UNIWERSYTET MEDYCZNY
IM. PIASTÓW ŚLĄSKICH WE WROCŁAWIU

Praca doktorska:

Związek między agresją i autoagresją a inteligencją emocjonalną oraz polimorfizmem genów związanych z neurotransmisją dopaminergiczną u młodzieży z zaburzeniami zachowania

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Halicka-Masłowska J, Szewczuk-Bogusławska M, Rymaszewska J, Adamska, A, Misiak B. (2021). From emotional intelligence to self-injuries: A path analysis in adolescents with conduct disorder. *Frontiers in Psychiatry* doi:10.3389/fpsyt.2020.556278

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Halicka-Masłowska J, Szewczuk-Bogusławska M, Pawlak-Adamska E, Adamska A, Misiak B. (2021). Effects of variation in dopaminergic genes on the level of aggression and emotional intelligence in adolescents with conduct disorder. *Arch.Psychiatr.Psychother* doi:10.12740/APP/128451

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Halicka-Masłowska J, Szewczuk-Bogusławska M, Adamska A, Misiak B. (2020). Neurobiology of the association between non-suicidal self-injury, suicidal behavior and emotional intelligence: A review. *Arch.Psychiatr.Psychother* 2: 25–35 doi:10.12740/APP/117705

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Oświadczenia współautorów prac określające indywidualny wkład każdego z nich zawiera załącznik nr 2

Spis treści

1. Streszczenie

Agresja charakteryzuje młodzież, u której rozpoznano zaburzenia zachowania. Autoagresja (samouszkodzenia, próby samobójcze) często współwystępuje z zachowaniami agresywnymi. Współwystępowanie agresji i autoagresji potwierdzono także w grupie adolescentów z diagnozą zaburzeń zachowania. Istotną funkcją tych zachowań jest regulacja emocjonalna, z którą wiąże się funkcjonowanie układu dopaminergicznego. Ponadto, wykazano, że niski poziom inteligencji emocjonalnej (ang. *emotional intelligence*, EI) może wiązać się z wyższym ryzykiem agresji i autoagresji. Zmienione neuroprzekaźnictwo dopaminergiczne może przyczyniać się do częstszego podejmowania agresji i autoagresji oraz upośledzenia EI. Do tej pory, dokładne mechanizmy leżące u podstaw oddziaływania polimorfizmów w genach kodujących białka neurotransmisji dopaminergicznej na poziom agresji, autoagresji oraz EI nie zostały szczegółowo poznane.

Cel: Celem pracy doktorskiej była analiza związku pomiędzy inteligencją emocjonalną, agresją, samouszkodzeniami a polimorfizmami genów katecholo-O-metylotransferazy (*COMT*, rs6277) i receptora dopaminergicznego D2 (*DRD2,* rs4680) w grupie adolescentów z zaburzeniami zachowania. Dodatkowym celem była ocena związku pomiędzy EI a ryzykiem samouszkodzeń w omawianej grupie adolescentów.

Metoda: Grupę badaną stanowiły 144 osoby (w wieku 14,8 ± 1,2 lat, 56,6% kobiet) będące wychowankami Młodzieżowego Ośrodka Socjoterapii nr 2 we Wrocławiu. Do badania wykorzystano narzędzia diagnostyczne i kwestionariusze psychologiczne: MINI-Kid (ang. Mini International Neuropsychiatric Interview for Children and Adolescents), INTE (ang. Emotional Intelligence Questionnaire; Kwestionariusz Inteligencji Emocjonalnej), PEIQ (ang. Popular Emotional Intelligence Questionnaire; Popularny Kwestionariusz Inteligencji Emocjonalnej; Popularny Kwestionariusz Inteligencji Emocjonalnej), BPAQ (ang. Buss-Perry Aggression Questionnaire; Kwestionariusz Agresji Bussa-Perry'ego), CDI2 (ang. Child and Adolescent Depression Assessment Questionnaire; Kwestionariusz do Diagnozy Depresji u Dzieci i Młodzieży), STAI (ang. State-Trait Anxiety Inventory; Inwentarz Stanu i Cechy Lęku), SES (ang. Rosenberg Self-Esteem Scale; Skala Samooceny Rosenberga), IVE (ang. Impulsivity Questionnaire; Kwestionariusz Impulsywności Eysencka), QADS (ang. Questionnaire for the Assessment of Disgust Sensitivity; Kwestionariusz Oceny Wrażliwości na Wstręt). Analizę polimorfizmów w genach *COMT* i *DRD2* przeprowadzono w DNA wyizolowanym z leukocytów krwi obwodowej (genotypowanie metodą dyskryminacji alleli z użyciem zwalidowanych sond typu TaqMan).

Wyniki: Nie stwierdzono istotnego statystycznie związku między polimorfizmami *COMT* rs4680 i *DRD2* rs6277 a inteligencją emocjonalną i agresją. Wykazano jednak, że osoby dokonujące autoagresji mają znacznie wyższy poziom depresji, lęku i impulsywności, a także znacznie niższy poziom EI i samooceny. Wyższy poziom EI wiązał się ze znacznie wyższym poziomem samooceny i empatii, a także znacznie niższym poziomem depresji, lęku i impulsywności. Dalsza analiza wykazała, że lęk oraz samoocena są całkowitymi mediatorami związku między EI a autoagresją w badanej grupie.

Wnioski: Przeprowadzone analizy wskazują, że lęk i samoocena mogą pośredniczyć w związku między EI a samouszkodzeniami u adolescentów z zaburzeniami zachowania. W celu potwierdzenia tego wniosku potrzebne są badania podłużne na większej próbie. Uzyskane wyniki badań nie potwierdzają związku pomiędzy polimorfizmem genów *COMT* i *DRD2* a poziomem agresji i EI.

1. Abstract

Introduction: Aggression characterizes adolescents diagnosed with conduct disorder. Autoaggression (self-harm, suicide attempts) often co-occur with aggressive behavior. An important function of these behaviors is emotional regulation associated with the functioning of the dopaminergic system. Moreover, it has been shown that a low level of emotional intelligence (EI) may be associated with a higher risk of aggression and self-harm. Altered dopaminergic neurotransmission may contribute to more frequent aggression and self-harm and EI impairment. Until now, the exact mechanisms underlying the influence of polymorphisms in genes encoding dopaminergic neurotransmission proteins on the level of aggression, self-harm, and EI have not been elucidated in detail.

Aim: The aim of the dissertation was to analyze the relationship between emotional intelligence, aggression, self-harm, and polymorphisms of catechol-O-methyltransferase genes (*COMT*, rs6277) and the dopaminergic D2 receptor (*DRD2*, rs4680) in a group of adolescents with conduct disorders. An additional goal was to assess the relationship between EI and the risk of self-harming behavior in the discussed group of adolescents.

Method: The study group consisted of 144 people (14.8 ± 1.2 years old, 56.6% women) who were charges of the Youth Sociotherapy Center No. 2 in Wrocław. The study used diagnostic tools and psychological questionnaires: MINI-Kid (Mini International Neuropsychiatric Interview for Children and Adolescents), INTE (Emotional Intelligence Questionnaire), PKIE (Popular Emotional Intelligence Questionnaire), CDI2 (Child and Adolescent Depression Assessment Questionnaire), STAI (State-Trait Anxiety Inventory), SES (Rosenberg Self-Esteem Scale), IVE (Impulsivity Questionnaire), QADS (Questionnaire for the Assessment of Disgust Sensitivity). The analysis of polymorphisms in *COMT* and *DRD2* genes was performed in DNA isolated from peripheral blood leukocytes (genotyping by allele discrimination using validated TaqMan probes).

Results: There was no statistically significant relationship between the *COMT* rs4680 and *DRD2* rs6277 polymorphisms and emotional intelligence, aggression, and self-harm. However, it has been shown that self-harming individuals have significantly higher levels of depression, anxiety, and impulsivity, as well as significantly lower levels of EI and self-esteem. Higher levels of EI were associated with significantly higher levels of self-esteem and empathy, as well as significantly lower levels of depression, anxiety, and impulsivity. Further analysis showed that anxiety and self-esteem are complete mediators of the relationship between EI and self-harm in the study group.

Conclusions: The conducted analyzes indicate that anxiety and self-esteem may mediate the relationship between EI and self-harm in adolescents with conduct disorders. A larger sample longitudinal study is needed to confirm this conclusion. The obtained results do not confirm the relationship between the polymorphism of *COMT* and *DRD2* genes and the level of aggression and EI.

2. Wstęp

Zaburzenia zachowania (F91.0, F91.1, F91.2 wg klasyfikacji ICD-10) występują u około 5% adolescentów [1]. W zależności od przyjętych kryteriów i definicji agresji, zachowania agresywne podejmuje ok. 22-93% młodzieży z zaburzeniami zachowania [2, 3]. Zachowania autoagresywne często współwystępują w tej grupie adolescentów. Typowymi zachowaniami autoagresywnymi są: samobójstwa, próby samobójcze oraz samouszkodzenia. Aktualne dane epidemiologiczne wskazują, że w skali globalnej w ciągu roku samobójstwo popełnia miliony ludzi [4]. Samobójstwo jest drugą przyczyną śmierci wśród osób w wieku 15-19 lat i stanowi 8,5% wszystkich zgonów w tej grupie wiekowej [5]. Rozpowszechnienie prób samobójczych wynosi 2,7% w USA [6], 10,5% w Europie [7]. Częstość występowania samouszkodzeń u młodzieży szacuje się na 17-18% w populacji ogólnej [8] i u 40-61% u adolescentów hospitalizowanych psychiatrycznie [9]. U młodzieży z zaburzeniami zachowania, częstość występowania autoagresji szacuje się na 15,5-62,5% [10].

Wiele czynników psychologicznych, psychiatrycznych, genetycznych i demograficznych zostało wcześniej zbadanych w celu oceny czynników agresji i autoagresji. Jedną ze zmiennych psychologicznych wpływających na dokonywanie autoagresji jest poziom inteligencji emocjonalnej (ang. *emotional intelligence*, EI). EI odpowiada za skuteczność regulacji emocjonalnej, której funkcjonowanie jest czynnikiem ochronnym przed agresją i autoagresją. Według Golemana, IE to zbiór umiejętności społecznych, które odnoszą się do możliwości rozumienia emocji własnych oraz innych osób, a także zarządzania nimi i ich kontroli [11]. Meta-analiza badań dotyczących autoagresji wykazała, że objawy zaburzeń eksternalizacyjnych (62,9%) są częstsze niż zaburzeń internalizacyjnych (51,7%) u osób podejmujących zachowania autoagresywne [12]. Częstsze dokonywanie agresji i autoagresji zostało stwierdzone u osób z niskim poziomem EI. Jednak dokładne mechanizmy leżące u podstaw agresji i autoagresji oraz IE u osób z zaburzeniami zachowania nie zostały jeszcze dokładnie poznane.

Szacuje się, że 65% wariancji w występowaniu zachowań agresywnych można przypisać czynnikom genetycznym, a resztę czynnikom środowiskowym [13]. Coraz więcej danych wskazuje na to, że zmienność genów dopaminergicznych może znacząco wpływać na zachowanie agresywne i autoagresywne. Uważa się, że układ dopaminergiczny prążkowia pośrednio wpływa na występowanie impulsywności i innych cech temperamentu, co sugeruje, że różne warianty genów zaangażowanych w neurotransmisję dopaminergiczną mogą modulować wzorzec zachowań agresywnych oraz autoagresywnych. Wyniki badań wskazują na związek pomiędzy allelem *COMT* Met158 a agresją jako cechą osobowości u dorosłych, agresją w schizofrenii, zaburzeniach zachowania i agresją u dzieci [14-16]. Jak wiadomo, produkt ekspresji genu *COMT* wpływa na aktywność dopaminy w korze przedczołowej. U homozygot *COMT* 158Val/Val, aktywność dopaminy jest czterokrotnie niższa ze względu na wysoką aktywność *COMT*. Wiąże się to ze zmniejszoną wrażliwością na czynniki stresowe, wyższym progiem wrażliwości na ból oraz obniżeniem możliwości przetwarzania informacji [17].

Aktywność dopaminy jest regulowana również poprzez funkcjonowanie receptora dopaminergicznego D2 (kodowanego przez gen *DRD2*). Badania wskazują na wpływ polimorfizmu *DRD2* A241G TaqIA oraz rs1079598 na agresji u dzieci [18]. Wiadomo, że polimorfizm rs6277 zmniejsza aktywność dopaminy, jednakże, nie wiadomo w jaki sposób oddziałuje na agresję i autoagresję. Co więcej, badania wykorzystujące techniki neuroobrazowania wykazały, że istnieją wspólne obszary mózgu aktywowane podczas zachowań agresywnych, autoagresywnych i przetwarzania emocjonalnego.

W dostępnej literaturze nie ma jednak wyników, w których poddawano analizie związek genów układu dopaminergicznego z wzorcem zachowań agresywnych i autoagresywnych.

3. Cele szczegółowe poszczególnych części cyklu pracy doktorskiej

Część I:

Halicka-Masłowska J, Szewczuk-Bogusławska M, Adamska, A, Misiak B. (2020) Neurobiology of the association between non-suicidal self-injury, suicidal behavior and emotional intelligence: A review. Arch.Psychiatr.Psychother 2: 25–35 doi:10.12740/APP/117705

Celem artykułu przeglądowego było przedstawienie obecnego stanu wiedzy na temat neurobiologicznego podłoża EI i autoagresji (samouszkodzeń oraz samobójstw), a także wykazanie związku między nimi. Dokonano krytycznej oceny literatury oraz przedstawiono perspektywę dalszych badań, które stały się również podstawą rozważań przeprowadzonych w ramach pracy doktorskiej.

Część II:

Halicka-Masłowska J, Szewczuk-Bogusławska M, Pawlak-Adamska E, Adamska A, Misiak B. (2021). Effects of variation in dopaminergic genes on the level of aggression and emotional intelligence in adolescents with conduct disorder. Arch.Psychiatr.Psychother doi:10.12740/APP/128451

Celem pracy było zbadanie wpływu polimorfizmów w genach neurotransmisji dopaminergicznej (*COMT* rs6277 i *2 DRD2* rs4680) na poziom agresji i IE u młodzieży z zaburzeniami zachowania. W dotychczas przeprowadzonych badanach wykazano, wpływ neurotransmisji dopaminergicznej na zachowania agresywne i autoagresywne.

Część III:

Halicka-Masłowska J, Szewczuk-Bogusławska M, Rymaszewska J, Adamska A, Misiak B. (2021). From emotional intelligence to self-injuries: A path analysis in adolescents with conduct disorder. Frontiers in Psychiatry doi:10.3389/fpsyt.2020.556278

Celem tego badania było sprawdzenie, czy i w jaki sposób wybrane zmienne kliniczne, takie jak: poziom agresji, depresji, samooceny, impulsywności, lęku i wstrętu pośredniczą w związku między EI a ryzykiem samouszkodzeń u nastolatków z zaburzeniami zachowania.

4. Materiał i metody

1. Grupa badana:

Badanie zostało przeprowadzone wśród wychowanków Młodzieżowego Ośrodka Socjoterapii nr 2 we Wrocławiu. Do badania zakwalifikowano 144 osoby (85 kobiet i 61 mężczyzn) w wieku 13-18 lat z diagnozą zaburzeń zachowania F91.0, F91.1, F90.2. Z powodu braku danych niezbędnych do wykonania z analiz wykluczono 8 osób. Ostateczna próba obejmowała 136 osoby (77 kobiet i 59 mężczyzn).

Kryteriami wykluczenia były: niepełnosprawność intelektualna, majaczenie, zatrucie lub objawy odstawienne, zaburzenia psychotyczne oraz zaburzenia ze spektrum autyzmu. Wszyscy uczestnicy oraz ich przedstawiciele ustawowi wyrazili pisemną zgodę na wszystkie procedury badawcze.

Dane były zbierane od września 2016 r. do sierpnia 2019 r. Dbając o komfort badanych, badanie podzielono na trzy części, z których każda trwała około godziny. W pierwszej części zebrano wywiad dotyczący samouszkodzeń, w drugiej dokonano diagnozy zaburzeń psychicznych, a w części trzeciej dane kliniczne zebrane za pomocą wystandaryzowanych narzędzi psychologicznych. Od każdej osoby pobrano 10 ml krwi obwodowej celem przeprowadzenia badań genetycznych.

Na przeprowadzenie badania uzyskano zgodę Komisji Bioetycznej Uniwersytetu Medycznego we Wrocławiu nr KB532/2017.

2. Narzędzia diagnostyczne oraz kwestionariusze psychologiczne:

2.1. Kwestionariusz własny:

Częściowo ustrukturalizowany wywiad dotyczył dokonywania zachowań autoagresywnych. Zawierał pytania dotyczące liczby i częstotliwości dokonywania samouszkodzeń i prób samobójczych.

2.2. MINI-Kid (ang. Mini International Neuropsychiatric Interview for Children and Adolescents):

MINI-Kid jest ustrukturyzowanym wywiadem diagnostycznym, opracowanym przez psychiatrów i klinicystów w Stanach Zjednoczonych i Europie według kryteriów DSM-IV i ICD-10. W aktualnym badaniu narzędzie to posłużyło do postawienia diagnozy zaburzeń zachowania [19].

2.3. PEIQ (ang. Popular Emotional Intelligence Questionnaire; Popularny Kwestionariusz Inteligencji Emocjonalnej):

PEIQ jest narzędziem do oceny poziomu inteligencji emocjonalnej. Składa się z 94 pozycji o charakterze samoopisowym, przy użyciu pięciostopniowej skali Likerta. Zawiera następujące podskale: akceptacja (wyrażanie i używanie własnych emocji), empatia (rozumienie i rozpoznawanie emocji innych ludzi), kontrola (kontrola nad własnymi emocjami) i rozumienie (rozumienie i świadomość własnych emocji) [20]. Współczynnik alfa Cronbacha w badanej próbie wyniósł 0,89.

2.4. BPAQ (ang. Buss and Perry Aggression Questionnaire; Kwestionariusz Agresji Bussa-Perry'ego):

BPAQ jest kwestionariuszem mierzącym agresję u nastolatków i dorosłych. BPAQ składa się z 29 pozycji, 252 podzielonych na cztery czynniki: agresję fizyczną, agresję werbalną, złość i wrogość [21]. Współczynnik alfa Cronbacha w badanej próbie wyniósł 0,80, dla agresji fizycznej 0,77, dla 254 agresji słownej 0,73, dla złości 0,62 i dla wrogości 0,77.

2.5. CDI2 (ang. Children's Depression Inventory 2; Kwestionariusz do Diagnozy Depresji u Dzieci i Młodzieży 2):

CDI2 jest narzędziem do badania objawów depresji u dzieci i młodzieży. Kwestionariusz zawiera skale: problemy emocjonalne, problemy związane z codziennym funkcjonowaniem, samoocena, negatywny nastrój/objawy somatyczne, brak efektywności, problemy interpersonalne. Kwestionariusz składa się z 28 pozycji. Do obliczenia spójności wewnętrznej skali wykorzystano współczynnik alfa Cronbacha, a wyniki wskazały, że zgodność wewnętrzna wszystkich podskal jest na zadowalającym poziomie [22]. Współczynnik alfa Cronbacha dla CDI2 w badanej próbie wyniósł 0,94.

2.6. STAI (ang. State-Trait Anxiety Inventory; Inwentarz Stanu i Cechy Lęku):

STAI służy do badania lęku rozumianego jako względnie stała cecha osobowości oraz poziom lęku związanego z określonymi sytuacjami. Każda z dwóch podskal (lęk jako cecha i lęk jako stan) składa się z 20 pytań diagnostycznych ocenianych w czterostopniowej skali [23]. Współczynnik alfa Cronbacha dla całkowitego wyniku w badanej próbie wyniósł 0,89 oraz 0,92 dla lęku jako cechy i 0,94 dla lęku jako stanu.

2.7. SES (ang. Rosenberg Self-Esteem Scale; Skala Samooceny Rosenberga):

SES jest jednowymiarowym narzędziem do badania poziomu samooceny - rozumianej jako względnie trwała, świadoma, postawa (pozytywna lub negatywna) wobec siebie. Kwestionariusz składa się z 10 pytań diagnostycznych. Każde pytanie opiera się na czterostopniowej skali Likerta ilustrującej stopień, w jakim dana osoba zgadza się z twierdzeniami [24]. Współczynnik alfa Cronbacha dla całkowitego wyniku SES w badanej próbie wyniósł 0,89.

2.8. IVE (ang. Eysenck's Impuslivity Questionnaire; Inwentarz Impulsywności Eysencka):

IVE służy do oceny impulsywności i obejmuje następujące podskale: impulsywność, skłonność do ryzyka i empatia. Składa się z 63 pytań diagnostycznych w dwustopniowej skali [25]. Współczynnik alfa Cronbacha dla każdej podskali kształtował się następująco: 0,75 (dla impulsywności), 0,66 (dla skłonności do ryzyka) i 0,65 (dla empatii).

2.9. QADS (ang. Questionnairefor the Assessment of Disgust Sensitivity; Kwestionariusz Oceny Wrażliwości na Wstręt):

QADS służy do oceny nasilenia wstrętu. Składa się z 37 stwierdzeń, w których badany ocenia stopień nasilenia wstrętu na pięciostopniowej skali Likerta. Wrażliwość na wstręt odnosi się do cech osobowości jednostki i opisuje predyspozycję do reagowania z obrzydzeniem na określone sytuacje i czynniki. W kwestionariuszu występują trzy podskale: wstręt podstawowy, wstręt do natury zwierzęcej i wstręt zakażenia / interpersonalny [26]. Współczynnik alfa Cronbacha dla całkowitego wyniku QADS w badanej próbie wyniósł 0,94 oraz 0,88 dla wstrętu podstawowego, 0,90 dla wstrętu do natury zwierzęcej i 0,79 dla wstrętu zakażeń / interpersonalnego.

3. Badania genetyczne:

Od wszystkich uczestników pobrano 10ml krwi obwodowej, z której wyizolowano DNA przy użyciu zestawu Maxwell® 16 LEV Blood DNA Kit (Promega Corporation, Madison, USA) zgodnie z protokołem producenta.

Polimorfizmy zlokalizowane w genach: *COMT* rs4680 oraz *DRD2* rs6277 oznaczone zostały techniką dyskryminacji alleli (ang. *allelic discromination*) z wykorzystaniem swoistych zestawów TaqMan®SNP Genotyping Assays (C__25746809_50 i C__11339240_10, odpowiednio).

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Neurobiology of the association between non-suicidal self-injury, suicidal behavior and emotional intelligence: A review

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Summary

Non-suicidal self-injuries (NSSI) and suicidal behaviours (SB) are common causes of serious medical problems leading to hospitalization or death in adolescents and young adults. The prevalence of NSSI in adolescents is estimated at 17-18% in general population and in 40% of psychiatric hospitalized patients. Nearly one million people worldwide die from suicide each year. The epidemiological data show that suicide is the fourth cause of death among children between 10 and 14 years old, the third cause among people aged 15-19 years old, the first or second among people 14-25 years old and the second among people 25-34 years old. Many psychological, psychiatric, genetic and demographic factors have been previously studied in order to assess risk factors leading to NSSI and SB. One of psychological factors influencing the engagement in NSSI and SB is emotional intelligence (EI), which is defined as collection of social skills. More frequent NSSI and SB have been found in individuals with low EI in previous studies. The relationship between SB, NSSI and underlaying neurotransmission and brain structures have been also extensively studied. Studies applying neuroimaging techniques show correlation between alterations of brain areas which are responsible for involving in self-injourous acts and suicidal behaviours and regions key to EI levels. Thus we aimed to review the neurobiological background of emotional intelligence and self-harm and discuss the current state of knowledge on its relationship

emotional intelligence, suicide, adolescence, suicide attempt, non-suicidal self-injury

BASIC DEFINITIONS AND EPIDEMIOLOGICAL ISSUES

In order to describe self-injury behaviours, researchers usually use the following terms: suicidal behaviour (SB) and non-suicidal self-injury (NSSI). The first one includes thoughts of suicide, suicidal attempt (SA) and committed suicide (suicide, S). NSSI is a deliberate self-harm behaviour without intent of death.

Suicide traits include: purposefulness, intentionality, independence, voluntary, consciousness and planned [1]. According to current WHO data, suicides are among the twenty most common causes of death among the general population, the third leading cause of death among adolescents aged 15-19 years and the fourth leading cause of death in children aged 10-14 years [2]. In 2001, the number of people who died as a result of suicide was about one

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million, exceeding the number of murder and war victims (500.000 and 230.000 respectively). According to current estimates, the number of suicide victims may reach as much as 1.5 million in 2020. The suicide rate varies from country to country per year, ranging from 0.1 (Egypt) to over 40 (Hungary, Russia, Lithuania) per 100.000 inhabitants [2]. In Poland, this indicator has oscillated around 15 per 100.000 people in recent years [3].

Due to the epidemiological importance of the problem, research is underway into the identification of risk factors for suicide acts. Despite the fact, that in DSM-5 the following criteria for SBD have been established, this diagnostic construct is still included in the section "conditions for further study".

Suicidal behavior disorder (SBD) can be diagnosed when:

1. at least one suicide attempt occurred in the last two years,
2. suicide attempt does not meet the criteria for self-mutilation,
3. diagnosis does not apply to suicidal thoughts, suicidal plans or preparation for suicide,
4. suicidal behaviour is not related to disturbances of consciousness,
5. the suicide act is not connected with political or religious motivation [4].

NSSI is a specific form of self-injury connected with the destruction of the body tissue occurring without the intention of death. These are activities in which the individual aims at causing immediate damage to himself, which has no effect and no suicidal intent. These behaviours are undertaken in order to damage the body, induce bleeding, bruising or physical pain. NSSI is most commonly performed as a result of cutting the skin continuity – cutting and incision (70-90%), as well as striking (21-44%), burning (15-35%), biting, bruising or scratching the skin, interfering with wound healing (e.g. scratching wounds and scabs), sticking, sticking needles, sticking objects under the nails, pulling hair out [5]. NSSI coexists with emotional and developmental disorders of different etiologies or personality disorders. According to DSM-5, these behaviours are pereceived as a separate nosological entity. The ICD-10 and DSM-IV classifications include self-mutilations as one of the symptoms of borderline personality disorders.

The DSM-5 non-suicidal self-injury disorder diagnostic criteria include [4]:

A. Intentionally committed physical damage, but without suicidal intent, on five or more days in the last year;
B. These acts should be made for at least one of the following reasons:
   1. to free oneself from negative thoughts or feelings,
   2. to solve an interpersonal problem,
   3. in order to evoke positive feelings or emotions;
C. Such behaviour is associated with one or more of the following factors:
   1. negative thoughts, feelings or interpersonal problems that immediately precede the self-mutilations,
   2. absorbing self-mutilations, which is hard to resist,
   3. frequent coercion to perform self-mutilations;
D. Behaviour is not socially sanctioned and is more significant than biting nails or scratching wounds (scabs);
E. It causes clinically significant suffering or damage;
F. This behaviour does not only occur in the context of another disorder and cannot be explained by another mental disorder or condition.

The NSSI is being undertaken by 17-18% of adolescents in the world. After the adolescent period, the prevalence of these behaviors decreases, reaching 6% in adults [6]. In adolescents staying in care and educational centres, this behaviour occurs in 30-40% of cases [7, 8]. The intensity of NSSI is related to developmental stage. The onset of NSSI appears around 12-15 years of age. [5]. These behaviours become particularly intense in the middle of puberty. The highest prevalence of self-mutilations is observed among 15-year-old adolescents (12.7%) [9]. Among younger teenagers (13-15 years of age), NSSI is chronic (12-month period) in 2.5% – 7.5% [10].

It should be noted that the are certain differences in prevalence rates of NSSI acts between girls and boys. The prevalence of NSSI among

girls ranges from 13.5% to 24.3% and among boys from 4.3% to 8.5% [10]. Zetterqvist et. al. [11] describe that DSM-5 self-mutilations frequency criterion among girls (11.1%) than among boys (2.3%).

The risk factors of self-injury include: co-occurrence of mental disorders or psychopathological symptoms (especially affective disorders or borderline personality disorder), substance use, post-traumatic stress disorder, impulsiveness, externalizing disorders, attention deficit disorder with or without hyperactivity and behavioural disorders [1, 5-11]. One of the factors not related to psychopathology, influencing SB and NSSI, is the level of emotional intelligence (EI).

What is EI and how is it related to self-injuring?

EI is a clustering of social skills, and according to Goleman [12], it is the ability to understand, direct, control, and empathize with oneself and one's own emotions. There are two main models describing EI: Trait Emotional Intelligence (TEI) and Abilities Emotional Intelligence (AEI). TEI – capturing EI as a permanent personality trait [13] and is evaluated by questionnaires, e.g. The Trait Meta-Mood Scale [14]. AEI that captures EI as ability [14] and is assessed in objective tests, e.g. The Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) [15].

One of the variables influencing self-injurious behaviour is the level of EI. The results of many studies indicate a correlation between low EI level and more frequent self-destructive actions: both self-injuries and suicide attempts [16-18]. Studies using different brain imaging methods show that differences in the structure and function of similar brain areas are responsible for differences in self-injurious responses as well as differences in EI levels.

Search strategy

The aim of this research is to discuss the neurobiological background of EI, NSSI and SB on the structural, functional and molecular levels and to present the current knowledge on the relationship between EI and suicide risk and deliberate self-harm. Reviewed brain-imaging modalities included structural magnetic resonance imaging, diffusion tensor imaging, positron-emission tomography, single-photon emission computed tomography, resting-state functional imaging, and functional magnetic resonance imaging. Studying these brain-imaging modalities of suicide behaviour have provided crucial information on the neural circuitry associated with EI, NSSI and SB.

For this purpose, from February to April 2019 a review of the latest literature from the years 2015-2019 was carried out, to included only the latest data.

The following data bases were used to review the literature: Scopus, PubMed, Web of Science and Google Scholar. Initially, the combination of keywords was: "emotional intelligence" AND "suicide" OR "suicide attempt" OR "suicidal behavior" OR "suicidality" OR "non-suicidal self-injury", to ensure that the review covers a wide range of research issues related to the problem of co-existence of NSSI and SB with EI. To found neurobiological background in the next searches were added keywords like: "brain structure" OR "structural neuroimaging" OR "functional neuroimaging" OR "molecular neuroimaging" OR "magnetic resonance imaging" OR "neuroimaging" OR "diffusion tensor imaging" OR "positron emission tomography"OR "positron-emission tomography" OR "single-photon emission computed tomography". Then these keywords were added in various combinations to select the articles that are focused on the theme of the brain imaging: Another criterion of selection were the years of publication: 2015-2019. The language of the publication was not a criterion.

Brain structure and its functioning of the self-harming people

The first reports on neurobiological changes concerning people after suicide attempts have their origin in the middle of 1960's. One of the first such studies was conducted by Bunney and Fawcett. They observed elevated levels of 17-hydroxycorticosteroid (a cortisol breakdown product) in urine samples of the suicidal attempters, before suicide [19]. So far, several neurobiological models have been developed to explain the

etiology of suicide. The most common explanation is a model explaining stress-diathesis. It advocates interaction between life stressors and biological susceptibility to suicide. According to this model, susceptible individuals show abnormal or exaggerated reactions to neutral stimuli [20]. There is now more neurobiological data explaining the phenomenon of NSSI and SB that includes the structural, functional and molecular changes in the brain [21-30].

Among methods used to study suicidal brain we can mention: structural imaging, functional imaging and molecular imaging. These methods allowed researchers to explore the relationship between the cognitive, emotional and behavioral components of suicide and the altered neuroanatomy and neural function. The majority of data comes from the results of the magnetic resonance imaging (MRI).

Structural changes mainly concern the areas of the limbic system and are associated with a reduction in the volume of some anterior and temporal parts of the brain [23, 24]. In suicide attempters, a reduction in the volume of brain structures has been demonstrated: the brain cortex (CC, *cerebral cortex*) – mainly *prefrontal cortex* (PFC) and *orbitofrontal cortex* (OFC) [31**],***insula*, *hippocampus*, grey matter (GM, *substantia grisea*), white matter (WM, *substantia alba*), *lentiform nucleus* and *corpus callosum*. Neuroanatomical changes in such individuals are also associated with reducing the volume of the left *angular gyrus* and the right *gyrus rectus* [32]. Some studies have also shown reduction the *anterior cingulate cortex* (ACC), *nucleus caudatus* and reduced volume of the *globus pallidus* on both sides [31-33]. These structural changes appear to be in general particularly involved in the processing of the relevant aspect of the event and can therefore mediate behavioral planning on the basis of negative stimuli [32]. OFC is involved as a key area of the brain for the regulation of emotions and impulses. Studies applying neuroimaging techniques in patients with suicidal behavior demonstrate an increased activity in the *temporal gyrus*, *nucleus caudatus* and *gyrus cinguli* [34], however lower activity is observed in the right *anterior cingulate cortex* (ACC) [30]. Structural changes are present even on the cellular level: the decrease of dendritic branches, decrease in the number of glial cells, especially astrocytes and dendrocytes [31, 32] and the dysfunction of astrocytes [33]. Neurotrophins such as brain-derived neurotrophic factor (BDNF) and fibroblast growth factor (FGF) play a regenerative role and are involved in neuroplastic processes. Among the self-injurious patients, a decrease in the number of these molecules is observed.

Therefore, self-injurious acts seems to be associated as well with neuroanatomical dysfunctions of the brain on a structural level, as with its hyperactivity in some areas and low activity in others. The results of the study indicate that the frontostriatal circuitry is a part of SA etiology. This network regulates emotional stimulation between grey matter and prefrontal cortex. GM and WM are part of the frontostriatal circuitry, which is up to eight times more common among the SA patients than these without SA [30].

Reisch and others [35] have demonstrated, in addition to the aforementioned activation of some areas responsible for emotional control, increased work in the hippocampus area while recalling experiences of suicide attempts with simultaneously cortical inhibition. This suggests deactivation of the frontal areas during the most severe mental pain, inhibiting the emotional control system, leading to suicide attempts or NSSI, and thus to the relief of a mental pain. PFC and ACC are areas activated not only during self-harming behaviour, but also during target-oriented activities. However, focusing on goals can protect from SA, as mental pain is one of the factors of self-mutilation [36,37]. Therefore, this finding can be useful in the therapy of suicidal patients.

The majority of changes in the brain structures of the self-injuring individuals are related to dopamine and serotonergic systems. Suicide etiopathogenesis is associated with a lower level of 5-HT (5-hydroxytryptophan) and 5-HIAA (5-hydroxyindoloacetic acid) in the brainstem [38] and a higher level of TPH2 (tryptophanic hydroxylase 2, an enzyme limiting the rate of serotonin synthesis) among the suicidal attempters. Considering the evidence indicating a general decrease in 5-HT neurotransmission in both cortical and subcortical regions of the individuals after SA, an increase in the amount of TPH2, TPH proteins and mRNA levels can be attributed to the compensation of 5-HT central transmission mitigation mechanism and stress

response [38]. A low level of noradrenaline in the *locus coeruleus* was also found in suicide victims [39].

Brain structures related to EI

The key role in the process of regulation of emotional impulses, processing of emotional information, emotional perception and emotional decision making [19] is played by the limbic system and cerebral cortex (CC, *cerebral cortex*). The limbic system consists mainly of the following brain structures: cingulate cortex, entorhinal cortex (EC, *cortex entorhinalis*) and perirhinal cortex, thalamus, hypothalamus, hippocampus, fornix, mammillary body (*corpus mamillare)* and amygdala.

Due to the complexity and diverse nature of emotional processes, the integration of AEI and TEI constructs is necessary to understand their creation. In persons with higher AEI rates, low activation of aMPFC (*anterior medial prefrontal cortex*) in the context of social understanding is observed [21]. A similar relationship of TEI is observed in adolescents in the context of emotional understanding [22], which suggests the involvement of common nervous pathways for both EI models. On the other hand, it has been demonstrated that only high AEI results are associated with increased activation of vmPFC (*ventromedial prefrontal cortex*), amygdala and insula during emotional responses [23, 24]. High TEI patients were characterized by increased activity of mFC (medial frontal cortex), ACC [25] and default mode network (DMN). Moreover, persons with high TEI results show low correlation between DMN and TPN (dorsal-lateral areas of PCF) [26]. The relationship between the height of TEI and activation of these areas increases during developmental changes – from childhood to early adulthood [27]. Among the patients with high AEI level, increased activity of RSN (resting state network) and decreased activity of DMN and the main emotional processing areas (e.g. insular cortex and orbitofrontal cortex, abdominal corpus striatum, amygdala) are observed [21]. Patients with low Ability EI show a strong basal ganglia network BGN activation [23]. Due to strong connections between the areas responsible for emotional regulation and BGN, people with low AEI have difficulties with emotional self-regulation [11]. Therefore, one of the neurobiological mechanisms underlying the low AEI is ineffective BGN modulation by prefrontal control areas. Moreover, Li et al. [26] have discovered less functional connections between the regions involved in emotion regulation and the antero-medial region of the brain in people with severe depression. They also found a significant correlation between MSCEIT results and functional connectivity in the abdominal prefrontal cortex. The results indicate that people with severe depressive disorder have difficulty in perceiving, understanding and managing emotions.

It was found that the volume of grey matter (GM, *substantia grisea*) in the right upper temporal gyrus, another important region of social emotion processing, decreased in the group of people attempting suicide with major depressive disorder (MDD). It was considered as evidence to support the suggestion that emotional and impulse dysregulation may be associated with suicidal behavior [20]. Several lines of evidence obtained from MRI studies indicated that the frontostriatal network was involved in the etiology of suicide. This network includes a pathway for regulating emotions between subcortical GM areas (e.g. basal ganglia) and PFC. Another area where dysfunction is observed in both self-injurious patients and these with low EI level is ACC. Deficiency in the antero-ACC-striatum system may contribute to impaired decision-making and dysfunctional patterns of emotional regulation [42]. ACC is associated with control of impulsive behaviour and the caudate nucleus is also involved in dopamine rewarding processes [43]. Structural changes in the frontostriatal pathway may lead to impaired behavioral and emotional control, leading to suicidal behavior.

The latest data on the analysis of brain structures related to SB and EI

Study on emotional intelligence and suicidal behaviour is becoming increasingly popular among scientists, but there is still little known about the neurobiological mechanisms of these

behaviors. There are also few meta-analyses of this studies – one of them was created by Balcioglu [21], which reviewed articles studies of suicide behavior published from 1990 to 2017. Analyzed brain-imaging data included structural magnetic resonance imaging, diffusion tensor imaging, positron-emission tomography, single-photon emission computed tomography, resting-state functional imaging, and functional magnetic resonance imaging. This review concerned only neuroimaging studies of suicide behavior, not including NSSI and EI. The effect of this review was to describe the nerve circuits associated with the SB risk: the frontostriatal network, frontal-limbic structures and serotonergic system.

The highest number of data in SB literature is related to studies utilizing neuroimaging techniques which allow to indicate abnormalities in brain morphology and neuronal activity. These studies, however, are often conducted on patients with additional psychopathologies: with diagnosed MDD, BD, SCZ, BPD, which makes the identification of biomarkers specific for SB more difficult. Cox Lippard et al. have performed a review of 57 original studies utilizing diverse neuroimaging methods in the context of the aspects of suicidial behavior. According to the results, there is a noticeable decrease in the volume of gray matter (GM) in the orbitofrontal cortex (OFC) area in the case of MRI attempters in the patient population with MDD, BD, schizophrenia, BPD, as well as white matter hyperintensities in young/mid-adult MDD and BD attempters [44].

In a literature review from 2018 [45] the authors summarize the results of 33 studies related to identifying brain regions associated with SB in patients with various mental disorders. The phenomena with the largest amount of coverage involve volume loss or cortical thinning, especially in the temporal cortex area of suicidal patients in MDD, schizophrenia, BPD. Other areas where cortical reduction has been observed include the frontal, limbic, orbitofronal, and insular lobes.

An interesting meta-analysis of molecular studies on the functioning of the serotonergic system, as the one most strongly involved in the etiopathogenesis of suicide, was carried out by Wang et al. [46] The findings provide an indication of connection between pathological functioning of this system with some subtypes of 5-HT receptors and suicidal behaviours. The authors conclude that the decreased binding of the 5-HT1A receptor was associated with depression pathology in various regions of the brain, which may be associated with suicidal behaviour.

There is a smaller number of reports dealing with changes in the noradrenergic system and dopaminergic system (reduction in the dopamine turnover). These involve dysfunctions at the level of receptors and enzymes [47]. Noradrenergic studies in the postmortem brains of suicide victims indicate increased protein expression of tyrosine hydroxylase (TH) as well as a rise in the amount of α_2 – and β_2-adrenergic receptors [48].

The table below shows the results of the latest reports on brain structures associated with EI and SB (SA and NSSI). It presents the most important publications of oryginal research carried out on N>50 groups of recent years (2015-2019) concerning neurobiological determinants of EI and self-injury/suicidal bahaviour.

Table 1. The table presents the most important publications of oryginal research from 2015-2019 regarding the analysis of brain structural and cellular changes among the self-injurious and suicidal patients.

Author(s)	The aim of the study	Sample characteristics	Measures	Results
Quarto et al. [33].	Analysis of brain structures associated with AEI.	N = 63; age 29.4 (6.3) the number of women 34; Hollingshead 41.8 (16.7); Handedness0.8 (0.4); IQ=112 (12.3).	fMRI; AEI behavioral test.	High AEI results are associated with increased activation of vmPFC, amygdala and insula in emotional reactions; low AEI results are associated with strong BGN activation [26].

Killgore et al. [34].	Analysis of brain structures associated with EI (TEI and AEI).	N=70; women32; age 18-45 years(average age = 30,9 years; SD = 8,4 years).	fMRI; Bar-On Emotional Quotient Inventory (EQ-i); MSCEIT.	High AEI results are associated with insula activity and vmPFC and deactivation of BGN and DMN.
Linget al. [43].	Analysis of brain structures associated with EI.	N=105.	resting-state functional magnetic resonance imaging (rs-fMRI); MSCEIT.	The region strongly associated with EI is the superior left parietal lobe (SPL).
Oliveira et al. [49].	Analysis of brain structures associated with cognitive and emotional intelligence.	N=50; age 24-37years (average for women (SD) = 29,4 (4,5), average for men (SD) = 30,2 (4.6); 50% women).	fMRI; Empathy self report measure.	High activity of DMN in cognitive processing, low activity in emotional processing.
Duarte et al. [27].	Analysis of brain structures associated with SB and ChAD.	N=59.	fMRI.	In persons with SB an increased activation of PFC and ACC is observed.
Zhang et al [40].	Search differences in DMN activity which could be related to SB.	N=100.	rs-fMRI.	Depressive adolescents involved in SA are characterized by abnormal functional connectivity in some DMN regions, and abnormal connectivity in the PCC/precuneus and left cerebellum.
Groschwitz et al. [50].	Analysis of brain structures associated with NSSI.	N=53; average age u15,2 years (SD = 1,8).	fMRI.	In persons with NSSI an increased activation of mPFC, vrPFC, right *ventrolateral prefrontal cortex*) and vlPFC, *left ventrolateral prefrontal cortex*) is observed.
Pu et al. [51].	Association between prefrontal function and suicidal ideation in MDD.	N= 134; 67 MDD patients (31 suicidal ideators) vs. 67 healthy controls.	Near-infrared spectroscopy with verbal fluency task: regional cerebral oxy-hemoglobin measurement.	Smaller hemodynamic changes during the task in the right dorsolateral PFC, the OFC, and the frontopolar cortex in the MDD patients with suicidal thoughts.
Tsujii et al. [52].	Fontotemporal hemodynamic responses in depressed patients with a history of suicide attempts using 52-channel NIRS.	N=108; 68 MDD patients (30 attempters) vs. 40 healthy controls.	Near-infrared spectroscopy with verbal fluency task: regional cerebral oxy-hemoglobin measurement.	Smaller hemodynamic changes during the task in the left precentral gyrus in the attempters.

Cao et al. [53].	The relationship between abnormalities involving local brain function and suicidal attempts in depressed youths.	N=100; 53 MDD patients (35 attempters) vs. 47 health controls (young adults).	fMRI: Resting-state activity.	Increased activity in the right superior temporal, left middle temporal, and left middle occipital gyri; decreased activity.
Cyprien et al. [24].	The impact of suicidal behavior on CC integrity in mood disorders.	N=121; 91 patients with mood disorders (45 attempters) vs. 30 healthy controls.	MRI: DTI.	Significantly lower FA value of the splenium part of the corpus callosum in attempters.
Colle et al. [54].	The association between hippocampal volumes and suicide attempts in MDD.	N=63 MDD patients (24 attempters).	MRI: volumetric analysis of the hippocampus.	Smaller hippocampal volumes in the attempter group.
Lee et al. [55].	The relationship between alterations in brain white matter (WM) and suicidal behavior in people with schizophrenia or schizophreniform disorder.	N=56 patients with schizophrenia/ schizophreniform disorder (15 attempters).	MRI: DTI.	Higher FA values in various parts of the fronto-temporolimbic circuits in attempters.
Baek et al. [56].	Tested the hypothesis that suicidal behavior is associated with heightened aversion to risk and loss, which might produce negative predictions about uncertain future events.	N=167; 92 MDD patients (45 attempters) vs. 75 healthy controls.	fMRI: Monetary risk and loss aversion tasks.	Disrupted neural responses to potential gains and losses in the subgenual ACC, insular cortex and left amygdala.
Sullivan et al. [57].	To determine the relationships between brain serotonin(1A) binding and suicidal behavior in vivo in major depressive disorder (MDD).	N=91 MDD patients (29 with a history of suicide attempt).	PET ([11C]-WAY-100635): 5-HT1A binding potential.	Greater serotonin 1A binding potential in the raphe nuclei predicted higher suicidal ideation and more lethal suicidal behavio

SUMMARY

The aim of this paper was to give a clear indication of the association between the EI level and NSSI/SB but also presenting the findings on the structural, functional and molecular levels. NSSI/SB associated with the different functioning of limbic system and CC [e.g. 25, 27, 30, 42, 54]. Reviewed data support the conclusion that dysregulation of emotional states under-

lies NSSI and SB in many individuals [21-30]. Many data indicate that individuals with high EI are less involved in NSSI and SB compared to those with low EI [16, 18]. Alterations in the brain structures are related to the modified neurotransmission of dopamine, serotonin and noradrenaline[40, 42, 45-47].

Our ability to predict NSSI and SB is still not much better than a chance, although, there has been a welcome focus on suicide prevention interventions (both at the public health and clinical level), many gaps in our knowledge remain. Better knowledge about the brain areas activated during emotional processes and NSSI and SB will allow a better understanding of these phenomena and facilitate the development of more effective pharmacologic interventions as well as preventive and therapeutic programs. Future research should seek to better understand NSSI and SB risk factors, taking into account gender differences. We assume that more detailed information about biological markers associated with NSSI and SB will allow to reduce this behaviour.

REFERENCES

1. O'Connor RC, Nock MK.The psychology of suicidal behaviour. Lancet Psychiatry.2014;1(1):73-85.
2. The World Health Report. 2001: Mental health: new understanding, new hope. World Health Organization. Genewa. 2001: 1-169.
3. Pużyński S.: Samobójstwa i zachowania psychiczne (zwłaszcza depresje). In: Psychiatria, (red. Bilikiewicz A.). Wydawnictwo Lekarskie PZWL. Warszawa. 2004. p. 428-433.
4. DSM-5 (Diagnostic and Statistical Manual of Mental Disorders).
5. Nock MK. (red.): The Oxford Handbook of Suicide and Self-Injury. New York: Oxford University: Press; 2014. p. 7—20.
6. Swannell SV, Martin GE, Page A, Hasking P, St John NJ. Prevalence of nonsuicidal self-injury in nonclinical samples: Systematic review, meta-analysis and meta-regression. Suicide Life—Threat. Behav. 2014; 44: 273–303.
7. Lewandowska A, Śmigielski J, Gmitrowicz A. Rodzinne czynniki ryzyka a samouszkodzenia u młodzieży szkolnej. Psychiatr. Psychol. Klin. 2004; 4: 224–233.
8. Warzocha D, Gmitrowicz A, Pawełczyk T. Związek samouszkodzeń wśród młodzieży hospitalizowanej psychiatrycznie z rodzajem zaburzeń psychicznych i wybranymi czynnikami środowiskowymi. Psychiatr. Pol. 2010.
9. Muehlenkamp JJ, Peat CM, Claes L, Smits D. Self-injury and disordered eating: Expressing emotion dysregulation through the body. Suicide Life-Threat. Behav. 2012; 42: 416–425.
10. Klonsky ED. The functions of deliberate self-injury: A review of the evidence. Clinical, Psychology Review. 2007; 27, 226–239.
11. Zetterqvist M. The DSM-5 diagnosis of nonsuicidal self-injury disorder: a review of the empirical literature. Child and Adolescent Psychiatry and Mental Health.20159:31.
12. Goleman D, Boyatzis R. Social intelligence and the biology of leadership. Harv Bus Rev. 2008; 86(9):74-81, 136.
13. Petrides K.V, Pita R, Kokkinaki F. The location of trait emotional intelligence in personality factor space. British Journal of Psychology. 2007; 98, 273–289.
14. Schutte NS, Malouff JM, Bhuller N: The Assessing Emotions Scale. In: Stough C, Saklofske DH, Parker JDA (Eds) Assessing Emotional Intelligence. Theory, Research, and Applications. 2009.
15. Palmer BR, Gilles E, Gignac GE, Manocha R, Stough KK. A psychometric evaluation of the Mayer–Salovey–Caruso Emotional Intelligence Test Version 2.0. Intelligence. 2005; 33(3):285-305.
16. Brackett M, Mayer J, Warner R: Emotional intelligence and its relation to everyday behaviors. Personality and Individual Differences. 2004; 36(6):1387–1402.
17. Thayer JF, Rossy LA, Ruiz-Paidal E, Johnsen BH. Gender differences in the relationship between emotional regulation and depressive symptoms. Cognitive Therapy and Research 2003; 27(3):349–364.
18. Downey L, Johnston P, Hansen K, Schembri R, Stough C, Tuckwell V, et al. The relationship between emotional intelligence and depression in a clinical sample. European Journal of Psychiatry. 2008; 22(2):93–98.
19. Bunney W, Fawcett J. Possibility of a biochemical test for suicidal potential. Arch Gen Psychiatry. 1965;;13: 232–239.
20. van Heeringen K. Stress–Diathesis Model of Suicidal Behavior. The Neurobiological Basis of Suicide. Frontiers in Neuroscience. 2012; 6.
21. Johnston JAY, Wang F, Liu J. Multimodal neuroimaging of frontolimbic structure and function associated with suicide attempts in adolescents and young adults with bipolar disorder. Am J Psychiatry. 2017; 174:667–675.
22. Balcioglu YH, Kose S. Neural substrates of suicide and suicidal behaviour: from a neuroimaging perspective. Psychiatry and Clinical Psychopharmacology. 2018.
23. Sudol K, Mann JJ. Biomarkers of suicide attempt behavior: towards a biological model of risk. Curr Psychiatry Rep. 2017; 19:1259.
24. Cyprien F, de Champfleur NM, Deverdun J, et al. Corpus callosum integrity is affected by mood disorders and also

by the suicide attempt history: a diffusion tensor imaging study. J Affect Disord. 2016; 206:115–124.

25. Wagner G, Koch K, Schachtzabel C, Schultz CC, Sauer H, Schlösser RG. Structural brain alterations in patients with major depressive disorder and high risk for suicide: evidence for a distinct neurobiological entity? Neuroimage. 2011; 15;54(2):1607-14.
26. Li W, Wu B, Batrachenko A, Bancroft-Wu V, Morey RA, Shashi V. Differential developmental trajectories of magnetic susceptibility in human brain gray and white matter over the lifespan. Human Brain Mapping. 2014.
27. Duarte DGG, Neves MCL, Albuquerque MR, Turecki G, Ding Y. Structural brain abnormalities in patients with type I bipolar disorder and suicidal behavior. Psychiatry Res Neuroimaging. 2017; 30;265:9-17.
28. Hercher C, Canetti L, Turecki. Anterior cingulate pyramidal neurons display altered dendritic branching in depressed suicides. J Psychiatr Res. 2010; 44:286–293.
29. Ernst C, Deleva V, Deng X. Alternative splicing,methylation state, and expression profile of tropomyosin-related kinase B in the frontal cortex of suicide completers. Arch Gen Psychiatry. 2009; 66:22–32.
30. Rizvi SJ, Iskric A, Calati R. Psychological and physical pain as predictors of suicide risk. Curr Opin Psychiatry. 2017; 30:159–167.
31. Killgore WDS, Weber M, Schwab ZJ.Gray matter correlates of Trait and Ability models of emotional intelligence. Neuroreport. 2012; 23, 551–5.
32. van Heeringen K, Wu GR, Vervaet M, et al. Decreased resting state metabolic activity in frontopolar and parietal brain regions is associated with suicide plans in depressed individuals. J Psychiatr Res. 2017; 84:243–248.
33. Quarto T, Blasi G, Maddalena C. Association between ability emotional intelligence and left insula during social judgment of facial emotions. PLoS One. 2016; 11.
34. Killgore WDS, Smith R, Olson OA, Weber M, Rauch SL Nickerson LD. Emotional intelligence is associated with connectivity within and between resting state networks. Social Cognitive and Affective Neuroscience. 2017; 1624–1636.
35. Reisch T, Seifritz E, Esposito F. An fMRI study on mental pain and suicidal behavior. J Affect Disord. 2010; 126:321–325.
36. Takeuchi H, Taki Y, Sassa Y. Regional gray matter density associated with emotional intelligence: evidence from voxel-based morphometry. Human Brain Mapping. 2011; 32, 1497–510.
37. Furczyk K, Schutová B, Michel TM, Thome J. The neurobiology of suicide – A Review of post-mortem studies. J Mol Psychiatry. 2013; 1(1): 2.
38. Arango V, Underwood MD, Mann J. Chapter 35 Serotonin brain circuits involved in major depression and suicide. Progress in rain Research. 2002.
39. Currier D, Mann JJ. Stress, Genes and the Biology of Suicidal Behavior. Psychiatr Clin North Am. 2008; 31(2): 247–269.
40. Zhang S, Chen J-M, Kuang L, Cao J, Zhang H, Ai M, et al. Association between abnormal default mode network activity and suicidality in depressed adolescents. BMC Psychiatry 2016; 16:337.
41. Fritz J, Dreisbach G. Conflicts as aversive signals: conflict priming increases negative judgments for neutral stimuli. Cogn Affect Behav Neurosci. 2013;13:311– 317. doi:10.3758/s13415-012-0147-1.
42. Li CS, SinhaR. Inhibitory Control and Emotional Stress Regulation: Neuroimaging Evidence for Frontal-Limbic Dysfunction in Psycho-stimulant Addiction. Neurosci Biobehav Rev. 2008; 32(3): 581–597.
43. Ling G, Lee I, Guimond G, Lutz O, NawazNTU. Individual variation in brain network topology is linked to emotional intelligence NeuroImage. 2019.
44. Domínguez-Baleón C, Gutiérrez-Mondragón LF, Campos-González AI, Rentería ME. Neuroimaging Studies of Suicidal Behavior and Non-suicidal Self-Injury in Psychiatric Patients: A Systematic Review. Front Psychiatry. 2018; 9:500.
45. Cox Lippard ET, Johnston JAY, Blumberg HP. Neurobiological Risk Factors for Suicide. American Journal of Preventive Medicine. 2014; 47:S152–62.
46. Wang L, Zhou C, Zhu D, et al. Serotonin-1A receptor alterations in depression: a meta-analysis of molecular imaging studies. BMC Psychiatry. 2016; 16:449.
47. Furczyk K, Schutova B, Michel TM, Thome J, Buttner A. The neurobiology of suicide – A Review of post-mortem studies. J Mol Psychiatry. 2013; 1:2.
48. Pandey GN, Dwivedi Y. Noradrenergic Function in Suicide. Archives of Suicide Research 2007;11:235–46.
49. Oliveira SP, Maia L, Coutinho J, Frank B, Soares JM i in. Empathy by default: Correlates in the brain at rest. Psicothema. 2018 Feb;30(1):97-103. doi: 10.7334/psicothema2016.366.49.
50. Groschwitz RC, Plener PL, Groen G, Bonenberger M, Abler B. Differential neural processing of social exclusion in adolescents with non-suicidal self-injury: An fMRI study. J Affect Disord. 2017; 15;221:47-55.
51. Pu S, Nakagome K, Yamada T, et al. Suicidal ideation is associated with reduced prefrontal activation during a verbal fluency task in patients with major depressive disorder. J Affect Disord. 2015;181:9–17.
52. Tsujii N, Mikawa W, Tsujimoto E, et al. Reduced left precentral regional responses in patients with major depressive disorder and history of suicide attempts. PLoS One. 2017; 12:e0175249.
53. Cao J, Chen X, Chen J, et al. Resting-state functional MRI of abnormal baseline brain activity in young depressed patients with and without suicidal behavior. J Affect Disord. 2016; 205:252–263.

54. Colle R, Chupin M, Cury C, et al. Depressed suicide attempters have smaller hippocampus than depressed patients without suicide attempts. J Psychiatr Res. 2015; 61:13–18.
55. Lee SJ, Kim B, Oh D, et al. White matter alterations associated with suicide in patients with schizophrenia or schizophreniform disorder. Psychiatry Res – Neuroimaging. 2016; 248:23–29.
56. Baek K, Kwon J, Chae J-H, et al. Heightened aversion to risk and loss in depressed patients with a suicide attempt history. Sci Rep. 2017; 7:53.
57. Sullivan GM, Oquendo MA, Milak M, et al. Positron emission tomography quantification of serotonin 1A receptor binding in suicide attempters with major depressive disorder. JAMA Psychiatry. 2015;72:169.

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

Dear Joanna Halicka-Masłowska,

I am pleased to inform you that your manuscript, entitled: Effects of variation in dopaminergic genes on the level of aggression and emotional intelligence in adolescents with conduct disorder, has been finally accepted for publication in our journal.

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

Type

Study

Keywords

genetics, dopamine, Conduct Disorder, neurotransmitter, externalizing behaviors

Abstract

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.

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 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 (χ^2 = 1.95, p = 0.162). However, there was a significant deviation from the HWE for the *DRD2* rs6277 genotypes (χ^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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References

1. Antisocial behaviour and conduct disorders in children and young people: recognition and management. NICE Clinical Guidelines, No. 158 London: National Institute for Health and Care Excellence (UK); 2013 Mar 27. ISBN-13: 978-1-4731-0055-8.

2. Dell'Agnello G, Maschietto D, Bravaccio C, Calamoneri F, Masi G, Curatolo P, et al. Atomoxetine hydrochloride in the treatment of children and adolescents with attention-defi cit/hyperactivity disorder and comorbid oppositional defi ant disorder: a placebo-controlled Italian study. European Neuropsychopharmacology. 2009;19:822–34.

3. Duica L, Antonescue, Pirlog M, Purnichi T, Szakac J, et al: Clinical and Biochemical Correlations of Aggression in Young Patients with Mental Disorders. Revista de Chimie Bucharest. 2018; 69(6) DOI: 10.37358/RC.18.6.6365.

4. Vorobyeva E, Fatima Hakunova F, Irina Skirtach I, Kovsh E: A review of current research on genetic factors associated with the functioning of the perceptual and emotional systems of the brain. 2019; DOI: 10.1051/shsconf/20197009009.

5. Vorobyeva E: Psychogenetics (Publ. h. of the SFU, Rostov-on-Don, 2014)

6. Zai CC, Ehtesham S, Choi E, Nowrouzi B, de Luca V, Stankovich L: Dopaminergic system genes in childhood aggression: Possible role for DRD2. The World Journal of Biological Psychiatry. 2012; 13(1):65–74.

7. Eisenberg D, MacKillop J, Modi M, Beauchemin J, Dang D, Lisman SA, et al: Examining impulsivity as an endophenotype using a behavioral approach: A DRD2 TaqI A and DRD4 48-bp VNTR association study. Behavior and Brain Functions. 2007; 3(e2), e2. Doi:10.1186/1744-9081-3-2.

8. Hirvonen M, Lumme V, Hirvonen J, Pesonen U, Någren K, Vahlberg T, Scheinin H, Hietala J: C957T polymorphism of the humandopamine D2 receptor gene predicts extrastriatal dopamine receptor availability in vivo. Progress in Neuro-Psychopharmacology and Biological Psychiatry. 2009; 33(4) 630-636.

9. Hirvonen M, Laakso A, Någren K, Rinne JO, Pohjalainen T, Hietala J: C957T polymorphism of the dopamine D2 receptor (DRD2) gene affects striatal DRD2 availability in vivo. Molecular Psychiatry. 2004; 9, 1060–1061. https://doi.org/10.1038/sj.mp.4001561.

10. Goleman D, Boyatzis R: Social intelligence and the biology of leadership. Harv Bus Rev. 2008; 9: 74-81.

11. Mayer JD, Salovey P, Caruso DR: Emotional intelligence: Theory, findings, and implications. Psychological Inquiry. 2004; 15: 197–215.

12. Klonsky ED: The functions of deliberate self-injury: A review of the evidence. Clin Psychol Rev. 2007; 27(2):226-39.

13. Nock MK, Joiner TE, Gordon KH, Lloyd-Richardson E, Prinstein MJ: Non-suicidal self-injury among adolescents: diagnostic correlates and relation to suicide attempts. Psychiatry Res. 2006; 144 (1):65–72.

14. Domínguez-García E, Fernández-Berrocal P: The Association Between Emotional Intelligence and Suicidal Behavior: A Systematic Review. Front Psychol. 2018; 9: 2380. doi:10.3389/fpsyg.2018.02380.

15. Gohier B, Senior C, Radua J, El-Hage W, Reichenberg A, Proitsi P, Surguladze

SA, Eur. Psych. 2014; 29, 4, 197-202. doi.org/10.1016/j.eurpsy.2013.03.003.

16. Jiang W, Shang S, Su Y: Genetic influences on insight problem solving: the role of catechol-O-methyltransferase (COMT) gene polymorphisms Front. in psych 2015; 6, 1569 doi.org/10.3389/fpsyg.2015.01569.

17. Thompson JM, Sonuga-Barke EJ, Morgan AR, Cornforth CM, Turic D, Ferguson LR, et al: The catechol- O- methyltransferase (COMT) Val158Met polymorphism moderates the effect of antenatal stress on childhood behavioural problems: longitudinal evidence across multiple ages. Developmental Medicine & Child Neurology. 2015; 54(2), 148-154. doi.org/10.1111/j.1469- 8749.2011.04129.x

18. George D, Mallery P: IBM SPSS Statistics 23 Step by Step: A Simple Guide and Reference. New York, NY: Routledge; 2016.

19. Adamowska S, Adamowski, T, Frydecka D, Kiejna A: Diagnostic validity of the Polish language version of the questionnaire MINI-KID (Mini International Neuropsychiatry Interview for Children and Adolescent, Comprehensive Psych. 2016; Apr; 66: 219-219. doi: 10.1016/j.comppsych.2015.09.013

20. Salamone JD, Correa M, Nunes EJ, Randall PA, Pardo M: The Behavioral Pharmacology of Effort-related Choice Behavior: Dopamine, Adenosine and Beyond. J Exp Anal Behav. 2012; Jan; 97(1): 125–146. doi: 10.1901/jeab.2012.97-125.

21. Matczak, A. (red.): Inteligencja emocjonalna – kierunki i metody badań. Psychologia, Edukacja i Społeczeństwo. 2007; 4.

22. Buss AH, Perry M: The aggression questionnaire. Journal of personality and social psychology. 1992; 63(3), p. 452.

23. Tucholska S: Pomiar agresji: Kwestionariusz Agresji A. Bussa i M. Perry'ego. Studia z Psychologii w Katolickim Uniwersytecie Lubelskim, 9, 1998; 369-378.

24. Bae Y: Test Review: Children's Depression Inventory 2 (CDI 2). Journal of Personality Assessment 90(3):280-5 2011; 32, 12, 2327. e7-2327. E19.

25. Vigneau F, Cormier S, The Factor Structure of the State-Trait Anxiety Inventory: An Alternative View. 2008. doi:10.1080/00223890701885027

26. Pitulaj A, Rajba B, Anrzejewska B, Kiejna A, Dominiak M: Psychometric validation of Corah's Dental Anxiety Scale in the Polish population. Advances in Clincal and Experimental Medicine. 2020. 29(1); 45-49. doi: 10.17219/acem/111818

27. Hohmann S, Zohsel K, Buchmann AF, Blomeyer D, Holz N, Boecker-Schlier R., et al: Interacting effect of MAOA genotype and maternal prenatal smoking on aggressive behavior in young adulthood. J. Neural Transm. 2016; 123, 885–894. doi: 10.1007/s00702-016-1582-x.

28. Hygen BW, Belsky J, Stenseng F, Lydersen S, Guze IC, Wichstrom L: Child exposure to serious life events, COMT, and aggression: testing differential susceptibility theory. Dev. Psychol. 2015; 51, 1098–1104. doi: 10.1037.

29. Zhang Y, Ming QS, Yi JY, Wang X, Chai QL, YaoS Q: Gene-gene-environment interactions of serotonin transporter, monoamine oxidase a and childhood maltreatment predict aggressive behavior in Chinese adolescents. Front. Behav. Neurosci. 2017; 11:17. doi: 10.3389/fnbeh.2017.00017

30. Wang M, Li H, Deater-Deckard K Zhang W: Interacting Effect of Catechol-O-Methyltransferase (COMT) and Monoamine Oxidase A (MAOA) Gene Polymorphisms, and Stressful Life Events on Aggressive Behavior in Chinese Male. 2018; doi: 10.3389/fpsyg.2018.01079

31.Tuvblad C, Narusyte J, Comasco E, Andershed H, Andershed AK, Colins OF, et al: Physical and verbal aggressive behavior and COMT genotype: sensitivity to the environment. Am. J. Med. Genet. B Neuropsychiatr. Genet. 2016; 171, 708–718. doi: 10.1002/ajmg.b.32430

32. Zalsman G, Huang Y, Oquendo MA, Brent DA: No Association of COMT Val158Met Polymorphism with Suicidal Behavior or CSF Monoamine Metabolites in Mood Disorders. Arch Suicide Res. 2008; 12(4): 327–335. doi:10.1080/13811110802324912.

33. Tovilla-Zárate C, Juárez-Rojop I, Ramón-Frias T, Villar-Soto M, Pool-García S, et al: No association between COMT val158met polymorphism and suicidal behavior: metaanalysis and new data. BMC Psychiatry. 2011; 11:151

34. Strous RD, Bark N, Pasia SS, Volavka J, Lachman HM: Analysis of a functional catechol-O-methyltransferase gene polymorphism in schizophrenia: evidence for association with aggressive and antisocial behavior. Psychiatry Res. 1997; 69, 71-77. http://dx.doi.org/10.1016/S0165-1781(96)03111-3

35. Soyka M, Zill P, Koller G, Samochowiec A, Grzywacz A, Preuss UW. Val158Met COMT polymorphism and risk of aggression in alcohol dependence. Addict. Biol. 2015; 20, 197-204. doi.org/10.1111/adb.12098. (b) Volavka, J.; Kennedy, J.L.;

36. Lachman HM, Nolan KA, Mohr P, Saito T, Volavka J: Association between catechol O-methyltransferase genotype and violence in schizophrenia and

schizoaffective disorder. Am. J. Psychiatry. 1998; 155, 835-837.

37. Fegert J, Findling RL, Fisman S, Greenhill LL, Huss, M, et al: International consensus statement on attention-deficit/hyperactivity disorder (ADHD) and disruptive behaviour disorders (DBDs): clinical implications and treatment practice suggestions. Eur. Neuropsychopharmacol., 2004; 14, 11-28. http://dx.doi.org/10.1016/S0924-977X(03)00045-2

38. Kotowicz K, Frydecka D, Gawęda Ł, Prochwicz K, Kłosowska J, et al: Effects of traumatic life events, cognitive biases and variation in dopaminergic genes on psychosis proneness. 2019; DOI: 10.1111/eip.12925

39. Beaver KM, Gibson CL, DeLisi M, Vaughn MG, Wright JP: The interaction between neighborhood disadvantage and genetic factors in the prediction of antisocial outcomes. Youth Violence and Juvenile Justice. 2012; 10(1), 25–40. doi:10.1177/ 1541204011422085.

40. Boardman JD, Menard S, Roettger ME, Knight KE, Boutwell BB, Smolen A. Genes in the dopaminergic system and delinquent behaviors across the life course the role of social controls and risks. Criminal Justice and Behavior. 2014; 41, 713–731. doi:10.1177/0093854813514227.

41. Nederhof E, Belsky J, Ormel J, Oldehinkel A. J: Effects of divorce on Dutch boys' and girls' externalizing behavior in gene 9 environment perspective: Diathesis stress or differential susceptibility in the Dutch tracking adolescents' individual lives survey study? Development and Psychopathology. 2012 24(03), 929–939. doi:10.1017/S0954579412000454

42. Weeland J, Overbeek G, de Castro BO, Matthys W: Underlying Mechanisms of Gene–Environment Interactions in Externalizing Behavior: A Systematic Review and Search for Theoretical Mechanisms: Clin Child Fam Psychol Rev. 2015; 18:413–442 DOI 10.1007/s10567-015-0196-4

43. Munafo M, Bowes L, Clark T, Flint J: Lack of association of the COMT (Val158/108 met) gene and schizophrenia: A meta-analysis of case–control studies. Molecular Psychiatry. 2005;10(8), 765–770. doi:10.1016/j.biopsych.2007.08.016.

44. Costas J, Sanjua´n J, Ramos-Rı´os R, Paz E, Agra S, Ivorra JL, et al: Heterozygosity at catechol-O-methyltransferase Val158Met and schizophrenia: New data and meta-analysis. Journal of Psychiatric Research. 2011; 45(1), 7–14. doi:10.1016/j.jpsychires.2010.04.021.

45. Mier D, Kirsch P, Meyer-Lindenberg A: Neural substrates of pleiotropic action of genetic variation in COMT: A meta-analysis. Molecular Psychiatry,.2009; 15(9), 918–

927. doi:10.1038/mp.2009.36.

46. Jokela M, Ferrie J, Kivima¨ki M: Childhood problem behaviors and death by midlife: the British National Child Development Study. Journal of the American Academy of Child and Adolescent Psychiatry. 2009; 48(1), 19–24. doi:10.1097/CHI. 0b013e31818b1c76.

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From Emotional Intelligence to Self-Injuries: A Path Analysis in Adolescents With Conduct Disorder

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Objective: Self-harm acts are highly prevalent among adolescents with conduct disorder. It has been shown that low level of emotional intelligence (EI) might be related to a higher risk of self-injuries. However, the exact mechanisms underlying this association are still unclear. The purpose of this study was to explore whether psychopathological symptoms and selected psychological processes mediate the association between EI and self-harm risk in adolescents with conduct disorders.

Method: Out of 162 adolescents with conduct disorder approached for participation, 136 individuals (aged 14.8 ± 1.2 years, 56.6% females) were enrolled and completed the questionnaires evaluating the level of EI, depression, anxiety, impulsiveness, empathy, venturesomeness, self-esteem, and disgust.

Results: Individuals with a lifetime history of self-injuries had significantly higher levels of depression, anxiety and impulsivity as well as significantly lower levels of EI and self-esteem. Higher levels of EI were associated with significantly higher levels of self-esteem, venturesomeness and empathy as well as significantly lower levels of depression, anxiety and impulsivity. Further analysis revealed that trait and state anxiety as well as self-esteem were complete mediators of the association between EI and self-harm risk.

Conclusions: Our findings indicate that anxiety and self-esteem might mediate the association between EI and a risk of self-injuries in adolescents with conduct disorder. However, a cross-sectional design of this study limits conclusions on the direction of causality. Longitudinal studies are needed to test validity of our model.

Keywords: emotional intelligence, self-harm, self-injuries, conduct disorder, adolescent

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INTRODUCTION

Non-suicidal self-injuries (NSSI) are increasingly being recognized as a highly prevalent aspect of psychopathology in young people. Recent epidemiological studies have shown that self-harm occurs in 17–18% of adolescents in the general population (1) and 40–80% of psychiatric patients (2). It has been estimated that even 92% of people consulted at the general hospital due to self-injuries might have one or more mental disorders (3, 4). Self-injuries are listed among the diagnostic criteria for borderline personality disorder [DSM-5; (5)]; however, they can appear in patients with other mental disorders. The Diagnostic and

Statistical Manual Version 5 (DSM-5) (5) has pointed out "non-suicidal self-injury disorder" (NSSID) as a problem to further study that extends current diagnostic boundaries (1). According to the International Society for the Study of Self-Injury, NSSI can be defined as the deliberate, self-inflicted damage to body tissue without suicidal intent and for purposes not sanctioned by society or culture (6). It has been reported that self-injuries might be associated with a number of negative outcomes that include repetitive self- injuries (7) and suicide (8).

It is now widely accepted that self-harm may occur in the context of various mental disorders and psychopathological symptoms. To date, several mental disorders that might develop in adolescence have been associated with self-harm risk, including attention deficit hyperactivity disorder, anxiety and depressive disorders, and conduct disorder (9). It has been noted that depression is a risk factor for self-harm, with affective disorders, such as bipolar disorder and depression being the most common primary diagnoses of patients who engage in self-harm acts (72%) and commit suicide (45%) (3, 10). Based on a meta-analysis, Fox et al. (11) found that the possibility of externalizing disorder symptoms is higher than the one of internalizing disorder symptoms among individuals who engage in non-suicidal self-injuries. The study by Nock et al. (12) estimated the prevalence of any externalizing disorder at 62.9%, and the presence of any internalizing disorder at 51.7% in adolescents who engage in self-harm the prevalence. In turn, prevalence rates of self-harm acts in adolescents with conduct disorder have been estimated at 15.5–62.5% (13). For instance, our group has recently reported that almost 53% of adolescent girls with conduct disorder have a history of self-injuries (14).

It has been shown that emotional intelligence (EI) can be associated with a risk of self-harm. Indeed, EI provides effective ways of balancing negative affect in adolescence and protecting from the aftermaths of self-harm. According to Goleman (15), it is a set of social skills that refer to the capacity to understand own emotions, manage and control them as well as the ability to empathize. EI may be perceived as a tool to encompass a personality dimension and also as the means to comprehend, process, and use affect-laden information gained by monitoring other's and one's own emotions. EI relies upon the ability to take suitable action to overcome the problem (16).

It has been reported that lower EI is associated with higher risk of internalizing disorders, including depression and anxiety, as well as substance use and less efficient coping (17). Petrides and Furnham (18) reported that in people with a higher level of EI, it serves as a protective factor for suicidal attempts and ideation (17). However, emerging evidence indicates that EI is not directly associated with suicide risk. The recent study by Quintana-Orts et al. (19) showed that depressive symptoms mediate the association between low level of EI and suicide risk among people who were bullied. This mediation appeared to be stronger among girls. At least theoretically, other processes might also mediate the association between EI and self-harm risk.

Individuals engaging in self-harm experience a variety of negative emotions. The most common categories of unpleasant emotional states declared by these individuals include feelings of guilt, anger, frustration, fear, sadness, shame, tension, anxiety and contempt (20). Apart from these emotions, there is evidence that disgust often occurs in this group of individuals, and in contrast to most other emotions, it does not tend to decrease after self-harming. It can be recognized as one of trait-dependent aspects of those who are prone to engage in self-harm acts (21). Another important aspect connected to self-harm is "impulsivity." It refers to actions that are risky, unduly hasty, and damaging (22). Higher levels of impulsivity have been reported in subjects with a history of self-harm (23). Moreover, higher levels of impulsivity and aggression have been associated with lower levels of EI (24). Finally, there is evidence that lower self-esteem might be related to higher risk of self-harm. In this regard, self-dislike in adolescents can be perceived as the way of punishing oneself and developing self-injurious behaviors (25). On the other hand, a significant positive relationship between the levels of EI and self-esteem has been demonstrated (26).

A majority of previous studies have investigated single correlates of psychological constructs associated with EI and self-harm. In light of findings mentioned above, we aimed to investigate as to whether psychopathology and selected psychological processes mediate the association between EI and self-harm risk in adolescents with conduct disorder. More specifically, we tested the hypothesis that depressive and anxiety symptoms, aggression, impulsivity, self-esteem as well as disgust mediate this association in adolescents with conduct disorder.

We decided to focus on adolescent patients due to the highest prevalence of NSSI among people at this age. A broad spectrum of negative emotions leading to aggressive behavior is typical for conduct disorder. One of the key functions of NSSI is to relieve negative feelings. Thus, we decided to assess this specific group of patients because of co-occurrence of NSSI and emotional dysregulation which play important roles as triggers of NSSI.

METHOD

Participants

Participants were enrolled among the students of the Youth Sociotherapy Centre (YSTC) No. 2 in Wroclaw, Poland. YSTCs in Poland have been designed by the Ministry of National Education to provide comprehensive pedagogical, educational and psychological support for children and adolescents with different problems or disorders (developmental, learning or social) who are at risk of social maladjustment. Adolescents, being admitted to the YSTC No 2 in Wrocław (Poland), mainly present with conduct disorder (mild or moderate severity of symptoms). Residents of YSTCs receive accommodation and attend school at these facilities. Students are recruited to YSTCs based on the opinion stating special education needs issued by professionals from the psychological and pedagogical counseling centers. According to the DSM-V criterion F of non-suicidal self-injury disorder (NSSID), participants were excluded if they had presented with intellectual disability, delirium, intoxication or withdrawal symptoms, psychotic disorder or autism spectrum disorders. Out of 162 individuals approached for participation (all individuals residing in the YSTC at the time of the study), 144 adolescents were enrolled (3 individuals and/or their legal guardians refused to participate and 15 individuals were

transferred to another institution). Due to a lack of necessary data to perform analyses, eight participants were excluded. The final sample included 136 adolescents (77 females and 59 males).

Procedures

The data were collected from September 2016 to August 2019 by a psychologist and a psychiatrist. Taking care of the comfort of the subjects, the study was divided into three parts, each lasting about an hour. During the first part, data on self-inflicted injuries were collected. A semi-structured questionnaire was administered to confirm a history of self-harm. This questionnaire recorded the information regarding the frequency of self-injuries and suicidal behaviors (suicidal thoughts and attempts) that had occurred at different time periods (lifetime as well as the preceding year, month, and week).

During the second part, all participants underwent psychiatric examination using the MINI-Kid interview. The MINI-Kid is a structured diagnostic tool, developed together by European and American psychiatrists and clinicians, for the DSM-IV and the ICD-10 criteria (27). This measure was used to establish a diagnosis of conduct disorder and comorbid mental disorders. Apart from the MINI-Kid, a diagnosis of CD was confirmed based on participants' psychiatric examination, medical records and psychological opinion. Furthermore, a diagnosis of potential comorbid mental disorders listed as exclusionary diagnoses of NSSID in the DSM-5 (criterion F), except for intellectual disability, was carried out. All students were assessed regarding intellectual functions before admission to YSTC by psychologists from the psychological and pedagogical counseling centers. After psychiatric examination, participants were divided into two groups – adolescents with a positive lifetime history of self-injuries and those who had never engaged in self-harm acts. We decided to focus our analyses on this categorization due to controversies around operationalization of the severity of self-injuries. For instance, although the NSSID has been developed in the DSM-5 as a new diagnostic category for further studies, there are studies showing insufficient validity of the NSSID frequency criterion (1, 14).

During the third part, emotional intelligence and concomitant psychopathology were assessed. Questionnaire data regarding self-esteem, impulsivity, depressive symptoms, anxiety and aggression levels and disgust sensitivity were collected using standardized self-reports. Self-reports were administered in the following order: (1) The Popular Emotional Intelligence Questionnaire (PEIQ); (2) The Buss-Perry Aggression Questionnaire (BPAQ); (3) The Children's Depression Inventory 2 (CDI2); (4) The State-Trait Anxiety Inventory (STAI); (5) The Rosenberg Self-Esteem Scale (SES); (6) The Eysenck's Impulsivity Inventory (IVE) and (7) The Questionnaire for the Assessment of Disgust Sensitivity (QADS).

The study was approved by the Bioethics Committee of Wroclaw Medical University, Poland. All participants and their statutory representatives gave written consent to all procedures carried out as the part of this study.

Self-Report Measures

The Popular Emotional Intelligence Questionnaire (PEIQ)

It 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 one's emotions), and understanding (understanding and awareness of own emotions) (28). The Cronbach's alpha for the PEIQ was estimated at 0.89 in our sample.

The Buss-Perry Aggression Questionnaire (BPAQ)

The BPAQ is a self-report measure of aggression in adolescents and adults. The BPAQ has 29 items, subdivided into four factors: physical aggression, verbal aggression, anger, and hostility (29). The Cronbach's alpha for the BPAQ total score in our sample was 0.80, for physical aggression 0.77, for verbal aggression 0.73, for anger 0.62 and for hostility 0.77.

The Children's Depression Inventory 2 (CDI2)

This measure 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 behavior efficacy, interpersonal problems, emotional problems and problems in functioning (30). The Cronbach's alpha for the CDI2 was 0.94 in our sample.

The State-Trait Anxiety Inventory (STAI)

This measure consists of two subscales measuring anxiety as a relatively stable personality component (state anxiety subscale) and the level of transient anxiety attributable to specific situations (trait anxiety subscale). Each subscale consists of 20 items which the subject answers by selecting one of four pre-categorized answers (31). The Cronbach's alpha for our sample was 0.94 for state anxiety and 0.99 for trait anxiety.

The Rosenberg Self-Esteem Scale (SES)

This tool consists of 10 diagnostic questions. Each question is based on a four-point Likert scale illustrating the level of agreement with the statements. The SES is a one-dimension tool which measures the level of overall self-esteem—approximately consistent disposition understood as conscious attitude—positive or negative toward the self (32). The Cronbach's alpha for the SES total score in our sample was 0.89.

The Eysenck's Impulsivity Inventory (IVE)

This measure consists of 63 diagnostic questions, using a two-point scale. The IVE consists of the following subscales: impulsivity, venturesomeness, and empathy (33). The Cronbach's alpha for each subscale was as follows: 0.75 (for impulsivity), 0.66 (for venturesomeness), and 0.65 (for empathy).

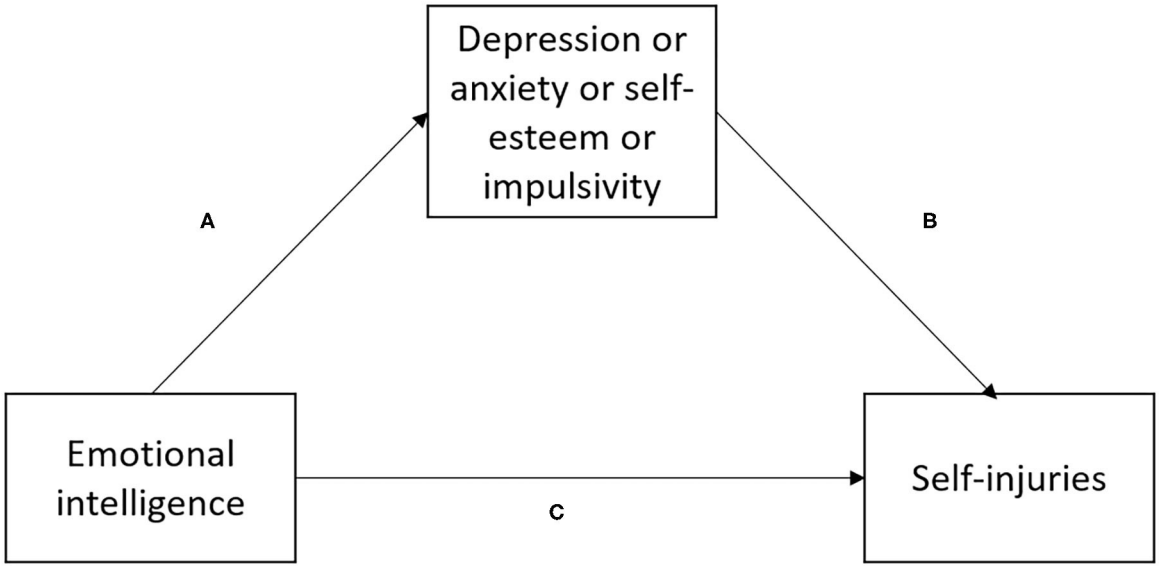

FIGURE 1 | A simple mediation model tested in this study. **(A)** effect of emotional intelligence on mediator, **(B)** effect of mediator on self-injuries, **(C)** direct effect of emotional intelligence on self-injuries.

The Questionnaire for the Assessment of Disgust Sensitivity (QADS)

This measure consists of 37 statements, in which the severity of disgust is assessed on a Likert five-point scale. Disgust sensitivity refers to individual personality traits and describes a predisposition to react to specific situations and materials with disgust. There are three subscales in the questionnaire: Core Disgust, Animal-Reminder, and Contamination-Interpersonal (34). Animal – Reminder disgust sensitivity addresses these aspects of human functioning which are shared with animals i.e., death, sex, a lack of hygiene, and damage to the body surface. The Cronbach's alpha for the QADS total score in our sample was 0.94.

Statistical analysis

The chi^2 test was applied to evaluate sex differences as well as differences in the rates of comorbid mood and anxiety disorders between participants with lifetime history of self-ham and those who did not engage in self-harm acts. Due to non-normal distribution, the Spearman rank correlation coefficients and the Mann-Whitney U test were used to analyze continuous variables. Results of bivariate tests were considered statistically significant if their p-value was <0.05. Simple mediation was analyzed using the PROCESS Macro Model 4 (35). Separate models for specific mediators were analyzed to avoid potential multicollinearity (**Figure 1**). The PEIQ score was inputted as an independent variable while a history of self-injuries was an outcome variable. One of main assumptions underlying mediation analysis is that the mediator must be associated with the independent variable and the outcome variable. Therefore, potential mediators were selected from the measures that were significantly associated with the PEIQ score and lifetime history of self-injuries. Age and sex were added as co-variates. The bootstrap calculation with 5,000 samples was applied to check direct and indirect effects. Mediation was considered significant if the 95% CI of indirect effect did not include zero. All analyses were conducted using the Statistical Package for Social Sciences, version 20 (SPSS Inc., Chicago, Illinois, USA).

RESULTS

The comparison of adolescents with a positive history of self-harm and those who had never engaged in self-injuries was provided in **Table 1**. Females were overrepresented in the subgroup of adolescents who reported engaging in self-injuries. Individuals with a positive history of self-harming presented with significantly lower levels of EI (PEIQ – total score and scores of acceptance, control and understanding) and self-esteem as well as significantly higher levels of depression, state and trait anxiety and impulsivity.

Table 2 shows bivariate correlations between EI and other measures tested in this study. There were significant negative associations between the level of EI (PEIQ – total score and scores of acceptance and control) and the scores of depressive symptoms and anxiety. Lower level of the PEIQ control subscale was related to significantly higher levels of core disgust. In turn, higher levels of the PEIQ acceptance subscale were associated with significantly higher levels of physical and verbal aggression, anger, hostility, venturesomeness as well as empathy. There were also significant negative correlations between the levels of impulsivity and the total PEIQ score as well as scores of two PEIQ subscales (control and understanding). Finally, higher levels of empathy (IVE) were significantly associated with the PEIQ total score and the scores of three PEIQ subscales (acceptance, control and empathy).

TABLE 1 | General characteristics of the sample.

	Self-harm (+) $n = 78$	**Self-harm (–), $n = 58$**	**Statistics**
Age, years	14.6 ± 1.1	15.1 ± 1.3	$U = 1{,}814.5$, $r = -0.16$, $p = 0.055$
Sex, F/M (%)	57/21 (73.1/26.9)	20/38 (34.5/65.5)	**$\chi^2 = 20.2$, $p < 0.001$**
Age of self-harm onset, years	10.7 ± 4.4	–	–
Lifetime number of self-harm acts	179.3 ± 362.5	–	–
The number of self-injuries in the preceding year	35.8 ± 77.6	–	–
Comorbid mood and/or anxiety disorder, *n* (%)	28 (35.9%)	12 (20.7)	$\chi^2 = 3.71$, $p = 0.054$
CDI2 – depression	20.4 ± 13.4	12.9 ± 11.0	**$U = 601.5$, $r = 0.29$, $p = 0.024$**
STAI – trait anxiety	47.0 ± 13.2	36.8 ± 9.5	**$U = 3{,}120.5$, $r = 0.46$, $p < 0.001$**
STAI – state anxiety	50.3 ± 12.7	37.7 ± 8.9	**$U = 3{,}328.5$, $r = 0.55$, $p < 0.001$**
PEIQ – EI (total score)	297.0 ± 29.2	309.8 ± 33.2	**$U = 1{,}585.0$, $r = -0.21$, $p = 0.022$**
PEIQ – acceptance	46.0 ± 9.0	50.1 ± 10.7	**$U = 1{,}563.0$, $r = -0.21$, $p = 0.017$**
PEIQ – empathy	65.1 ± 12.1	61.9 ± 10.4	$U = 2{,}400.0$, $r = 0.14$, $p = 0.123$
PEIQ – control	30.0 ± 6.8	33.8 ± 5.6	**$U = 1{,}301.5$, $r = -0.32$, $p < 0.001$**
PEIQ – understanding	27.3 ± 6.9	29.9 ± 4.8	**$U = 1{,}535.0$, $r = -0.22$, $p = 0.011$**
SES – self-esteem	23.8 ± 6.4	28.6 ± 5.4	**$U = 1{,}188.0$, $r = -0.35$, $p < 0.001$**
QADS - disgust (total score)	123.1 ± 31.5	118.4 ± 32.6	$U = 2{,}209.5$, $r = 0.06$, $p = 0.457$
QADS – core disgust	54.5 ± 15.2	54.3 ± 13.6	$U = 2{,}070.5$, $r = 0.02$, $p = 0.787$
QADS – animal reminder	25.6 ± 10.1	28.8 ± 9.6	$U = 1{,}634.0$, $r = -0.16$, $p = 0.069$
QADS – contamination-intepersonal	42.0 ± 12.8	39.7 ± 10.3	$U = 2{,}278.5$, $r = 0.11$, $p = 0.205$
BPAQ – physical agression	19.4 ± 7.1	20.9 ± 7.3	$U = 1{,}622.0$, r = −0.09, p = 0.295
BPAQ – verbal agression	13.8 ± 5.3	12.4 ± 4.7	$U = 2{,}082.0$, r = 0.12, p = 0.185
BPAQ – anger	18.4 ± 6.3	19.5 ± 6.1	$U = 1{,}648.5$, r = −0.09, p = 0.363
BPAQ – hostility	19.0 ± 8.1	17.8 ± 7.7	$U = 1{,}985.0$, r = 0.07, p = 0.410
IVE – adventuresomeness	8.9 ± 3.4	8.9 ± 3.2	$U = 1{,}931.0$, r = −0.01, p = 0.886
IVE – empathy	12.3 ± 3.3	11.4 ± 3.5	$U = 2{,}232.5$, r = 0.12, p = 0.179
IVE – impulsivity	10.7 ± 4.2	8.5 ± 3.8	**$U = 2612.5$, $r = 0.29$, p < 0.001**

Data expressed as mean ± SD or n (%).

Significant differences ($p < 0.05$) were marked with bold characters.

BPAQ, the Buss-Perry Aggression Questionnaire; CDI2, the Children's Depression Inventory 2; EI, emotional intelligence; IVE, the Eysenck's Impulsivity Inventory; PEIQ, the Popular Emotional Intelligence Questionnaire; QADS, the Questionnaire for the Assessment of Disgust Sensitivity; Self-harm (+), adolescents with a positive lifetime history of self-harm; Self-harm (–), adolescents with a negative lifetime history of self-harm; SES, the Rosenberg; Self-Esteem Scale; STAI, the State-Trait Anxiety Inventory.

Results of mediation analysis were presented in **Table 3**. There were significant direct effects of EI on the level of depression (PEIQ – total score, PEIQ – acceptance score and PEIQ - control score), state and trait anxiety (PEIQ – total score, PEIQ – acceptance score and PEIQ - control score), impulsivity (PEIQ – total score, PEIQ – understanding score and PEIQ - control score) as well self-esteem (PEIQ – total score, PEIQ – acceptance score and PEIQ - control score). Similarly, direct effects of self-esteem, state and trait anxiety on a history of self-injuries were also significant in these models. No significant effects of depressive symptoms as mediators were found. Self-esteem, state and trait anxiety mediated the association between EI and a history of self-injuries in the models with the PEIW total scores as well as the scores of two subscales, including control and acceptance (significant indirect effects). Direct effects of EI on a history of self-injuries were non-significant in these models. Therefore, these results indicate that self-esteem, trait and state were complete mediators.

DISCUSSION

Results of this study imply that individuals with conduct disorder and positive lifetime history of self-injuries present with significantly lower levels of EI and self-esteem together with higher levels of depressive and anxiety symptoms as well as impulsivity. Previous studies have also shown that on the one hand depression is associated with a higher risk of self-harm (36) as well as with lower level of EI on the other hand. A negative correlation between the level of EI or its components and depressive symptoms score has been replicated in early, middle and late adolescence (37–39). Regarding anxiety, similar results have been shown. In a cross-sectional study conducted in over 12,000 adolescents from 11 European countries, it was demonstrated that not only depression but also anxiety symptoms are significantly associated with self-harm risk (40). Furthermore, self-reported EI was negatively correlated with anxiety severity, social anxiety and the level of stress in adolescent samples (41, 42). Moreover, consistent findings have been

TABLE 2 | Correlations between the level of emotional intelligence and other measures recorded in this study.

	PEIQ – total score	PEIQ - acceptance	PEIQ – control	PEIQ – empathy	PEIQ - understanding
CDI2 - depression	$r = -0.350^{b}$	$r = -0.519^{c}$	$r = -0.369^{b}$	$r = 0.156$	$r = -0.130$
STAI - trait anxiety	$r = -0.248^{b}$	$r = -0.350^{c}$	$r = -0.297^{b}$	$r = 0.086$	$r = -0.077$
STAI - state anxiety	$r = -0.423^{c}$	$r = -0.477^{c}$	$r = -0.400^{c}$	$r = 0.103$	$r = -0.205$
SES - self-esteem	$r = 0.345^{c}$	$r = 0.382^{c}$	$r = 0.300^{b}$	$r = -0.068$	$r = 0.128$
QADS - disgust (total score)	$r = 0.071$	$r = 0.124$	$r = -0.142$	$r = 0.038$	$r = -0.076$
QADS – core disgust	$r = -0.048$	$r = -0.002$	$r = -0.268^{b}$	$r = 0.103$	$r = -0.160$
QADS – animal reminder	$r = 0.018$	$r = 0.021$	$r = -0.167$	$r = 0.014$	$r = -0.119$
QADS – contamination/interpersonal	$r = -0.023$	$r = 0.056$	$r = -0.126$	$r = 0.100$	$r = -0.172$
BPAQ - physical agression	$r = 0.162$	$r = 0.282^{b}$	$r = -0.077$	$r = 0.095$	$r = -0.143$
BPAQ - verbal agression	$r = 0.122$	$r = 0.191^{a}$	$r = -0.082$	$r = 0.171$	$r = -0.052$
BPAQ - anger	$r = 0.102$	$r = 0.230^{a}$	$r = 0.009$	$r = 0.081$	$r = 0.013$
BPAQ - hostility	$r = 0.077$	$r = 0.219^{a}$	$r = -0.062$	$r = 0.121$	$r = -0.075$
IVE - venturesomeness	$r = 0.177^{a}$	$r = 0.204^{a}$	$r = -0.034$	$r = 0.171$	$r = 0.051$
IVE - empathy	$r = 0.266^{b}$	$r = 0.192^{a}$	$r = -0.182^{a}$	$r = 0.518^{c}$	$r = -0.168$
IVE - impulsivity	$r = -0.184^{a}$	$r = -0.073$	$r = -0.437^{c}$	$r = 0.111$	$r = -0.328^{c}$

Spearman rank correlation coefficients were shown.
[a]$p < 0.05$.
[b]$p < 0.01$.
[c]$p < 0.001$.
BPAQ, the Buss-Perry Aggression Questionnaire; CDI2, the Children's Depression Inventory 2; EI, emotional intelligence; IVE, the Eysenck's Impulsivity Inventory; PEIQ, the Popular Emotional Intelligence Questionnaire; QADS, the Questionnaire for the Assessment of Disgust Sensitivity; SES, the Rosenberg Self-Esteem Scale; STAI, the State-Trait Anxiety Inventory.

reported with respect to impulsivity. Chamberlain et al. (4) found that self-harm dimensions are associated with impulse control disorders. A higher level of impulsiveness has previously been found in subjects with a history of self-injuring (12, 23). Finally, people with higher levels of EI are characterized by less frequent engagement in self-harm acts (20, 40), less frequent suicide attempts (43) and better overall social functioning (40). These observations appear to be consistent and independent of age (40), cultural context (12), nationality (44) or self-harm method (2). Therefore, high EI level might be perceived as a protective factor for self-harm.

One of the most important variables associated with self-harm risk is self-esteem. Greydanus and Shek (45) found that adolescents with low levels of self-esteem are at higher risk of engaging in self-injuries. A large number of previous reviews have consistently shown links between self-harm behaviors and low levels of self-respect among adolescents (7, 9). Hodgson (46) demonstrated that those who reported self-harm have also more problems with self-criticism and self-denigration. Moreover, they tend to present lower levels of self-esteem in contrast to adolescents who never engaged in self-harm acts. Increased self-dislike also advocates for the concept of self-harm as a way of punishing oneself and growing self-hatred of one's own body (25).

We also found that higher levels of EI are related to higher levels of self-esteem, venturesomeness and empathy, and at the same time with lower levels of depressive symptoms, anxiety and impulsivity in adolescents with conduct disorder. High levels of EI have been reported in correlation with a lower severity of symptoms related to mood and anxiety disorders (17, 27). High level of EI has been related to a subjective perception of well-being and satisfaction with life as well as higher levels of self-esteem (47). In some studies, lower self-esteem has been associated with a higher frequency of self-injuries (46). Importantly, self-esteem has also been found to mediate the association between childhood maltreatment and self-injuries in adolescents (47).

Similar results have been reported with respect to impulsiveness. It has been found that higher levels of impulsiveness are linked with a risk of self-harm. Moreover, there is evidence that self-injuries are driven by a wish to lessen emotional distress, and increased negative affect may precede episodes of self-harm (48). Higher level of impulsivity has been identified in individuals with self-harm history, because they worry less about the long-term consequences (e.g., discomfort, scarring, stigmatization). They can also be encouraged to self-injurious behavior by the promise of the immediate benefits (e.g., relief) (49, 50). Notably, we did not find any significant association between self-injuries and disgust sensitivity. Higher levels of core disgust were weakly associated with lower levels of control over one's emotions. It was previously demonstrated in college students that another type of disgust referred to as self-disgust plays a role as a mediator between depressive symptoms and NSSI (51). However, this category of disgust was not included in our study.

Our path analysis demonstrated that trait and state anxiety as well as impulsivity completely mediate the association between EI and a lifetime history of self-injuries in this group of adolescents (non-significant direct effects on a history of self-injuries with significant indirect effects). Previous studies have also shown that EI is not directly related to a risk of self-injuries or suicide. For instance, a recent study by Quintana-Orts

TABLE 3 | Results of mediation analysis.

Mediator	Effect	Predictor			
		PEIQ – total score	**PEIQ - acceptance**	**PEIQ - control**	**PEIQ – understanding**
CDI2 - depression	Effect of EI on mediator (a)	B = −0.104[a], SE = 0.047, 95% CI = −0.189 to −0.007	B = −0.523[c], SE = 0.137, 95% CI = −0.750 to −0.253	B = −0.582[a], SE = 0.202, 95% CI = −0.928 to −0.122	–
	Effect of mediator on self-injuries (b)	B = 0.001, SE = 0.002, 95% CI = −0.004 to 0.003	B = 0.004, SE = 0.007, 95% CI = −0.011 to 0.018	B = 0.006, SE = 0.006, 95% CI = −0.005 to 0.017	–
	Direct effect of EI on self-injuries (c)	B = −0.001, SE = 0.009, 95% CI = −0.018 to 0.016	B = 0.019, SE = 0.037, 95% CI = −0.047 to 0.085	B = −0.018, SE = 0.047, 95% CI = −0.110 to 0.075	–
	Indirect effect (ab)	B = −0.002, SE = 0.001, 95% CI = −0.005 to 0.001	B = −0.006, SE = 0.004, 95% CI = −0.014 to 0.002	B = −0.016, SE = 0.006, 95% CI = −0.029 to 0.004	–
STAI - state anxiety	Effect of EI on mediator (a)	B = −0.145[c], SE = 0.032, 95% CI = −0.208 to −0.082	B = −0.480[c], SE = 0.095, 95%CI = −0.659 to −0.288	B = −0.579[c], SE = 0.170, 95% CI = −0.919 to −0.259	–
	Effect of mediator on self-injuries (b)	B = 0.015[b], SE = 0.005, 95% CI = 0.006 to 0.025	B = 0.016[b], SE = 0.005, 95% CI = 0.007 to 0.026	B = 0.014[b], SE = 0.005, 95% CI = 0.005 to 0.024	–
	Direct effect of EI on self-injuries (c)	B = 0.001, SE = 0.001, 95% CI = −0.003 to 0.003	B = 0.001, SE = 0.005, 95% CI = −0.008 to 0.010	B = −0.049, SE = 0.034, 95% CI = −0.111 to 0.023	–
	Indirect effect (ab)	**B = −0.013, SE = 0.007, 95% CI = −0.032 to −0.004**	**B = −0.045, SE = 0.023, 95% CI = −0.104 to −0.015**	**B = −0.049, SE = 0.027, 95% CI = −0.118 to −0.012**	–
STAI - trait anxiety	Effect of EI on mediator (a)	B = −0.093[b], SE = 0.030, 95% CI = −0.149 to −0.031	B = −0.332[b], SE = 0.003, 95% CI = −0.531 to −0.118	B = −0.444[b], SE = 0.009, 95% CI = −0.780 to −0.130	–
	Effect of mediator on self-injuries (b)	B = 0.010[a], SE = 0.004, 95% CI = 0.002 to 0.019	B = 0.010[a], SE = 0.004, 95% CI = 0.002 to 0.019	B = 0.009[a], SE = 0.004, 95% CI = 0.002 to 0.018	–
	Direct effect of EI on self-injuries (c)	B = −0.002, SE = 0.001, 95% CI = −0.004 to 0.001	B = −0.003, SE = 0.004, 95% CI = −0.011 to 0.006	B = −0.012, SE = 0.006, 95% CI = −0.024 to 0.001	–
	Indirect effect (ab)	**B = −0.006, SE = 0.004, 95% CI = −0.016 to −0.001**	**B = −0.021, SE = 0.014, 95%CI = −0.057 to −0.003**	**B = −0.026, SE = 0.019, 95% CI = −0.076 to −0.002**	–
IVE - impulsivity	Effect of EI on mediator (a)	B = −0.025[a], SE = 0.011, 95% CI = −0.046 to −0.004	–	B = −0.218[b], SE = 0.001, 95% CI = −0.328 to −0.111	B = −0.219[c], SE = 0.058, 95% CI = −0.328 to −0.101
	Effect of mediator on self-injuries (b)	B = 0.019, SE = 0.011, 95% CI = −0.002 to 0.040	–	B = 0.012, SE = 0.012, 95% CI = −0.011 to 0.035	B = 0.016, SE = 0.011, 95% CI = −0.006 to 0.039
	Direct effect of EI on self-injuries (c)	B = −0.002, SE = 0.001, 95% CI = −0.004 to 0.001	–	B = −0.014, SE = 0.007, 95% CI = −0.029 to 0.001	B = −0.010, SE = 0.007, 95% CI = −0.023 to 0.003
	Indirect effect (ab)	B = −0.002, SE = 0.002, 95% CI = −0.007 to 0.001	–	B = −0.014, SE = 0.014, 95% CI = −0.044 – 0.016	B = −0.017, SE = 0.014, 95% CI = −0.050 to 0.007
SES - self-esteem	Effect of EI on mediator (a)	B = 0.058[a], SE = 0.022, 95% CI = 0.013 to 0.101	B = 0.206[b], SE = 0.064, 95% CI = 0.078 to 0.329	B = 0.231[a], SE = 0.099, 95% CI = 0.033 to 0.423	–
	Effect of mediator on self-injuries (b)	B = −0.018[b], SE = 0.007 95% CI = −0.031 to −0.004	B = −0.019[b], SE = 0.006, 95% CI = −0.032 to −0.005	B = −0.017[a], SE = 0.007, 95% CI = −0.030 to −0.003	–
	Direct effect of EI on self-injuries (c)	B = −0.001, SE = 0.001, 95% CI = −0.004 to 0.001	B = −0.002, SE = 0.005, 95% CI = −0.011 to 0.008	B = −0.011, SE = 0.007, 95% CI = −0.025 to 0.001	–
	Indirect effect (ab)	**B = −0.005, SE = 0.003, 95% CI = −0.013 to −0.001**	**B = −0.017, SE = 0.011, 95% CI = −0.046 to −0.003**	**B = −0.019, SE = 0.013, 95% CI = −0.049 to −0.003**	–

[a] $p < 0.05$.
[b] $p < 0.01$.
[c] $p < 0.001$.
CDI2, the Children's Depression Inventory 2; EI, emotional intelligence; IVE, the Eysenck's Impulsivity Inventory; SES, the Rosenberg Self-Esteem Scale; STAI, the State-Trait Anxiety Inventory. Significant indirect effects were marked with bold characters.

et al. (19) demonstrated that depressive symptoms mediate the association between suicide risk and EI among victims of bullying. This effect was moderated by sex, and appeared to be stronger in girls compared to boys. It is important to note that we did not find that depressive symptoms mediate the association between EI and a risk of self-injuries. However, to the best of our knowledge, our study is the first which was performed in adolescents with conduct disorder and we focused on a risk of self-injuries. Similarly, another study demonstrated that the level of psychological distress mediates the relationship between EI and suicide risk in adults (52). In turn, (53, 54) revealed that recognition and expression of emotions mediate the association between mindfulness and distress. The same study provided evidence that emotional recognition and expression as well as emotional management and control mediate the association between mindfulness and depression in adolescents.

There are some limitations of this research that need to be addressed. Our sample was rather small and a type II error cannot be ignored. Similarly, type I error should be taken into consideration due to a large number of estimated effects and a lack of correction for multiple testing. Therefore, our findings should be perceived as exploratory and requiring independent verification. Moreover, a cross-sectional study design does not support causal associations. Indeed, it has been demonstrated that the cross-sectional approaches can generate biased estimates of associations that are hypothesized to have a temporal ordering (55). Moreover, our findings cannot be generalized to other clinical populations with high prevalence of self-injuries. Although previous studies indicate that various psychological processes and low emotional abilities precede depressive symptoms, anxiety and self-harm behaviors, longitudinal studies are needed to investigate validity of the model tested in our study. Another limitation is that two subscales of the IVE (venturesomeness and empathy) had questionable internal consistency. Finally, investigating our hypotheses in a specific group of adolescents with conduct disorder limits generalization of findings to other populations.

In conclusion, main findings of our studies indicate that EI is not directly associated with a risk of self-injuries in adolescents with conduct disorder. Anxiety and self-esteem might serve as complete mediators of this association. However, longitudinal studies are required to confirm direction of causality. Results of our study hold a great promise for developing specific interventions that aim to target or prevent self-injurious behaviors. In light of our findings, one of potential approaches would be to target emotional competences of vulnerable individuals (43–50). Moreover, focusing on the development of self-esteem and reducing the level of anxiety seems to have an important role.

DATA AVAILABILITY STATEMENT

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

ETHICS STATEMENT

The studies involving human participants were reviewed and approved by Ethics Committee of Wroclaw Medical University, Poland. Written informed consent to participate in this study was provided by the participants' legal guardian/next of kin.

AUTHOR CONTRIBUTIONS

JH-M collected data and wrote the first draft of the manuscript. MS-B participated in data analysis and manuscript writing. JR participated in manuscript writing. AA participated in data collection and manuscript writing. BM performed data analysis and participated in manuscript writing. All authors contributed to the article and approved the submitted version.

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REFERENCES

1. Zetterqvist M. The DSM-5 diagnosis of nonsuicidal self-injury disorder: a review of the empirical literature. *Child Adolesc Psychiatry Ment Health.* (2015) 9:31. doi: 10.1186/s13034-015 0062-7
2. Selby EA, Bender TW, Gordon KH, Nock MK, Joiner TE. Non-suicidal self-injury (NSSI) disorder: a preliminary study. *Personal Disord.* (2012) 3:167–75. doi: 10.1037/a0024405
3. Haw C, Hawton K, Houston K, Townsend E. Psychiatric and personality disorders in deliberate self-harm patients. *Br J Psychiatry*. (2001) 178:48–54. doi: 10.1192/bjp.178.1.48
4. Chamberlain SR, Leppink EW, Redden SA, Grant JD. Associations between self-harm and distinct types of impulsivity. *Psychiatry Res*. (2017) 250:10–6. doi: 10.1016/j.psychres.2017.01.050
5. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. (2013). doi: 10.1176/appi.books.9780890425596
6. International Society for the Study of Self-Injury. *Self-injury*. (2020). Available online at: https://itriples.org/category/about-self-injury (accessed April 22, 2020).
7. Bergen H, Hawton K, Waters K, Cooper J, Kapur N. Epidemiology and trends in non-fatal self-harm in three centres in England: 2000–2007. *Br J Psychiatry*. (2010) 197:493–8. doi: 10.1192/bjp.bp.110.077651
8. Hawton K, Zahl D, Weatherall R. Suicide following deliberate self-harm: long-term follow-up of patients who presented to a general hospital. *Br J Psychiatry*. (2003) 182:537–42. doi: 10.1192/bjp.182.6.537
9. Waddell C, Offord DR, Shepherd CA, Hua JM, McEwan K. Child psychiatric epidemiology and Canadian public policy-making: the state of the science and the art of the possible. *Br J Psychiatry*. (2002) 47:825–32. doi: 10.1177/070674370204700903
10. NCISH. *Making Mental Health Care Safer: Annual Report and 20 Year Review*. Manchester: University of Manchester (2016).

11. Fox KR, Franklin JC, Ribeiro JD, Kleiman EM, Bentley KH, Nock MK. Meta analysis of risk factors for nonsuicidal self-injury. *Clin Psychol Rev*. (2015) 42:156–67. doi: 10.1016/j.cpr.2015.09.002
12. Nock MK, Joiner TE, Gordon KH, Lloyd-Richardson E, Prinstein MJ. Nonsuicidal self-injury among adolescents: diagnostic correlates and relation to suicide attempts. *Psychiatry Res*. (2006) 144:65–72. doi: 10.1016/j.psychres.2006.05.010
13. Ilomaki E, Rasanen P, Viilo K, Hakko H. Suicidal behavior among adolescents with conduct disorder–the role of alcohol dependence. *Psychiatry Res*. (2006) 150:305–11. doi: 10.1016/j.psychres.2006.02.011
14. Szewczuk-Bogusławska M, Kaczmarek-Fojtar M, Moustafa AA, Mahlberg J, Frydecka D, Oleszkowicz A, et al. Assessment of the frequency criterion for the diagnosis of non-suicidal self-injury disorder in female adolescents with conduct disorder. *Psychiatry Res*. (2018) 267:333–9. doi: 10.1016/j.psychres.2018.05.054
15. Goleman D, Boyatzis R. Social intelligence and the biology of leadership. *Harv Bus Rev*. (2008) 9:74–81.
16. Hargus E, Hawton K, Rodham K. Distinguishing between subgroups of adolescents who self-harm. *Suicide Life Threat Behav*. (2009) 39:518–37. doi: 10.1521/suli.2009.39.5.518
17. Thimm JC. Personality and early maladaptive schemas: a five-factor model perspective. *J Behav Ther Exp Psy*. (2010) 41:373–80. doi: 10.1016/j.jbtep.2010.03.009
18. Petrides KV, Funham A. Trait emotional intelligence: behavioural validation in two studies of emotion recognition and reactivity to mood induction. *Eur J Pers*. (2003) 17:39–57. doi: 10.1002/per.466
19. Quintana-Orts C, Rey L, Mérida-López S, Extremera N. What bridges the gap between emotional intelligence and suicide risk in victims of bullying? A moderated mediation study. *J Affect Disord*. (2019) 245:798–805. doi: 10.1016/j.jad.2018.11.030
20. Rowe AD, Fitness J. Understanding the role of negative emotions in adult learning and achievement: a social functional perspective. *Behav Sci*. (2018) 8:27. doi: 10.3390/bs8020027
21. Abdul-Hamid S, Denman C, Dudas RB. Self-relevant disgust and self-harm urges in patients with borderline personality disorder and depression: a pilot study with a newly designed psychological challenge. *PLoS ONE*. (2014) 9:e99696. doi: 10.1371/journal.pone.0099696
22. Klonsky ED. The functions of deliberate self-injury: a review of the evidence. *Clin Psychol Rev*. (2007) 27:226–39. doi: 10.1016/j.cpr.2006.08.002
23. Kleindienst N, Bohus M, Ludascher P, Limberger MF, Kuenkele K, Ebner-Priemer U. et al. Motives for nonsuicidal self-injury among women with borderline personality disorder. *J Nerv Ment Dis*. (2008) 196:230–6. doi: 10.1097/NMD.0b013e3181663026
24. Daruna JH, Barnes PA. A neurodevelopmental view of impulsivity. In: McCown WG, Johnson JL, Shure MB, editors. *The impulsive Client: Theory, Research, and Treatment. Am Psychop*. Washington (1993). p. 23–37
25. Janis IB, Nock MK. Are self-injurers impulsive?: results from two behavioral laboratory studies. *Psychiat Res*. (2009) 169:261–7. doi: 10.1016/j.psychres.2008.06.041
26. Coccaro EF, Zagaja C, Chen P, Jacobson K. Relationships between perceived emotional intelligence, aggression, and impulsivity in a population-based adult sample. *Psychiat Res*. (2016) 246:255–60. doi: 10.1016/j.psychres.2016.09.004
27. Sheehan DV, Lecrubier Y, Sheehan KH, Amorim P, Janavs J, Weiller E, et al. The mini-international neuropsychiatric interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *J Clin Psychiatry*. (1998) 59 (Suppl. 20):22–33.
28. Matczak A. Inteligencja emocjonalna – kierunki i metody badań. *Psychol Edukacja i Społeczeństwo*. (2007) 4:3–8.
29. Buss AH, Perry M. The aggression questionnaire. *J Pers Soc Psychol*. (1992) 63:452–9. doi: 10.1037/0022-3514.63.3.452
30. Bae, Y. (2011). Test review. children's depression inventory 2 (CDI 2). *J Pers Assess* 90:280–5. doi: 10.1177/0734282911426407
31. Vigneau F, Comier S. The factor structure of the state-trait anxiety inventory: an alternative view. *J Pers Assess*. (2008) 90:280–5. doi: 10.1080/00223890701885027
32. Rosenberg M. *Society and Adolescent Self-Image*. New York, NY: Princeton University Press (1989).
33. Eysenck SBG, Eysenck HJ. Impulsiveness and venturesomeness: their position in a dimensional system of personality description. *Psychol Rep*. (1978) 43:1247–55. doi: 10.2466/pr0.1978.43.3f.1247
34. Szewczuk-Bogusławska M, Słowińska A, Bak O, Oleszkowicz A, Kasibowska-Kuzniar K, Dudek K, et al. The study of the Polish version of the questionnaire for the assessment of disgust sensitivity (QADS). *Psychiatr Pol*. (2015) 49:145–57. doi: 10.12740/PP/34043
35. Hayes AF. *Introduction to Mediation, Moderation, and Conditional Process Analysis. A Regression-Based Approach*. New York, NY: The Guilford Press (2017).
36. Singha A, Ross J, Seminog O, Hawton K, Goldacre MJ. Risk of self-harm and suicide in people with specific psychiatric and physical disorders: comparisons between disorders using English national record linkage. *J R Soc Med*. (2014) 107:194–204. doi: 10.1177/0141076814522033
37. Bredemeier H, Bredemeier K, Thompson RJ, Boden MT. Worry, anhedonic depression, and emotional styles. *Cogn Ther Res*. (2012) 36:72–80. doi: 10.1007/s10608-010-9329-8
38. Salguero JM, Palomera R, Fernandez-Berrocal P. Perceived emotional intelligence as predictor of psychological adjustment in adolescents: a 1-year prospective study. *Eur J Psychol Educ*. (2012) 27:21–34. doi: 10.1007/s10212-011-0063-8
39. Stange JP, Alloy LB, Flynn M, Abramson LY. Negative inferential style, emotional clarity, and life stress: integrating vulnerabilities to depression in adolescence. *J Clin Child Adoles*. (2013) 42:508–18. doi: 10.1080/15374416.2012.743104
40. Brunner R, Kaess M, Parzer P, Fischer G, Carli V, Hoven CW, et al. Life-time prevalence and psychosocial correlates of adolescent direct self-injurious behavior: a comparative study of findings in 11 European countries. *J Child Psychol*. (2013) 55:337–48. doi: 10.1111/jcpp.12166
41. Fernandez-Berrocal P, Alcaide R, Extremera N, Pizarro D. The role of emotional intelligence in anxiety and depression among adolescents. *Indiv Psychol*. (2006) 4:16–27.
42. Cejudo J, Rodrigo-Ruiz D, López-Delgado ML, Losada L. Emotional intelligence and its relationship with levels of social anxiety and stress in adolescents. *Int Environt Re Pu*. (2018) 15:1073. doi: 10.3390/ijerph15061073
43. Domínguez-García E, Fernández-Berrocal P. The association between emotional intelligence and suicidal behavior: a systematic review. *Front Psychol*. (2018) 9:2380. doi: 10.3389/fpsyg.2018.02380
44. Moayedi F, Haji Alizadeh K, Khakrah M, Theshnizi SH. Emotional intelligence in suicide commiters. *Life Sci J*. (2014) 11:65–8. doi: 10.7537/marslsj1102s14.12
45. Greydanus DE, Shek D. Deliberate self-harm and suicide in adolescents. *J Med*. (2009) 58:144–51. doi: 10.2302/kjm.58.144
46. Hodgson S. Cutting through the silence: a sociological construction of self-injury. *Soc. Inq*. (2004) 74:162–79. doi: 10.1111/j.1475-682X.2004.00085.x
47. Muehlenkamp JJ, Swanson JD, Brausch AM. Self-objectification, risk taking, and self-harm in college women. *Psychol Women Quart*. (2005) 29:24–32. doi: 10.1111/j.1471-6402.2005.00164.x
48. Resurrección DM, Salguero JM, Ruiz-Aranda D. Emotional intelligence and psychological maladjustment in adolescence: a systematic review. *J Adolesc*. (2014) 37:461–72. doi: 10.1016/j.adolescence.2014.03.012
49. Wang S, Xu H, Zhang S, Wan Y, Fangbiao T. Mediating effects of self-esteem in the relationship between childhood maltreatment and non-suicidal self-injury among adolescents: the roles of sex and only-child status. *Soc Sci Med*. (2020) 249:112847.
50. da Fonseca NP, Silva CA, Araújo LM, Botti NC. Auto lesão sem intenção suicida entre adolescentes. *Arquivos Brasileiros Psicol*. (2018) 70:246–58.
51. Smith NB, Steele AM, Weitzman ML, Trueba AF, Meuret AE. Investigating the role of self-disgust in nonsuicidal self-injury. *Arch Suicide Res*. (2015) 19:60–74. doi: 10.1080/13811118.2013.850135
52. Allen KJD, Fox KR, Schatten HT, Hooly JM. Frequency of nonsuicidal self-injury is associated with impulsive decision-making during criticism. *Psychiatry Res*. (2019) 271:68–75. doi: 10.1016/j.psychres.2018.11.022

53. Foster B, Lomas J, Downey L, Stough C. Does emotional intelligence mediate the relation between mindfulness and anxiety and depression in adolescents? *Front Psychol*. (2018) 9:2463. doi: 10.3389/fpsyg.2018.02463
54. Hamza CA, Willoughby T, Heffer T. Impulsivity and nonsuicidal self-injury: a review and meta-analysis. *Clin Psychol Rev*. (2015) 38:13–24. doi: 10.1016/j.cpr.2015.02.010
55. Mérida-LópezS, Extremera N, Rey L. Understanding the links between self-report emotional intelligence and suicide risk: does psychological distress mediate this relationship across time and samples? *Front Psychiatry*. (2018) 9:184.doi: 10.3389/fpsyt.2018.00184

Conflict of Interest: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

6. Wyniki

Część I:

Halicka-Masłowska J, Szewczuk-Bogusławska M, Adamska A, Misiak B. (2020). Neurobiology of the association between non-suicidal self-injury, suicidal behavior and emotional intelligence: A review. Arch.Psychiatr.Psychother 2: 25–35 doi:10.12740/APP/117705

U osób podejmujących działania o charakterze autoagresji występują zmiany strukturalne, funkcjonalne i molekularne mózgu [27-29]. Większość zmian w strukturach mózgu osób dokonujących autoagresji jest związana układem dopaminergiczny i serotoninergicznym. Zmiany zauważalne są na poziomie układu limbicznego i występują w obszarach: kory mózgowej (głównie przedczołowej, oczodołowo-czołowej) [30], wyspy, hipokampu, istoty szarej, istoty białej, jądra soczewkowatego i ciała modzelowatego [31]. Zmiany strukturalne są obecne także na poziomie komórkowym i dotyczą obniżenia liczby komórek glejowych i rozgałęzień dendrytycznych [32]. Analiza struktur mózgu związanych z przetwarzaniem emocjonalnym i rejonami mózgu aktywowanymi u osób które popełniły samobójstwo i osób, które dokonywały samouszkodzeń wykazała wiele powiązań między tymi obszarami [33-35].

Część II:

Halicka-Masłowska J, Szewczuk-Bogusławska M, Pawlak-Adamska E, Adamska A, Misiak B. (2021). Effects of variation in dopaminergic genes on the level of aggression and emotional intelligence in adolescents with conduct disorder. Arch.Psychiatr.Psychother doi:10.12740/APP/128451

W wyniku przeprowadzonej analizy stwierdzono brak powiązań między polimorfizmami genów układu dopaminergicznego *COMT* rs6277 i *DRD2* rs4680 a poziomem agresji i EI. Nie stwierdzono istotnej statystycznie różnicy pomiędzy osobami z dodatnim wywiadem w kierunku samouszkodzeń a grupą adolescentów, która nigdy nie podejmowała samouszkodzeń, w odniesieniu do rozkładu genotypów *COMT* rs6277 (Val/Val: 22,3% i Val/ Met + Met/Met: 77,7% vs. Val/Val: 20,0% i Val/Met + Met/Met: 80,0%, p = 0,763) i *DRD2* rs4680 (TT: 18,8% i CC + CT: 81,2% vs. TT: 12,8% i CC + CT: 87,2%, p = 0,385). Aby potwierdzić te wyniki, potrzebne są badania podłużne na większej próbie.

Część III:
Halicka-Masłowska J, Szewczuk-Bogusławska M, Rymaszewska J, Adamska A, Misiak B. (2021). From emotional intelligence to self-injuries: A path analysis in adolescents with conduct disorder. Frontiers in Psychiatry doi:10.3389/fpsyt.2020.556278

Osoby, które w ciągu całego życia dokonały samouszkodzeń, miały znacznie wyższy poziom depresji, lęku i impulsywności, a także znacznie niższy poziom EI i samooceny. Ponadto, wyniki badania wskazują na to, że wysoki poziom EI wiąże się z wysoką samooceną oraz empatią i niskim poziomem depresji, lęku i impulsywności. Dalsze analizy wykazały, że lęk (jako stan i cecha) i samoocena są całkowitymi mediatorami związku między EI a ryzykiem samouszkodzeń. Wobec tego lęk i samoocena mogą pośredniczyć w związku między EI a ryzykiem samouszkodzeń u nastolatków z zaburzeniami zachowania.

7. Wnioski

Przeprowadzone badania pozwalają na wyciągnięcie następujących wniosków:

1. Uzyskanie wyniki nie potwierdzają związku pomiędzy polimorfizmem genów *COMT* i *DRD2* a poziomem agresji i EI.

2. Badania strukturalne wskazują na potencjalne istnienie wspólnego molekularnego podłoża zachowań autoagresywnych i przetwarzania emocjonalnego.

3. Lęk i samoocena mogą pośredniczyć w związku między EI a ryzykiem samouszkodzeń u nastolatków z zaburzeniami zachowania.

4. Planując przyszłe badania należałoby zbadać większą grupę, poddać analizie większą liczbę wariantów polimorficznych w genach układu dopaminergicznego. Należałoby rozważyć również wprowadzenie grupy kontrolnej.

8. Piśmiennictwo

1. Vorobyeva E, Hakunova F, Skirtach I, Kovsh E. (2019). A review of current research on genetic factors associated with the functioning of the perceptual and emotional systems of the brain. doi: 10.1051/shsconf/20197009009

2. Biel K. (2011). Rozwój agresji u dzieci w kierunku niedostosowania społecznego, [w:] Dziecko zagrożone wykluczeniem. Elementy diagnozy, działania profilaktyczne i pomocowe, red. K. Biel i J. Kusztal, Wydawnictwo WAM-WSF-P Ignatianum, Kraków

3. Jiménez TI, Estévez E. (2017). School aggression in adolescence: Examining the role of individual, family and school variables. Int J Clin Hlth Psyc. doi: 10.1016/j.ijchp.2017.07.002

4. World Health Organization: WHO. http://www.who.int/topics/suicide/en/

5. Kloves K, De Leo D. (2014). Regions with the highest suicide rates for children and adolescents – some observations. J. Child Adolesc. Behav 2.2.

6.Nock MK, Kessler RC. (2008). Prevelance of and risk factors for suicide aatempts versus suicide gestures: Analysis of the Nationaln Comobidity Suvey. Epidemiol. Rev. , 30{1), 133-154

7. Kokkevi V, Rotsika A, Arapaki C, Richardson. (2012). Adolescents' self-reported suicide attempts, self-harm thoughts and their correlates across 17 European countriesJ Child Psychol Psychiatry 53(4):381-9.doi: 10.1111/j.1469-7610.2011.02457.x

8. Zetterqvist M. (2015). The DSM-5 diagnosis of nonsuicidal self-injury disorder: a review of the 532 empirical literature. Child Adolesc Psychiatry Ment Health. 9:31.doi: 10.1186/s13034-015-0062-7 533 534

9. Nixon MK, Cloutier PF, Aggarwal S.J. (2002). Affect regulation and addictive aspects of repetitive self-injury in hospitalized adolescents Am Acad Child Adolesc Psychiatry 41(11):1333-41. doi: 10.1097/00004583-200211000-00015.

10. Nock, MK, Joiner TE, Gordon KH, Lloyd-Richardson E, Prinstein MJ. (2006). Nonsuicidal 581 self-injury among adolescents: diagnostic correlates and relation to suicide attempts. Psychiatry Res. 582 144:65–72. doi: 10.1016/j.psychres.2006.05.0

11. Goleman D, Boyatzis R. (2008). Social intelligence and the biology of leadership. Harv Bus Rev 594 2008; 9: 74-81.

12. Ilomaki E, Rasanen P, Viilo K, Hakko H. (2006).Suicidal behavior among adolescents with 585 conduct disorder–the role of alcohol dependence. Psychiatry Res. 150:305–11. doi: 586 10.1016/j.psychres.2006.02.011

13. 2. Dell'Agnello G, Maschietto D, Bravaccio C, Calamoneri F, Masi G, Curatolo P, et al. Atomoxetine hydrochloride in the treatment of children and adolescents with attention-defi cit/ hyperactivity disorder and comorbid oppositional defi ant disorder: a placebo-controlled Italian study. European Neuropsychopharmacology. 2009;19:822– 34.

14.Wang M, Li H, Deater-Deckard K, Zhang W. (2018). Interacting Effect of Catechol-*O*-Methyltransferase (*CO*MT) and Monoamine Oxidase A (MAOA) Gene Polymorphisms, and Stressful Life Events on Aggressive Behavior in Chinese Male Adolescents. Front Psychol 3;9:1079. doi: 10.3389/fpsyg.2018.01079

15. Hirata Y, Zai CC, Nowrouzi B, Beitchman JH, Kennedy JL. (2013). Study of the catechol-o-methyltransferase (COMT) gene with high aggression in children. Aggress Behav39(1):45-51.

16. Lachman H, Nolan K, Mohr P, Saito T, Volavka J. (1998). Association between catechol-Omethyltransferase genotype and violence in schizophrenia and schizoaffective disorder: Brief Reports. Am. J. Psychiatry155: 835–837.

17. Vorobyeva E, Hakunova F, Skirtach I, Kovsh E. (2019). A review of current research on genetic factors associated with the functioning of the perceptual and emotional systems of the brain. doi: 10.1051/shsconf/20197009009.

18. Zai CC, Ehtesham S, Choi E, Nowrouzi B, de Luca V, Stankovich L. (2012). Dopaminergic system genes in childhood aggression: Possible role for DRD2. The World Journal of Biological Psychiatry 13(1):65–74.

19. Adamowska S, Adamowski, T, Frydecka D, Kiejna A. (2016). Diagnostic validity of the Polish language version of the questionnaire MINI-Kid Mini International Neuropsychiatry Interview for Children and Adolescent, Comprehensive Psych. 66: 219-219. doi: 10.1016/j.comppsych.2015.09.013

20. Matczak A. (2007). Inteligencja emocjonalna – kierunki i metody badań. Psychologia, Edukacja i 650 Społeczeństwo, 4 651 652

21. Buss AH, Perry M. (1992). The Aggression Questionnaire. J Pers Soc Psychol 63(3), 452–741 459. doi.org/10.1037/0022-3514.63.3.452 742 33. Vigneau, F., Comier, S., (2008). The Factor Structure of the State-Trait Anxiety Inventory: An 743 Alternative View doi: 10.1080/00223890701885027

22. Bae Y. (2011). Test Review. Children's Depression Inventory 2 (CDI 2). J Pers Assess 90(3):280- 653 5. 32, 12, 2327. e7-2327. E19. 654

23. Vigneau F, Comier S, (2008). The Factor Structure of the State-Trait Anxiety Inventory: An 738 Alternative View J Pers Assess doi: 10.1080/00223890701885027 739 740

24. Rosenberg M. (1965). Society and adolescent self-image. New York: Princeton University Press. 746 Rosenberg, M. (1989). 747 748

25. Eysenck SBG, Eysenck HJ. (1978). Impulsiveness and venturesomeness: Their position in a 749 dimensional system of personality description. Psychol Rep 43(3), 1247-1255. 750 doi:10.2466/PR0.43.7.1247-1255 751 752

26. Szewczuk-Bogusławska M, Słowińska A, Bąk O, Oleszkowicz A, Kasibowska-Kuźniar K, 753 Dudek, K, et al. (2015). The study of the Polish version of the Questionnaire for the Assessment of 754 Disgust Sensitivity (QADS). Psychiatr. Pol 49(1):145–1571 doi: 10.12740/ PP/34043 755 36. Haye

27. Wagner G, Koch K, Schachtzabel C, Schultz CC, Sauer H, Schlösser RG. (2011). Structural brain alterations in patients with major depressive disorder and high risk for suicide: evidence for a distinct neurobiological entity? Neuroimage. 15;54(2):1607-14.

28. Li W, Wu B, Batrachenko A, Bancroft-Wu V, Morey RA, Shashi V. (2014). Differential developmental trajectories of magnetic susceptibility in human brain gray and white matter over the lifespan. Human Brain Mapping

29. Duarte DGG, Neves MCL, Albuquerque MR, Turecki G, Ding Y. (2017). Structural brain abnormalities in patients with type I bipolar disorder and suicidal behavior. Psychiatry Res Neuroimaging. 30;265:9-17

30. Killgore WDS, Weber M, Schwab ZJ. (2012). Gray matter correlates of Trait and Ability models of emotional intelligence. Neuroreport. 23, 551–5.

31. van Heeringen K, Wu GR, Vervaet M, et al. (2017). Decreased resting state metabolic activity in frontopolar and parietal brain regions is associated with suicide plans in depressed individuals. J Psychiatr Res. 84:243–248.

32. Hercher C, Canetti L, Turecki. (2010). Anterior cingulate pyramidal neurons display altered dendritic branching in depressed suicides. J Psychiatr Res. 44:286–293.

33. Domínguez-Baleón C, Gutiérrez-Mondragón LF, CamposGonzález AI, Rentería ME. (2018). Neuroimaging Studies of Suicidal Behavior and Non-suicidal Self-Injury in Psychiatric Patients: A Systematic Review. Front Psychiatry. 9:500.

34. Furczyk K, Schutova B, Michel TM, Thome J, Buttner A. (2013). The neurobiology of suicide – A Review of post-mortem studies. J Mol Psychiatry. 1:2.

35. Pandey GN, Dwivedi Y. (2007). Noradrenergic Function in Suicide. Archives of Suicide Research. 11:235–46.

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Podpis

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OŚWIADCZENIE

Oświadczam, że w pracy Halicka-Masłowska, J., Szewczuk-Bogusławska, M., Rymaszewska, J., Adamska, A., Misiak, B. (2021) From emotional intelligence to self-injuries: A path analysis in adolescents with conduct disorder. *Frontiers in Psychiatry* mój udział polegał na pisaniu tekstu manuskryptu, jego korekcie i ostatecznej akceptacji.

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Podpis

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Wrocław

Oświadczam, że w pracy:

Halicka-Masłowska, J., Szewczuk-Bogusławska, M., Pawlak-Adamska, E., Adamska, A., Misiak, B.k (2021) Effects of variation in dopaminergic genes on the level of aggression and emotional intelligence in adolescents with conduct disorder. *Arch.Psychiatr.Psychother* doi 10.12740/APP/128451

mój udział polegał na: analizie i interpretacji danych genetycznych , a także tworzeniu tekstu manuskryptu, jego korekcie oraz ostatecznej akceptacji.

Podpis

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Wykształcenie

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10.2016 - obecnie - Studia Doktoranckie; Katedra Psychiatrii; Uniwersytet Medyczny im. Piastów Śląskich we Wrocławiu

10.2016 - 06.2016 - Studia Podyplomowe; Przygotowanie Pedagogiczne; Wyższa Szkoła Gospodarki w Bydgoszczy

10.2013 - 06.2016 - Studia Licencjackie; Biologia; Uniwersytet Wrocławski

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Przebieg kariery zawodowej

08.2020 - obecnie - Psycholog i terapeuta w trakcie certyfikacji uzależnień; Ośrodek Leczenia, Terapii i Rehabilitacji Uzależnień dla Dzieci i Młodzieży we Wrocławiu

12.2018 - obecnie - Biegły Psycholog Sądowy; Sąd Okręgowy we Wrocławiu

09.2018 - obecnie - Psycholog; Szkoła Podstawowa im. Skarbów Dolnego Śląska w Malczycach

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09.2018 - 12.2018 - Psycholog; Przedszkole Integracyjne nr 35 we Wrocławiu

09.2017 - 09.2018 - Psycholog, kierownik świetlicy oraz nauczyciel biologii; Niepubliczna Szkoła Podstawowa Montessori we Wrocławiu

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Dorobek naukowy

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Halicka-Masłowska J, Szewczuk-Bogusławska M, Pawlak-Adamska E, Adamska A, Misiak B. (2021). Effects of variation in dopaminergic genes on the level of aggression and emotional intelligence in adolescents with conduct disorder. Arch.Psychiatr.Psychother doi 10.12740/APP/128451
Pkt. MNiSW/KBN: 20.000

Halicka-Masłowska J, Szewczuk-Bogusławska M, Adamska A, Misiak B. (2020). Neurobiology of the association between non-suicidal self-injury, suicidal behavior and emotional intelligence: A review. Arch.Psychiatr.Psychother 2: 25–35 doi: 10.12740/APP/117705
Pkt. MNiSW/KBN: 20.000

Makara-Studzińska M, Somasundaram SG, Halicka J, Madej A, Leszek J, Rehan M, Ashraf GM, Gavryushova LV, Nikolenko VN, Mikhaleva LM, Muresanu C, Kirkland CE, Avila-Rodriguez M, Aliev G. (2020). Suicide and Suicide Attempts in the Elderly Patients: An Epidemiological Analysis of Risk Factors and Prevention. Current Pharmaceutical Design doi: 10.2174/1381612826999201126202008
IF: 2.208; Pkt. MNiSW/KBN: 70.000

Halicka J, Kiejna A. (2018). Non-suicidal self-injury (NSSI) and suicidal: criteria differentiation Adv.Clin.Exp.Med. 27(2): 257-261. doi: 10.17219/acem/66353.
IF= 1.514; Pkt. MNiSW/KBN: 40.000

Halicka J. (2017). Ewersja kresomózgowia - ewolucyjna specyfika mózgu ryb promieniopłetwych (Actinopterygii). Kosmos 66(3):441-447.
Pkt. MNiSW/KBN: 12.000

Halicka J, Kiejna A. (2015). Differences between suicide and non-suicidal self-harm behaviours:a literary review. Arch.Psychiatr.Psychother 17(3):59-63 doi: 10.12740/APP/58953
Pkt. MNiSW/KBN: 20.000