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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).

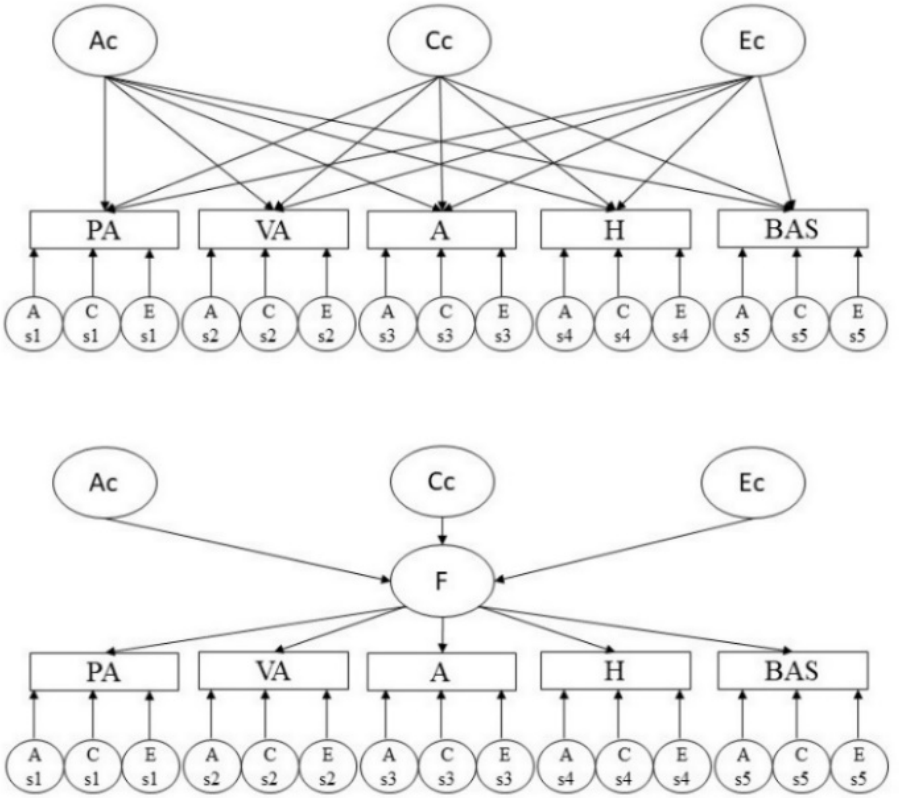

Figure 1. ACE independent pathway model (top pannel), and ACE common pathway model (bottom panel) for four BPAQ dimensions and BAS scale.
Note. PA – physical aggression, VA – verbal aggression, A – anger, H – hostility, BAS – behavioral activation system, *Ac* – common additive genetic variance, *Cc* – 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