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Effects of variation in dopaminergic genes on the level of aggression and emotional intelligence in adolescents with conduct disorder

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Summary

It has been reported that altered dopaminergic neurotransmission may contribute to the development of aggressive behaviors and emotional intelligence (EI) impairment. However, less is known about the impact of polymorphisms in dopaminergic genes on the level of aggression and EI. Therefore, we aimed to investigate the association between the catechol-O-methyltransferase (COMT) rs6277 gene polymorphism and the dopamine 2 receptor (DRD2) rs4680 gene polymorphism as well as the level of aggression and EI in adolescents with conduct disorder. Participants were 144 adolescents with conduct disorder recruited at the youth sociotherapy centre. The Buss-Perry Aggression Questionnaire (BPAQ) was administered to record the level of aggression while the Popular Emotional Intelligence Questionnaire (PEIQ) and the Schutte Self-Report Inventory (SSRI) were used to assess EI. We found no significant associations between selected polymorphisms and the scores of BPAQ, PEIQ and SSRI. Our findings do not support the role of the COMT and the DRD2 gene polymorphisms in shaping aggressive behaviors and EI in adolescents with conduct disorder. Longitudinal studies on larger populations are needed to confirm these results.

genetics, dopamine, Conduct Disorder, neurotransmitter, externalizing behaviors

INTRODUCTION

Aggression represents one of typical clinical characteristics of conduct disorders in adolescents. Children who are diagnosed with conduct disorders significantly violate social norms and the rights of other people. Conduct disorders, which occur in about 5% of children during adolescence, are a serious medical and social problem, due to the consequences for the patient, his family and the society [1].

Although the exact mechanisms underlying conduct disorders and aggression remain unclear, the role of biological factors, including genetic backgrounds, is increasingly being recognized. It has been estimated that 65% of variance in the prevalence of aggressive behaviors can be attributed to genetic factors, while the rest is attributable to environmental insults [2]. Many genes are thought to be responsible for the development of aggression. For instance, there is a growing interest in the role of variation in dopaminergic genes as risk factors for aggressive

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behaviors. It is believed that the dopaminergic system of the striatum indirectly affects the occurrence of impulsiveness and it has been suggested that different variants of the genes involved in dopaminergic neurotransmission may modulate the pattern of aggressive behaviour.

More specifically, high dopamine levels been identified in impulsive individuals and attributed to variation in the catechol-*O*-methyltransferase (*COMT*) gene [3]. Carriers of the Met allele of the Val158Met polymorphism have a longer dopamine firing time in the prefrontal cortex, also they have increased vulnerability to stress factors, a lower threshold of pain sensitivity, and more efficient information processing [4]. On the contrary, in the *COMT* 158Val/Val homozygotes, the duration of dopamine activity in the prefrontal cortex is lower due to high activity of the COMT. In addition, these individuals are characterized by higher stress resistance and increased threshold of pain sensitivity [5].

Other way to look at dopamine function is to consider the polymorphism of the dopamine D2 receptor (*DRD2*) gene. Among children, the *DRD2* gene polymorphisms have been linked to aggression such as anger expression, bullying, and cruelty. For instance, it has been found, that aggressive children are significantly more likely to be a carrier for the G allele of the *DRD2* A241G polymorphism and the T allele of the *DRD2* TaqIA polymorphism. Moreover, this study revealed overrepresentation of the *DRD2* rs1079598 CC genotype among aggressive children [6]. The TaqI A1 allele has also been associated with impulsivity [7]. However, less is known about the impact of the *DRD2* rs6277 polymorphism, also known as the 957C > T transition, on aggressive behaviors. It has been found that this polymorphism decreases binding activity of the *DRD2* in the striatum and extrastratial areas [8, 9].

It has been reported that a regulation of emotions plays an important role in shaping aggressive behaviors. It is believed that the level of emotional intelligence (EI) is one of the factors that can affect the occurrence of autogression in adolescents. Importantly, according to Goleman [10], the EI is a set of social skills that provide the capacity to understand yourself and own emotions, manage and control them, and the ability to empathize. The EI depends on the ability to take adequate action to adapt or solve the problem [11]. Nowadays the concept of EI is widely used in applied research (psychiatry, developmental psychology, engineering psychology, behavioral economics, etc.). Considering psychological mechanisms of autoimmunity of aggression in adolescents, one should pay attention to their common feature – reduction of basic emotional and interpersonal competences [12] and ineffective regulation of the physical level of arousal [13]. In adolescents showing aggressive behaviors, the ability to deal with negative emotions is more often observed, as well as difficulties in regulating emotions and the transmission and reception of emotional than in the group of non-aggressive youth. The high level of EI is the overriding protection factor against aggression [14].

There is a scarcity of studies investigating the association between variation in dopaminergic genes and the EI. Some studies have shown that variation in the *COMT* gene is associated with the success of the recognition of negative emotions [15], which is a component of EI. Carriers of the Met allele of Val158Met polymorphism have been found to present with more efficient emotional information processing [5] and higher level of insight problem solving [16]. According to another study the *COMT* Met/Met homozygotes [17], have an increased risk of behaviors and emotional problems in childhood compared to heterozygous or homozygous carriers of Val158Met polymorphism, but only if they were born with reduced body weight and were subjected to prenatal stress. To our knowledge, results of studies investigating the association between the *DRD2* gene polymorphisms and the EI have not been published so far. In light of these research gaps, we aimed to investigate the association between two single nucleotide polymorphisms in dopaminergic genes (the *COMT* rs4680 polymorphism and the *DRD2* rs6277 polymorphism) and the measures of aggression and the EI in adolescents with conduct disorder.

PARTICIPANTS AND MEASURES

The study was conducted among the students of the Youth Sociotherapy Centre No. 2 in Wroclaw, Poland. It was approved by the Bioethics

Committee of Wroclaw Medical University. All participants and their statutory representatives gave written consent to all procedures carried out as the part of this study.

There were following inclusion criteria: diagnosis of conduct disorders and written consent of the patient and statutory representative to participate in the study. A total of 144 adolescents (85 girls aged 13-18 years and 61 boys aged 13-18 years) were found to be eligible for participation.

The following diagnostic tools and psychological questionnaires were used in this study:

1) *The Mini International Neuropsychiatric Interview for children and adolescents (MINI-Kid)* is the structured diagnostic interview, developed jointly by psychiatrists and clinicians in the United States and Europe, for the DSM-IV and the ICD-10 criteria. This tool was used to establish a diagnosis of conduct disorder and exclude individuals with other mental disorder [18, 19].
2) *The Schutte's Self-Report Emotional Intelligence Test (SSEIT)* is a measure of general EI. It includes a 33 self-report items that are based on a 5-point Likert scale ranging from 1 (strongly agree) to 5 (strongly disagree). This questionnaire consists of four sub-scales: emotion perception, utilizing emotions, managing self-relevant emotions, and managing others' emotions. The SSRI is based on the EI model developed by Salovey and Mayer (1990) [20]. Cronbach's alpha in our sample was 0.90
3) *The Popular Emotional Intelligence Questionnaire (PEIQ)* also measures EI and consists of 94 items of self-descriptive nature, using a five-point Likert scale. The PEIQ consists of the following subscales: acceptance (expressing and using own emotions), empathy (understanding and recognizing emotions of other people), control (control over ones' emotions), and understanding (understanding and awareness of own emotions) [21]. The Cronbach's alpha for the PEIQ was estimated at 0.89 in our sample.
4) *The Buss-Perry Aggression Questionnaire (BPAQ)* is a 29-item self-report measure of aggression. It has been designed to assess four dispositional components of aggression: physical aggression, verbal aggression, anger, and hostility [22]. The standardization study [23] confirmed sufficient internal compliance rates. The Cronbach's alpha was 0.80.
5) *The Children's Depression Inventory 2 (CDI2)* includes 28 items. It is a measure which allows for a comprehensive assessment of depressive symptoms in children and adolescents. The questionnaire also includes scales measuring emotional problems and problems related to everyday functioning; in addition the self-rating version includes subscales measuring negative mood/somatic symptoms, low self-esteem, lack of behaviour efficacy and interpersonal problems [24]. The Cronbach alpha coefficient has been used to calculate the internal consistency of the scale, and the results indicated that internal consistency for all subscales was at a satisfactory level. The Cronbach's alpha for CDI2 was 0.94 in our sample.
6) *The State-Trait Anxiety Inventory (STAI)* consists of two subscales measuring anxiety understood as a transient and situationally determined state of the individual (trait anxiety subscale) and anxiety understood as a relatively stable personality component (state anxiety subscale). Each subscale consists of 20 items which the subject answers by selecting one of four pre-categorized answers. Both subscales have high internal consistency and stability [25]. The standardization study [26] Cronbach's alpha in our sample was 0.94 for state anxiety and 0.99 for trait anxiety.

GENOTYPING

Venous blood samples were collected from all participants. Genomic DNA was obtained from peripheral white blood cells as described previously with use of the Maxwell® 16 LEV Blood DNA Kit (Promega Corporation, Madison, USA) according to the manufacturer's protocol.

The single-nucleotide polymorphisms were genotyped: the *COMT* rs4680 polymorphism (Val158Met) and the *DRD2* rs6277 polymorphism (957C > T) using the Allelic discrimination (AD) technique with appropriate TaqMan®SNP Genotyping Assays (C__25746809_50, and

C__11339240_10, respectively). In the AD assay, a unique pair of fluorescent dye detectors was used (two unique allele-specific TaqMan®MGB probes that target a SNP site) and the change in fluorescence of the dyes associated with the probes was measured. All the Assays were validated and predesigned. Reaction components and amplification parameters were based on the manufacturer's instructions. The ABI Prism® 7300 (ThermoFisher Scientific Inc., USA) sequence-detection system was used for amplification for TaqMan®SNP Genotyping Assay plates. The SDS, version 2.1 software (ThermoFisher Scientific Inc., USA) was used for data acquisition and analysis. The same software was used for the allelic discrimination-analysis module.

Plate genomic control DNA samples (with defined genotypes) and non-template controls (Nuclease-free water) were included for each reaction plate. The TaqMan®SNP Genotyping Assay was controlled (25% of randomly chosen samples from both groups) to check for genotyping accuracy. Identical genotypes were identified in all repeated samples.

STATISTICAL ANALYSIS

Descriptive statistics were presented as mean and standard deviation. Agreement of genotype distribution with the Hardy-Weinberg equilibrium (HWE) was tested by comparing expected and observed distributions using the χ^2 test. We conducted statistical analyses using the Statistical Package for Social Sciences, version 20 (SPSS Inc., Chicago, Illinois, USA). Due to non-normal distribution of continuous variables (assessed using the Kolmogorov-Smirnov test), a series of Mann-Whitney U tests were performed to test between-group differences. Differences were considered statistically significant if the p-value was less than 0.05.

RESULTS

General characteristics of the sample were shown in Table 1. The distribution of the *COMT* rs4680 genotypes was in agreement with the HWE ($\chi^2 = 1.95$, $p = 0.162$). However, there was a significant deviation from the HWE for the *DRD2* rs6277 genotypes ($\chi^2 = 15.67$, $p < 0.001$). Altogether, 28.5% of the sample met criteria for a diagnosis of any mood and/or anxiety disorder.

Tables 2 and 3 present differences in the levels of aggression and EI between with respect to the *COMT* rs4680 and the *DRD2* rs6277 allele status. There were no significant differences in the level of various aggression categories and EI between the *DRD2* rs6277 TT homozygotes and the C allele carriers (Table 2). Similarly, no significant differences in these measures were found between the *COMT* rs4680 Val/Val homozygotes and the Met allele carriers. However, there was trend toward significantly higher level of acceptance among the *COMT* rs4680 Val/Val homozygotes compared to the Met allele carriers ($p = 0.079$).

Table 1. General characteristics of the sample.

Variable	Mean ± SD or n (%)
Sex	60 (41.7) / 84 (58.3)
Age	14.85 ± 1.22
CDI 2 – total score	16.61 ± 12.76
STAI – state anxiety	42.78 ± 12.67
STAI – trait anxiety	45.02 ± 12.9
BPAQ – total score	70.26 ± 23.551
BPAQ – physical aggression	20 ± 7.14
BPAQ – verbal aggression	13.19 ± 5.064
BPAQ – anger	18.7 ± 6.257
BPAQ – hostility	18.41 ± 7.901
PEIQ – total score	304.96 ± 34.007
PEIQ – empathy	64,5 ± 11,782
PEIQ – acceptance	48,31 ± 10,201
PEIQ – control	31,66 ± 91
PEIQ – understanding	28,48 ± 6,147
SSRI – total score	109,9 ± 25,7

Data expressed as mean ± SD

Abbreviations: CDI 2 – Children's Depression Inventory 2; BPAQ – the Buss-Perry Aggression Questionnaire, PEIQ – The Popular Emotional Intelligence Questionnaire; SSRI – The Schutte Self-Report Inventory.

Table 2. The measures of aggression and EI with respect to the *DRD2* rs6277 polymorphism.

Variable	TT (n = 18)	CC + TT (n = 91)	p-value
BPAQ – total score	67.44 ± 25.53	68.88 ± 23.926	0.816
BPAQ – physical aggression	18.44 ± 9.624	19.67 ± 6.738	0.762
BPAQ – verbal aggression	12.44 ± 5.044	13.11± 5.295	0.375
BPAQ – anger	16.72 ± 6.596	18.82 ± 6.164	0.213
BPAQ – hostility	19.5 ± 7.618	17.59 ± 8.188	0.391
PEIQ – total score	294.25 ± 23.702	307.08 ± 36.8	0.115
PEIQ – empathy	64.65 ± 12.21	64.16 ± 12.275	0.649
PEIQ – acceptance	44.85 ± 9.922	48.64 ± 10.449	0.216
PEIQ – control	30.5 ± 5.577	32.31 ± 7.147	0.239
PEIQ – understanding	26.65 ± 5.976	28.92 ± 6.205	0.173
SSRI – total score	106.32 ± 21.331	111.01 ± 25.812	0.373

Data expressed as mean ± SD

Abbreviations: BPAQ – the Buss-Perry Aggression Questionnaire, PEIQ – The Popular Emotional Intelligence Questionnaire; SSRI – The Schutte Self-Report Inventory.

Table 3. The measures of aggression and EI with respect to the *COMT* rs4680 polymorphism.

Variable	Val-/Val (n = 24)	Met-/Val + Met-/Met (n = 82)	p-value
BPAQ – total aggression	68.67 ± 26.189	68,27 ± 23,886	0.816
BPAQ – physical aggression	19.13 ± 7.64	19,48 ± 7,242	0.762
BPAQ – verbal aggression	13.04 ± 5.473	12.8 ± 5.17	0.375
BPAQ – anger	19.04 ± 7.123	18.3 ± 6,081	0.213
BPAQ – hostility	17.17 ± 8.899	18.05 ± 7.891	0.391
PEIQ – total score	311.52 ± 36.361	303.18 ± 35.022	0.115
PEIQ – empathy	65.48 ± 12.686	64.13 ± 12.149	0.649
PEIQ – acceptance	51.64 ± 10.132	47.02 ± 10.505	0.216
PEIQ – control	31.8 ± 7.681	31.86 ± 6.811	0.239
PEIQ – understanding	28.92 ± 6.952	28.48 ± 6.121	0.173
SSRI – total score	115.25 ± 22.305	109.37 ± 26.038	0.373

Data expressed as mean ± SD

Abbreviations: BPAQ – the Buss-Perry Aggression Questionnaire, PKIE – The Popular Emotional Intelligence Questionnaire; SSRI – The Schutte Self-Report Inventory

DISCUSSION

In this study, we failed to find any significant associations between variation in dopaminergic genes and the levels of aggression and EI in adolescents with conduct disorder. The mesolimbic dopaminergic innervations have an important modulating role in aggressive behaviors. Dysfunctions in this system can contribute to conduct disorders [5]. The current study explored the role of dopaminergic system genes in the etiology of aggressive behaviour in adolescents with conduct disorders. The aim of this study was characterize the the impact of the *COMT* Val158Met polymorphism and the *DRD2* gene polymorphism on EI and aggressive behaviors. This functional variant of the *COMT* gene has been found to account for a four-fold reduction

enzymatic activity resulting in increased dopamine levels. To our knowledge, this is the first study addressing the association between polymorphisms in the *COMT* and *DRD2* genes, aggressive behavior and the EI level in adolescents with conduct disorder.

The dopaminergic system is a complex structure encoded by many genes. The majority of previous studies have demonstrated that any single gene polymorphism is related to aggressive behavior [27-31]. Our results are in agreement with recent reports showing no association between the *COMT* gene polymorphism and other dysfunctional behaviors, such as suicidal behavior [28, 29]. However, both the *COMT* gene polymorphism [30-36], and the *DRD2* gene polymorphism [37, 38] have been associated with susceptibility to specific mental disorders, including attention deficit hyperactivity disorder (ADHD), schizophrenia, schizoaffective disorder, alcohol dependence or mood disorders.

One of potential directions for this field would be to test the effects of potential gene x environment interactions on the level of aggression and EI. Indeed, interactions between variation in the *DRD2* gene, family dysfunction and adolescent behavioral disorders have been found [39, 40]. More specifically, have been reported to be greater among the A1-allele carriers. In another study, no significant effects of interaction between the *DRD2* gene polymorphisms and early separation on aggression in adolescents was found [41]. Discordant findings between these studies can therefore be explained by differences in the conceptualization of externalizing behavior and/or family adversity. The *DRD2* genotype in adolescents might not affect the relation between parental separation, which might not necessarily correlate with the experience of aggressive behavior and family dysfunction, while it may affect the relationship between adverse familial events, such as the experience of having an incarcerated father or a lack of family closeness, and delinquency [42]. Alternatively, variation in the *DRD2* gene might interact with family adversity in predicting aggressive behavior in adolescent, but not in predicting other or broader forms of behavior. Moreover, a meta-analysis carried out by Weeland, et al. [43] showed no direct associations between the *COMT* gene polymorphism and externalizing psychopathology [43], but it was proposed that heterozygosity might be a protective factor for psychopathology [44]. The existing data are contradictory: some studies have shown that the effect of family adversity is greater among the Met allele carriers while other studies have shown this effect among the Val allele carriers. For example, Thompson et al. [17] demonstrated that the effect of maternal stress on behavioral disorders is greater in homozygous children with the Met allele than in children with the Val allele. In turn, Hygen et al. [28] found that children who had to deal with many serious life events and were Val homozygotes are more aggressive than their Met allele-carrying counterparts. In particular, in the absence of serious life events, the Val allele homozygotes have been demonstrated to display significantly lower aggression scores than the Met allele carriers. In the case of the *COMT* gene polymorphism, this apparent contradiction might be explained by a cognitive/emotional compromise [10], in which the Met allele is associated in cognitive processing and the Val allele is related to an advantage in emotional processing [45]. At the same time, this allele might create a genetic predisposition to increased emotional agitation and lower emotional control. This might further mean that a lower level of EI can contribute to emotional dysregulation, aggressive behavior as consequences reported in the Met/Met homozygotes. Two different alleles may therefore function both as genetic risk and/or protective factor in different environments. These findings might suggest that the individuals with the *COMT* gene alleles, leading to decreased enzymatic activity, are more sensitive to stressful life events in terms of developing aggressive behavior. Importantly, this does not mean that adolescents with other genotypes are not susceptible to the effects of environmental exposures but rather that they might respond to different levels or types of environmental exposures. Whether adolescents will develop aggressive behaviors may depend on the combined effect of genes and environmental factors on dopamine activity in the brain.

There are certain limitations of this study that need to be addressed. Firstly, our sample was not large and thus a type II error cannot be excluded. The small sample size does not provide sufficient data to detect a significant statistical

difference, and the power of this study to detect genetic associations might be insufficient. Similarly, some clinical correlations might have been overlooked due to small sample size. In this regard, our results should be considered preliminary and warrant further studies in larger samples. Another downside of this study is the lack of a control group. When planning future research, one should consider comparing the results of the study group with randomly selected peers.

Moreover, it should be noted that genes encoding proteins involved in dopaminergic neurotransmission are highly polymorphic. Therefore, assessment of two single nucleotide polymorphisms provides a limited insight into genetic variability of the dopaminergic system. Examining additional polymorphisms across these genes is required to provide more comprehensive insights. Finally, caution should be taken as to the way our results with respect to the *DRD2* rs6277 polymorphism are being interpreted. Indeed, the distribution of the *DRD2* rs6277 polymorphism did not follow the HWE. This might be due to population stratification as our study was based on individuals with conduct disorder. Similar disagreement was also reported in one of our previous studies based on a different population [39].

Moreover, testing gene x environment interactions by taking into account the effects of various environmental exposures, such as early-life stress, might provide more comprehensive insight into the role of variation in dopaminergic genes in shaping aggressive behaviors. Finding explanations for behavioral disorders and their aggressive behaviour during adolescence is particularly important because they are known to be a strong predictor of psychopathological outcomes later in life [46].

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