

Proceeding Paper

# Aggression, Genetics, and Adverse Childhood Experiences in a University Sample <sup>†</sup>

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**Abstract:** The literature shows that aggression in adulthood is associated with adverse childhood experiences and genetics. This research aims to study the phenomenon of aggression in adulthood and analyse its relationship with genetics (dopamine and serotonin polymorphisms) and adverse childhood experiences. The sample was collected as part of the research project “Aggression and Genetics in a University Context” and consists of 93 individuals, 81 women, and 12 men. Participants completed a protocol consisting of a sociodemographic questionnaire, the Reactive–Proactive Aggression Questionnaire (RPQ), the Short Form of the Buss–Perry Aggression Questionnaire (BPAQ-SF), and the Childhood History Questionnaire (ACE). The most important results indicate that adverse childhood experiences are correlated with aggression in adulthood.

**Keywords:** aggression; adverse childhood experiences; serotonin; dopamine



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## 1. Introduction

Human aggression is a controversial concept, with different perspectives on its origins. Aggression is a maladaptive expression of human behaviour directed at others intending to harm, injure, or harass [1,2]. According to the General Model of Aggression (GAM), human aggression is influenced by knowledge structures with implications in various sociocognitive phenomena, including perception, interpretation, decision making, and behaviour [2]. Knowledge structures are formed from experience and influence perception at different levels, from simple object perception to complex interpersonal perception. GAM thus integrates a social, cognitive, and biological aspect of the development of aggression, divided into two main processes: distal and proximal processes. Distal processes underlie each episode of proximal processes and explain, through modifiers, how genetic and environmental factors can influence personality through changes in knowledge structures. The proximal processes describe how personal and situational factors influence aggressive thoughts, feelings of anger, and arousal levels, which in turn affect appraisal and decision-making processes, resulting in aggressive or non-aggressive behaviour [2].

The study of human aggression is very complex. It is crucial to understand how aggression is influenced at the genetic level [3]. Our behaviour can be influenced by two neurotransmitters, dopamine (DA) and serotonin (5-hydroxytryptamine or 5-HT) [4]. The proper functioning of neurotransmitters depends on the balance of [3], which, when changed at the genetic level, significantly dysregulate the DA and 5-HT system [5]. These changes at the genetic level are called genetic polymorphisms, which differ from genetic mutations in that, to be considered polymorphic, they must have a frequency of more than

1% in the chromosomes of the general population [6]. The changes at the genetic level may appear during childhood and may occur in individuals with a history of adverse experiences at this stage of life [7].

Adverse childhood experiences (ACEs) result from a series of adverse situations to which individuals are exposed that, over time, become a limitation to normative human development [8]. Changes at the genetic level can occur during, and especially in, childhood, and can occur in individuals who exhibit ACE at this stage of life [2], which in turn is considered a predictor of human aggression [8]. This study is relevant to psychology and genetics as it attempts to study a complex phenomenon like aggression by combining two areas to understand the human aggression. The present research aims to study aggression in adulthood, its relation with genetics, through the identification of serotonin and dopamine polymorphisms (5-HTTLPR and DAT1) and with adverse childhood experiences.

## 2. Materials and Methods

The sample consists of university students ( $M = 20.95$ ;  $DP = 2.98$ ). The data in this study were collected as part of the research project “Aggression and Genetics in a university context”. It is, therefore, a sample of volunteer university students who participated in the overall project.

The sample consists of 93 individuals, of whom 12 are male (12.9%) and 81 are female (87.1%). Data were collected online through a battery of psychological tests. The instruments used were as follows: (1) Buss–Perry Aggression Questionnaire—Short Form (BPAQ-SF), adapted for the Portuguese population [9]. It assesses four aggression subscales: physical aggression, verbal aggression, anger, and hostility. (2) The Reactive–Proactive Aggression Questionnaire (RPQ), adapted for the Portuguese population [10], is a self-report measure that distinguishes between reactive and proactive aggression. (3) Adverse Childhood Experiences (ACEs), Portuguese version [11], is a self-report questionnaire for adults that aims to assess the occurrence of adverse childhood experiences. It can be grouped into 2 major categories: (1) experiences against the individual and (2) the family environment. To calculate the total, it is necessary to create a variable: adversity total. First, it is necessary to recalculate all the categories, i.e., each category is transformed into a dichotomous value category, where the value ‘zero’ is given if the subject does not report this form of adversity, or the value ‘one’ if this adversity is reported [11].

The analysis of the genetic material revealed two polymorphisms, one of serotonin (5-HTTLPR) and the other of dopamine (DAT1). The 5-HTTLPR polymorphism has been divided into three different groups: heterozygous (ID); homozygous for insertion (II); and homozygous for deletion (DD). When we refer to homozygous for insertion or homozygous for deletion, it means that there has been an insertion or deletion of one or more nucleotides in a genetic sequence.

## 3. Results

Chi-squared tests were run to analyse the association between the polymorphisms (5-HTTLPR and DAT1) with the ACE subscales. A significant association was obtained between the 5-HTTLPR (serotonin polymorphism) and the emotional abuse scale ( $\chi^2 = 6.756$ ;  $p = 0.034$ ). Thus, heterozygotes (ID) (63.6%) are the ones with more indicators of the presence of a history of the emotional abuse scale, followed by deletion homozygotes (DD) (27.3%) and insertion homozygotes (II) (9.1%).

To verify the relationship between the aggression scales' scores (BPAQ-SF and RPQ) and ACE scores, Pearson correlation analysis was used, as shown in Table 1.

**Table 1.** Correlations between the indicators of aggression (BPAQ-SF and RPQ) and ACE (*n* = 93).

	Anger	Hostility	Total BPAQ	Reactive Aggression	Proactive Aggression	Total RPQ
Experiences Against Individual	0.22 *	0.45 **	0.37 **	0.30 **	0.25 *	0.30 **
Dysfunctional Family Environment	-	0.23 *	-	-	-	-
Total ACE	-	0.39 **	0.30 **	0.22 *	-	0.22 *

\* *p* < 0.05; \*\* *p* < 0.01.

#### 4. Discussion

It was possible to verify the existence of a significant relationship between the 5-HTTLPR polymorphism and emotional abuse, with the percentage of individuals who are heterozygous (ID) having a higher number of indicators of the presence of a history of emotional abuse compared to those who are homozygous. This polymorphism is mainly involved in inhibiting impulsive aggression [12,13]. This finding is supported in the literature, with several studies showing that adverse childhood experiences and the 5-HTTLPR are related [12].

No association was found between dopamine and the other variables. It was not possible to confirm, as reported in the literature, that the presence of the DAT1 polymorphism in individuals with ACEs presents higher levels of aggression [11]. Studies conducted in university students on the differences between men and women showed that the effects of polymorphisms on aggression were significant only for men [14].

It was also found that the greater the number of experiences against the individual (e.g., types of abuse and neglect), the greater the level of aggression in adulthood. This finding is consistent with what has been explained in the literature [15].

It was found that the dysfunctional environment was only correlated with hostility, i.e., the more significant the exposure to the dysfunctional environment (e.g., experiencing domestic violence), the higher the level of hostility in the future. A meta-analysis study shows that both anger and hostility are associated with exposure to an event such as domestic violence [16].

Finally, the fourth outcome found that adverse childhood experiences were not associated with anger or proactive aggression, so this finding does not support the literature [17].

When examining the results of the present research, it is possible to identify an association with the distal processes of the GAM. In this case, environmental modifiers assessed as adverse childhood experiences correlate with the forms and functions of aggression as explained by the model. Biological modifiers, assessed in this research as serotonin and dopamine, were only correlated with environmental modifiers and, according to the GAM, these act together to influence personality, thus modifying personal (and situational) factors and consequently influencing aggressive thoughts and feelings of anger, which in turn affect appraisal and decision-making processes, resulting in aggressive or non-aggressive behaviour [18].

The research carried out has several limitations. The first is related to the sample, as it is small. The second limitation relates to the lack of heterogeneity (there are more women than men), which is reflected in the low level of aggression, as men show higher levels of aggression.

#### 5. Conclusions

In summary, the results show that the significant relationship between serotonin polymorphism and adverse childhood experiences are correlated with aggression in adulthood. Through this result, we can verify the importance of how a childhood free of adverse experiences can be associated with a low risk of being aggressive in the future.

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**Data Availability Statement:** The data presented in this study are available on request from the corresponding author. The data are not publicly available due to privacy and ethical restrictions.

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