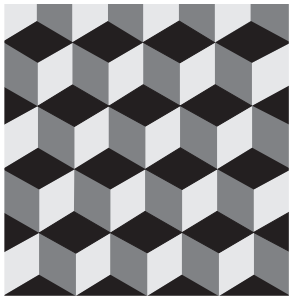

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Special issue:
Behavioral Genetics: Relation to Psychological Science

No4, 2018

Sadržaj

Contents

INTRODUCTION TO THE SPECIAL ISSUE

The nature versus nurture debate is one of the oldest in psychology, and has been a central issue in theorizing about the causes of individual differences. As early as 1582, Richard Mulcaster, who worked as a teacher, used the words "nature" and "nurture" in attempt to describe the factors that influence children's development. However, it was not until 1869 that Francis Galton conducted the first studies which aimed to investigate this problem in a scientific manner. Galton was prone to believe that heredity was a far more powerful agent in human development than nurture. Despite his failure to consider environmental factors in the familial clustering, Galton is regarded to be the starting point in the long process of establishing the clear view about genetic and environmental backgrounds of psychological phenomena.

Nowadays, the basic debate of whether nature prevails over nurture or vice versa, seems to be resolved: both are important for psychological development. Nonetheless, questions about the contributions of genes and experiences to trait origins continue to intrigue the researchers. The modern field of behavioral genetics takes two different approaches to the study of how genes contribute to behavior. The first one, which is developed within the field of biology, uses population genetics methods to explore the way genes contribute to behavior. This approach also characterizes a subdiscipline of psychology known as quantitative behavioral genetics. Second, newer approach has emerged of molecular biology, and is focused on exploring the structure and function of biological molecules such as DNA. This approach is known as molecular behavioral genetics.

Over a century after Galton's work, twin studies remain a favorite tool of behavioral geneticists. This design is based on comparing the observed similarity of members of monozygotic (identical) twin pairs to the observed similarity of members of fraternal (dizygotic) twin pairs. Prominent similarity among the identical twins indicates that genetic factors contribute to the observed similarity, rather than environment. This method is being used to estimate the heritability of traits, i.e., the percentage of variance in a population due to genes. The modern twin studies also intend to quantify the effect of a person's shared environment (family) and unique environment (the individual events that shape a life) on a specific trait.

This special issue of Primenjena psihologija presents a selection of papers which came out from two different twin studies conducted in Germany and Serbia. German study represents Twin Life study, i.e., a longitudinal twin family study that examines more than 4000 same-sex twin pairs and their family's representative for twin families in Germany. In this study, a Nuclear Twin Family Design (NTFD) is being used. Within this type of design, data of parents and available siblings of the twins were collected in addition to data of monozygotic and dizygotic twins reared together. Serbian twin study, on the other hand, is conducted by the Centre

for Behavioral Genetics, which was founded in 2013 as a separate organizational unit of the Faculty of Philosophy, University of Novi Sad. It is the only research centre for behavioral genetics in Serbia, which count over 350 investigated twin pairs, both mono- and dizygotic.

The papers included in this volume illustrate application of the twin study on the wide range of psychological topics such as: achievement motivation, personality traits and satisfaction with life, emotion regulation and satisfaction with life, sensation seeking and risky behavior, aggressiveness and impulsiveness. Finaly, the issue is completed with the paper that describes the validation procedure of zygosity assessment by a self-report questionnaire.

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GENETIC AND ENVIRONMENTAL FACTORS IN EMOTION REGULATION AND LIFE SATISFACTION: A TWIN STUDY[2]

An expansion of the mental health research at the end of the 20th century largely places well-being in the focus of interest of contemporary psychological science. However, the state of the art results of behavioral-genetic studies provide a complete framework of the factors that influence the indicators of subjective well-being through the specification of etiology of their relationship. The main aim of this study is to evaluate genetic and environmental factors that contribute to connections among life satisfaction measures and emotional regulation. The study included 182 pairs of twins of both sexes (121 monozygotic and 61 dizygotic twin pairs), aged 18-48. The proportion of individual sources of covariance between the examined phenotypes was tested with a multivariate biometric method. Genetic factors explained a slightly higher variance of life satisfaction (53%), while the environmental factors had a significant role in explaining different types of emotional regulation. General genetic factors were potentially important only in the explanation of the cognitive reappraisal of negative emotions. In other cases, the environmental factors were of the greater importance. An insight into the phenotypic correlations suggests these constructs have low to moderate intercorrelations; likewise genetic factors have a potential significance (45%) merely in the case of two types of cognitive reappraisal of emotions.

Key words: cognitive reappraisal, emotion regulation, emotional suppression, satisfaction with life, twin study

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Introduction

In the psychology of mental health, contemporary theories of well-being are based on two different approaches. The first approach emphasizes a subjective appraisal and personal experience, uses hedonism as the main determinant of well-being affected by positive and negative emotions, as well as a subjective evaluation of satisfaction with one's own life (Diener et al., 2010). The second approach derives from the eudaimonistic conceptualization of well-being, explaining it throughout the growth and development of human potential through autonomy, relationships with other people, self-acceptance, finding the meaning of life, and personal progress (Ryff, 1989). Researchers often combine these two approaches, and, in many cases, consequently do not use subjective and psychological well-being as exclusive constructs. In this view, current models of subjective well-being largely rely on the idea that the experience of well-being depends on both cognitive and affective components. Considering the results of previous physiological studies, it can be concluded that the affective and cognitive processes are linked, both through a common neurohumoral basis (Ledoux & Phelps, 2008), and the emotional regulation, like perception process, attention and decision-making (Storbeck & Clore, 2007). It seems that studying the well-being concepts at the behavioral-genetic level is of utmost importance for determining the influence of genetic and environmental factors on the development of different cognitive and emotional potentials, such as life satisfaction and emotional regulation.

Satisfaction with Life and Emotion Regulation: Operationalization and Relations

Diener (Diener, 1984) defines the concept of the subjective well-being as tripartite: life satisfaction, a high level of positive affect, and a low level of negative affect. The satisfaction with life is a cognitive component of the subjective well-being, refering to the perception and evaluation of an individual about the quality of one's own life (Diener, Suh, Lucas, & Smith, 1999). Although there are dilemmas about the operationalization of this construct (Diener, Lucas, & Oishi, 2002), most researchers agree with the statement that satisfaction with life as a construct has an evaluation character, through which every individual globally assesses his/her own life. The results of the previous studies indicate that the satisfaction with life is of great importance for mental health, regarding a positive relationship with marriage and partnerships (Powdthavee, 2009), a lower level of stress (Argyle, 1999), general health status (Diener & Chan, 2011), and other indicators of subjective well-being.

The emotional regulation is a key aspect of emotional processes that mediate between different preconditions for reporting emotions and the actual emotional response, also affecting psychological and physiological functioning (Gross & John, 2003). According to Gross (1998), the emotional process has a temporal

character, and begins either by some activating external event, or by the mental representation a person evaluates, regardless of whether an activating event in the external environment has actually occurred. Evaluation of the event or the mental representation further triggers a series of behavioral and physiological responses that can be regulated before the real manifestation of an emotion (Gross & Thompson, 2007), through processes that are carried out before a full development of an emotional response, and directed to an activating, emotionally generating situation, but also through the reduction of physiological response, and a reduction of inadequate response modes after developing a complete emotional response (Gross & John, 2003).

According to the model, the authors distinguish two time-distanced and qualitatively different types of the emotional regulation: a cognitive reappraisal and an emotional suppression (Gross & John, 2003). While the cognitive reappraisal is a process of reinterpretation of an activating event that changes the emotional response in an adaptive way, the emotional suppression represents a process of regulating one's own emotional reaction in order to avoid its manifestation (Gross, 1998). When it comes to the emotional suppression, a series of research results point to its dysfunctional nature. For example, the emotional suppression causes an increase of inadequate physiological response and a reduction in memory capacity (Richards & Gross, 2000), as well as a lower quality of interpersonal relationships (Srivastava, Tamir, McGonigal, John, & Gross, 2009). At the same time, the level of the emotional suppression is partly modulated by both cultural factors and / or environmental influences (Kim et al., 2011). The cognitive reappraisal, which is directed to regulation of activating and usually negative situations, most often has positive outcomes of the emotional regulation for a consequence, such as improving the work performance, increasing the enthusiasm (Leroy, Grégoire, Magen, Gross, & Mikolajczak, 2012), healthier functioning of the cardiovascular system (Mauss, Cook, Cheng, & Gross, 2007), reduction of the distress and physiological reactions (Wolgast, Lundh, & Viborg, 2011), as well as the establishment of more functional social relationships (Gross & John, 2003). It is important to note that the cognitive reappraisal refers not only to the reinterpretation of negative emotions, but it can also be a process directed to positive emotions in order to increase their effects (Mauss & McRae, 2016). According to the concepts of Mauss and McRae (Mauss & McRae, 2016), a distinction between the cognitive reappraisal of positive and negative emotions is also based on different levels of activation of their common physiological bases.

Results of numerous studies aiming to explore connections of satisfaction with life with the emotional regulation point to majority of conclusions about thier positive relationships with cognitive reappraisal (Gross & John, 2003; Haga, Kraft, & Corby, 2009; Perrone-McGovern, Simon-Dack, Beduna, Williams, & Esse, 2015; Yiğit, Özpolat, & Kandemir, 2014), and negative relationships with emotional suppression (Gross, Richards, & John, 2006; Haga et al., 2009; Randal, Rickard, & Vella-Brodrick, 2014; Soto, Perez, Kim, Lee, & Minnick, 2011). However, some

researchers conclude that there are no significant relationships (e.g., Ciuluvica, Amerio, & Fulcheri, 2014). When it comes to the impacts of two types of emotional regulation on life satisfaction, the results are largely consistent with the results of exploring basic relationships (e.g., Yiğit et al., 2014). This conclusion is supported by Hua and associates in a meta-analysis study (Hu, Zhang, & Wang, 2015), but there are also findings on the absence of these effects (Liliana & Nicoleta, 2014).

Behavioral Genetics Perspective of Satisfaction with Life and Emotion Regulation

Subjective well-being is a relatively new and unexplored construct in behavioral-genetic studies compared to some other psychological constructs (e.g., the intelligence or personality traits). The expansion of behavioral-genetic research of subjective well-being has began just at the end of the 20th century and the beginning of the 21st century. The contribution of genetic factors to the manifestation of various indicators of subjective well-being has varied from 0% for the positive affect (Baker, Cesa, Gatz, & Mellins, 1992) to 62% for the ability to achieve positive interpersonal relationships (Gigantesco et al., 2011). The most common finding is that genetic factors explain about 50% of variance in the manifestation of the general subjective well-being (Røysamb, Harris, Magnus, Vittersø, & Tambs, 2002; Røysamb, Tambs, Reichborn-Kennnerud, Neale, & Harris, 2003). In addition, genes may have a qualitatively different form of influence on these constructs: protective or plastic (e.g., Belsky et al., 2009). The results of various behavioral-genetic studies suggest that the genetic contribution to life satisfaction varies from 25% to 55% (Diener & Diener, 1996; Whisman, Rhee, Hink, Boeldt, & Johnson, 2014). Moreover, results of a Dutch study regarding the life satisfaction report on a contribution of 38% to the genetic factors (Stubbe, Posthum, Boomsma, & De Geus, 2005), similar to the findings in Bartels (Bartels, 2015) meta-analysis. In the same studies, the majority of the remaining variance in the life satisfaction is explained by the nonshared envionmental influences. Other genetic studies also emphase an importance of genetic factors in explaining the time stability of the life satisfaction (Lykken & Tellegen, 1996; Nes, Røysamb, Tambs, Harris, & Reichborn-Kjennerud, 2006; Pavot & Diener, 1993). The main results of the study that includes the largest number of indicators of subjective well-being suggest that the gene contribution to life satisfaction is 31%, while 69% of the variances are explained by the nonshared enviormental factors (Gatt, Schofield, Bryant, & Williams, 2014). Shared environmental factors do not contribute significantly in the manifestation of the life satisfaction.

Results of a behavioral-genetic study with experimental design have pointed out coefficients from 45% to 55% for genetic contributions to the emotional regulation (Weinberg, Venabes, Proudfit, & Patrick, 2014), and similar findingshave have been obtained in the research using self-assessment questionnaires (Canli, Ferri, & Dunman, 2009). In the study of Gat and associates (Gatt et al., 2014), the

genetic contribution to the manifestation of the emotional suppression is 34%, and of the cognitive reappraisal is 19%, suggesting that the emotional component of regulation is more hereditary than the cognitive component. However, it seems that the emotional regulation in the light of hereditary and envionmental factors needs to be considered from a developmental perspective. At an early age, the manifestation of emotional regulation is mostly contributed by the nonshared environment, while the influence of genetic factors is negligible (Soussignan et al., 2009). With age, the genetic contribution is growing (Wang & Saudino, 2013), but the nonshared environmental influences remain dominant. Genetic factors play an important role in the manifestation of maladaptive patterns of the emotional regulation (Kanakam, Raoult, Collier, & Treasure, 2013). The results of some molecular-genetic studies (e.g., Ford, Mauss, Troy, Smolen, & Hankin, 2014; Grossman et al., 2011) provide support for behavioral-genetic studies on the heritability of various processes of emotional regulation. Genetic and enviornmental influences in the manifestation of cognitive reappraisal of positive and negative emotions have not been investigated so far. However, longitudinal studies have confirmed the stability of positive and negative affects, as well as personality dimensions (Canli, Silvers, Whitfield, Gotlieb, & Gabrieli, 2002; Chunningham, Van Barel, & Johnson, 2008; Kim & Hamann, 2007; Schwartz et al., 2003), and executive functions (e.g., Ochsner, Silver, Buhle, 2012) related to their manifestation, which support the thesis of the heritability of these constructs. It is therefore possible that different types of cognitive reappraisal have a certain hereditary component, but so the contextual factors in explaining their etiology cannot be ignored either.

The Present Study

Since the satisfaction with life and the emotional regulation are both important determinants of the subjective well-being, it seems that understanding of ethiology and nature of their relationships is essential in the field of mental health. Also, the two types of cognitive reappraisal remain, until now, unexplored in the light of hereditary and central factors, as well as the evidence of their different physiological bases (Mauss & McRae, 2016). This provides an additional need to specify etiology of their interrelations, as well as with other well-being indicators. The main aim of this study is to assess genetical and environmental factors that contribute to the connection among life satisfaction measurements and the emotional regulation. The contribution of certain sources of variance in the manifestation of these constructs is tested with a multivariate biometric method (Neale & Maes, 2004).

Method

Sample and Procedure

The study involved 364 twins (242 monozygotic and 122 dizygotic) who have grown up together. The respondents were 18 to 48 years old, and the average age was 24.59 (*SD* = 7.11). Out of the total sample, 23 pairs of monozygotic twins and 9 pairs of dizigotic twins were male, 98 pairs of monozygotic twins and 24 pairs of dizigotic twins were female, and 28 dizigotic pairs of twins were of different sexes. The zygosity of twin pairs was determined by DNA analysis of buccal swab. The sample included twins from the whole territory of Serbia, with a slightly higher number of twins from Vojvodina. Respondents were recruited as a part of the national project. A call for participation in the research was published through the media and press. Data collection was carried out in Novi Sad, Niš, Novi Pazar, Zrenjanin and Belgrade. The participation of the twins was voluntary, and each respondent signed an information consent for participation.

Instruments

Emotion Regulation Questionnaire (ERQ: Gross & John, 2003). ERQ contained 10 items that measured two strategies of emotional regulation: Cognitive Reappraisal and Emotional Suppression (e.g., *I keep my feelings for myself*). The Cognitive Reappraisal strategy consiseds of two specific scales (Popov, Dinić, & Janičić, 2016): cognitive reappraisal of negative emotions (Negative Cognitive Reappraisal; e.g., *When I face a stressful situation, I make myself think about it in a way that helps me stay calm*), and cognitive reappraisal of positive emotions (Positive Cognitive Reappraisal; e.g., *I control my emotions by changing the way I think about the situation I'm in*). The responses to each item ranged from 1 - *Generally I do not agree* to 7 - *I completely agree*. The Cronbach reliability coefficient was .72 for the scale of Emotional Suppression, .63 for the Negative Cognitive Reappraisal, and .66 for the Positive Cognitive Reappraisal. In the previous research in Serbian sample (Popov et al., 2016), the ERQ also showed satisfactory psychometric characteristics.

Satisfaction With Life Scale (SWLS: Diener, Emmons, Larsen, & Griffin, 1985; Serbian version: Vasić, Šarčević, & Trogrlić, 2011). This scale was used to assess the cognitive component of satisfaction with life. The responses to each of the five items (e.g., *In most ways my life is close to my ideal*) range from 1 - S*trongly disagree*, to 7- S*trongly agree*. This scale was widely used, and it showed good psychometric properties in previous research. Cronbach's alpha coefficient for SWLS (.83) was also acceptable.

Results

Descriptive Statistics and Gender Differences

The preliminary analysis involved a partialisation of the gender effect, as well as the linear and quadratic partialization of the age effect. Partialization of these effects was conducted by using the standard regression procedures which were proposed by McGue and Bouchard (McGue & Bouchard, 1984). Table 1 shows a descriptive statistics for the used variables. According to Tabachnick & Fidell (2013), it can be concluded that all variables, except satisfaction with life, are normally distributed (skewness and kurtosis are lower/higher than 1.50/-1.50). The measure of satisfaction with life has been normalized by Tuckey transformation. Gender differences are detected only on the dimension of Emotional Suppression in favour of males ($t = 3.57$, $p < .01$, $\eta^2 = .14$).

Table 1
Descriptive statistic for the used variables

	Monozygotic				Dizygotic			
	M	*SD*	*Sk*	*Ku*	*M*	*SD*	*Sk*	*Ku*
Satisfaction with Life	4.92	1.13	.68	7.01	4.84	1.00	0.07	2.28
Emotional Suppression	3.40	1.19	0.20	-0.54	3.45	0.93	0.13	-0.55
Positive Cognitive Reappraisal	4.34	1.17	-0.37	0.04	4.42	0.94	-0.60	-0.28
Negative Cognitive Reappraisal	5.27	0.87	-0.29	-0.20	4.04	0.92	0.67	0.99

Note. M – mean, *SD* – standard deviation, *Sk* – skewness, *Ku*– kurtosis.

Relations between Emotion Regulation Strategies and Satisfaction with Life: Cross Twin – Cross Trait Correlations

Table 2 presents the coefficients of intraclass correlations, as well as cross twin-cross trait correlations. Both types of correlation coefficients have been calculated separately for the MZ and DZ group. According to Rijsdijk & Sham (2002), intraclass correlation represents a more adequate measure of similarities between twins than ordinary Pearson's correlations. The MZ-DZ correlation pattern indicates a relative share of different sources of variance in the design of the tested constructs.

Table 2
Intraclass and cross twin – cross trait correlations

	Satisfaction With Life		Emotional Suppression		Positive Cognitive Reappraisal		Negative Cognitive Reappraisal	
	MZ	DZ	MZ	DZ	MZ	DZ	MZ	DZ
Satisfaction With Life	.54**	.42**						
Emotional Suppression	.11	.16	.38**	.20*				
Positive Cognitive Reappraisal	.03	.19	-.05	.04	.28**	.26**		
Negative Cognitive Reappraisal	.25**	.07	.07	.22*	.07	.07	.25**	.01

Notes. MZ – monozygotic twins, DZ – dizygotic twins. Diagonal numbers represent intra-class, while the remaining ones represent cross twin-cross trait correlation coefficients.
* $p < .05$. ** $p < .01$.

When the tested correlation between MZ twins was twice high as the tested correlation between DZ twins, then the influence of genetics was more crucial for the formation of phenotype, and probably both types of genetic effects shaped the examined construct. In both samples, the cross twin – cross trait correlations in the group of monozygotic twins were consistently higher than the correlation of the variables in the group of dizygotic twins. This correlation pattern provided evidence that genetic factors were likely to significantly contribute to covariance between emotion regulation strategies and satisfaction with life. The largest correlation difference was detected in the case of Negative Cognitive Reappraisal ($\Delta r = .24$), while the smallest correlation difference was detected in the case of Positive Cognitive Reappraisal ($\Delta r = .02$).

Genetic Structural Modeling: Comparison of the Multivariate Models

For the purpose of specifying the form of the observed covariants among the emotion regulation strategies and satisfaction with life, multivariate Independent Pathway Models and Common Pathway Models were tested. A comparison of the two groups of models, as well as the comparison between full (ACE, ADE) and reduced (AC, AE) models, was carried out by using several fit indicators. Analysis parameters were calculated by using the method of maximum likelihood. Model evaluation was conducted based on the Akaike Information Criterion (AIC: Akaike, 1973), Bayesian Information Criterion (BIC; Schwarz, 1978), comparative fit

index and the Tucker–Lewis Index (CFI and TLI – optimal values higher than .95, acceptable higher than .90), the root mean square error of approximation (RMSEA - optimal values lower than .05, acceptable lower than .08) and the quotient $\chi 2/df$ (recommended < 2) (Ching–Yun, 2002; Kline, 2010). By testing different models of genetic and environmental impacts on the constructs related to life satisfaction and emotional regulation, it was found that the best fit had an independent AE model ($\chi^2(56) = 67.95$, $p = .13$, CFI = .95, TLI = .94, RMSEA = .03, AIC = 99.95, BIC = 7335.7). The estimation of the parameters of the independent AE model is given in Table 3.

Table 3
Parameters estimation of the AE independent model

	Satisfaction with Life	Emotional Suppression	Positive Cognitive Reappraisal	Negative Cognitive Reappraisal
Ac^2	.13 (.08 -.23)	.01 (.01 -.02)	.08 (.03-.18)	.28 (.10 -.56)
As^2	.40 (.20-.81)	.36 (.23 -.52)	.17 (.13 -.29)	.00 (.00-.00)
ΣA	.53	.37	.25	.28
Ec^2	.01 (.00-.01)	.04 (.00 -.11)	.75 (.38 -.84)	.10 (.05 -.20)
Es^2	.46 (.18 -.67)	.59 (.40-.75)	.00 (.00 -.03)	.62 (.29 -.86)
ΣE	.47	.63	.75	.72

Note. Ac^2 - common genetic factor, As^2-unique genetic factor, ΣA^2 - total genetic variance, Ec^2–common nonshared environmental factor, Es^2– unique nonshared environmental factor, ΣE^2 - total environmental variance.

Heritability is higher only in the case of life satisfaction (53%), while in other cases the environmental influence is crucial for manifestation of the investigated phenotypes. Heritability on the most of tested variables refers to specific genetic factors, except in the case of negative cognitive reappraisal, where the overall variance of heredity is explained by general (common) genetic factors. Only in the case of positive cognitive reappraisal, the general factor has a greater impact than the specific factors of the nonshared environment.

Table 4
Genetic and non shared environmental contributions to phenotypic correlations between life satsfaction and different types of emotion regulation

Sources of variance	r_f	*Ac(%)*	*Ec(%)*
Satisfaction with life X emotional suppresion	.06	64	36
Satisfaction with life X negative cognitive reappraisal	.22	86	14
Satisfaction with life X positive cognitive reappraisal	.19	53	47
Emotional suppresion X positive cognitive reappraisal	.20	14	86
Emotional suppresion X negative cognitive reappraisal	.12	35	65
Negative cognitive reappraisal X positive cognitive reappraisal	.43	45	55

Note. r_f – coefficient of phenotypic correlations, *Ac* - common genetic factor, *Ec* - common nonshared environmental factor.

Phenotypic correlations between life satisfaction and various manifestations of emotional regulation are low ($.06 \leq r \leq .22$), and the share of genetic factors in the covariance of these measures ranges from 53% to 86% (Table 4). Nonshared environmental factors explain significantly less of covariations, except in the case of Positive Cognitive Reappraisal where genetic and environmental factors are almost equally meritorious for correlation of these measures. Phenotype correlations between the measures of emotional regulation are low to moderate ($.12 \leq r \leq .43$) and in all cases the nonshared environmental factors contribute more to their covariation (55% - 86%) than genetic factors. Genetic factors potentially have a marginal role only in the case of covariation between the two types of cognitive reappraisal (45%).

Discussion

The main aim of this study was to explore the nature of the relationship between life satisfaction and various types of the emotional regulation by using multivariate genetic analysis.

It was found that genetic factors explained 53% of the life satisfaction variance. The specific genetic factors explained about 81% of its total heredity, while the general genetic factor explained 19% of the heredity of the construct. Such findings were consistent with several results of the previous behavioral-genetic studies of the life satisfaction (Diener & Diener, 1996; Whisman et al., 2014). According to some researchers (Lykken & Tellegen, 1996; Pavot & Diener, 1993), gene-based basis of life satisfaction was to be found in more stable constructs, such as personality traits, which represented a more time-firing disposition than the life satisfaction itself, and determined its baseline level. The impact of nonshared environmental factors on the level of the life satisfaction (47%) was

manifested almost completely through the influence of specific factors in the nonshared environment. Due to the fact, it seemed that variable enviornmental factors significantly influenced the manifestation of life satisfaction, but their nature could not be precisely defined. They depended on individual experiences of an individual, while life events had a moderate moderation effect on life satisfaction. More precisely, the variable characteristics of nonshared environmental factors affected the level of life satisfaction, but not for a long time, i.e. only in certain time frames during which there were strong consequences of life events on functioning of an individual (Pavot & Diener, 1993). After this acute period, during which there were strong consequences of life events, the satisfaction with life returned to its basic level.

It has also been found that the nonshared environmental factors have the strongest influences (63%) on emotional suppression. This finding is in line with the assumptions of Kim and the associates (Kim et al., 2011) who argue that the everyday environment is the most important for the way of expressing emotions. If an individual, inclined to express emotions, approaches a specific environment that does not support emotional exchange, there is a greater chance of reporting the suppression of different emotions with the aim of not expressing it. The specific genetic influence in the manifestation of emotional suppression is not negligible (36%, or almost 100% of the total genetic influence), and explanations can firstly be found in relations between activities of certain physiological structures (e.g., amygdala) during emotional processing, and personality traits, such as extraversion and neuroticism (Canli et al., 2002), or inhibition of temperament (Schwatrz et al., 2003). Therefore, the genetical contribution to the emotional suppression seems to be explained first by genetic bases that it partly shares with the personality traits, or through the partial moderation effects of personality traits, such as stable dispositions, on manifestation of the emotional regulation.

Previous behavioral-genetic studies were not conducted for the purpose of specifying genetic and environmental factors of different types of cognitive reappraisal of emotions. In the present study, higher effects of nonshared environmental factors were determinate for both types of the cognitive reappraisal (75% and 72%), while genetical contributions were somewhat lower (27% and 26%). The most noticeable difference between these two types of cognitive reappraisal were contributions of general genetic factors, since its influence on the reformulation of positive emotions was low, while the influence on the reformulation of negative emotions was high (100% of total genetic impact). This difference could be explained in the light of various neural processes, having the same physiological basis. Namely, the amygdala played an important role for both types of the cognitive reappraisal. It was activated both in the cases of cognitive reappraisal of positive and negative emotions. However, some researchers argued that in the case of cognitive reappraisal of positive emotions, the amygdala was sensitive to new and positive events affecting an individual, and therefore the greater activity of the amygdala was recorded, while in situations of negative events this was not

the case (Chunningham et al., 2008; Kim & Hamann, 2007). This is in accordance with the obtained finding that specific nonshared environmental influences better explains the cognitive reappraisal of positive emotions than of negative emotions. It is possible that certain physiological structures are basically reactive to emotional stimuli, but that these structures are further stimulated, due to everyday positive and pleasant stimuli from the environment that are specific to the individual. Therefore, for the greater activity of these structures in the process of the cognitive reappraisal of positive emotions, environmental factors play a "plastic" role, while in the case of the reappraisal of negative emotions, the same neural structure has a predisposed "protection" effect. Furthermore, that is in line with the claims of Belsky and associates (Belsky et al., 2009) that genetic factors influence different indicators of well-being through "protection effects" or "effects of plasticity" in the context of their sensitivity to negative or positive environment. Findings of this study support the discussion on genetic influences: genetic factors potentially have significance in the case of covariation between the cognitive reappraisal of positive and negative emotions (45%). In addition, it seems that the general genetic influence on manifestation of the cognitive reappraisal of negative emotions has the connection with different executive functions and their neural correlates (e.g., Ochner et al., 2012), and thus greater specific genetic influences to its manifestation. However, these assumptions should be taken with reserve, since the variables mentioned have not been a part of this study.

An insight into phenotypic correlations of the life satisfaction and the emotional regulation suggests that these are low-correlation constructs. The same finding is obtained if only specific forms of the emotional regulation are considered, except in the case of the cognitive reappraisal of positive and negative emotions, where a moderate coefficient of correlation is detected. A potential explanation for this result can be found in personalty factors, which significantly affect life satisfaction and the emotional regulation (Canli et al., 2002; Diener & Diener, 1996), and which are genetically determined to a large extent.

The obtained findings suggest a certain kind of assumption that can be considered as potentially important guidelines in the field of mental health. Namely, it seems that genetic factors are more important than environmental ones in explaining life satisfaction, and it is likely that various interventions carried out in order to increase the level of global satisfaction and well-being need to be focused on indirect factors that determine the satisfaction with life. On the other hand, some future studies of genetic and environmental factors of the emotional regulation could continue focusing on neural structures that are responsible for the emotion of emotions. However, at the same time, it is important to pay attention to the environmental factors involved in shaping the way of processing and overcoming emotions that cause an expression of strong positive or negative affect.

With this in mind, the need for more extensive and complete testing of these constructs in a behavioral-genetic paradigm can be identified. This would include other potential determinants of subjective well-being, such as personality traits

or exquisite functions. Still, some of the limitations of the presented study would relate to the chosen type of design, a transversal one. Longitudinal tracking would enable stability of genetic and environmental influences to be detected, as well as as their changes over time. Besides usage of somewhat more reliable measures of emotional regulation, another addition to this research would be a sample of a larger age range, since we can not assume an equal manifestation of the level of life satisfaction in different developmental periods.

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GENSKI I SREDINSKI ČINIOCI EMOCIONALNE REGULACIJE I ZADOVOLJSTVA ŽIVOTOM: BLIZANAČKA STUDIJA

Ekspanzija istraživanja indikatora mentalnog zdravlja krajem XX veka velikim delom stavlja faktore blagostanja u fokus interesovanja savremene psihološke nauke. Međutim, tek noviji rezultati bihejvioralno-genetičkih studija pružaju potpunu sliku o faktorima koji utiču na indikatore subjektivnog blagostanja kroz specifikaciju etiologije njihovog odnosa. Osnovni cilj ovog istraživanja usmeren je na procenu genskih i sredinskih činilaca koji utiču na kovariranje među merama zadovoljstva životom i emocionalne regulacije. Istraživanje je obuhvatalo 182 parova blizanaca (121 para monozigotnih i 61 para dizigotnih), oba pola, u starosnoj dobi 18 - 48 godina. Udeo pojedinih izvora kovariranja između ispitivanih fenotipova testiran je multivarijatnim biometrijskim metodom. Genski činioci objašnjavaju nešto veći deo varijanse zadovoljstva životom (53%), dok sredinski činioci imaju većinski udeo u objašnjenju različitih tipova emocionalne regulacije. Opšti genski činioci potencijalno su značajni samo u objašnjenju kognitivne preformulacije negativnih događaja, dok u ostalim slučajevima veća važnost pripada sredinskim faktorima. Uvid u fenotipske korelacije navedenih mera ukazuje na to da je reč o konstruktima koji ostvaruju niske do umerene korelacije, te da genski činioci imaju potencijalni značaj (45%) samo u slučaju kovariranja dve vrste kognitivne preformulacije događaja.

Ključne reči: blizanačka studija, emocionalna regulacija, emocionalna supresija, kognitivna preformulacija, zadovoljstvo životom

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VALIDATION OF ZYGOSITY ASSESSMENT BY A SELF-REPORT QUESTIONNAIRE IN A SAMPLE OF ADULT SERBIAN TWINS[2]

Validation of a twin zygosity-estimating questionnaire, The Questionnaire of Twins' Physical Resemblance, created by Oniszczenko et al. and used in European and Serbian twin studies, was carried out on a sample of 222 pairs (176 monozygotic, 46 dizygotic) of adult twins (average age 24.6). Four discriminant functions, use of different sets of indicators (zygosity questionnaire items), were applied in order to obtain the most correct and accurate estimates of zygosity. The first function was a predefined function used in European twin studies, the following two functions contained sets of 18 and 24 freely estimated indicators respectively, while the last one utilized the items with most consistent contributions to zygosity prediction. The analytic procedure included cross-validation, whereby the sample was randomly split into two subsamples, comprising 107 and 115 twin pairs. The results pointed to successful (over 90% correct) identification of monozygotic twins, and sizeably lower correctness in identifying dizygotic twins. Overall correctness of estimation exceeded 90%, with the small set of best-performing indicators. The results encourage questionnaire estimation of zygosity, and raise the issue of improving the classification procedure in dizygotic twins.

Key words: behavioral genetics, questionnaire assessment of zygosity, twin studies

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Introduction

Correct estimation of twins' zygosity is a crucial prerequisite for the validity of results of the twin studies (Lenau et al., 2017). Currently, several procedures are available for the assessment of zygosity. Undoubtedly most efficient are the DNA analytic procedures, which reduce estimation error to less than 1% (Becker et al., 1997; Lenau et al., 2017). However, the cost of such analyses (Lenau et al., 2017) may still be a challenge for large studies or studies in underprivileged regions. Therefore, besides DNA analyses, or instead of them, the questionnaire assessment of zygosity is often used as an alternative (Joseph, 2004). Technical aspects of the questionnaire-based zygosity estimation in twin studies imply identification of indicators with best discriminant power (by means of discriminant analysis or similar analytic procedures), and the use of the extracted discriminant function in subsequent estimations.

The efforts to improve the accuracy of zygosity questionnaires were evident in recent decades. In adult twin samples, overall classification rates in most cases exceeded 90%, and occasionally amounted to approximately 98% (e.g., Jarrar et al., 2018; Joseph, 2004; Lenau et al., 2017; Ooki, Yamada, Asaka, & Hayakawa, 1990; Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998), whereby one or more zygosity questionnaires were applied.

In order to reliably determine twins' zygosity, the questionnaires employ a number of indicators, which are assumed to discriminate well between monozygotic and dizygotic twins (Oniczenko, Angleitner, Strelau, & Angert., 1993). Such indicators are sometimes labelled as biological and physical characteristics (Lenau et al., 2017). The aforementioned set of indicators includes "objective" estimates such as height, eye colour, natural hair colour, blood type, earlobe shape, etc. Indicators based on "subjective" assessment are also considered, and they mostly refer to mistaking twins for one another: in childhood, by family, friends, acquaintances, teachers, colleagues, strangers, in photographs taken recently, etc. Relevant are also the data about chronic and acute medical conditions. These features have been shown to be reliable indicators of zygosity in self- and peer- ratings. Reportedly, the questions referring to twins being as similar as "peas in the pod" have been particularly informative (Joseph, 2004).

However, classification of dizygotic twins may still be a challenge. While there are findings that suggest similar or equal precision of MZ and DZ twin's classification, or even better classification rates for DZ twins (Jarrar et al., 2018; Lenau et al., 2017), there are results that point to the contrary (Lenau et al., 2017; Ooki et al., 1990).

A zygosity questionnaire most frequently used in European twin studies is the The Questionnaire of Twins' Physical Resemblance by Oniszczenko et al. (1993). The questionnaire has been successfully applied in BilSat (Kandler et al., 2012), JetSSA (Stößel, Kämpfe, & Riemann, 2006), GOSAT (Spinath, Angleitner, Borkenau, Riemann, & Wolf, 2002), and TwinLife (Lenau et al., 2017) studies.

Within and beyond these studies, the questionnaire has demonstrated excellent classification rates (Lenau et al., 2017).

The Oniczenko et al. (1993) questionnaire has also been applied in the Serbian twin study, conducted within the research project "Psychological foundations of mental health: hereditary and environmental factors" (e.g., Nikolašević, Bugarski - Ignjatović, Milovanović, & Raković, 2014). The zygosity classification function used in the BilSat study (Kandler et al., 2012), similarly, but not identically to the function applied in cohorts 3 and 4 of the TwinLife study (Lenau & Hahn, 2017), has been used in Serbia so far. However, the predictive validity of the measure has not been validated yet in a sample of Serbian twins. Despite robustness of the phenomenon, it is not warranted that the Bilsat/TwinLife classification procedure will be as efficient in Serbian culture as it is in its original form. A number of issues should be addressed: does the original classification procedure (as shown in Lenau & Hahn, 2017) discriminate well between adult MZ and DZ twins in Serbia? Would an extended set of indicators perform better? Would it be possible to select the most discriminative items and develop a brief, but efficient classification tool? All these questions are subordinate to the principal aim of the current study: to identify the set of indicators, which most efficiently discriminate between monozygotic and dizygotic twins in the adult Serbian sample. The results are expected to help in future self-report zygosity assessment in behavioral genetic studies.

Method

Sample and Procedure

A sample of 222 twins (111 twin pairs; 70% female participants in total), whose average age was 24.6 (*SD* = 7.64), took part in the study. The DNA test results suggested that 176 twin pairs were monozygotic, while 46 twin pairs were dizygotic. Prior to the analyses, 37 undoubtedly dizygotic (different-sex) twin pairs were excluded from the study. For the purposes of cross-validation, following, but not mirroring the procedure used in the reference study of Lenau et al. (2017), the sample was randomly split into two sub-samples. The first subsample included 87 monozygotic and 20 dizygotic twin pairs (average age 24, *SD* = 7.92), while the second one included 89 monozygotic and 26 dizygotic twin pairs (average age 25.16, *SD* = 7.36). The data were collected from 2011 to 2018, by administering the questionnaire to participants (twins) in a form of a standardized interview, with the standard clause of confidentiality. A smaller number of twins who were not able to attend the interview completed the questionnaires at their homes and returned them by mail. The zygosity questionnaire was not administered to the twin pairs of different sexes. Twins were recruited as a part of the wide Serbian national project "Psychological Foundations of Mental Health: Hereditary and Environmental Factors".

Instrument

The Questionnaire of Twins' Physical Resemblance (Oniszczenko et al., 1993). This questionnaire is a self-report measure containing 31 sets of items (plus 19 demographic questions) referring to the above mentioned biological and physical indicators. The questionnaire can be applied as the standard self-report, paper-pencil format, or in the form of a standardized interview. The measure, scoring procedures, and the discriminant functions used for classification, are described in detail in Lenau & Hahn (2017). Certain indicators, carrying the extensions 1 and 2, have been calculated in two different variants, and entered as such in the functions (for details see Lenau et al., 2017; Lenau & Hahn, 2017).

Data analysis

The criterion used for the validation of the zygosity questionnaire was the result of DNA zygosity estimation, carried out by method of micro-satellites (Becker et al., 1997).

According to the standard procedure, all indicators used in the analyses were calculated from "raw" responses to questionnaire items. The procedure was primarily based on the calculation of differences in responses by the twins from each pair. The final indicators values ranged from 0 to 1, whereby the scoring was such that the value 1 points to monozygosity, value 0 to dizygosity, while the value 0.5 was assigned to the cases where zygosity could not be estimated with sufficient reliability. Thus, although most labels of indicators in Tables 3, 5 and 8 contained the word "differences" (for the sake of comparability with other studies), the reader should interpret them according to the scoring procedure described above.

Classification procedures were carried out by discriminant function analyses, using the "MASS" (Venables & Ripley, 2002) package in R 3.5.1 (R Core Team, 2018). The "lda" function from the "MASS" package performed linear discriminant analysis, with the possibility of cross-validation. Thus, the functions developed in the first subsample were applied in the second, and vice versa. In the entire sample, the function was estimated independently from the ones derived and cross-validated in the subsamples. A linear discriminant analysis was used according to methodology presented in Lenau et al. (2017), assuming that such decision would facilitate the comparability of results. Prior probabilities were set to 80:20 for MZ and DZ twins respectively.

The following discriminant / classification functions were tested:

a) The function developed in the BilSat study (Kandler et al., 2012), similar to the function applied in the cohorts 3 and 4 of the TwinLife study (Lenau et al., 2017). The function was weighted according to the BilSat original function (Kandler et al., 2012; weights for the TwinLife cohorts 3 /4 function is presented in Lenau & Hahn, 2017);

b) the function comprising the indicators used in the BilSat study (Kandler et al., 2012), whereby the discriminant coefficients (weights) were freely estimated;
c) the function freely estimated in the sample of adult Serbian twins, based on the extended set of indicators described in Lenau et al. (2017) and Lenau and Hahn (2017), including twins' own belief about their zygosity, as well as the "peas in the pod" statement;
d) the discriminant function based on the best-discriminating items selected from the previous functions. The criterion for the selection was the following: items which standardized discriminant coefficients were stable across samples were chosen to be included in the analysis.

Results

Weighted BilSat Function: Classification Rates

In the first step of the study, classification rates of the discriminant function obtained from the BilSat study were estimated (Table 1).

Table 1
Function 1: BilSat - classification rates

	Subsample 1	Subsample 2	Total
MZ (%)	98.63	96.20	97.37
DZ (%)	55.88	63.89	60.00
Total (%)	85.05	86.09	85.59

Note. MZ – monozygotic twins, DZ – dizygotic twins.

The results show excellent classification rates for monozygotic twins, and unsatisfactory rates for dizygotic twins. Correct classification rates are approximately 85% to 86%. These results suggest that the adjustment of the "original" classification procedure to Serbian sample would be recommended. Nevertheless, having in mind that the study has identified Serbian monozygotic twins with almost perfect correctness (Table 1), we cannot dispute validity of the indicators, and tend to see this result as corroborating the robustness of the phenomenon.

BilSat Function: Freely Estimated Coefficients

With the discriminant coefficients estimated freely, correct classification rates improve substantially, with correct classification rates approximating 90% (Table 2).

Table 2
Function 2: Classification- freely estimated coefficients and group centroids

				Group centroids		
	Subsample 1	Subsample 2	Total	Subsample 1	Subsample 2	Total
MZ (%)	95.29	92.31	94.32	-0.45	-0.75	-0.55
DZ (%)	72.73	79.17	67.39	2.27	2.13	2.22
Total (%)	90.65	89.57	88.74	-	-	-

Note. MZ – monozygotic twins, DZ – dizygotic twins.

Still, the correctness in identifying dizygotic twins is still unsatisfactory. At the same time, instability of indicators' contributions over subsamples is evident, with some of the standardized discriminant coefficients varying not only regarding size, but also regarding the sign (Table 3).

Table 3
Function 2: Standardized discriminant coefficients

	Subsample 1	Subsample 2	Total
difference in height	-0.17	-0.04	-0.05
difference in hairiness	-0.10	-0.33	-0.23
difference in skin colour	0.07	0.08	0.07
difference in sweating	-0.09	-0.13	-0.11
difference in eye colour	-0.05	0.05	-0.01
difference in blood type	-0.15	0.15	0.02
difference in hair type	-0.36	-0.34	-0.32
difference in eye colour 2	-0.69	-0.04	-0.37
difference in ear lobes	0.28	0.28	0.24
parent's effort to keep apart	-0.09	-0.14	-0.06
difference in sickness	-0.05	-0.24	-0.09
mistaken in childhood	-0.29	-0.40	-0.34
mistaken by siblings 1	-0.08	-0.09	-0.08
mistaken by teachers 1	-0.20	-0.17	-0.13
mistaken by people meeting first time 1	-0.49	-0.58	-0.53
mistaken by parents 2	0.30	-0.21	0.01
mistaken by teachers 2	0.07	-0.09	-0.06
mistaken in a photograph	-0.29	0.07	-0.05

While this result could be partly attributed to the sample size and somewhat unfavourable conditions for cross-validation, it also suggests that modification of the function (either its extension or reduction) may improve its correctness in classification.

Extended Set of Predictors – Freely Estimated Coefficients

The results suggest that the extension of predictor set have not significantly improved classification rates (Table 4). They also show that the problem encountered in Function 2, namely 'the instability of indicators' contributions across functions, remains.

Table 4
Function 3: Classification based on extended set of predictors and group centroids

				Group centroids		
	Subsample 1	Subsample 2	Total	Subsample 1	Subsample 2	Total
MZ (%)	93.18	92.22	94.89	-0.47	-0.74	-0.58
DZ (%)	73.68	76.00	69.57	2.37	2.29	2.34
Total (%)	89.72	88.70	89.64	-	-	-

Note. MZ – monozygotic twins, DZ – dizygotic twins.

Standardized, freely estimated discriminant coefficients on the extended set of predictors are presented in Table 5.

Table 5
Function 3: Standardized discriminant coefficients – extended set of items

	Subsample 1	Subsample 2	Total
difference in height	-0.14	-0.05	-0.07
difference in hairiness	-0.14	-0.46	-0.29
difference in skin colour	0.03	0.06	0.09
difference in sweating	-0.14	-0.19	-0.11
difference in eye colour	-0.03	0.22	0.00
difference in blood type	-0.21	-0.26	-0.27
difference in rhesus factor	0.11	0.48	0.39
difference in hair type	-0.36	-0.37	-0.33
difference in eye colour 2	-0.67	-0.15	-0.38
difference in ear lobes	0.27	0.28	0.27
parent's effort to keep apart	-0.06	-0.01	0.04
difference sickness	0.00	-0.27	-0.08
mistaken in childhood	-0.23	-0.82	-0.40
mistaken by parents 1	-0.21	-0.31	-0.23
mistaken by siblings 1	0.06	-0.09	-0.02
mistaken by friends 1	-0.18	0.30	0.03
mistaken by teachers 1	-0.20	-0.14	-0.14
mistaken by people meeting first time 1	-0.65	-1.07	-0.62
mistaken by parents 2	0.43	-0.14	0.08
mistaken by siblings 2	-0.23	0.00	-0.05
mistaken by friends 2	0.34	-0.17	0.06
mistaken by teachers 2	0.02	-0.09	-0.06
mistaken by people meeting first time 2	-0.08	0.82	0.03
mistaken in photograph	-0.24	0.05	-0.07
peas in a pod	0.16	0.05	0.10
own belief	-0.21	0.12	-0.03

Among the salient indicators that remain invariant or "partially invariant" across samples, there are physical features such as differences in eye colour, blood group, and hairiness, but also indicators of mistaking twins by parents, teachers, people met for the first time. Curiously, some of the physical indicators appear to be indicative of dizygosity, such as the difference (or similarity) in rhesus factor

and ear lobes. This also applies to "peas in the pod" similarity statement, which contribution is modest (even negligible), but with consistently positive sign.

Selected Items

In the final phase of the study, ten "stable" indicators were selected according to their coefficients in Function 3, and entered into the analysis. Selection of the best-discriminating items apparently contributed not only to coefficient stability (with some exceptions, such as difference in hairiness and rhesus factor), but also to classification correctness, with correctness rate in subsamples around 91%, and the overall correctness in the entire sample also being 91%. Although these results were favourable, the problem of correctly identifying dizygotic twins remained, with correctness approximating 80%, but not exceeding it (Table 6).

Table 6
Function 4: Ten best - discriminating items and group centroids

				Group centroids		
	Subsample 1	Subsample 2	Total	Subsample 1	Subsample 2	Total
MZ (%)	94.32	94.32	93.82	-0.44	-0.63	-0.54
DZ (%)	78.95	77.78	79.55	2.10	2.07	2.17
Total (%)	91.59	90.43	90.99	-	-	-

Note. MZ – monozygotic twins, DZ – dizygotic twins.

Standardized, freely estimated discriminant coefficients on the selected set of items are presented in Table 7.

Table 7
Function 4: Standardized discriminant coefficients based on 10 selected items

	Subsample 1	Subsample 2	Total
difference in height	-0.22	-0.07	-0.08
difference in hairiness	0.02	-0.31	-0.19
difference in blood type	-0.09	0.11	0.02
difference in hair type	-0.36	-0.25	-0.31
difference in eye colour 2	-0.63	-0.01	-0.30
mistaken in childhood	-0.18	-0.52	-0.39
mistaken by parents 1	-0.04	-0.29	-0.19
mistaken by people meeting first time 1	-0.55	-0.50	-0.51
difference sweating	-0.05	-0.12	-0.10
mistaken by teachers 1	-0.10	-0.19	-0.12

Discussion

This study was conducted in order to validate "The Questionnaire of Twins' physical resemblances" (Onisczenko et al., 1993), which might be regarding as something of a standard in European behavioral genetic studies. Generally, the questionnaire and classification procedures based on it performed well in our sample, but certain adjustments were nevertheless necessary. The results of this research were nearly in line with the results of the previous research that spoke in favour of high classification rates (>90%) of zygosity questionnaires (e.g., Jarrar et al., 2018; Joseph, 2004; Lenau et al., 2017; Ooki et al., 1990; Peeters et al., 1998).

One of the most important, though expected, findings concerns better performance of the "freely estimated" functions compared to the predefined function derived in the BilSat / TwinLife studies. Although this result may be regarded as self-explanatory, still it is important to mention that the "predefined" classification procedure has performed satisfactorily in identification of monozygotic twins. Whether this result is due to cultural factors, specific self-assessment of Serbian dizygotic twins, or relatively small number of dizygotic twin pairs in this study, is yet to be resolved.

What seems to be a persistent issue is the assessment of dizygotic twins. In this study, correctness of their classification has not been up to our expectations, despite prior probabilities set to fairly liberal 80:20 in favour of monozygotic twins, reflecting the sample structure. A possible hypothesis based on this result could be that the Serbian dizygotic twins tend to accentuate their similarities, instead of differences. Therefore, qualitative or quantitative examination of their responses on the zygosity questionnaire may help clarify this issue. Nevertheless, the classification of dizygotic twins has been improved by modification of the discriminant function, suggesting that further work in this area may yield more favourable results. What could be recommended for the future studies would be the application of less traditional classification procedures, such as learning-based algorithms or discriminant analysis based on different estimation methods. However, in case of Serbian twin samples, it would be highly recommendable to apply these procedures with larger samples of dizygotic twins.

One of the crucial limitations of the study is the sample size. This is the issue that cannot be resolved quickly, however further validity checks of the questionnaire are expected as the number of participants increases. In this study, we relied on the traditional classification procedure based on discriminant function analysis, deliberately choosing not to apply more recent or (arguably) more sophisticated procedures. This decision was in accordance with the aims of this study: as the first validation study of this sort in Serbia, its goal was to test the existing methodology before making any recommendations for future studies.

The study has shown that (at least in Serbian twins) reasonably correct estimation is possible with a relatively small number of reliable indicators which are contained in The Questionnaire of Twins' Physical Resemblance (Onisczenko

et al., 1993). This does not mean that more elaborate sets of indicators are unnecessary or unwelcome (it is quite the opposite, for the sake of reliability and validity of estimation). Rather, this result suggests that an experienced researcher, whenever DNA analyses are unavailable, could rely on a small set of features to estimate zygosity with an acceptable error rate. Although this study does not bring a definitive solution to the problem of the questionnaire estimation of zygosity in Serbian twins, it at least highlights the risks that the researchers should be aware of and take into account.

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VALIDACIJA PROCENE ZIGOTNOSTI UPITNIČKIM PUTEM NA UZORKU ODRASLIH BLIZANACA IZ SRBIJE

Najprecizniji metodi kojima se u bihejvioralno - genetičkim studijama procenjuje zigotnost blizanaca jesu procedure DNK analize. Medutim, budući da su za sprovođenje ovakvih analiza potrebna nezanemarljiva finansijska sredstva, istraživači se cesto odlučuju na nesto manje preciznu, ali finansijski znatno manje zahtevnu alternativu - primenu upitnika za procenu zigotnosti. Tačnost klasifikacije, odnosno tačnog prepoznavanja monozigotnih i dizigotnih blizanaca u velikom broju studija prevazilazi 90%, što je dovoljan razlog za njihovu široku primenu. U srpskoj bihejvioralno - genetičkoj studiji za procenu zigotnosti primenjuje se upitnik Oniščenka i saradnika, nazvan ,,Upitnik fizičkih sličnosti medu blizancima". Pored fizičkih karakteristika, ovaj instrument obuhvata biološke markere zigotnosti i markere koji se odnose na ,,mešanje" blizanaca (pogrešno prepoznavanje jednog kao drugog od strane bliskih i nepoznatih osoba). Osnovni cilj istraživanja prikazanog u ovom radu jeste validacija ovog instrumenta. Osnovna istrazivačka pitanja koja se tom prilikom postavljaju odnose se na primenljivost ,,predefinisanih" diskriminativnih funkcija (s unapred određenim ponderima za indikatore), korišćenih u inostranim studijama, i na mogućnost identifikacije optimalnog seta prediktora zigotnosti na srpskom uzorku. Pri tome, kriterijum za procenu predstavljaju rezultati procene zigotnosti DNK analizom, koji se smatraju maksimalno pouzdanim. U istraživanju su učestvovala 222 para blizanaca istog pola, starosti približno 24 godine, ispitana u okviru blizanačke studije u periodu 2011 - 2018. Kao osnov za procenu zigotnosti, korišćen je upitnik Oniščenka i saradnika, a analitička procedura obuhvatala je evaluaciju kvaliteta predikcije zigotnosti na osnovu četiri diskriminativne funkcije: predefinisane funkcije razvijene u okviru BilSat studije u Nemačkoj, dve slobodno procenjene funkcije s 18, odnosno 24 prediktora, kao i funkcije koja obuhvata indikatore koji su u ovim analizama pokazali najveću diskriminativnu moć. Svaka analiza podrazumevala je unakrsnu validaciju na dva nasumično formirana poduzorka, (N1 = 107, N2 = 115), pri čemu su oba obuhvatila približno 80% monozigotnih i približno 20% dizigotnih blizanaca. Rezultati upućuju na visoku uspešnost predefinisane funkcije u identifikaciji monozigotnih, ali ne i dizigotnih blizanaca. Tačnost klasifikacije povećava se primenom ,,slobodno procenjenih" funkcija, mada procenat tačno

identifikovanih dizigotnih blizanaca i dalje nije zadovoljavajući. Skup najboljih indikatora daje najuspešniju predikciju generalno, pri čemu tačnost prelazi 90%, ali prepoznavanje dizigotnih blizanaca pokazuje se kao problem koji tek očekuje zadovoljavajuće rešenje.

Ključne reči: bihejvioralna genetika, blizanačka studija, upitnička procena zigotnosti

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WHY DO PEOPLE DIFFER IN THEIR ACHIEVEMENT MOTIVATION? A NUCLEAR TWIN FAMILY STUDY[2]

Although many previous studies have emphasized the role of environmental factors, such as parental home and school environment, on achievement motivation, classical twin studies suggest that both additive genetic influences and non-shared environmental influences explain interindividual differences in achievement motivation. By applying a Nuclear Twin Family Design on the data of the German nationally representative of TwinLife study, we analyzed genetic and environmental influences on achievement motivation in adolescents and young adults. As expected, the results provided evidence for the impact of additive genetic variation, non-additive genetic influences, as well as twin specific shared environmental influences. The largest amount of variance was attributed to non-shared environmental influences, showing the importance of individual experiences in forming differences in achievement motivation. Overall, we suggest a revision of models and theories that explain variation in achievement motivation by differences in familial socialization only.

Key words: achievement motivation, behavioral genetics, Nuclear Twin Family Design

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Introduction

Motivation gives to the people's behavior direction, intensity and persistence (Spinath, Toussaint, Spengler, & Spinath, 2008). Achievement motivation is an important key qualification in a modern society due to its central role for learning and career success, as well as for lifelong learning in general (Looser, 2011; Röhr-Sendlmeier, & Kröger, 2014). It has been defined as the striving to increase or to keep, as high as possible, one's own capabilities in all activities in which a standard of excellence is thought to apply, and where the execution of such activities can therefore either succeed or fail (Heckhausen, 1967). Due to its high relevance, personality research has been examining the factors that influence the development of individual differences in achievement motivation (Heckhausen & Heckhausen, 2010).

On the one hand, achievement motivation has been investigated from a behavioral genetic perspective, and it has been found to be genetically influenced (e.g., Spinath, 2001; Spinath et al., 2008). On the other hand, most theories and models that attempt to explain differences in the motivation to perform focus on education and socialization, emphasizing the role of school and parental home for the development of individual differences in achievement motivation (Deci & Ryan, 2004; Heckhausen & Heckhausen, 2010; Wigfield & Eccles, 2000).

Influences of Social Contexts

Previous studies have shown a connection between children's achievement motivation and home, as well as parental factors (Mansour & Martin, 2006). The intellectual and performance-related stimulating value in the parental home, as well as a connection with the parental performance pressure, were shown to be correlated with the achievement motivation of children (Heckhausen & Heckhausen, 2010). Additionally, cross-sectional and longitudinal correlations between achievement motivation and children's social integration in their family were found (Looser, 2011; Looser, 2017): Specifically, achievement motivation in adolescence correlated with perceived quality of the parent-child relationship, a consistent parenting style, an authoritative educational style in the parent-child interaction, the perceived well-being at home, and the parent-child intensity of conversation. Negative correlations were found between achievement motivation and frequent conflicts at home, and an inconsistent parenting style.

Furthermore, factors of the school setting correlated with different levels of achievement motivation: Positive correlations were found between achievement motivation and the teacher-student relationship, the feeling of well-being at school, the perception of the teacher's appreciation, emotional affection and attribution concerning aptitudes, the feeling of competence within the class and the recognition by classmates. Negative correlations were found between achievement motivation and school norm violations (Looser, 2011). Additional

supporting factors in the school context were education aimed at the interests and lifestyles of students, an appreciative teacher-student-relationship, an educational leadership style of schools, and a combination of high performance-related expectations and positive social relationships in general (Looser, 2017; Wigfield, Eccles, Schiefele, Roeser, & Davis-Kean (2006). An importance of the school setting for the development of achievement motivation was emphasized by studies showing correlations between teachers' reference orientation and students' motivation (Heckhausen & Heckhausen, 2010; Schlag, 2013).

Lastly, the development of individual differences in achievement motivation is affected by leisure activities and peer relationships (Nelson & DeBacker, 2008; Heckhausen & Heckhausen, 2010) and correlates with socio-cultural backgrounds (Röhr-Sendlmeier, Jöris, & Pache, 2012). All in all, individual differences in achievement motivation seem to be explainable partly by influences of the parental home, school, gender, school type, peer-relationships, and socio-economic background, as shown by previous research.

Behavioral Genetic Studies

Next to studies considering only environmental influences on achievement motivation, Röhr-Sendlmeier and Kröger (2014), as well as Bergold, Röhr-Sendlmeier, Heuser, Bieling and Burdorf (2014), have found significant correlations between parents' and adolescent children's achievement motivation. These family correlations may point to learning influences and /or genetic transmission from parents to the offspring. Behavioral genetic studies allow distinguishing both pathways of intergenerational similarity.

The most frequently used research design in behavioral genetics is the Classical Twin Design (CTD; see Knopik, Neiderhiser, DeFries, & Plomin, 2017). The CTD compares the covariance of MZ twins who share 100% of their segregating genes with the covariance of DZ twins sharing 50% of their segregating genes. Structural equation modeling of these covariance matrices allows estimation of additive genetic influences, the net effect of both non-additive and shared environmental influences, as well as non-shared environmental effects on individual differences.

Spinath (2001) used the CTD on achievement motivation in a sample of German adult twins reared together. Additive genetic influences were found to explain 41% of the phenotypic variance, while the remaining variance could be explained by non-shared environmental effects. Kovas et al. (2015) used data from over 13,000 twins aged 9 to 16, from six different twin studies in six different countries. Almost identical to the results of Spinath, they found that about 40% of the variance could be attributed to genetic factors and non-shared environmental influences (60%). These findings clearly pointed out a moderate genetic influence on motivational personality traits, such as achievement motivation, and thus they were not in line with current popular motivation theories which explained individual differences in achievement motivation by environmental factors, such

as factors of the parental home and school environment only (Deci & Ryan, 2004; Heckhausen & Heckhausen, 2010; Wigfield & Eccles, 2000).

There is a seeming inconsistency in the results of family studies, which report correlations between characteristics of the family and genetically informative that do not indicate an effect of the environment shared by family members. A possible explanation is that variables such as the parent-child-relationship, parenting behavior and school variables, although usually regarded as examples of shared environments, affect siblings in the same family differently. Thus, the effects of these variables are correctly identified as non-shared (Bleidorn et. al., 2018; Knopik et al., 2017).

In addition, the CTD is not optimally suited for investigating these influences, because it is based on strict assumptions which need to be met in order to obtain accurate estimates (Keller, Medland, & Duncan, 2010). The CTD tries to estimate three or four parameters by using MZ twin and DZ twin variance-covariance matrices: Additive genetic influences (a^2), non-additive genetic influences (i^2), and shared environmental influences (c^2), which are mutually confounded in the CTD and only two of these parameters can be estimated. Since parameters of non-additive genetic influences cannot be estimated in the presence of additive genetic effects and shared environmental effects, either i^2 or c^2 are fixed to 0. If the assumption is violated, parameters for additive genetic effects are overestimated, and parameters for non-additive genetic effects and shared environmental effects are be underestimated (Kandler & Papendick, 2017).

Another assumption of the CTD is that assortative mating does not have an effect on the examined trait (Keller et al., 2010). Assortative mating describes the fact that people choose their partners according to their own genetically influenced characteristics. If this is the case, the parents of twins are more similar to each other than it would be expected under random mating, which would raise the genetic relatedness of DZ twins, but obviously not the perfect genetic correlation of MZ twins as well. Consequently, no considering assortative mating results in overestimating shared environmental influences and underestimating genetic influences (Knopik et al., 2017).

Furthermore, genetic and environmental influences are rarely independent of each other. However, the CTD relies on the assumption that gene-environment correlation and interaction have no influence on the trait under study (Keller et al., 2010). If this assumption is violated though, it would also result in biased parameter estimates. Moreover, the CTD does not provide detailed information about the origin of shared environmental effects (Bleidorn et al., 2018).

Many of these shortcomings can be overcome, if data from additional family members are available. In the current study, the Nuclear Twin Family Design (NTFD) was used. Data of parents and available siblings of the twins were collected in addition to data of MZ and DZ twins reared together. These additional measurements increased statistical power and allowed estimation of more parameters that are less biased (Bleidorn et al., 2018; Keller et al., 2010).

Method

Sample

This study uses the data of the TwinLife study, a longitudinal twin family study that examined more than 4,000 same-sex twin pairs and their family representative for twin families in Germany (Hahn et al., 2016). The first wave of data collection took place between 2014 and 2015 (Brix et al., 2017). The twins and their families were grouped in four age cohorts. This analysis used the data from the two oldest twin cohorts (C17; age 17 and C23; age 23). The data were collected by means of interviews in the participants' homes. Table 1 provides an overview of sample sizes and age distributions.

Table 1
Age distribution of sample

		M	Range
C17	MZ	17.01	16-18
	DZ	17.02	16-18
	Siblings	18.65	5-44
	Mothers	47.74	34-63
	Fathers	50.53	34-73
C23	MZ	23.06	21-25
	DZ	23.03	21-25
	Siblings	24.82	7-50
	Mothers	52.59	41-69
	Fathers	55.25	42-79

Note. C17 - younger cohort, C23 - older cohort, MZ - monozygotic twins, DZ - dizygotic twins, *M* -mean.

Measurement

Zygosity. The zygosity of the twins was determined by using a self-report zygosity questionnaire (Oniszczenko, Angleitner, Strelau, & Angert, 1993). This questionnaire consisted of three parts: Items to determine the similarity of the external appearance of the twins, items to determine the frequency with which the twins were confused by others, and items to assess the zygosity of the twins by the parents. The results of the zygotic questionnaire were validated and corrected by using genetic fingerprinting (Hahn et al., 2016).

Achievement Motivation. Achievement motivation was measured for over 16-year olds by two items (*Good achievements mean a lot to me* and *In order to get ahead in life, I am prepared to put in great efforts*), and a sum score was built. Table 2 shows the descriptive statistics and reliability estimates (Cronbach's α) for different sample groups. Scores have been corrected for linear age and gender differences, as these can distort parameter estimates (McGue & Bourchard, 1984).

Table 2
Descriptive statistics for achievement motivation

	N	*M*	*SD*	α
MZ (T1)	1015	8.38	1.32	.59
MZ (T2)	1020	8.34	1.43	.66
DZ (T1)	1019	8.30	1,34	.57
DZ (T2)	1016	8.18	1.48	.65
Siblings	633	8.18	1.41	.65
Mothers	1898	8.03	1.37	.64
Fathers	1159	8.15	1.39	.73

Note. MZ - monozygotic twins, DZ - dizygotic twins, T1 - first-born twin, T2 - second-born twin, *M* – mean, *SD* - standard deviation; α - Cronbach's alpha.

Analyses

The NTFD model was fitted to the data with AMOS Version 24 (Arbuckle, 2014) by using the full information maximum likelihood algorithm. Since the NTFD included the data of the twins, full siblings, and biological parents, the design allowed the decomposition of the variance in achievement motivation into various genetic and environmental components. The model is depicted in Figure 1.

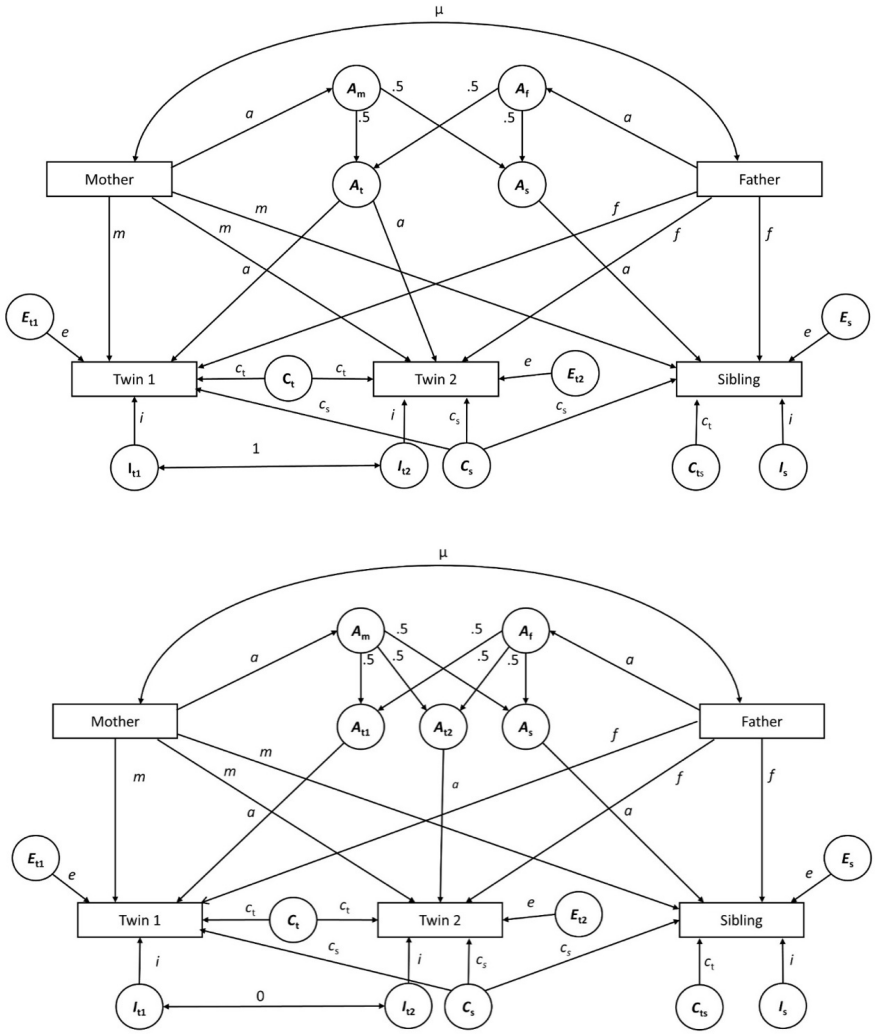

Figure 1. Nuclear Twin Family Model for monozygotic twins (the upper figure) and dizygotic twins (the lower figure). *a* - additive genetic effects; *e* - non-shared environmental effects incl. measurement error, *i* – epistasis-effects, *f* - environmental transmission from fathers to offspring, *m* - environmental transmission from mothers to offspring, *s* - shared environmental effects between siblings, *t* - shared environmental effects between siblings, μ - phenotypic correlation of parents.

The NTFD model specified additive genetic effects (a^2), non-additive genetic effects (i^2; epistasis), non-shared environmental effects confounded with measure-

ment error (e^2), and shared environmental effects that were further partitioned: The inclusion of a non-twin sibling allowed to separate environmental influences shared among all children in a family (cs^2) from environmental influences that were exclusively shared by the twins (ct^2). By including data of the mothers and fathers of the twins, parameters for parental environmental transmission from a mother to offspring (m^2), parental environmental transmission from a father to offspring (f^2), and from both parents to offspring ($m^2 + f^2 + 2mf\mu$) could be estimated, while considering the influence of the correlation between the parents (i.e. assortative mating, μ). In addition, the model we applied allowed us to estimate the influence of passive gene-environment-correlation ($a^2m[1+\mu]+a^2f[1 + \mu]$; Bleidorn et al., 2018). Passive gene-environment-interaction occurred when parents created environmental conditions that matched the child's genetic predisposition due to genetic correspondence with the child (Knopik et al., 2017).

In this NTFD model, non-additive genetic effects and environmental effects shared by all children of the families could not be estimated in the presence of each other (Kandler, Gottschling, & Spinath, 2016). We chose the model $cs = 0$ as a baseline model. This model allowed the estimation of non-additive genetic effects instead of sibling-specific environmental effects, and it was chosen because the correlations provided indication for non-additive genetic influences (see Table 4, a model with $i = 0$, yielded a poorer fit and resulted in a parameter estimate of $cs = 0$). We reduced the baseline model by testing whether a model fixing $m = 0$ and $f = 0$ parameters, and an even more parsimonious model ($m=f=ct=0$), resulted in significantly poorer model fit without any effects of the environment shared by family members. For nested model comparisons, we used the χ^2-difference test. Further goodness-of-fit indices which were considered, were the comparative fit index (CFI; Bentler, 1990), where values close to 1 indicated a good fit, the root mean square of approximation (RMSEA; Browne & Cudeck, 1992), where values close to 0 indicated a good fit and the Akaike information criterion (AIC; Akaike, 1969, 1970), where smaller values indicated a better fit.

Results

Family Correlations

Correlations of different family-dyads are shown in Table 3. The correlation for the MZ twins was more than twice as high as the correlation for the DZ twins. Moreover, the correlation of the MZ twins was substantially higher than in all the other family dyads. This indicated that both additive and non-additive genetic influences might play a role in explaining individual differences in achievement motivation. The average parent-child and twin-sibling correlations were lower than the correlation of the MZ twins, which indicated relevant environmental influences on differences in achievement motivation, which were shared only by the

twins, and not with siblings or parents. The correlations between a mother and a father were not significant, and therefore they provided no evidence for assortative mating.

Table 3
Achievement motivation correlations between dyads of twin family members (for z-standardized residuals/corrected for age and sex differences)

Dyads	*N*	*r*	95% C.I.	*p*
MZ T1 and MZ T2	1013	.37	[.32 - .43]	.00
DZ T1 and DZ T2	1015	.18	[.11 - .25]	.00
Sibling and T1	632	.14	[.06 - .22]	.00
Sibling and T2	631	.11	[.04 - .18]	.00
Mother and T1	1893	.08	[.03 - .12]	.00
Mother and T2	1893	.10	[.05 - .15]	.00
Mother and sibling	598	.01	[-.06 - .09]	.79
Father and T1	1152	.04	[-.02-.10]	.16
Father and T2	1151	.05	[.00 -.10]	.08
Father and sibling	387	.14	[.03 - .24]	.00
Father and mother	1025	.03	[-.03 - .09]	.32

Notes. MZ - monozygotic twin, DZ - dizygotic twin, T1 - first-born twin, T2 - second-born twin. *r* - Pearson-Correlation, C.I. - confidence interval, *p* - two-sided significance.

Results of the Nuclear Twin Family Model

Fit indices for the NTFD are given in Table 4, and results of the model comparisons can be found in Table 5.

Table 4
Nuclear Twin Family Design: Model comparison tests and fit-statistics

Model	χ^2	*df*	*p*	CFI	RMSEA	AIC
cs = 0	28.98	23	.18	.97	.011	62.98
cs = 0 *m* = 0 *f* = 0	32.12	24	.15	.97	.012	62.12
cs = 0 *m* = 0 *f* = 0 *ct* = 0	38.05	26	.06	.94	.015	66.05

Note. cs - environmental effects shared by siblings, *m* - environmental transmission from a mother to offspring, *f* - environmental transmission from a father to offspring, *ct* - environmental effects shared by twins, *p* - two-sided significance, CFI - Comparative Fit Index, RMSEA - Root Mean Square of Approximation, AIC - Akaike Information Criterion.

The model dropping effects of the environment shared by all siblings (*cs*) of a family and the parental paths (*m* and *f*) represented the best compromise between model fit and parsimony. In addition, we tested for cohort differences in the etiology of achievement motivation, by estimating a four-group model allowing for cohort (age) specific parameter estimates. This model did not significantly improve the fit indicating that parameter estimates could be generalized across the age range studied here.

Table 5
NTFD Model comparison: χ^2-difference test

Model	χ^2	*df*	$\Delta\chi^2$	Δdf	*p*
cs = 0	28.98	23			
cs = 0 *m* = 0 *f* = 0	32.12	25	3.15	2	.20
cs = 0 *m* = 0 *f* = 0	32.12	25			
cs = 0 *m* = 0 *f* = 0 *ct* = 0	38.05	26	5.93	1	.01
cs = 0 *m* = 0 *f* = 0	32.12	25			
cs = 0 *m* = 0 *f* = 0 with C. D.	68.45	51	36.33	26	.08

Note. cs = 0 - no environmental effects shared by siblings, *m* = 0 - no environmental transmission from a mother to offspring, *f* = 0 - no environmental transmission from a father to offspring, *ct* = 0 - no environmental effects shared by twins, C. D. - with cohort-differentiation between cohort 3 and 4; for the model *cs* = 0 *m* = 0 *f* = 0 with C.D. each parameter (except μ = phenotypic correlation of the parents) was estimated, *p* - two-sided significance.

The selected model provided evidence for additive genetic influences (a^2), non-additive genetic influences (i^2), twin-specific shared environmental influences (ct^2) and non-shared environmental influences (e^2). Standardized path coefficients and standardized variance components are shown in Table 6.

Table 6
NTFD: Standardizes path coefficients and variance components of the best-fitting model

Standardized path coefficients								
Model	*A*	*i*	μ	*m*	*f*	*cs*	*ct*	*e*
cs = 0 *m* = 0 *f* = 0	.41	.37	.03	.00	.00	.00	.28	.79
Standardized variance components								
Model	a^2	i^2		*COV*	*PAR*		ct^2	e^2
cs = 0 *m* = 0 f = 0	.17	.13		.00	.00		.08	.62

Note. *a* - additive genetic effects, *i* - epistasis-effects, μ - phenotypic correlation between the parents, *m* - environmental transmission from a mother to offspring, *f* - environmental transmission from a father to offspring, c*s* - shared environmental effects between siblings, c*t* - shared environmental effects between twins, *e* - non-shared environmental effects (incl. measurement error), *COV* - passive gene-environment-correlation, *PAR* - environmental transmission from both parents to offspring.

Additive genetic influences explained 17% of the variance in achievement motivation, whereas epistasis-effects explained 13%. Moreover, the results provided evidence for significant twin-specific shared environmental influences, which accounted for about 8% of individual differences in achievement motivation. These environmental influences were specific to twins (ct^2), and not shared with non-twin siblings or parents. The largest portion of the variance (62%) could be attributed to non-shared environmental influences (e^2). There was no evidence for sibling-specific and parent-specific shared environmental effects, effects of assortative mating, and effects of passive gene-environment correlation.

Discussion

This study used data from a large representative twin family sample to examine genetic and environmental contributions to the development of individual differences in achievement motivation using an NTFD model. To the best of our knowledge, this was the first study of achievement motivation that included data of full siblings and biological parents in addition to using the data of twins only, and thus could test additional parameters that were less biased. The results of the NTFD analyses suggested a significant influence of genetic effects ($a^2 + i^2 = 30\%$), which was, however, smaller than that found in previous studies by using the CTD (Kovas et al., 2015; Spinath, 2001; Spinath et al., 2008). Furthermore, the current study showed a significant influence of environmental experiences shared by twins ($ct^2 = 8\%$). The largest part of variance could be attributed to non-shared

environmental effects (e^2 = 62%), just like it was shown in the previous studies using the CTD (Kovas et al., 2015; Spinath, 2001; Spinath et al., 2008).

Consistent with the previous research, this study did not provide any evidence for shared environmental influences shared by parents and offspring, as well as by twins and their non-twin-siblings. Thus, at first glance, the results contradicted studies that could demonstrate a significant relation between achievement motivation and socio-cultural background, family climate and performance expectations, role model effects, and parenting style (Heckhausen & Heckhausen, 2010; Looser, 2011; Röhr-Sendlmeier et al., 2012; Röhr-Sendlmeier & Kröger, 2014).

Two points are important to note. First, studies correlating (even over time) parental characteristics, or characteristics of the home environment, with offspring's characteristics are not informative of the connecting path, which may be environmental or genetic. Our study emphasizes the importance of a genetic path. Second, as outlined above, our results do not imply that parental or family influences are irrelevant to achievement motivation. However, these characteristics might differentially affect children reared in the same family. For example, the parent-child-relationship, which was shown to be correlated with achievement motivation (Looser, 2011), might differ between the children in one family.

The small, but significant effect of the twin-specific environment implies influences of shared demographics, age-specific experiences, peer-groups, and social experiences. Previous studies have confirmed that leisure activities and peer-relationships supply social contacts and opportunities of interaction that can influence a person's achievement motivation (Heckhausen & Heckhausen, 2010). Moreover, compared to siblings of different ages, twins are more likely to attend the same school and classes, and thus make similar experiences at the same age that might affect achievement motivation. Those experiences in the school context might be, for example, the reference orientation, the classroom management of the teachers, and the educational leadership style of the school, which demonstrably influence the achievement motivation of students (Heckhausen & Heckhausen, 2010; Looser, 2017; Wigfield et al., 2006). Finally, twins share the timing of events and changing environmental conditions. For example, an economic situation of the family may improve over the years to the effect that an older sibling grows up in tight economic conditions, whereas family finances are more relaxed for younger siblings.

As mentioned above, the largest amount of variance (62%) could be attributed to non-shared environmental effects (including a measurement error). Individual experiences, such as different peer-relationships, parent-child and teacher-student relationships, experiences in the family and in the school context, as well as individual life events might therefore be of great importance for the emergence of individual differences in achievement motivation (Bakadorova & Raufelder, 2014; Mansour & Martin, 2006, Martin, Marsh, McInerney, Green, & Dowson, 2007; Nelson & DeBacker, 2008).

Limitations

Though the NTFD requires less stringent assumptions than the CTD, allowing a more precise and detailed analysis of genetic and environmental influences on individual differences, it also has its limitations. Firstly, although the NTFD allows to determine the impact of passive gene-environment correlation, it does not obtain enough information to investigate other types of gene-environment interplay, such as active or reactive gene-environment correlations or interaction (Bleidorn et al., 2018; Keller et al., 2010;). Active and reactive gene-environment correlations are confounded with the genetic variance component, and can therefore lead to an overestimation of heritability coefficients, when not taken into account (Bleidorn et al., 2018). Likewise, ignoring gene-environment-interactions could also lead to biased estimates (Kandler & Papendick, 2017).

When interpreting the results of this study, it should be also noted that achievement motivation was only surveyed with two items, and had a moderate internal consistency. Influences of a measurement error could lead to an underestimation of heritability coefficients, since they increased the dissimilarity of monozygotic and DZ twins, and are thus reflected in the variance component of non-shared environmental influences.

Implications and Future Directions

Despite the aforementioned limitations, our study contributes to the research on achievement motivation. The results show that 30% of the individual differences in achievement motivation are influenced by genetic (additive and non-additive) factors, to a small degree by environmental factors which the twins share, and to the biggest part by aspects that are specific for each individual and not shared among family members. This could imply that the family environment of adolescents and young adults plays only a minor role in establishing individual differences in the motivation to perform, and thus contradicts classical educational theories and models. Those non-shared environmental components might well derive from true individual experiences, such as friends and partners. However, it might also reflect experiences that are objectively shared between the children of a family, but perceived differently, such as parenting style. Nevertheless, though we do not challenge the importance of the familial home, our results underpin the necessity to focus on individual aspects of young people in order to understand why they differ in their achievement motivation.

There are obviously several questions left open by our results: What are the environmental influences that contribute to individual differences in achievement motivation? Which of these influences contribute to the similarity of twins, but not non-twin siblings? Detailed measurement of characteristics of the environment in longitudinal genetically informative studies is an obvious way to answer these questions. From a developmental perspective, it is further important to

study both stability and change in the relative contribution of genetic and environmental influences over the lifespan, as well as the contributions of genes and (measured) environments to the stability and change of achievement motivation (Kandler et al. 2010). To answer these questions, it would be helpful to conduct twin studies by using a broader range of age and a longitudinal design to investigate stability and change in variance components over the lifespan (Bleidorn et al., 2018; Kandler & Papendick, 2017). Lastly, we are convinced that future research will profit from epigenetic analyses that provide a novel tool to track environmental influences.

Conclusion

In this study we used the NTFD to derive a detailed picture of the etiology of individual differences in achievement motivation. Like previous studies relying on the classical twin design, we found that variance in achievement motivation was primarily explained by (additive and non-additive) genetic and non-shared environmental influences. In addition, variation could also be explained by environmental factors shared among the twins, albeit to a small degree. Thus, we suggest a revision of models and theories that answer the question of why people differ in their achievement motivation by differences in socialization only.

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ZAŠTO SE LJUDI RAZLIKUJU U MOTIVACIJI KA POSTIGNUĆU? BLIZANAČKA PORODIČNA STUDIJA

Iako mnoge prethodne studije naglašavaju doprinose sredinskih činilaca, poput roditeljskog doma i školskog okruženja, motivaciji ka postignuću, klasične blizanačke studije sugerišu da i aditivni genetski i nedeljeni sredinski uticaji mogu da objasne individualne razlike u oblikovanju ovog fenotipa. Primenom nuklearnog porodičnog dizajna na podatke nemačke nacionalne TwinLife studije, analizirani su genski i sredinski doprinosi ispoljavanju motivacije ka postignuću kod adolescenata i mladih odraslih osoba. Kao što se očekivalo, rezultati su ukazali na značajne uticaje aditivne i neaditivne genetske komponente, kao i na značajne uticaje deljene sredine. Najveći procenat varijanse objašnjen je nedeljenim sredinskim uticajima, ukazujući tako na važnost individualnih iskustava u formiranju razlika u motivaciji ka postignuću. Rezultati ovog istraživanja ukazuju na potrebnu reviziju modela i teorija koje objašnjavaju varijacije u motivaciji ka postignuću isključivo kroz razlike u porodičnoj socijalizaciji.

Ključne reči: bihejvioralna genetika, motivacija ka postignuću, nuklearni porodični dizajn

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TWIN STUDY OF AGGRESSIVENESS AND IMPULSIVENESS RELATIONSHIP[2]

Aggressive and impulsive behaviors have shown sufficient genetic influences and high co-occurrence, thus the question is whether dispositions for these behaviors share unique genetic or environmental contributions. The aim of this research was to explore etiology of phenotypic relationships between aggressiveness and impulsiveness. More precisely, we tested which component of aggressiveness (affective, behavioral, or cognitive) shared the most underlying genetic and environmental influences with impulsiveness. There were applied Serbian adaptation of the Buss-Perry Aggression Questionnaire as a measure of three aggressiveness components, and Behavioral Activation System scale from the Revised Sensitivity Theory Questionnaire as a measure of impulsiveness, on a sample of 208 adult twin pairs (132 pairs were monozygotic). Results of a multivariate biometric method showed that the aggressiveness and impulsiveness could be explained by the common additive genetic (6% of impulsiveness and 16-31% of aggressiveness components), and common non-shared environmental contributions (1% of impulsiveness and 11-47% of aggressiveness components), but those contributions were rather small. An affective component of aggressiveness (anger) showed the most genetic similarity with impulsiveness, indicating that the lack of anger and behavior regulation shared partially the same genetic basis. However, aggressiveness and impulsiveness contained a larger proportion of the specific genetic and environmental effects, which confirmed a distinction between these phenomena.

Key words: aggressiveness, biometric model, genetic and environmental effects, impulsiveness, twin study

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Introduction

Aggression and impulsivity are the most common co-occurring symptoms of psychopathology (Seroczynski, Bergeman, & Coccaro, 1999). Previous research on the etiology of aggression and impulsivity has indicated that both genetic and environmental influences are important for the development of each of these characteristics (e.g., DiLalla, 2002; Plomin, Nitz, & Rowe, 1990). However, the question is whether these characteristics share the same genetic and environmental influences, and whether traits related to the tendency towards these behaviors, i.e. aggressiveness and impulsiveness, share the same genetic and environmental influences. Aggressiveness is a complex trait with specific affective (anger), behavioral (aggressive behavior or aggression), and cognitive components (hostility), called ABC components (see Martin, Watson, & Wan, 2000). Out of all three components, anger shows the higher relationships with impulsivity, indicating the lack of behavioral control in both characteristics (e.g., Grcía-Forero, Gallardo-Pujol, Maydeu-Olivares, & Andrís-Pueyo, 2009). However, the other components of aggressiveness are also related to impulsivity, but to a lesser extent (e.g., Grcía-Forero et al., 2009). These relationships raise the question whether some specific component of aggressiveness share the same genetic and environmental influences with the impulsiveness.

Genetic and Environmental Influences of Aggressiveness

In order to explore genetic and environmental influences on aggressiveness components, Coccaro, Bergeman, Kavoussi, and Seroczynski (1997) conducted a study on adults, using only subscales from Buss-Durkee Hostility Inventory (BDHI) that constituted aggressiveness factor: direct assault (physical aggression), verbal assault (verbal aggression), indirect assault (indirect aggression, such as a malicious gossip, but also an inhibition of temper tantrums), and irritability (quick temper, grouchiness, and exasperation). They showed that genetic influences explained 47% of direct assault, 40% of indirect assault, 37% of irritability, and 27% of verbal assault, while non-shared environmental influences explained 53-72% of the rest of the variance. In study by Sluyter et al. (2000), results showed that there was a distinction in genetic end environmental factors between the affective component of aggressiveness (which included a type A personality, anger, irritability, and resentment), and behavioral component (which included assault, negativism, and verbal hostility). Moreover, in the same study, environmental factors were remarkably higher for indirect hostility, anger, and verbal hostility (77%, 75%, and 61%, respectively), while the genetic factors for the assault and irritability (48% and 46%, respectively) were almost the same as environmental factors (52% and 54%, respectively). Later research on children, which measured indirect aggression only as a social aggression without temper tantrums and similar, showed that physical aggression was largely explained by the genetics factors, while the social aggression was explained by the non-shared

environmental factors. However, both types of aggression shared overlapping genes to a large extent, and overlapping environmental factors only to a small extent (Brendgen et al., 2005).

Vernon, McCarthy, Johnson, Lang, and Harris (1999) used an improved measure of aggressiveness among adults, Buss-Perry Aggression Questionnaire (BPAQ), which captured all ABC components and comprised of four subscales: physical and verbal aggression as motor or behavioral components, anger as an affective component, and hostility as a cognitive component. They showed that 49% of physical aggression, 36% of anger, and 36% of hostility could be explained by the genetic effects, while verbal aggression was explained only by environmental effects. Results on other measures of physical aggression were similar, with 27-39% of variance explained by genetic effects. However, there were inconsistent results regarding verbal aggression, which showed 36-42% of genetic effects, when it was measured via other than BPAQ measure (Vernon et al., 1999). In a joined factor analysis of several measures of aggressiveness and related constructs, impulsivity was loaded on the same factor as anger and hostility, but it was not loaded on the factor which constituted physical and verbal aggression. However, the genetic influences in both factors were the same (52%, see Vernon et al., 1999). Based on this research, we could conclude that physical aggression was largely influenced by the genetic factors, while the other aggressiveness components were influenced mostly by environmental factors, as well as by the genetic factors to a lesser extent.

Besides a distinction by ABC components, there is the distinction of aggressive behavior based on its function (e.g., Bushman & Bartholow, 2010), which is also important in the context of relationship with impulsivity. Based on the functions, aggression could be reactive or proactive. Reactive aggression refers to aggressive behavior as a response to real or perceived provocation and threat, and it is aimed to harm another person. Reactive aggression has been characterized as involving high emotional arousal, anger, hostility, and lack of behavioral and affect control, and therefore it is more related to impulsivity (e.g., Merk, Orobio de Castro, Koops, & Matthys, 2005; Raine et al., 2006). On the contrary, proactive aggression is instrumental, aimed to achieve other goals, such are money, social status, justice, etc., and it is related to positive expectations about the outcomes of aggression, and problems with impulse and affect control to the lesser extent (e.g., Merk et al., 2005; Raine et al., 2006). Previous studies on children and adolescents have shown that the genetic effects are higher in proactive aggression later in adolescence, compared to reactive aggression, although environmental factors, especially non-shared ones, explain greater or almost equal proportion of variance as genetic factors in both aggression types (e.g., Tuvblad, Raine, Zheng, & Baker, 2009).

Genetic and Environmental Influences of Impulsiveness

Like aggressiveness, impulsiveness is also a complex construct. Although impulsiveness is commonly defined as a predisposition toward rapid, unplanned reactions

without regard to the consequences of these reactions (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001), there is still no consensus on the definition, theoretical, and operational status of this trait (Congdon & Canli, 2008). Thus, there is no consensus regarding its components, and it seems that behavioral or motor component is dominant in describing the impulsiveness (e.g., reduced inhibitory control, rapid reactions), followed by the cognitive component (decreased sensitivity to negative consequences, lack of planning, see Barratt, 1993). Instead of ABC components, dimensions of impulsiveness are rather described in terms of functional factors (tendency to act with relatively little forethought, e.g., fast and with willingness to take advantage of a particular moment) or dysfunctional factors (tendency to act with less forethought than most people with equal ability when this tendency is a source of difficulty, see Dickman, 1990), or factors such as urgency, lack of premeditation, lack of perseverance, and sensation seeking (Whiteside & Lynam, 2001).

The most consistent result in various twin studies shows robust evidence of heritability of impulsiveness, confirming the influences of additive genetic factors (e.g., Andoet al., 2004; Eaves et al., 2000; Hur & Bouchard, 1997; Jang, Livesley, Angleitner, Riemann, & Vernon, 2002), or non-additive or dominant genetic factors (e.g., Hur & Bouchard, 1997; Pedersen, Plomin, McClearn, & Frisberg, 1988; Seroczynski et al., 1999). In spite of the partial disagreement among the findings of the research on genetic contribution to the personality traits related to impulsiveness, the results of a large number of twin and adoptive studies have shown heritability rates that range from 20% to 62%. (e.g., Gustavson, Miyake, Hewitt, & Friedman, 2014; Niv, Tuvblad, Raine, Wang, & Baker, 2012; Seroczynski et al., 1999).

A recent meta-analysis (Bezdjian, Baker, & Tuvblad, 2011), which was systematically examined the heritability of impulsivity across twins and adoptive studies of infants, children, adolescents, and adults, estimated overall 38% of additive genetic, 12% of non-additive genetic, and 50% of non-shared environmental influences of impulsiveness. Although overall genetic influences were 50%, the relative importance of these effects varying across different subdimensions of impulsiveness. Genetic effects for the lack of persistence was 69%, for sensation seeking it was 47%, and for lack of planning it was 41%, while remaining of the variance was captured by the non-shared environmental effects (Bezdjian et al., 2011). However, the authors concluded that even though impulsiveness was a multidimensional construct, the genetic and environmental influences on the different subtraits seemed to have similarities in the magnitude of genetic and environmental effects.

The Present Study

Due to co-occurrence and overlapping between some aspects of aggressiveness and impulsiveness, the aim of this study was to explore etiology of the phenotypic relationships between these characteristics among adult twin sample. Considering the multidimensionality of aggressiveness, the question was which its component (affective, behavioral, or cognitive) shared underlying influences contributing to

impulsiveness. There were only a few previous studies addressed to this problem. For example, the study by Seroczynski et al. (1999) showed that irritability, as the aggressiveness component, mostly related to anger, while impulsivity had a greater portion of shared genetic and environmental factors, compared to the others components of aggressiveness, such as direct, verbal, or indirect assault. However, in the mentioned study, no distinction between three main ABC components of aggressiveness was made. Therefore, in this study we attempted to overcome this limitation by using measure of ABC components of aggressiveness. Previous research showed that aggressiveness components showed different heritability pattern, with large variation in genetic contribution (e.g., Vernon et al., 1999). Unlike aggressiveness, a distinction between ABC components could not be made in impulsiveness. Moreover, subdimensions of impulsivity showed similar contribution of the genetic and environmental influences (Bezdjian et al. 2011), and impulsiveness was threatened in this study as a one-dimensional construct.

Method

Sample and Procedure

The sample consisted of 416 twins, were 264 twins were monozygotic (MZ), and 152 of them were dizygotic (DZ). Out of 132 MZ twin pairs, 29 were males and 103 were females. From 76 DZ twin pairs, 11 were males, 31 were females, and 34 pairs were of different gender. Zygosity was determined on the basis of DNK analysis for 94.5% (197) of twin pairs. Zygosity estimation for the remaining 11 (5.3%) twin pairs was computed from the Twins Physical Resemblance Questionnaire (Oniszczenko, Angleitner, Strelau, & Angeri, 1993). This questionnaire included a series of questions about similarities and dissimilarities between two twins, within the twin pair (e.g., eye color, body weight, body height, etc.). Zygosity estimation based on this questionnaire was reliable in 95% of cases in previous researches (Reed et al., 2005; Spitz et al., 1996). Participants age ranged from 18 to 58 years old (M = 24.56, SD = 7.47). This study included twin pairs from the entire territory of the Republic of Serbia, with a slightly higher number of twins who currently lived in Novi Sad and Belgrade. Participants were recruited in the period from 2011 to 2018. The invitation for participation in the research was sent via media, press, website, and social networks, and applications for the participation were made through the website (www.blizanci.rs), or via telephone contact. Data collection was mostly done at the Faculty of Philosophy in Novi Sad, while a small part of the sample was collected at the Faculty of Philosophy in Belgrade, Niš, and Novi Pazar. Some participants filled out questionnaires at home via online platform. As the research involved the assessment of phenomena in various fields of psychology and medicine, the session lasted from 3 to 5 hours, with a break for a meal and refreshments. The participation in the research was voluntarily, and

the research was approved by the Ethics Commission of the Faculty of Philosophy, University of Novi Sad, Serbia, which was the Second Instance Commission of the Ethical Committee within the Serbian Psychological Society.

Instruments

Buss-Perry Aggression Questionnaire (BPAQ; Buss & Perry, 1992, for Serbian adaptation see Dinić & Janičić, 2012). BPAQ consists of 29 items with five-point response scale (from 1 - *strongly disagree* to 5 - *strongly agree*), which measure four dimensions of the aggressiveness: Physical Aggression (9 items; α = .77), Verbal Aggression (5 items; α = .61), Anger (7 items; α = .74), and Hostility (8 items; α = .76). The Anger represents an affective component, Hostility represents a cognitive component, and Physical and Verbal Aggression represents motor or behavioral components of aggressiveness.

The Reinforcement Sensitivity Questionnaire (RSQ; Smederevac, Mitrović, Čolović, & Nikolašević, 2014) - Behavioral activation system (BAS) scale. RSQ is a measure of the dimensions from the revised Gray's model of personality: Behavioral inhibition system - BIS, Behavioral activation system - BAS, and Fight, Flight, and Freeze system. The items are rated on a 4-point scale, ranging from 1 - *completely disagree* to 4 - *completely agree*. For the purpose of this research, only the BAS scale has been used as the measure of impulsivity (6 items; α = .76). BAS refers to impulsivity, i.e., sensitivity to signals of reward (e.g., *When I want something, I never think about possible obstacles*), and preferring new and exciting situations (e.g., *I readily accept new and exciting situations*).

Data Preparation and Analysis

Missing values were replaced by using the expectation maximization (EM) algorithm. The use of the EM algorithm was justified by the insignificant Little MCAR test, for each BPAQ dimensions and the BAS scale (*p* values ranged from .078 to .744). Replacement of missing values, descriptive statistical parameters, correlations, and α coefficient, were calculated in the SPSS v.21 software (IBM corp., 2012). The scores on the BPAQ and BAS scale were partialized for sex and age.

Phenotypic similarities between MZ and DZ were examined in each dimension by using a structural equation modeling (SEM), or more precisely, a univariate biometric method. In this method, the total variation of the phenotype could be explained by two types of genetic variance (additive – A, and non-additive – D), and two types of environmental variance (shared environmental variance – C, non-shared environmental variance, and measurement error - E). It was possible to test several models: ACE, ADE, AE, CE, and E. An important specificity of the biometric model was to fix the values of certain parameters. Parameter A was fixed at 1.00 for MZ, since they shared 100% of the genes, while this parameter was fixed to 0.50 for DZ, since they shared about 50% of their genes on average. Pa-

rameter C was fixed to 1.00 in both MZ and DZ, due to the assumption that twins shared 100% of the shared environmental variance. If there was an identical form of genetic and environmental effects for variables in the univariate models, then the multivariate biometric model was applied. Two multivariate models were tested: an independent pathway model and a common pathway model (Rijsdijk & Sham, 2002). In both models there were specific (s) and common (c) genetic and environmental sources of variance, but in the case of the independent model, the sources interacted independently, while in the case of the common pathway model, a common mechanism of decomposition of variance was introduced as an additional latent variable within the model (see Figure 1).

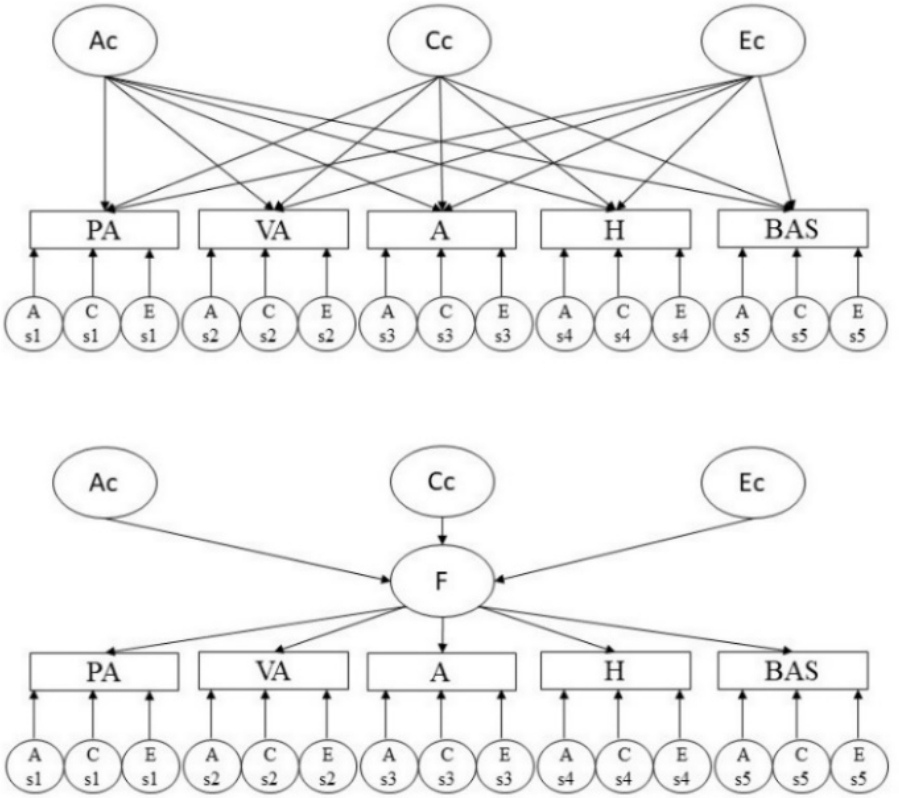

Figure 1. ACE independent pathway model (top pannel), and ACE common pathway model (bottom panel) for four BPAQ dimensions and BAS scale.
Note. PA – physical aggression, VA – verbal aggression, A – anger, H – hostility, BAS – behavioral activation system, *Ac* – common additive genetic variance, *Cc* – common shared environmental variance, *Ec* – common non-shared environmental variance and measurement error, F - common factor, A, C, and E refer to specific additive genetic, shared environmental, and non-shared environmental variances, respectively.

The overall model fit was estimated through several indicators: Comparative fit index (CFI) and Tucker-Lewis index (TLI), with acceptable values above .90, the standardized root mean square residual (SRMR), which acceptable value was below .08, and the root mean square error of approximation (RMSEA), which acceptable values were below .10 (Hu & Bentler, 1999; MacCallum, Browne, & Sugawara, 1996). In addition, Bayesian information criteria (BIC) was used to compare the model, with a lower value indicating better fit. Univariate and multivariate SEM were carried out in the "lavaan" R package (Rosseel, 2012).

The parameter estimates from the best-fitting model could be used to calculate the extent to which phenotypic correlations were due to common genetic (*Ac*) vs. common environmental factors (*Ec* or *Cc*). For example, in order to calculate the total phenotypic correlation between BAS and anger from AE multivariate model, first the A_C pathways for BAS and anger were multiplied together, as well as the E_C pathways for BAS and anger. These products were then added to calculate the total phenotypic correlation. To estimate to what extent genetic factors contribute to this correlation, the product of the A_C pathways was divided by the total phenotypic correlation.

Results

Descriptive Statistics and Correlations

Based on the values of skewness and kurtosis (Table 1), it could be seen that the data were normally distributed. They did not come out of the recommended range of ± 1.5 (see Tabachnick & Fidell, 2013). Correlations between MZ twins were consistently higher than correlations between DZ twins. Correlations between MZ twins were positive, significant, and moderately strong on all five measures. Correlations between DZ twins were positive, significant, and moderately strong for physical aggression and hostility, while they were not significant for the remaining dimensions.

Table 1
Descriptive statistics and correlations

	Scale	*M*	*SD*	*Sk*	*Ku*	r_{MZ}	r_{DZ}
Whole sample (*N* = 416)	Behavioral activation system	16.81	3.33	-0.17	0.12	.48**	.09
	Physical aggression	16.58	5.65	1.13	1.27	.50**	.31**
	Verbal aggression	13.96	3.31	0.19	0.09	.32**	.20
	Hostility	19.62	5.73	0.50	0.02	.55**	.29*
	Anger	16.48	4.95	0.49	0.00	.26**	.18
MZ twins (*n* = 254)	Behavioral activation system	16.88	3.44	-0.06	-0.09		
	Physical aggression	16.41	5.67	1.15	1.21		
	Verbal aggression	13.76	3.38	0.17	-0.03		
	Hostility	19.51	5.88	0.64	0.26		
	Anger	16.27	4.77	0.51	-0.14		
DZ twins (*n* = 140)	Behavioral activation system	16.60	3.32	-0.37	0.27		
	Physical aggression	17.10	5.64	1.09	1.51		
	Verbal aggression	14.19	3.24	0.38	0.39		
	Hostility	19.86	5.52	0.28	-0.37		
	Anger	16.86	5.27	0.51	0.18		

Notes. M – mean, *SD* – standard deviation, *Sk* – skewness, *Ku* – kurtosis, r_{MZ}– correlations between monozygotic twins, r_{DZ}– correlations between dizygotic twins. * $p < .05$. ** $p < .01$.

Biometrical Models

The results of the univariate genetic modeling are shown in Table 2. Based on the BIC criteria, the AE model stands out as the most optimal in the case of the dimensions of physical aggression, hostility, and anger, as well as in the case of the BAS. In the case of the verbal aggression, the AE and CE model have almost identical BIC values, but the AE model is retained, in line with results from the other BPAQ scales, as well as with the previous studies in which verbal aggression has shown genetic influences, although to a small extent (Coccaro et al., 1997; Vernon et al., 1999). The remaining fit indices are within acceptable boundaries for all AE models. Additive genetic effects are stronger for physical aggression (A = .51, E = .49) and hostility (A = .54, E = .46), while the effects of the shared environment

are stronger for the BAS (A = .45, E = .54), verbal aggression (A = .31, E = .69), and anger (A = .29, E = .71).

Table 2
Fit indices for univariate models

	Model	$\chi^2(df)$	BIC	CFI	TLI	RMSEA (95% CI)	SRMR
BAS	ACE	1.91(3)	1127.25	1.000	1.022	0.000 (0.000 – 0.143)	.060
	AE	**1.91(4)**	**1121.97**	**1.000**	**1.032**	**0.000 (0.000 – 0.106)**	**.060**
	CE	8.21(4)	1128.27	0.871	0.936	0.103 (0.000 – 0.205)	.098
	E	35.87(5)	1150.64	0.058	0.623	0.250 (0.177 – 0.330)	.172
PA	ACE	3.60(3)	1103.03	0.986	0.990	0.045 (0.000 – 0.182)	.075
	AE	**4.04**(4)	**1098.18**	**0.999**	**1.000**	**0.010 (0.000 – 0.153)**	**.078**
	CE	6.16(4)	1100.30	0.949	0.974	0.074 (0.000 – 0.182)	.080
	E	47.03(5)	1135.89	0.000	0.599	0.292 (0.219 – 0.371)	.213
VA	ACE	0.48(3)	1133.89	1.000	1.117	0.000 (0.000 – 0.057)	.032
	AE	**0.83(4)**	**1128.95**	**1.000**	**1.111**	**0.000 (0.000 – 0.040)**	**.032**
	CE	**0.81(4)**	**1128.93**	**1.000**	**1.111**	**0.000 (0.000 – 0.037)**	**.039**
	E	16.79(5)	1139.62	0.175	0.670	0.155 (0.077 – 0.239)	.129
H	ACE	1.67(3)	1098.60	1.000	1.017	0.000 (0.000 – 0.136)	.061
	AE	**1.88(4)**	**1093.53**	**1.000**	**1.021**	**0.000 (0.000 – 0.105)**	**.060**
	CE	5.45(4)	1097.10	0.972	0.986	0.061 (0.000 – 0.173)	.086
	E	55.22(5)	1141.58	0.026	0.611	0.319 (0.247 – 0.398)	.220
AN	ACE	3.59(3)	1133.10	0.941	0.960	0.045 (0.000 – 0.181)	.084
	AE	**3.68(4)**	**1127.91**	**1.000**	**1.016**	**0.000 (0.000 – 0.147)**	**.086**
	CE	4.22(4)	1128.45	0.977	0.989	0.024 (0.000 – 0.156)	.082
	E	15.44(5)	1134.38	0.000	0.578	0.146 (0.067 – 0.231)	.136

Note. BAS – behavioral activation system, PA – physical aggression, VA – verbal aggression, H – hostility, AN – anger, A – additive genetic variance, C – shared environmental variance, E – non-shared environmental variance and measurement error. Models with the best fit indices are bolded.

As an identical mechanism of genetic and environmental effects was identified for all tested measures in univariate models, multivariate genetic modeling was applied. Based on the BIC criteria, the best model for both common and independent pathway models was the AE model. Fit indices for both AE models were within acceptable boundaries, except SRMR, which was slightly above .08.

Although both AE independent and common multivariate models had the best fit and similar genetic, as well as environmental contributions, we presented contributions only in parsimonious independent AE model. For comparison, contributions in common AE model could be found in Appendix.

Table 3
Fit indices for multivariate models

	Model	χ^2(*df*)	BIC	CFI	TLI	RMSEA (95% CI)	SRMR
Independent	ACE	102.7 (80)	5239.2	.962	.957	.054 (.009 - .082)	.084
	AE	**111.5 (90)**	**5195.2**	**.964**	**.964**	**.049 (.000 - .077)**	**.085**
	CE	124.3 (90)	5208.0	.943	.943	.062 (.032 - .087)	.091
	E	242.5 (100)	5273.3	.762	.785	.120 (.101 - .140)	.168
Common	ACE	115.8 (87)	5215.4	.952	.950	.058 (.024 - .084)	.088
	AE	**116.2 (93)**	**5184.0**	**.961**	**.962**	**.050 (.000 - .077)**	**.089**
	CE	130.6 (93)	5198.5	.937	.939	.064 (.035 - .089)	.095

Note. A – additive genetic variance, C – shared environmental variance, E – non-shared environmental variance and measurement error. Common E model has not converged. Models with the best fit indices were bolded.

Results from the independent pathway model suggest that the genetics effects were higher in the case of hostility, while the genetic and environmental effects were equally contributed in the case of physical aggression. For all other dimensions environmental effects were stronger than genetic effects. Although BAS and other dimensions of aggressiveness share some of the common genetic contributions, it's noticeable that specific genetic contribution is higher for BAS then in the other aggressiveness dimensions. It is also noticeable that anger does not have a specific genetic contribution, but only common genetic contribution, while verbal aggression has a very low specific genetic contribution.

Table 4
Genetic and environmental contributions for AE independent multivariate model

Scale	*Ac*	*As*	h^2	*Ec*	*Es*	e^2
Behavioral activation system	0.06	0.39	0.45	0.01	0.54	0.55
Physical aggression	0.24	0.27	0.51	0.11	0.39	0.49
Verbal aggression	0.25	0.07	0.32	0.25	0.43	0.68
Hostility	0.16	0.36	0.52	0.15	0.33	0.48
Anger	0.31	0.00	0.31	0.47	0.22	0.69

Note. *Ac* – common genetic contribution, *As* – specific genetic contribution, *h*2 – total genetic contribution, *Ec* – common non-shared environmental contribution, *Es* – specific non-shared environmental contribution, e^2 – total non-shared environmental contribution.

Phenotypic correlations between aggressiveness components and impulsiveness were ranged between .12 (with hostility) and .18 (with anger, see Table 5). In all these correlations, the source of correlations was larger for the same genetic contribution, compared to the contribution of the non-shared environmental factors.

Table 5
Genetic and environmental contributions to the phenotypic correlations

Variance	r_g	r_e	r_f	*Ac(%)*	*Ec(%)*
Behavioral activation system X physical aggression	.25	.04	.14	85	15
Behavioral activation system X verbal aggression	.33	.00	.16	79	21
Behavioral activation system X hostility	.21	.05	.12	79	22
Behavioral activation system X anger	.37	.07	.18	76	24
Physical aggression X verbal aggression	.59	.30	.41	59	41
Physical aggression X hostility	.37	.28	.33	58	42
Physical aggression X anger	.68	.39	.49	54	46
Verbal aggression X hostility	.48	.36	.40	48	52
Verbal aggression X anger	.88	.51	.62	44	56
Hostility X anger	.55	.48	.49	44	56

Note. r_g – genetic correlation, r_e – environmental correlation, r_f –phenotypic correlation, *Ac* –genetic contribution to the phenotypic correlations, *Ec* – environmental contribution to the phenotypic correlations.

Discussion

The aim of this research was to explore which component of aggressiveness (affective, behavioral, or cognitive) shared underlying genetic and environmental influences with impulsiveness. The results of biometric modeling showed that aggressiveness and impulsiveness shared some additive genetic influences, ranged from 6% (BAS) to 31% (anger). As we could see, impulsiveness had the least contribution in shared additive genetic influences (BAS), with larger proportion of specific genetic variance (39%). Thus, although aggressiveness and impulsiveness shared some genetic basis, the results indicated that these two traits had unique genetic influences. In other words, aggressiveness and impulsiveness were mostly distinctive traits with specific patterns of genetic and environmental contributions. This was in line with previous results (Seroczynski et al., 1999), and we could assume that measurement assessment did not influence the results.

Although generally aggressiveness and impulsiveness were distinct traits from the aggressiveness components, anger showed the higher phenotypic correlation with the impulsiveness (.18), and this correlation was largely due the same genetic influences (76%). Thus, affective component of aggressiveness shared partially the same genetic basis as the impulsiveness. This was in line with previous studies which showed that impulsiveness was mostly related to the affective component of aggressiveness (e.g., Grcía-Forero et al., 2009; Vernon et al., 1999), and that they shared some genetic influences, compared to the relations between impulsiveness and other aggressiveness components (Seroczynski et al., 1999).

The explanation of shared genetic influences of anger and impulsiveness could be found in neurobiological studies. Brown, Manuck, Flory, and Hariri (2006) showed the synergistic relationship of inhibition- and arousal-related neural circuitry as they contributed to dispositional impulsivity. Results of this study suggested that the ability to modulate impulses, experiences, and responses (i.e., impulsiveness) was, at least in a part, determined by the functional interplay of corticolimbic arousal and control circuits. As well as in a case of impulsiveness, neurobiological markers that were most often associated with individual differences in aggressiveness were related to the activity of prefrontal cortex and limbic regions. More precisely, the prefrontal cortices played a key role in inhibiting limbic regions involved in the generation of the aggression. The anterior cingulate cortex might be involved in evaluating affectively charged stimuli, just as the amygdala responded to threat and provocative stimuli. At the level of neurotransmitters, an important neurotransmitter that was considered to have an important role in the regulation of affective conditions was serotonin. Serotonergic activity in the central nervous system correlated negatively with aggressiveness, impulsiveness, and anger-related personality traits in diverse clinical, forensic, and non-patient populations (Coccaro et al., 1989; Linnoila et al., 1983; Manuck et al., 1998). Moreover, reactive aggression, which was characterized by the impulsivity, appeared to be more governed by the serotonergic pathways, while instrumental

or proactive aggression appeared to be more governed by the dopaminergic pathways which mediateed in learning, motivation, and attaching the importance to the stimulus, including reward (Nelson & Trainor, 2007).

Among aggressiveness components, hostility seemed to be the most different from impulsiveness regarding the genetic basis. Hostility as a cognitive component of aggressiveness captured antagonistic and hostile attitude towards others, but in BPAQ it also captured lack of self-esteem, jealousy, bitterness, etc. (Buss & Perry, 1992). Regardless of specific operationalization of hostility, it was not related to any immediate expression of aggressive motives and impulses, but rather to covert or passive aggression, which was more subtle (Dinić, Mitrović, & Smederevac, 2010). In other words, hostility was not necessarily related to the lack of behavior control under state of anger and rage.

Although the results of this study offered an important contribution to the determination of the etiology of aggressiveness and impulsiveness, there were several limitations of this research. First, impulsiveness was also multidimensional trait. However, there was a lack of adequate measure of impulsiveness components in terms of sound psychometrics properties (e.g., Barratt Impulsiveness Scale, in Steinberg, Sharp, Stanford, & Tharp, 2013), or a distinction among ABC components (e.g., UPPS; Whiteside & Lynam, 2001). Second, the used BAS scale seemed closer to the functional impulsivity, while aggressiveness seemed closer to the dysfunctional impulsivity (Smillie & Jackson, 2006). Moreover, although in Reinforcement sensitivity theory and its revision, BAS was considered as impulsivity trait, and its correlated to different types of impulsivity (Quilty & Oakman, 2004), some research suggested that BAS also included a part of the variability with extraversion or positive emotionality (e.g., Smederevac et al., 2014; Smillie, Pickering, & Jackson, 2006). The question of the dimensionality of BAS was related to the problem of the distinction among sensitivity to signals of reward, which was associated with impulsive behavior, and sensitivity to reward itself, which was not necessarily associated with impulsivity. Thus, the used BAS scale from the RSQ captured various aspects of BAS contained in other scales of this construct. Third, all used measures were self-reported, so the shared method could also influence the correlations, as well as the social desirability, given that both traits were socially undesirable. Fourth, the sample structure might also bias the results, because our participants were in most cases young females. However, the sex and age effects were partialized out.

Taken together, the results have indicated that aggressiveness and impulsiveness have differences that are manifested in unique genetic contributions. Although these two traits are distinct, the aggressiveness component which is the closest to the impulsiveness is the affective component, i.e., anger. Thus, difficulties in anger regulation and behavioral control clearly share the same genetic basis in some part.

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Appendix

Table A
Specific and common genetic and environmental contributions for AE multivariate common model

Scale	*Ac*	*As*	h^2	*Ec*	*Es*	e^2
Behavioral activation system	0.02	0.42	0.44	0.02	0.54	0.56
Physical aggression	0.16	0.31	0.46	0.16	0.37	0.54
Verbal aggression	0.25	0.07	0.32	0.26	0.42	0.68
Hostility	0.15	0.36	0.51	0.16	0.33	0.49
Anger	0.36	0.00	0.36	0.37	0.26	0.64

Note. *Ac* – common genetic contribution, *As* – specific genetic contribution, h^2 – total genetic contribution, *Ec* – common non-shared environmental contribution, *Es* – specific non-shared environmental contribution, e^2 – total non-shared environmental contribution.

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BLIZANAČKA STUDIJA ODNOSA IZMEĐU AGRESIVNOSTI I IMPULSIVNOSTI

U objašnjenju agresivnih i impulsivnih ponašanja značajne doprinose ostvaruju genetski uticaji, a ujedno postoji i visok komorbiditet između ovih ponašanja. S obzirom na to, postavlja se pitanje da li su predispozicije za agresivna i impulsivna ponašanja pod uticajem istih genetskih i sredinskih činilaca. Osnovni cilj ovog istraživanja je ispitivanje etiologije fenotipske povezanosti agresivnosti i impulsivnosti. Preciznije, ispitano je koja komponenta agresivnosti (afektivna, bihejvioralna ili kognitivna) deli zajedničke genetske i sredinske činioce sa impulsivnošću. Na uzorku od 208 odraslih blizanačkih parova (132 monozigotnih blizanaca), srpska adaptacija Bas-Perijevog upitnika agresije je primenjena kao mera trikomponentne agresivnosti, i Skala bihejvioralnog sistema aktivacije iz Upitnika osetljivosti na potkrepljenje. Rezultati multivarijatnog biometrijskog metoda pokazuju da se agresivnost i impulsvinost mogu objasniti na osnovu zajedničke aditivne genetske (6% varijanse impulsivnosti i 16-31% varijanse komponenti agresivnosti) i zajedničke nedeljene sredinske varijanse (1% varijanse impulsivnosti i 11-47% varijanse komponenti agresivnosti), ali su ovi doprinosi mali. Afektivna komponenta agresivnosti (bes) pokazuje najviše genetske sličnosti sa impulsivnošću. Ovaj rezultat ukazuje na to da nedostatak regulacije besa i bihejvioralne kontrole dele, jednim delom, istu genetsku osnovu. Međutim, i agresivnost i impulsivnost sadrže veliki doprinos specifičnih genetskih i sredinskih efekata, što potvrđuje da su u pitanju različiti fenomeni.

Ključne reči: agresivnost, biometrijski model, blizanačka studija, genetski i sredinski efekti, impulsivnost

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SENSATION SEEKING AND RISKY BEHAVIORS IN LIGHT OF GENETIC AND ENVIRONMENTAL FACTORS[2]

The study was conducted on a sample of 171 pairs of adult twins with the aim of determining the level of the genetic and environmental influence on individual differences for an optimal level of arousal (Zuckerman's sensation seeking construct), and tendencies towards risky behavior (smoking and alcohol abuse). Sensation Seeking Scale and Personal Information Questionnaire were applied. The results have shown that sensation seeking subdimensions belong to the category of highly heritable personality traits (50% - 63%), as well as the smoking addiction (75%). An unshared environmental influence has also proven to be important for these variables. As for the alcohol abuse, it has been proven that it is mostly determined by the effect of the unshared environment (68%). Additionally, the additive genetic factor mainly contributes to covariation between sensation seeking constructs and different risky behaviors.

Key words: behavioral genetics, drinking, sensation seeking, smoking, twin study

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Introduction

According to the World Health Organization, alcoholism and smoking are ones of the most widespread addiction diseases. It is estimated that around 2 billion people consume alcohol all over the world, with 76.3 million people having alcohol addiction. Given the fact that the alcohol consumption is associated with more than 200 types of illnesses and injuries, it has been estimated that alcohol abuse caused more than 3 million deaths worldwide in 2016 (WHO, 2018).

In Serbia, 3.4% of the population consumes alcohol on a daily base, and almost 40% of the population drinks alcohol periodically. The largest percentage of people who drink is in the age group from 20 to 34 (WHO, 2018). Not only that alcohol abuse is a socially acceptable activity, but it is also favored to the level of compulsory rituals in many social situations (celebrations, weddings, weekend nights out, etc.). Therefore, the current situation in this field is characterized by a high incidence of alcohol consumption, especially among young people. Risky drinking exceeds 14 SD (standard drinks) per week or more than 4 SD during specific situations for men, while for women it is more than 7 SD per week, or more than 2 SD during specific situations (Institute of Public Health of Serbia, 2008).

Smoking is a risk factor for 6 to 8 diseases that are leading causes of death, such as malignant diseases and cardiovascular diseases. According to the World Health Organization, there were around 1.1 billion regular smokers in the world in 2016 (WHO, 2018). It is estimated that tobacco is responsible for the deaths of half its consumers, and that the number of deaths is 6 million per year. The assumption is that the number of deaths by 2030 will reach 8 million people per year.

Data from 2013 shows that about 30% of the population smokes in Serbia. Serbia is the second country in the world by the number of heart and brain strokes caused by smoking. The worrying fact is that about 10% of smokers belong to the population of young people between 13 and 15 years old. Also, about 77% of young people of that age live with someone who smokes in their presence (WHO, 2013).

Most of the previous studies have tried to determine factors that lead to the risky behavior and a habit of abusing these psychoactive substances through examining the biological factors (genetic, neurological, and biological basis of psychological functioning), psychological development in adolescence, interpersonal (a family system, peer relationships), and environmental factors (Despotović at al., 2013). Since behavioral geneticists can evaluate the degree in which genetic and environmental factors, as well as their interaction, contribute to the variability in phenotypic characteristics, it should provide the most comprehensive understanding of these behaviors.

Smoking and Drinking: Genetic and Environmental Factors

Based on the results of the previous studies, it can be concluded that individual differences in the development of nicotine dependence are predominantly determined by genes (Vink, Willemsen, & Boomsma, 2005). These studies have estimated that the gene influence determines up to 75% of the variance of smoking addiction. These results have been explained by the neurological basis of the dopaminergic system. The results suggest that the remaining variance can be explained by an unshared environment, i.e. events and environment characteristics specific to an individual. Findings that emphasize the importance of a shared, family environment are less consistent. This is probably due to the influence of the age cohort or the culture influence (Tsuang, Bar, Harley, & Lyons, 2001). However, when it comes to initial smoking, the influence of genes and the shared environment, such as the family environment and the influence of culture, i.e., the attitudes of the environment on the consumption of cigarettes, are primarily emphasized (True et al., 1997).

The genetic contribution to alcohol abuse and alcohol dependence have been examined in a few twin studies. The results suggest that 40-60% of the variance of propensity to drink alcohol can be explained by the genetic effect. The remaining variance is explained by an unshared environment, while the shared or family environment has not proved to be significant in explaining individual differences in alcohol abuse (Goldman, Oroszi, & Ducci, 2005; Prescott et al., 2006; Tsuang et al., 2001). It is important to note that the findings supporting the genetic explanation of alcohol abuse do not only refer to alcoholism as a diagnosed psychological disorder, but also to the inclination of a person to exaggerate in drinking alcohol.

Although the results of behavioral genetic studies show little influence of the shared or unshared environment, the results of recent studies suggest that the behavior of parents is significant, but insufficiently investigated perceiving or protective factor for the development of the habit of alcohol abuse and cigarette consumption for younger and older adolescents (Kaplan Napoles-Springer, Stewart, & Perez-Stable, 2001). Based on previous research, a wide spectrum of psychosocial impacts of parents on the development of these habits in behavior can be divided into three conceptualized behaviors: a parent support, a parental control, and parental attitudes towards these forms of behavior (Wood, Read, Mitchell, & Brand, 2004).

Smoking and Abuse of Alcohol

Research has shown that the use of some psychoactive substance is often associated with the use of another psychoactive substance. Researches have shown that nicotine and alcohol dependence is comorbid: 85% of alcoholics are smokers. Studies have been carried out to determine the existence of the same genetic or environmental factors for the development of these forms of behavior (Goldman

et al., 2005). In a study by Swan et al. (Swan, Carmelli, & Cardon, 1997), the obtained results suggest that about 50% of the genetic predisposition for nicotine dependence is shared with a genetic predisposition for alcoholism, while 15% of the genetic predisposition for alcoholism is common with nicotine dependencies. Tsuang and his associates (Tsuang et al., 2001) have found that about 25% of the total risk variability for the development of alcoholism has a common genetic basis with dependency on nicotine, and that about 2.4% of the risk factors of unshared environment for the development of alcoholism overlaps with the risk factors for the development of nicotine addiction.

In contrast to these findings, some studies (Koopmans, van Doornen, & Boomsma, 1997; Young, Rhee, Stallings, Corley, & Hewitt, 2006) have found very low shared influence of genetic and environmental factors. The reason for such variability among findings can be a different approach to the operationalization of measured behaviors, which points to the necessity of exploring the common genes of the neurobiological bases of these behaviors, that is, the identification of biological mechanisms that will enable the common risks to be explicitly explained through manifest processes (Goldman et al., 2005).

Sensation Seeking and Risky Behaviors

A comprehensive understanding of the evolution of habits in one's behavior should certainly include personality traits as a lasting determinant of the behavior of an individual (Terracciano & Costa, 2004). Zuckerman constructs of sensation seeking proves to be one of the most reliable predictors of the initial use of psychoactive substances, as well as for its abuse (Kaprara & Ćervone, 2003; Pihl & Suton, 2009; Shakra et al., 2018; Zuckerman, 2007). Sensation seeking is based on the theory of existence of individual differences in the optimal level of arousal, according to which everyone has a characteristic optimal level of excitement and stimulation for motor and cognitive activity. This depends on the age of the individual, learning, experience, environmental and day cycle.

Zuckerman has defined this psychobiological construct as a personality dimension characterized by the search for new, diverse, complex, and intense sensations from the environment, which involves accepting a certain level of risk in physical, social, legal, and financial areas. This construct contains four basic subdimensions. Thrill and Adventure Seeking (TAS) refers to engage in activities involving some physical danger or risk (extreme sports activities, speed driving, etc.). Experience Seeking (ES) measure the desire for new experiences through living in a nonconforming uncoventional lifestyle and travel. Disinhibition (DIS) operationalize the need to disinhibit behaviour in the social sphere by drinking, partying, and seeking variety in sexual partners. Aversion for rutine or repetitive experiences of any kind, as well as predictable people, are defined as a Boredom Susceptibility (BS), and there is a restless reaction when things are being unchanged. Research has shown that individuals with high scores on the sensation

seeking dimensions start to use different types of psychoactive substances at an early age. They use larger amounts of these substances, being more susceptible to developing addictions, and more likely to maintain abstinence during treatment (Roberti, 2004; Zuckerman, 2007).

In many studies, the correlation between the high scores on the sensation seeking dimension and use of alcohol has been confirmed (Hittner & Swickert, 2006; Martin et al., 2002). A dominant theoretical explanation of this association derives from research that indicates a negative correlation between this dimension and the level of monoamine oxidase enzyme (MAO). MAO dissolves dopamine and norepinephrine, and in that way it regulates their level (Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993). Numerous studies have confirmed that people who are prone to excessive alcohol consumption have a lower level of MAO enzyme than people who are not prone to these behaviors. The assumption is that an elevated dopamine level is responsible for developing these habits in a sensation seeker for two reasons: a) dopamine motivates behavior with an appetizing reward, in particular when the reward is biologically (substantially) stimulating (Zuckerman, 1994); and b) the use of alcohol stimulates the release of dopamine in the "pleasure center" (ventral striatum and nucleus accumbens) in the brain (Koob & Le Moal, 1997).

The positive correlation of cigarettes consumption and sensation seeking construct has also been proven in many studies (Dinn, Aycicegi, & Harris, 2004; Frankenberger, 2004; Kopstein, Crum, Celentano, & Martin, 2001; Roberti, 2004; Zuckerman, 2007). Studies with smokers have shown that sensation seeking is associated with the higher urge for cigarettes (Doran, Cook, McChargue, & Spring, 2009), stronger symptoms of negative affectivity, and anhedonia during nicotine abstinence (Carton, Le Houezec, Lagrue, & Jouvent, 2000; Leventhal et al., 2007), as well as higher recurrence rates after attempts of quitting (Kahler, Spillane, Metrik, Leventhal, & Monti, 2009). These findings suggest that sensation seeking plays an important role in initiating, escalating and maintaining this behavior. One of the explanations is the preference for unusual and intense taste and sensory stimulation characteristic for sensation seekers (Zuckerman, 1994, 2007). These studies have shown that even simple experimental manipulation with a new taste of cigarettes increases the intent for them to consume cigarettes.

Sensation Seeking: Genetic and Environmental Factors

Research has shown that biochemical mechanisms provide a significant support for the sensation seeking construct and related biological systems (Balada, Torrubia, & Arque 1993; Ballenger et al., 1983; Daitzman & Zuckerman, 1980; Dellu, Piazza, Mayo, Le Moal, & Simon 1996; Piazza et al., 1993). The assumption was that the construct with confirmed biological bases should have a significant influence on the genes in explaining the individual differences in its variability in the population.

Fulker et al. conducted the first major study of twins for assessing the heritability of sensation seeking construct (Fulker at al., 1980). The study was conducted on 422 pairs of adult twins, in collaboration with Zuckerman. The results of the study supported the assumption of a significant gene contribution to the existence of individual differences on this construct, given that the estimated heritability explained 58% of the variance. The resulting percentage of heritability was quite high for the estimation of the personality dimension, so the assumption of the researcher was that sensation seeking was predominantly determined by the additive genes (Fulker at al., 1980). The authors linked the estimated heritability to the level of MAO enzymes.

A significant contribution of genes was confirmed in the study by Koopmans and associates (Koopmans, Boomsma, Heath, & van Doornen, 1995). Their study with 1591 twin pairs confirmed high heritability. The study also showed that 58% of individual differences on this dimension could be explained by genes. Unlike previous studies, the researchers used a multivariate model for calculating the proportion explained by the variance among the subdimensions, so the covariance among variables was also calculated. The obtained results suggested that the genetic variation was the highest for subdimension Threats and Adventures (62% for men, 63% for women), and subdimension Disinhibition (62% for men, 60% for women), and the lowest for subdimension Boredom Sensitivity (48% for men, 58% for women). Estimated genetic variance for the Search for Experience was 56% on the male sample, and 58% on the female sample. Effects of the unshared environments were smaller, but also significant, while the effects of the shared environment did not appear to be significant. By testing the gender differences, no evidence was found that different genes affected the expression of this dimension in men and women.

The basic problem of the research is the determination of common and specific genetic and environmental factors of individual differences in optimal sensation (arousal) level, and tendency to risky behaviors: smoking and drinking. Based on previous research, a significant genetic contribution can be assumed. Also, it can be expected that the unshared environment will also be significant. Based on a previously confirmed relation between the dimension of sensation seeking and these types of risky behavior, the assumption is that the study results will show a significant overlapping of genetic and unshared environment variables.

Method

The Sample and Procedure

The sample included 171 pairs of twins from the general population in Serbia, aged between 18 and 60 (M = 24.80, SD = 7.61). Participants were 91 female and 21 male monozygotic twin pairs, and 27 female, 9 male, and 23 opposite sex

twin dizygotic pairs. We collected DNA samples by taking buccal swab in order to determine zygosity of twins. The data were collected in Novi Sad, Belgrade, Zrenjanin, Novi Pazar, and Niš in the period from 2011 to 2018. Participation of the twins was voluntary, and every respondent signed an information consent for participation in the research which was the part of Serbian wide national project.

Instruments

Sensation Seeking Scale form V (SSS-V; Zuckerman, 1994). SSS-V intend to measure individual differences in behavioral expression of sensation seeking. The scale consists 40 dichotomous items with forced choice of answering. The items are grouped in the four subdimensions: TAS - Thrill and Adventure Seeking, ES - Experience Seeking, DIS – Disinhibition, and BS - Boredom Susceptibility. Answers are coded as 1 (if item refers to some type od sensation seeking behavior), or 0 (if the item refers to other behaviors). The Cronbach reliability coefficient was .82 for TAS, .63 for ES, .67 for DIS, and .63 for BS. According to Loewenthal (2004), reliability coefficients above .60 could be considered as satisfactory.

Personal Information Questionnaire. By using this questionnaire we collected the information about frequency of cigarettes consumption, and frequency of alcohol drinking. The frequency values were determined in relation to the respondents' answers to the question of how many cigarettes they consumed per day (offered answers: up to 5, up to 20, up to 40, more than 40), and to the question of how often they got drunk (offered answers: once or twice a month; twice a week, several times a week, daily).

Results

Descriptive Statistics and Gender Differences

Table 1 shows descriptive statistics for the used variables. All SSS-V variables are normally distributed (skewness and kurtosis are lower/higher than 1.50/-1.50) according to Tabachnick & Fidell (2013). Frequency of cigarettes consumption had not been normally distributed, so we normalized it by using Tuckey data transformation. Gender differences were detected only in favour of males for Thrill and Adventure Seeking (t = 2.89, p < .01), Experience Seeking (t = 2.07, p < .05), Disinhibition (t = 3.32, p < .01), Boredom Susceptibility (t = 2.60, p < .01), and alcohol frequency (t = 3.35, p < .01).

Table 1
Descriptive statistics for sensation seeking sub-scales and risky behaviors

	Monozygotic twins				Dizygotic twins			
	M	*SD*	*Sk*	*Ku*	*M*	*SD*	*Sk*	*Ku*
Thrill and Adventure Seeking	6.09	2.91	-0.42	-0.92	6.33	2.67	-0.41	-0.81
Experience Seeking	4.76	2.16	-0.06	-0.53	5.25	2.25	-0.09	-0.81
Disinhibition	3.62	2.28	0.70	0.63	4.28	2.48	0.20	-0.66
Boredom Susceptibility	3.28	2.03	0.43	-0.26	3.49	2.18	0.56	0.26
Cigarettes consumption	1.34	0.74	1.95	2.49	1.41	0.85	1.91	2.33
Alcohol frequency	1.29	0.47	1.21	0.11	1.59	0.67	1.07	1.39

Note. M – mean, *SD* – standard deviation, *Sk* – skewness, *Ku* – kurtosis.

Relations between Different Risk Behaviours: Cross Twin – Cross Trait Correlations

Intraclass correlations and cross twin-cross trait correlations are presented in Table 2. Both types of correlation coefficients are calculated separately for the monozygotic and dizygotic group of twins.

Table 2
Intraclass and cross twin – cross trait correlations

	TAS		ES		DIS		BS		Cigarettes		Alcohol	
	MZ	DZ	MZ	DZ	MZ	DZ	MZ	DZ	MZ	DZ	MZ	DZ
TAS	.45**	.26*										
ES	.17	.11	.54**	.36**								
DIS	.21*	-.06	.32**	.19	.58**	.21*						
BS	.06	-.29*	.16	-.05	.33**	.28	.48**	.26*				
Cigarettes	-.04	-.06	.01	.04	-.04	.07	-.07	.15	.73**	.22*		
Alcohol	.07	-.01	.13	.13	.20	.37**	.13	-.04	.10	.19	.39**	.22*

Notes. MZ – monozygotic twins, DZ – dizygotic twins. TAS - Thrill and Adventure Seeking, ES - Experience Seeking, DIS – Disinhibition, BS - Boredom Susceptibility. Diagonal numbers represent intraclass, while the remaining ones represent cross twin-cross trait coefficients of correlation.
* $p < .05$. ** $p < .01$.

The cross twin – cross trait correlations in the group of monozygotic twins are signify higher than the correlation of the variables in the group of dizygotic

twins by both types of correlation. This provides evidence that genetic factors are likely to significantly contribute to covariance between all types of examined behaviours. The differences in correlations coefficients are most striking in the case of cigarettes consumption and Disinhibition, so it can be assumed that genetic factors will play a decisive role in shaping these phenotypes.

Multivariate Genetic Analysis

In order to assess the genetic and environmental influences in the dimension of seeking sensations, and the frequency of cigarettes consuming and drinking, a multivariate gene analysis was applied. Various full and reduced structural models (ACE, AE, CE), which represented the standard in genetic structural modeling (independent and common pathway), were compared by several fit criteria. Analysis parameters were calculated by using the ML method. Model evaluation was conducted based on the Bayesian Information Criterion (BIC), Akaike Information Criterion (AIC), Comparative Fit Index (CFI), Tucker–Lewis Index (TLI), the Root Mean Square Error of Approximation (RMSEA), and the quotient $\chi 2/df$ (Table 3). It was found that the best fit had an independent AE model ($\chi^2/df = 1.41$, $p = .27$, CFI = .92, TLI = .91, RMSEA = .06, AIC = 101.35, BIC = 7330.2).

Table 3
Parameters estimation of the AE independent model

	TAS	ES	DIS	BS	Cigarettes	Alcohol
Ac^2	.22 (.08 - .48)	.11 (.06 - .22)	.54 (.35 -.77)	.31 (.10 - .56)	.01 (.00 -.02)	.20 (.11 - .46)
As^2	.41 (.19 - .58)	.39 (.19 - .59)	.07 (.03 - .13)	.20 (.11 - .34)	.75 (.47 - .88)	.12 (.06 - .26)
ΣA	.63	.50	.61	.51	.76	.32
Ec^2	.02 (.00 - .03)	.31 (.13 - .51)	.04 (.02 - .10)	.02 (.00 - .06)	.01 (.00 - .02)	.00 (.00 - .03)
Es^2	.35 (.21 - .55)	.19 (.10 - .36)	.35 (.22 - .51)	.47 (.17 - .66)	.23 (.11 - .36)	.68 (.31 - .88)
ΣE	.37	.50	.39	.49	.24	.68

Note. TAS - Thrill and Adventure Seeking, ES - Experience Seeking, DIS – Disinhibition, BS - Boredom Susceptibility. Ac^2 – a common genetic factor, As^2 – a unique genetic factor, ΣA^2 - total genetic variance, Ec^2 – a common non-shared environmental factor, Es^2 – a unique non-shared environmental factor, ΣE^2 - total environmental variance.

Genetic factors contribute significantly to the manifestation of TAS, DIS, BS, and cigarette consuption, while the environmental factor is more important for drinking. Equal genetic and environmental variance is detectable in case of ES. Heritability of DIS mainly refers to common genetic factors (89%), as well as in the case of BS (61%), and drunking (63%), while heritability of cigarettes consumption (99%), ES (78%), and TAS (95%) mailny refers to specific genetic factor. A common non-shared environmental factor is more important for manifestation of ES (62%), while in other cases a specific non-shared environmental factor plays a more significant role.

Phenotypic correlations between different types of sensation seeking and risk behaviours are presented in Table 4.

Table 4
Genetic and nonshared environmental contributions to phenotypic correlations

Sources of variance	r_f	*Ac(%)*	*Ec(%)*
TAS X Cigarette consumption	.06	83	17
TAS X Drinking	.06	100	0
ES X Cigarette consumption	.09	33	67
ES X Drinking	.15	100	0
DIS X Cigarette consumption	.09	78	22
DIS X Drinking	.33	100	0
BS X Cigarette consumption	.07	86	14
BS X Drinking	.25	100	0
Cigarette consumption X Drinking	.05	100	0

Note. TAS - Thrill and Adventure Seeking, ES - Experience Seeking, DIS – Disinhibition, BS - Boredom Susceptibility. r_f – a coefficient of phenotypic correlations, *Ac* – a common genetic factor, *Ec* – a common non-shared environmental factor.

Phenotypic correlations between sensation seeking dimensions and risk behaviours are low to moderate ($.05 \leq r \leq .33$). Genetic factors have a main role in explainig the covariations between drinking and all other variables (100%), while environmental factors have more significant role in explaining the covariations between ES and Cigarette consumption (67%). In all other cases genetic factors explain a higher percentage of variance than environmental factors.

Discussion

The main question of the research was a coherence between the personality dimension and the risky forms of behavior. More specifically, the aim of the re-

search was to determine common and specific genetic and environmental factors of individual differences in an optimal sensation (arousal) level, and tendency to risky behaviors: smoking and drinking. The fact that the construct of sensation seeking was proven to be one of the most reliable predictors of the initial use and abuse of psychoactive substances (Kaprara & Ćervone, 2003; Pihl & Suton, 2009; Shakra et al., 2018; Zuckerman, 2007) gives a scientifically significant base for more detailed examination of the relationship of these variables. Also, since smoking and drinking were ones of the most frequent forms of risky behavior in the population (Institute of Public Health of Serbia, 2008; WHO, 2018), the research could have the significant practical value.

The results indicate the significance of gene effects, the effects of an unshared environment on the subdimensions of Sensation Seeking Scale, and the examined forms of risky behaviors. The results are in accordance with previous studies of sensation seeking (Fulker at al., 1980; Koopmans et al., 1995). People inherit a characteristic level of excitement and stimulation for motor and cognitive activity, so some individuals are predetermined to search for more new experience.

As Zuckerman has defined, and the previous studies confirm (Zuckerman, 2007), an optimal level of arousal for an individual also depends on the unique experience of a person (learning, life events etc). Influence of the unshared environment has been mostly smaller, but also significant in most of the research, while the effects of the shared environment have not been significant. The subdimension Experience Seeking has the same percentage of the influence of genes and unshared environment. Since this dimension relates to nonconforming unconventional lifestyle, "the need to live different from the environment", it could be concluded that the environment has a significant influence on forming this kind of attitude and lifestyle. As the impact of the environment is significant in our study, further studies should focus more on cultural differences.

The results implicate that genes dominantly (99%) affect the development and maintenance of the nicotine addiction, and that this addiction is mostly determined by specific genes. This leads to the conclusion that genes are not responsible for the correlation of the sensation seeking and this kind of addiction. Sensation seeking could have the important role in initiating, and maybe escalating addiction (Dinn et al., 2004; Frankenberger, 2004; Kopstein et al., 2001; Roberti, 2004; Zuckerman, 2007), but not in the maintenance of this kind of behavior. Therefore, the conclusion is that a high tendency for sensation seeking can provoke the initial consumption of cigarettes, which later, under the influence of neurological processes determined by specific genes, becomes an addiction.

The tendency of alcohol abuse is dominantly explained by an unshared environment. Drinking, in this case, does not refer to alcoholism as a disorder, so the assumption is that people in our sample practice this type of risk behavior because it is acceptable or desirable in a social situation. The alcohol is consumed because of the satisfaction, not because there is a physiological need based on the addiction. The high percentage of shared genetic influence (100%), with the

subdimensions of sensation seeking, can be explained with the determined correlation of the level of MAO enzymes within "sensation seekers" and people who tend to enjoy alcohol. The lower level of MAO enzymes is negatively correlated with dopamine levels, which is responsible for a sense of satisfaction during these types of behaviors.

The results lead to the conclusion that it is wrong to perceive sensation seeking as a predictor of nicotine dependence and alcohol abuse. However, high scores on this dimension should be considered as one of the main risk factors for developing these risky behaviors. Results have shown that the environment also has a significant influence on development and expression of these kinds of behavior, and thus there is a good possibility for preventive programs. The next study should be more focused on the factors of an unshared environment that could explain the individual differences in these variables. It could make a significant contribution to prevention programs.

Also, furder examination of this topic should involve more variance in the age of the participants. The participants are mostly in their twenties, with the average of 24 years old. The fact that sensation seeking is the highest in the early twenties (Zuckerman, 2007) makes this sample appropriate. Also, people in that age represent a very high percentage of the population who consumes cigarettes and abuse alcohol (Institute of Public Health of Serbia, 2008). However, there is still a question of the variability of this dimension and its correlation depending on the age cohort. The fact that a need for sensation is decreasing over the years of a person's life (Zuckerman, 1994) could influence its correlation with smoking and alcohol abuse, and also show significant changes in the level of influence of shared and specific genetics, and environmental factors on these behaviours.

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TRAŽENJE SENZACIJA I RIZIČNA PONAŠANJA U SVETLU GENSKIH I SREDINSKIH ČINILACA

Istraživanje sprovedeno na 171 paru odraslih blizanaca imalo je za cilj da odredi stepen doprinosa genskih i sredinskih činilaca formiranju optimalnog nivoa senzacija (Zakermanov konstrukt traženja senzacija), kao i sklonostima ka rizičnim oblicima ponašanja - konzumiranju cigareta i napijanju. U istraživanju je primenjena Skala traženja senzacija, kao i Upitnik o ličnim informacijama. Rezultati ukazuju na visoku heritabilnost pojedinačnih subdimenzija traženja sezacija (50% - 63%), kao i navike konzumiranja cigareta (75%). Doprinos nedeljene sredine se takođe pokazao kao značajan za objašnjenje individualnih razlika na ovim dimenzijama. Sa druge strane, na osnovu dobijenih rezultata može se zaključiti da nedeljena sredina ostvaruje najveći doprinos na razvoj sklonosti ka zloupotrebi alkohola (68%). Takođe, aditivni genski činioci ostvaruju većinski doprinos kovariranju mera traženja senzacija i različitih rizičnih ponašanja.

Ključne reči: bihejvioralna genetika, blizanačka studija, napijanje, pušenje, traženje senzacija

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BEHAVIORAL GENETICS FOUNDATIONS OF RELATIONS BETWEEN PERSONALITY TRAITS AND SATISFACTION WITH LIFE[2]

The long-term stability of subjective wellbeing has directed an attention to stable dispositions as the probable source of individual differences in the satisfaction with life (SWL). The main objective of this study was to examine the extent of genetic overlap between SWL and personality traits of the five-factor model (FFM). The sample consisted of 121 monozygotic and 61 dizygotic twin pairs (the average age was 24.59, SD = 7.11). Satisfaction with Life Scale and The Revised NEO Personality Inventory (NEO-PI-R) were applied. Multivariate genetic modeling was performed. The results show the most appropriate fit indices for Independent AE model(χ^2/df = 1.41, CFI = .92, TLI = .91, RMSEA = .07, AIC = 17400.81, BIC = 17558.68, SRMR = .10). SWL and all NEO-PI-R personality traits have a moderate to strong genetic bases, while the common genetic influences for SWL are 40%. The results show that unique environmental contributions are moderate to strong (from 61% for Neuroticism, 41% for SWL, to 23% for Conscientiousness). Genetically driven tendency common to Neuroticism, Extraversion, and Conscientiousness, underlines individual differences in SWL, and therefore a cognitive evaluation of SWL seems to be substantially based on emotional tendencies encompassed by the FFM. Also, SWL appears to be uniquely environmentally influenced, which implies benefits of wellbeing interventions through the process of learning or adopting a different life philosophy.

Key words: FFM, multivariate genetic modeling, satisfaction with life, twin study

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Introduction

In recent decades, there has been a growing consensus that mental health should be viewed not only as the absence of psychopathological symptoms, but rather as the presence of positive indicators (Keyes, 2005). This leads to the recognition of subjective wellbeing as an important aspect of mental health, as well as to the expansion of the research on happiness. A significant progress has been made in the research of subjective wellbeing and its correlates.

Subjective wellbeing is often defined as a cognitive and emotional evaluation of life (Diener, 2000). It comprises a hedonic balance, i.e. the balance between positive and negative affects, which constitutes its affective component, while satisfaction with life is assessed according to subjective standards, as its cognitive component. However, it has been proposed that cognitive component could, to some extent, rely on hedonic balance, because the overall assessment of satisfaction with life implies retrievement of pleasant and unpleasant experiences, with their ratio forming such judgment (Schimmack, Diener, & Oishi, 2002; Schimmack, Radhakrishnan, Oishi, Dzokoto, & Ahadi, 2002). Hence some personality traits can affect not only the level of positive and negative affects, but, indirectly, the assessment of satisfaction with life as well.

It has been well documented that, among many correlates of wellbeing, personality traits play a particularly important role (e.g., DeNeve & Cooper, 1998). Findings suggesting a long-term stability of subjective wellbeing have directed attention to stable dispositions as the probable important source of the individual differences in the overall sense of happiness and satisfaction with life. It has been suggested that personality traits could affect subjective wellbeing both directly and indirectly. More direct links derive from the fact that personality traits predispose individuals for certain emotional responses and experiences. However, personal dispositions could also lead to engagement in certain types of activities and situations more frequently. Since many of these activities and experiences might further promote or impair subjective sense of happiness, the personality traits can affect subjective wellbeing indirectly, through situational choices (e.g., Steel, Schmidt, & Shultz, 2008). Consequently, environments with reduced situational choice opportunities could decrease the role of stable dispositions in subjective wellbeing.

Findings point to Extraversion and Neuroticism as personality traits which are most consistently linked to subjective wellbeing, and which demonstrate that these two dimensions explain substantial amounts of variance in wellbeing. The findings regarding Conscientiousness are inconsistent, while Agreeableness and Openness seem to play a limited role in wellbeing (DeNeve & Cooper, 1998; Steel et al., 2008; Vitterso, 2001). Individuals with a high level of Extraversion and a low level of Neuroticism tend to be happier and more satisfied with their lives. Extraversion is often considered as the proneness to positive affective experiences, and Neuroticism as the tendency to experience negative affect. Therefore, a con-

nection between these traits and the affective component of subjective wellbeing is quite straightforward. However, research suggests that these traits are also correlated with measures of wellbeing which do not imply direct reports of affective experience, such as personal security and satisfaction with life (Costa & McCrae, 1980; Grevenstein & Bluemke, 2015). Some findings suggest that the influence of Extraversion and Neuroticism on satisfaction with life is largely mediated by hedonic balance (e.g., Schimmack et al., 2002).

The issue of genetic contributions to the individual differences in wellbeing are based on the well-established link with personality traits, which have already demonstrated substantial heritability (e.g., Butković, Hlupić, & Bratko, 2017; Johnson, Vernon, & Feiler, 2008), as well as from findings regarding a long-term stability of wellbeing. In accordance with popularity of the wellbeing phenomenon, a large number of behavioral genetic studies have been carried out in many countries and cultures, on samples of more than 80.000 twins and family members, covering the life span from early adolescence through senior years. These studies have tried to find out whether happiness is a hereditary predisposition, or it is associated with the process of learning and/or adopting a specific life philosophy (Archontaki, Lewis, & Bates, 2013; Bartels & Boomsma, 2009; Gigantesco et al., 2011; Hahn, Johnson, & Spinath, 2013; Kendler, Myers, & Keyes, 2011; Keyes, Myers, & Kendler, 2010; Nes, Roysamb, Tambs, Harris, & Reichborn-Kjennerud, 2006; Stubbe, Posthuma, Boomsma, & De Geus, 2005). Different definitions of wellbeing have contributed to the use of terms like wellbeing, satisfaction with life, happiness, or quality of life interchangeably (Layard, 2010). A large variety of definitions in wellbeing questionnaires, scales, subscales, and items makes a meta-analysis vulnerable to heterogeneity, complicating the estimation of genetic and environmental variance (Bartels, 2015). Nevertheless, meta-analysis conducted by Bartels (2015) has shown that individual differences in wellbeing and its components, such as satisfaction with life, happiness, and quality of life, are accounted for by both genetic and environmental factors. For overall wellbeing, heritability estimates, mainly represented by additive genetic effects, range from 17 to 56%, for satisfaction with life they range from 0 to 60%, for happiness they range from 22 to 41%, and for quality of life heritability estimates range from 22 to 42% (Bartels, 2015). These results indicate the unambiguous impact of hereditary factors on subjective wellbeing. However, Diener has emphasized that, although genetic effects are undoubtedly important, cultural and situational factors also influence subjective wellbeing, sometimes strongly (Larsen & Eid, 2008). Moreover, he has argued against the idea that subjective wellbeing is determined by genetic inheritance, providing evidence for environmental influences, such as differences in subjective wellbeing between young vs. old people (Diener & Suh, 1998), employed vs. unemployed people (Diener, Nickerson, Lucas, & Sandvik, 2002), married vs. unmarried women (Lucas, Clark, Georgellis, & Diener, 2003), the poorest vs. richest nations (Diener & Suh, 1999). Furthermore, behavioral genetic studies (Weiss, Bates, & Luciano, 2008) have demonstrated that there are no

genetic effects unique to subjective wellbeing. Namely, since subjective wellbeing is strongly linked to personality traits (DeNeve & Cooper, 1998), the basic question in behavioral genetic studies of subjective wellbeing is whether it shares the same additive genetic variance with personality traits.

On the other hand, results of behavioral genetic studies of personality traits (Bouchard, 1997; Butković, et al., 2017; Jang, Livesley & Vernon, 1996; Johnson et al., 2008; Rieman, Angleitner, & Strelau, 1997) have shown that approximately half of the variance in all FFM domains are genetically influenced. The results have indicated that the average heritability for personality traits is about 40% (Vukasović & Bratko, 2015), or even 48% and 49% (Johnson et al., 2008; van den Berg et al., 2014). By explaining relations between personality traits and subjective wellbeing, some studies have shown that personality and subjective wellbeing may be correlated, because they share the same genetics bases (e.g., Kendler, Gatz, Gardner, & Pedersen, 2006). Moreover, some authors have hypothesized that the heritable component of subjective wellbeing is entirely explained by the genetic architecture of the FFM (e.g., Weiss et al., 2008), suggesting that the genetic and environmental variance of subjective wellbeing may be explained in terms of personality.

Based on the strong evidence of a correlation between subjective wellbeing and personality traits, primarily Extraversion and Neuroticism, the first objective of the present study is to replicate the association of these constructs. Subsequently, we will examine the extent of a genetic overlap between subjective wellbeing and all FFM personality traits. Namely, we will decompose the genetic and environmental components of subjective wellbeing into those linked to personality and those specific to wellbeing, with main hypothesis that subjective wellbeing represents one of manifestations of personality traits, without an independent hereditary basis.

Method

Sample and Procedure

Participants in the present study were recruited from the Twin Registry, a voluntary based sample of Serbian twins. Twins were recruited as a part of the project "Psychological Foundations of Mental Health: Hereditary and Environmental Factors" during 2011-2018 period. A call for participation in the research was published through media, Internet and press. The participation in the research was voluntary, and every respondent signed an informed consent. Data collection protocol was approved by the Ethic Committee of Department of Psychology, Faculty of Philosophy, University of Novi Sad.

The sample consisted of 364 twins of whom 242 were monozygotic (76% female pairs of MZ twins) and 122 dizygotic (DZ). From 61 DZ twin pairs, 9 pairs

were males, 25 were females, and 27 were different gender. Participants ages ranged from 18 to 48 (M = 24.59, SD = 7.11). Zygosity was determined based on DNA analysis of the buccal swab, which was performed at the Institute of Forensic Medicine in Novi Sad, and John Jay College of Criminal Justice in New York. Data collection was carried out in Novi Sad, Belgrade, Niš, Zrenjanin, and Novi Pazar. A part of participants' data was collected by an online platform. Respondents did not receive any fee for participating in the research.

Instruments

Satisfaction with Life Scale (SWLS: Diener, Emmons, Larsen, & Griffin, 1985; Serbian version: Vasić, Šarčević, & Trogrlić, 2011). This scale was used to assess a cognitive component of subjective wellbeing. Answers to each of the five items (e.g., *The conditions of my life are excellent*) range from 1 - *strongly disagree*, to 7 - *strongly agree*. This scale was widely used, and it showed good psychometric properties in previous research. Cronbach's alpha coefficient of SWLS for our sample (.83) was acceptable according to Loewenthal (2004).

The Revised NEO Personality Inventory (NEO-PI-R: Costa & McCrae, 1990, Serbian version: Knežević, Džamonja Ignjatović, & Đurić Jočić, 2004). NEO PI-R consisted of 240 Likert-type items, and it was used to assess the Big Five personality traits: Openness to Experience (O), Conscientiousness (C), Extraversion (E), Agreeableness (A), and Neuroticism (N). Each item is rated on a five-point scale, from 1 - *strongly disagree*, to 5 - *strongly agree*. Cronbach's alpha coefficient for all scales (N = .81, E = .84; O = .80; C = .80; A = .81) was acceptable.

Data Analysis

For exploring the nature of the phenotypic associations between satisfaction with life and personality traits, multivariate twin modeling was we applied. The twin design compares the degree of phenotypic similarity between monozygotic twins, sharing 100% of their genes, with dizygotic twins, who shared 50% of their genes on average (Rijsdijk & Sham, 2002). Independent and common multivariate models were applied in order to estimate additive genetic factors (A); shared environmental (C), and non-shared environmental (E) factors. Different combinations of A, C and E (ACE, AE, E) were compared, and the best model was selected based on an optimal balance between goodness of fit and parsimony. A descriptive analysis and phenotypic correlations were carried out in the SPSS v.21 software (IBM corp., 2012), while the multivariate SEM modeling was conducted in the "lavaan" R package (Rosseel, 2012).

Results

Descriptive Statistics

The first step in the analysis was the partialization of the gender effect, as well as the linear and quadratic partialization of the age effect, conducted by using the standard regression procedures proposed by McGue and Bouchard (McGue & Bouchard, 1984). Table 1 shows descriptive statistics for all the variables. The results in Table 1 show that all variables are normally distributed (skewness and kurtosis are lower/higher than 1.50/-1.50; Tabachnick & Fidell, 2013). The measure of satisfaction with life has been normalized by Tuckey transformation.

Table 1
Descriptive statistic for SWLS and NEO-PI-R scales

	Monozygotic		Dizygotic			
	M	*SD*	*M*	*SD*	*Sk*	*Ku*
Satisfaction with Life	5.04	0.90	4.88	1.02	-0.79	-0.16
Neuroticism	2.72	0.59	2.79	0.59	0.19	0.02
Openness	3.45	0.48	3.44	0.47	-0.33	-0.27
Conscientiousness	3.66	0.47	3.56	0.50	-0.15	-0.20
Extraversion	3.49	0.50	3.40	0.53	-0.32	0.31
Agreeableness	3.45	0.70	3.46	0.68	-0.16	0.09

Note. M – mean, *SD* – standard deviation, *Sk* – skewness, *Ku*– kurtosis.

Intraclass and Cross-Twin – Cross-Trait Correlations

Table 2 presents the phenotypic correlations between SWLS and NEO-PI-R scales. Both types of correlation coefficients (intraclass and cross twin – cross trait) have been calculated separately for the monozygotic and dizygotic twins.

Table 2
Cross-twin within-trait (diagonal), and cross-twin cross-trait (off-diagonal) correlations

	N		E		O		C		A		SWL	
	MZ	DZ	MZ	DZ	MZ	DZ	MZ	DZ	MZ	DZ	MZ	DZ
N	.35**	.25*										
E	-.21*	-.20*	.60**	.40**								
O	-.03	-.16	.27**	.09	.56**	.21*						
C	-.20*	-.23*	.14	.17	.20*	.14	.63**	.41*				
A	.01	-.36**	-.17	.27*	-.03	-.09	.04	.22*	.52**	.19		
SWL	-.23*	-.30*	.11	.24*	.03	.08	.36**	.04	.01	.14	.54**	.42**

Notes. MZ – monozygotic twins, DZ – dizygotic twins. O - Openness to Experience, C - Conscientiousness, E - Extraversion, A - Agreeableness, N – Neuroticism, SWL – Satisfaction With Life.
* p <.05. ** p < .01.

Correlations between MZ twins are consistently higher than correlations between DZ twins on all variables. The biggest correlation difference is for Openness (Δr = .35), and the smallest one for Neuroticism (Δr = .10).

Multivariate Genetic Modeling: Model Comparison and Parameter Estimation

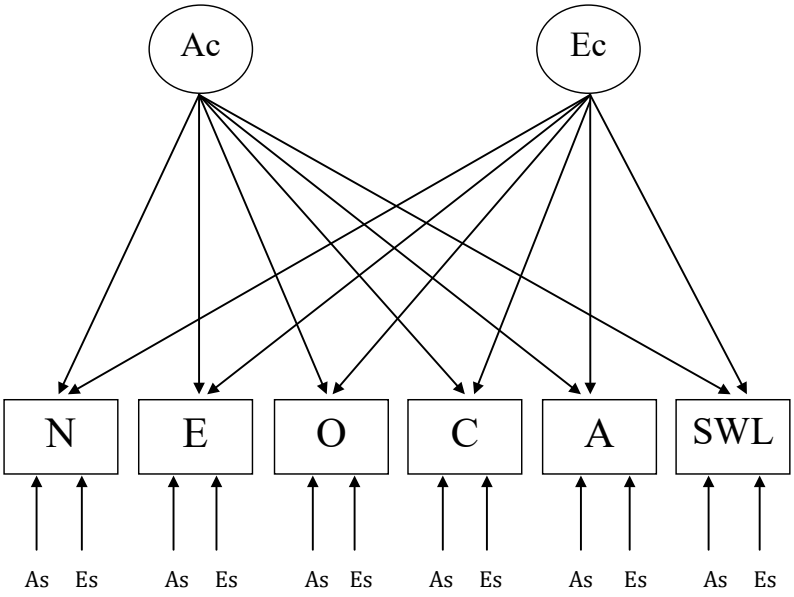
In order to specify the form of the observed covariates among the personality traits and satisfaction with life, multivariate Independent Pathway Models and Common Pathway Models were tested. A comparison of the two groups of models, as well as the comparison between full (ACE) and reduced (AE, CE) models, was carried out by using several fit indicators for all plausible models. Analysis parameters were calculated by using the method of maximum likelihood. Model evaluation was conducted based on the Akaike Information Criterion (AIC; Akaike, 1973), Bayesian Information Criterion (BIC; Schwarz, 1978), comparative fit index and the Tucker–Lewis index (CFI and TLI – optimal values higher than .95, acceptable higher than .90), the root mean square error of approximation (RMSEA - optimal values lower than .05, acceptable lower than .08), the standardized root mean square residual (SRMR), with acceptable value below .08 (Hu & Bentler, 1999), and the quotient $\chi 2/df$ (recommended < 2) (Kline, 2010).

Table 3
Fit indices for multivariate models

	Model	χ^2/df	CFI	TLI	AIC	BIC	RMSEA (95% CI)	SRMR
Independent	ACE	1.50	.89	.89	17411.89	17598.85	.07 (.05-.10)	.11
	AE	**1.41**	**.92**	**.91**	**17400.81**	**17558.68**	**.07 (.04-.10)**	**.10**
	CE	1.72	.84	.84	17440.81	17595.60	.09 (.07-.11)	.11
Common	ACE	1.59	.87	.87	17424.44	17588.85	.08 (.06-.10)	.11
	AE	1.56	.89	.89	17417.07	17566.05	.07 (.05-.10)	.11
	CE	1.76	.83	.83	17444.57	17586.54	.09 (.07-.11)	.11

Note. A – additive genetic variance, C – shared environmental variance, E – non-shared environmental variance and measurement error.

The most appropriate fit indices (Table 3) were for Independent AE model (Graph 1). All the indices were within acceptable boundaries, $\chi^2/df = 1.41$, CFI = .92, TLI = .91, RMSEA = .07, AIC = 17400.81, BIC = 17558.68, except SRMR (SRMR = .10). The estimation of the parameters of the independent AE model is showed in Table 4.



Graph 1. Independent AE model of satisfaction with life and personality traits.
Note. Ac – a common additive genetic factor, *As* – a unique additive genetic factor, *Ec* – a common non-shared environmental factor, *Es* – a unique non-shared environmental factor. N – Neuroticism, E – Extraversion, O – Openness to Experience, C –Conscientiousness, A –Agreeableness, SWL – Satisfaction With Life.

Table 4
Specific and common genetic and environmental contributions for AE multivariate models

Scale	Ac^2	As^2	ΣA^2	Ec^2	Es^2	ΣE^2
Satisfaction with life	.40	.12	.52	.07	.41	.48
Neuroticism	.19	.20	.39	.19	.42	.61
Extraversion	.09	.49	.58	.17	.25	.42
Openness to experience	.00	.50	.50	.15	.35	.50
Conscientiousness	.13	.44	.57	.22	.21	.23
Agreeableness	.00	.49	.49	.08	.43	.51

Note. Ac^2 – a common genetic factor, As^2- a unique genetic factor, ΣA^2 - total genetic variance, Ec^2– a common non-shared environmental factor, Es^2– a unique non-shared environmental factor, ΣE^2 - total environmental variance.

Results presented in Table 4 show that satisfaction with life and all personality traits have a moderate to strong genetic bases (from 58% for Extraversion to 39% for Neuroticism). Also, common genetic influences range from 40% (for satisfaction with life) to 0% (for Openness to Experience and Agreeableness). Unique genetic factors are most prominent in Openness (50%), Agreeableness (49%), Extraversion (49%) and Conscientiousness (40%). Common environmental impacts are generally low, foremost being Conscientiousness (22%) and Neuroticism (19%). However, unique environmental contributions are moderate to strong (from 61% for Neuroticism, Openness and Agreeableness - 50% and 51%, to 23% for Conscientiousness).

Table 5
Additive genetic and non-shared environmental contributions to phenotypic correlations of SWL and personality traits

Sources of variance	r_f	*Ac%*	*Ec%*
Satisfaction with life X neuroticism	.38	71	29
Satisfaction with life X extraversion	.29	66	34
Satisfaction with life X openness	.10	0	100
Satisfaction with life X conscientiousness	.31	61	39
Satisfaction with life X agreeableness	.07	0	100

Note. r_f – coefficient of phenotypic correlations, *Ac* – a common genetic factor, *Ec* – a common non-shared environmental factor.

Phenotypic correlations between satisfaction with life and different personality traits (Table 5) were low to moderate ($.07 \leq r \leq .38$), and the share of genetic factors in the covariance of these measures ranged from 0% to 71%. Non-shared environmental factors explained significantly less of co-variations, except for Openness and Agreeableness. For these two dimensions environmental factors determined 100% of phenotypic correlations with satisfaction with life. Genetic factors had the main role in the case of co-variation between satisfaction with life and Neuroticism (71%), Extraversion (66%) and Conscientiousness (61%).

Discussion

The first objective of this study was to replicate the association of FFM domain traits and satisfaction with life. Personality traits were well-established predictors of wellbeing in satisfaction with life in particular (Kandler et al., 2006; Kandler et al., 2007; Weiss et al., 2008). We set out to examine the etiological factors involved in the associations between personality and satisfaction with life: the role of genetic and environmental factors in the link between personality and satisfaction with life. The results were in line with previous studies showing that satisfaction with life was connected to all personality traits (DeNeve & Cooper, 1998; Steel et al., 2008; Vitterso, 2001), with higher correlations among monozygotic twin pairs, suggesting a potential genetic base.

We have also hypothesized that satisfaction with life represents one of the manifestations of personality traits, without independent genetic basis, in line with some previous studies (e.g., Weiss et al., 2008). An important finding of the current study is that satisfaction with life is genetically indistinct from personality traits, especially those reflecting emotional stability as low Neuroticism, social or physical activity as Extraversion, and constraint and self-discipline as Conscientiousness. The close genetic relationship between personality traits such as Emotional Stability, Extraversion and Conscientiousness, and so-called Happiness traits could be the key to understanding the comorbidity in psychopathology (Kandler et al., 2007). These findings show that general genetic variance, underlying individual differences in satisfaction with life, is indeed responsible for individual differences in Neuroticism, Extraversion, and Conscientiousness. Thus, the cognitive evaluation of satisfaction with life seems to be mostly based on emotional tendencies constituted in the five-factor model. A negligible unique genetic effect that contributes to variance in satisfaction with life suggests the importance of environmental factors for this phenomenon.

Moreover, findings have actually pointed to both genetic and environmental influences, yet with the unique environmental effect being the most important. As such, satisfaction with life appears to be environmentally influenced by life events, situations, social relationships, but also by genetically driven tendency common to most personality traits. Such interpretation implies potentials for

change in satisfaction with life, and benefits of wellbeing interventions through the process of relearning, social learning or adopting a different life philosophy (e.g., Archontaki et al., 2013; Hahn et al., 2013; Kendler et al., 2011). The present findings indicate that the relationship between subjective wellbeing and a range of health and social relationship factors may also be mediated by common genetic effects. In future twin studies, researchers could be interested in examining the relationships between subjective wellbeing and factors such as cognitive styles, important life events, controlling for personality, preferably at a behavior-genetic level. Such studies could determine whether these relationships are also moderated by common genetic effects.

While genetic factors seem to play a moderate role in the total variability in satisfaction with life, they appear to have a major role in the relations between distinct personality traits and satisfaction with life. Genetic factors are more important in explaining the correlation between Neuroticism, Extraversion, and Conscientiousness with satisfaction with life. More specifically, the genetic dispositions to experience a low degree of depression and anxiety, and a high degree of positive emotions and activity, as well as being constrained, self-efficient, achievement-strived and self-disciplined, contribute to a perception of life as good and satisfactory. Environmental factors fully explain the relationship between satisfaction with life and Openness and Agreeableness, which might be accounted by complex processes of social learning and individual experience.

These findings have potential implications for the set point theory of subjective wellbeing (Diener, 2000). Previous findings (e.g., Steel et al., 2008) have shown that both personality traits and environmental events bring up changes in the set point of wellbeing. A degree of adaptation to various situations and circumstances could be due to individual personality differences. Therefore, the genetic effects of personality may affect the rate that wellbeing returns to the set point after a misbalance and response to environmental factors. There are suggestions (e.g., Weiss et al., 2008) that personality may create an affective reserve, which can be called upon in times of stress and recovery. Moreover, a person with a strong tendency to experience positive emotions, activity, energy, self-efficiency, and self-discipline, combined with a low tendency to depression and anxiety, might recall a high number of pleasant life episodes and consequently summarize life as mostly positive. On the other hand, a person scoring low on these dimensions might have mental images comprising of situations in life that are less satisfactory.

The results of this study have implications for further molecular genetics studies of subjective wellbeing, which require focusing on searching for specific genes that influence personality, in order to understand how the complex processes starting with DNA-molecules end up with a personal evaluation of one's life as good and satisfactory.

The results of this study provide a confirmation of the previous research on satisfaction with life and personality traits. At the same time, the results provide guidance for future research in the field of behavior genetics. Besides a larger

sample, satisfaction with life additionally needs to be operationalized through an emotional component in order to gain more specific insights into the connection of cognitive and emotional aspects of satisfaction with life and personality traits, in light of genetic and environmental factors. Findings on the environmental impacts would be extended by a family design that would provide insights into the impact of the passive gene-environment correlation, in shaping of co-variation between satisfaction with life and personality traits.

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BIHEJVIORALNO-GENETIČKE OSNOVE RELACIJA OSOBINA LIČNOSTI I ZADOVOLJSTVA ŽIVOTOM

Rezultati savremenih istraživanja o vremenskoj stabilnosti subjektivnog blagostanja usmerili su pažnju istraživača na stabilne dispozicione karakteristike kao verovatan izvor individualnih razlika u zadovoljstvu životom. Glavni cilj ovog istraživanja usmeren je na ispitivanje stepena preklapanja genske varijanse zadovoljstva životom i osobina ličnosti iz modela Velikih pet (FFM). Uzorak su činili 121 par monozigotnih i 61 par dizigotnih blizanaca (prosečna starost 24.59 godina, SD = 7.11) sa teritorije Srbije. Na podacima prikupljenim pomoću Skale zadovoljstva životom i Revidiranog inventara ličnosti NEO-PI-R sprovedeno je multivarijatno genetsko modelovanje. Rezultati istraživanja ukazuju na to da najprikladnije indekse podesnosti ostvaruje AE model nezavisne putanje (χ^2/df = 1.41, CFI = .92, TLI = .91, RMSEA = .07, AIC = 17400.81, BIC = 17558.68, SRMR = .10). Zadovoljstvo životom i svih pet osobina ličnosti dele umerene do jake genske osnove, dok zajednički genski doprinos za zadovoljstvo životom iznosi 40%. Rezultati istraživanja ukazuju na to da se specifični doprinosi nedeljene sredine mogu opisati kao umereni do jaki (od 61% za Neuroticizam, preko 41% za zadovoljstvo životom, do 23% za Savesnost). Čini se da genske osnove koje su zajedničke Neurotizmu, Ekstraverziji i Savesnosti doprinose individualnim razlikama u zadovoljstvu životom, te da je kognitivna procena zadovoljstva životom u velikoj meri zasnovana na emocionalnim tendencijama obuhvaćenim FFM. Rezultat da jedinstveni sredinski činioci značajno oblikuju zadovoljstvo životom ukazuje na potencijalne dobiti od sprovođenja intervencija zasnovanih na učenju ili usvajanju određene životne filozofije.

Ključne reči: blizanačka studija, multivarijatno genetsko modelovanje, Petofaktorski model, zadovoljstvo životom

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Statistika. Rezultati statističkih testova treba da budu dati u sledećem obliku: $F(1, 9) = 25.35$, $p < .001$ i slično za druge testove (npr. $\chi^2(5, N = 454) = 5.311$, $p > .10$ ili $t(452) = 2.06$, $p < .05$) . Treba navoditi manji broj konvencionalnih nivoa p (npr: .05, .01, .001). Ukoliko je broj manji od 0, nula se ne stavlja ispred tačke. Po pravilu, nazivi statističkih

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