73.61	26.97	67.19	26.21	72.07	21.91	59.55	19.94	74.10	22.97

Note: MZ = Monozygotic Twins; DZ$_{SS}$ = Dizygotic Same-Sex Twins; DZ$_{OS}$ = Dizygotic Opposite-Sex Twins.

Table 4
Maximum likelihood estimates of correlations and 95% confidence intervals for the PIUS and its subscales.

Measure		Excessive use		Social comfort		Negative consequences		PIU-total	
Zygosity	n[a]	r	95% CI	r	95% CI	r	95% CI	r	95% CI
MZ_{FF}	49	.501**	.260–.683	.429**	.171–.631	.279*	.002–.517	.369**	.102–.586
MZ_{MM}	31	.421*	.089 –.671	.619**	.348–.795	.661**	.409–.820	.651**	.394–.815
MZ_{Total}	80	.470**	.281–.624	.549**	.376–685	.525**	.347–.666	.565**	.395–.697
$DZss\text{-}_{FF}$	57	.590**	.393–.736	.481**	.255–.657	.672**	.502–.793	.657**	.481–.782
$DZss\text{-}_{MM}$	41	.276*	–.028 to .535	.170	–.139 to .450	.002	–.301 to .305	.332**	.033–.577
$DZss_{Total}$	98	.475**	.307–.617	.358**	.173–.519	.196*	–.001 to .379	.548**	.393–.672
$DZos\text{-}_{FM}$	59	.108	–.149 to .352	.202	–.053 to .434	.165	–.091 to .402	.197	–.059 to .429

Note: MZ = Monozygotic Twins; DZss = Dizygotic Same-Sex Twins; DZ_{OS} = Dizygotic Opposite-Sex Twins; F = Female; M = Male.
* $p < .05$.
** $p < .001$.
[a] "*n*" represents the number of twin-pairs.

Table 5
Three categories for PIU scores and the percentage of $MZ\text{-}_{MM}$ and $DZss\text{-}_{MM}$ twins within each.

PIU levels	Range	$MZ\text{-}_{MM}$ (%)	$DZ\text{-}_{SS\text{-}MM}$ (%)
None to low	33–66	46.80	42.65
Low to moderate	67–99	36.95	45.15
Moderate to high	100–165	16.25	12.20

twin design. For male twin-pairs, we found that all the MZ correlations were higher than the DZ_{SS} correlations for the PIUS and its three subscales. However, for female twin-pairs, the MZ correlations were smaller than the DZ_{SS} correlations, showing that genetic factors did not play a role in female twin-pairs. With these findings, it seems reasonable to suggest that genetic influences are important for PIU in male twin-pairs. Besides, the opposite-sex DZ correlations tended to be much smaller than the same-sex DZ correlations. According to Boomsma, Busjahn, and Peltonen (2002), this situation indicates that different genes might influence the same trait in the two sexes. In other words, this is to say that there is a significant sex difference in the etiology of the PIU. These findings are consistent with the findings of available molecular genetics studies, mainly showing that a biological or genetic predisposition to PIU may exist especially in males (Han et al., 2007; Kim et al., 2006; Lee et al., 2008).

To estimate genetic and environmental contributions to PIU, we used the model-fitting analyses. The results of the model-fitting suggest that individual differences in the "*excessive use*" sub-dimension were mainly (61.9% of the variance) due to the non-shared environmental factors. Morahan-Martin (2008) highlighted that the importance of learning processes in the development of vulnerability to PIU/IA, and summarized that any behavioral addiction can be understood as learned adaptive or functional behavior in the context of personal or environmental factors. Thus, the features of a unique environment experienced by an individual twin may increase the level of their Internet use. According to Beard (2005), accessibility and modeling could play a role in this process, specifically, the greater the availability of the Internet, the increased chance that people will engage in Internet activities. In the literature, on the other hand, there is evidence of association between excessive Internet use and psychosocial problems such as low self-esteem and anxiety (Kim & Davis, 2009), social phobia (Yen et al., 2007), social isolation (Young & Rogers, 1998), and shyness (Odacı & Çelik, 2013). Therefore, future studies should try to identify specific sources of non-shared environmental contributions to excessive Internet use. Interestingly, one twin study concerning genetic and environmental influences on media use and communication behaviors revealed that individual differences in hours spent using the Internet in the previous 7 days were mainly (67% of the variance) due to the non-shared environmental influences, whereas the additive genetic contribution was only 10% (Kirzinger, Weber, & Johnson, 2012). Another twin study concerning the heritability of mobile phone use showed that the non-shared environmental influences on mobile phone talk and text messaging frequencies were modest to moderate, ranging from 28% to 45% (Miller, Zhu, Wright, Hansell, & Martin, 2012). The same study also showed that the additive genetic contributions to mobile phone talk and text messaging frequency ranged from 34% to 60%. Therefore, comparisons of genetic and environmental factors on the use frequency of different information and communication technologies deserve future examination.

For the "*social comfort/benefit*" sub-dimension, the $r_{MZ} = 0.619$ and the $r_{DZSS} = 0.170$ whereas for the "*negative consequences associated with the Internet use*" sub-dimension, the $r_{MZ} = 0.661$ and $r_{DZSS} = 0.002$, indicate evidence of substantial genetic non-additivity (i.e., dominance or epistasis). As is known, if non-additive genetic effects are important for a particular trait, the intra-pair correlation of DZ twins will be less than half the intra-pair correlation of MZ twins (Plomin, DeFries, & McClearn, 1990). In the current study, the $MZ\text{-}_{MM}$ intra-pair correlations both for "*social comfort/benefit*" and "*negative consequences associated with the Internet use*" sub-dimensions exceed twice the values of the $DZ_{SS}\text{-}_{MM}$ intra-pair correlations, indicating a genetic non-additivity. Thus this provides the best estimate of total genetic influence (broad-sense heritability). However, if non-additive effects are substantial, MZ and DZ twin correlation comparison overestimates genetic influence (Sherman et al., 1997). Therefore, the evidence for genetic non-additivity found in this study should be replicated using larger and more representative twin samples.

Based on above mentioned correlation coefficients, the model-fitting heritability estimates (the sum of a^2 and d^2) were 55.4% for the "*social comfort/benefit*" and 86.5% for the "*negative consequences associated with the Internet use*" sub-dimensions. The clear implication of these heritability estimates is that genes matter more than shared (family) environments in predicting how and why individuals use the Internet. The results of a study on PIU showed that individuals who are lonely tend to use the Internet for the purpose of social comfort (Davis et al., 2002). The results of another study on PIU also showed that the most strongly correlated psychosocial indicators of wellbeing with negative outcomes of the Internet use were loneliness and low self-esteem (Caplan, 2002). In the related literature, there are numerous studies showing that both loneliness (e.g., Boomsma, Willemsen, Dolan, Hawkley, & Cacioppo, 2005; McGuire & Clifford, 2000) and self-esteem (e.g., Kendler, Gradner, & Presscott, 1998; Raevuori et al., 2007) have a genetic component. Moreover, as mentioned earlier, it is argued that comorbidity of two disorders may indicate the casual relationship and/or common etiology shared by them (e.g., Mueser et al., 1998). Therefore, the investigation of whether

Table 6
Results of model fitting and parameter estimates for PIU in male twins.

PIUS	Model	Parameter estimates						Fit of model				
		a^2	c^2	e^2	d^2	Ep	−2LL	*df*	AIC	Δ−2LL	Δ*df*	*p*
Excessive use	SATM	–	–	–	–	10	876.85	134	608.85	–	–	–
	ADE	0.406	–	0.593	0.001	4	883.820	140	603.820	6.97	6	0.323
	ACE[a]	0.190	0.189	0.619	–	4	883.501	140	603.501	6.65	6	0.354
	AE	0.406	0	0.593	–	3	883.820	141	601.820	6.97	7	0.431
	CE	0	0.332	0.667	–	3	883.730	141	601.730	6.88	7	0.441
	E	0	0	1.000	–	2	892.181	142	608.181	15.33	8	0.053
Social comfort	SATM	–	–	–	–	10	1000.52	134	732.52	–	–	–
	ADE[a]	0.212	–	0.445	0.342	4	1006.332	140	726.332	5.81	6	0.444
	ACE	0.541	0.001	0.458	–	4	1006.573	140	726.575	6.05	6	0.417
	AE	0.541	0	0.459	–	3	1006.572	141	724.572	6.05	7	0.533
	CE	0	0.397	0.602	–	3	1009.961	141	727.964	9.43	7	0.222
	E	0	0	1.000	–	2	1022.327	142	738.327	21.81	8	0.005
Negative consequence	SATM	–	–	–	–	10	949.65	134	681.65	–	–	–
	ADE[a]	0.001	0	0.135	0.864	4	1018.007	140	738.007	68.36	6	0.000
	ACE	0.854	0.001	0.145	–	4	1028.801	140	748.801	79.16	6	0.000
	AE	0.854	0	0.145	–	3	1028.801	141	746.801	79.16	7	0.000
	CE	0	0.113	0.886	–	3	1038.929	141	756.929	89.28	7	0.000
	E	0	0	1.000	–	2	1039.859	142	755.859	90.21	8	0.000
PIUS-total	SATM	–	–	–	–	10	1295.3	134	1027.3	–	–	–
	ADE	0.594	–	0.405	0.001	4	1304.211	140	1024.21	8.91	6	0.178
	ACE[a]	0.417	0.167	0.415	–	4	1303.938	140	1023.938	8,64	6	0.194
	AE	0.595	0	0.405	–	3	1304.211	141	1022.211	8.91	7	0.258
	CE	0	0.492	0.508	–	3	1305.547	141	1023.547	10.25	7	0.174
	E	0	0	1.000	–	2	1325.453	142	1041.453	30.16	8	0.001

Note: SATM; Fully Saturated Model; a^2 = additive genetic, c^2 = shared environmental, e^2 = non-shared environmental, and d^2 = dominant genetic proportion of the variance; Ep = Estimated parameters; AIC: Akaike's Information Criterion.
[a] Indicates the best-fitting model for each PIU dimension.

pre-existing psychosocial problems or psychopathologies that are linked with PIU have an impact on the heritable part of PIU, is another research avenue that needs to be explored. In addition, Miller et al.'s (2012) study demonstrated that there are modest negative correlations between intelligence and mobile phone usage, showing that some of the same genetic variants that increase intelligence also tend to decrease mobile phone use frequency. Thus, cognitive factors such as intelligence and aptitude should also be examined in terms of their relationships with different aspects of PIU.

This study also found that individual differences in *Total-PIU* in male twin-pairs were due equally to both the additive genetic (41.7%) and non-shared (41.5%) environmental factors. The shared environmental factors, on the other hand, did not explain a significant portion of phenotypic variation. These findings are consistent with the findings of many other behavioral genetics studies showing that environmental influences on most behavioral traits are of the non-shared rather than shared variety (McGue & Bouchard, 1998). This is not to say that shared environmental factors may not be important in the development of PIU. Instead, the influence of shared environment may be intertwined with the genetic source of variance, especially in the classical twin design. Therefore, these findings should be replicated using the adoption and family designs.

This study examined the heritability of PIU within the context of Turkish culture. Because heritability is a statistic that describes genetic contributions to the phenotypic variance of a trait within a particular population of individuals, the heritability of any given trait may not necessarily be the same for all cultures (Saudino et al., 1999). Sherman et al. (1997) emphasized that heritability is a descriptive statistic of a trait in a particular population, not of a trait in an individual. Thus, heritability estimates like any statistics, can change over time and vary from population to population (McGue & Bouchard, 1998; Sherman et al., 1997). Therefore, future research should examine the heritability of PIU in different cultures. Cross-cultural studies can be especially useful to enhance understanding of the role of genetic factors on PIU. In addition, genetic and environmental influences on PIU can change throughout development. According to Plomin et al. (2001), the heritability of many behavioral traits tends to increase with age. Hence, researchers need to know much about whether genetic or environmental influences are more important for PIU in early childhood, adolescence, adulthood, or aged populations. Therefore, future research should investigate the heritability of PIU by using both cross-sectional and longitudinal research designs. In addition, it would be instructive and important to obtain comparative behavioral genetic analyses of PIU/IA especially measured by different valid and reliable PIU/IA scales or inventories or diagnostic criteria checklists to ascertain whether genetic and environmental influences vary or not by means of different measurement tools. Besides, in this study data collected using self-reports. The use of self-reports may have resulted in the common method variance (CMV). Therefore, future research could employ a multi-trait multi-method matrix (MTMT). MTMT is an approach that minimizes CMV (Campbell & Fiske, 1959).

5. Limitations

The major limitation of the study is the small sample-size. It should be noted that it was too difficult to find twins for this study, because there is neither clinical nor population-based twin registries in Turkey. The lack of a school-based twin registry also contributed to the difficulties encountered in collecting twin data. In addition, the relatively low rate of problematic or addictive Internet users in the general population made it hard to collect a large twin sample of problematic Internet users. Therefore, the results of the present study should be interpreted with caution since it has low power due to the relatively small sample-size (especially when fitting models to data for male twin-pairs). As Neale and Maes (2004) highlighted, smaller sample-size in twin studies can lead to finding larger effects. Thus, the model-fitting

results of the present study should be considered suggestive rather than definitive or conclusive. Future research with larger and more representative twin samples is needed to confirm the findings of the present study. Besides, studies based on both concordant twin design (in which each co-twin is identified as a problematic Internet user) and discordant twin design (in which one twin is identified as a problematic Internet user) should be conducted. These designs would permit a comparative exploration of psychosocial correlates, psychiatric comorbidities as well as other symptomatic behaviors regarding PIU. Future studies should also investigate the roles of possible traits that may mediate genetic and environmental contributions to the PIU, such as specific personality traits (e.g., high novelty seeking, harm avoidance, risk-taking, low self-directedness) and existing psychosocial problems (e.g., depression, loneliness).

6. Conclusion

The present study was designed to investigate the etiological basis of PIU. Although it is premature to draw strong conclusions from the findings of the present study due to its small sample size, it would help serve as a starting point for future research. Despite the fact that Internet use is highly widespread in both developed and developing countries, PIU appears to be on the increase around the world. Therefore, it is concluded that better understanding of its etiological components may lead to proper delineation and evaluation of those factors necessary in alleviating it.

Disclosure statement

The authors declare that they have no conflicts of interest.

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