International Journal of Neurorehabilitation

Popova et al., Int J Neurorehabilitation Eng 2017, 4:1
DOI: 10.4172/2376-0281.1000242

Research Article Open Access

Interrelation between Anxiety Level and Aggressiveness

Liudmyla D Popova*, Irina M Vasylyeva and Oxana A Nakonechnaya

Kharkiv National Medical University, Ukraine

***Corresponding author:** Liudmyla D Popova, Kharkiv National Medical University, Ukraine, Tel: +380984285463; E-mail: popova_ld@ukr.net

Received date: January 19, 2017; **Accepted date:** January 27, 2017; **Published date:** February 04, 2017

Abstract

High anxiety is the base not only for depression development but also to impulsive aggression manifestation. In the previous study we revealed the differences in neurohumoral status in animals with submissive and dominant behavioral types. Hypothalamic pituitary adrenal axis hyperactivity, the increase in noradrenaline and the decrease in serotonin levels in limbicocortical regions were observed in submissive male rats (high anxiety). Due to these results, we studied an interrelation between anxiety level and aggressiveness index and its components. The research involved 138 participants: 121 young men aged 18 to 22 years and 17 male adolescents within the age range 15-16 years. They were asked to answer Buss-Durkee Hostility Inventory, Spielberger State-Trait Anxiety Inventory and Eysenck Personality Inventory. The anxiety level was assessed in points. The aggressiveness index, physical, verbal and indirect aggressions were estimated in a percentage of the maximum level. No correlation between the anxiety level and the aggressiveness index was found in whole group of young men. Whole group was separated into three subgroups depending on anxiety level: with high, moderate and low anxiety levels. Strong positive correlation between anxiety level and aggression index in men with high anxiety level and negative correlation between these two parameters in men with low anxiety level were revealed. In last subgroup the correlation was statistically insignificant. In men with moderate anxiety level no correlation between anxiety level and aggression index was observed. This interrelation may be taken into account in anxiety treatment and in the prevention of impulsive aggression manifestation. In whole group of male adolescents no correlation between anxiety and aggressiveness index was found. Obtained data indicate the necessity of participants division depending on anxiety level and using the closed age groups to study the mechanisms of aggression development.

Keywords: Aggression; Anxiety; Men

Introduction

High anxiety is the base not only for depression development but also to impulsive aggression manifestation. Two main forms of human excessive aggression are described: impulsive and controlled. The first form of aggression is observed in patients with depression or intermittent explosive disorder. The second type can be revealed in patients with personality disorders, but may also be found in individuals without noticeable emotional or social deficits [1]. Many studies are devoted to the research of the aggression formation mechanisms. Many researchers focus their attention on monoaminergic brain systems, as these systems have a great influence on the emotions and behavior [1,2]. There is much evidence of disorders of neurotransmission of noradrenalin and serotonin systems in depression and anxiety states [3]. The literature on the role of monoamines in the development of aggression is rather contradictory [4,5]. According to some researchers, serotonin regulates impulsivity, providing an inhibitory control of aggression [5]. According to others, in contrast, increased serotonin transmission contributes to aggressiveness [6]. Serotonin facilitates the conditioned anxiety but inhibits unconditioned fear [7-9]. Noradrenalin initiates unconditioned fear, corticotrophin- releasing factor release [7], lateral hypothalamus stimulation. It mediates positively motivated exploratory and approach activities [10]. Serotonin and noradrenalin exert a significant influence on early brain development through the regulation of neurogenesis, migration, differentiation, plasticity and other key morphogenetic processes [11]. Most of studies have been carried out on animals. In the previous research we revealed the differences in neurohumoral status in animals with submissive and dominant behavioral types. Hypothalamic pituitary adrenal axis hyperactivity, the increase in noradrenalin and the decrease in serotonin levels in limbico cortical regions are observed in submissive male rats (high anxiety) [12,13].

The investigations on humans are limited by performing of psychological tests, blood plasma analysis, Magnetic Resonance Imaging of brain. Because temperamental characteristics of person are the reflection of neurohumoral status, the study of relationships between them and aggression in men and women of different age groups and different populations is very important for understanding mechanisms of aggression development. Due to the above mentioned, objective of work was the investigation of interrelation between anxiety and aggressiveness index and its components (physical, verbal and indirect aggressions).

Methods

The study involved 138 participants: 121 young men aged 18 to 22 years and 17 male adolescents within the age range 15-16 years. They were asked to answer Buss-Durkee Hostility Inventory, Spielberger State-Trait Anxiety Inventory and Eysenck Personality Inventory. The anxiety level was assessed in points. The aggressiveness index, physical, verbal and indirect aggressions were estimated in a percentage of the maximum level. Eysenck Personality Inventory provides to estimate the sincerity of answers. If the answers have not been sincere, they were not taken into account.

Statistical analysis of the results was carried out by methods of nonparametric statistics using the package "Statistica 6.0" and correlation analysis according to Spearman.

Results

Initially we calculated the correlation coefficients between anxiety and aggression index and its components (physical, verbal and indirect aggressions) in young men and male adolescents without separation of participants according to anxiety level.

Some differences between young men and male adolescents were observed. No correlations between anxiety and aggression index, anxiety and physical aggression, anxiety and indirect aggression were found both in young men and adolescents (Table 1). But unlike young men, in adolescents the high statistically significant negative correlation between anxiety and verbal aggression was observed.

	Young men	Male adolescents
	Anxiety	
Physical aggression	+0.121	-0.002
Verbal aggression	-0.049	-0.620*
Indirect aggression	-0.091	+0.164
Aggression index	-0.003	-0,021

Table 1: Correlation coefficients between anxiety and aggression index and its components (physical, verbal and indirect aggressions) in young men and male adolescents, *Correlation coefficient is statistically significant, P<0.05.

In group of young men the moderate statistically significant correlations between physical and verbal, physical and indirect, verbal and indirect aggressions were revealed (Table 2). In adolescents no correlations between these components were found.

	Young men		Male adolescents	
	Verbal aggression	Indirect aggression	Verbal aggression	Indirect aggression
Physical aggression	+0.438*	+0.337*	0.313	0.056
Verbal aggression	-	+0.439*	-	0.291

Table 2: Correlation coefficients between physical, verbal and indirect aggressions in young men and male adolescents, *Correlation coefficient is statistically significant, P<0.05

After combining groups of young men and adolescents the correlations were the same, as in young men. This indicates the importance of using groups with limited age ranges to obtain valid results.

We divided whole group of both young men and adolescent into three subgroups depending on anxiety level: with high, moderate and low anxiety levels. It should be noted, that the most of men had moderate anxiety level (69% in young men group and 71.4% in adolescent group). Persons with high anxiety level constituted 17% in group of young men and 14.3% in group of adolescents. A few men had low anxiety level (14% in young men group and 14.3% in adolescent group).

We revealed the existence of high positive correlation between anxiety level and aggression index, anxiety level and indirect aggression (Table 3), between physical and verbal, physical and indirect, verbal and indirect aggressions in young men with high anxiety level (Table 4). The negative but statistically insignificant correlations between anxiety level and aggression index, between anxiety and physical, anxiety and verbal aggressions, high statistically significant positive correlation between verbal and indirect aggressions were found in young men with low anxiety level.

	Young men with high anxiety	Young men with moderate anxiety	Young men with low anxiety
	Anxiety		
Physical aggression	0.404	0.07	-0.362
Verbal aggression	0.432	0.076	-0.449
Indirect aggression	+0.597*	-0.024	-0.112
Aggression index	+0.600*	0.041	-0.378

Table 3: Correlation coefficients between anxiety and aggression index and its components (physical, verbal and indirect aggressions) in young men with high, moderate and low anxiety, *Correlation coefficient is statistically significant, P<0.05.

In young men with moderate anxiety level no correlations between anxiety and aggression index, anxiety and physical aggression, anxiety and verbal aggression, anxiety and indirect aggression were observed. But in this subgroup of young men the moderate statistically significant correlations between physical and verbal, physical and indirect, verbal and indirect aggressions were found (Table 4).

	Verbal aggression	Indirect aggression
Young men with high anxiety		
Physical aggression	0.397	+0.757*
Verbal aggression	-	+0.719*
Young men with moderate anxiety		
Physical aggression	+0.483*	+0.286*
Verbal aggression	-	+0.335*
Young men with low anxiety		
Physical aggression	0.341	0.351
Verbal aggression	-	+0.606*

Table 4: Correlation coefficients between physical, verbal and indirect aggressions in young men with high, moderate and low anxiety, *Correlation coefficient is statistically significant, P<0.05.

We could not perform correlation analysis in subgroups of adolescents with high and low anxiety, because only 2 persons with high and 2 persons with low anxiety level were found in adolescent

group. We combined respective subgroups of young men and adolescents to increase the quantity of persons with high and low anxiety. In this case the same interrelations between anxiety and aggression index, between anxiety and physical, verbal, indirect aggressions were observed. But interrelations between physical, verbal, indirect aggressions were changed.

Discussion

A fear and an anxiety act as signals of danger, threat, or motivational conflict and trigger appropriate adaptive responses [14]. Some authors don't distinguish terms "fear" and "anxiety". According to others, the object of fear is real and anxiety origins are unclear [14]. However, when fear is disproportionally intensive, chronic or irreversible, or not associated with any genuine risk, it may be symptomatic of a debilitating anxious state: for example, social phobia, panic attacks or generalized anxiety disorder [15]. Anxiety- and depression-related disorders are often characterized by impaired social behaviors including excessive aggression and violence [1]. In patients with depression the impulsive aggression is manifested [16]. According to results obtained by Keck et al. [17], the rats with high and low anxiety demonstrate a high form of aggression.

In our study no correlation between anxiety and aggression index was shown in total group of young men. After a division of the participants into subgroups with high, moderate and low levels of anxiety the opposite interrelations between anxiety and aggression were observed in persons with high and low anxiety levels. In individuals with high anxiety level it was strong positive. In individuals with low anxiety level it was negative, although it was not statistically significant.

Due to these opposite tendencies and the existence of no correlation between anxiety and aggression in participants with moderate anxiety level, the study of neurohumoral status features in total group without division into subgroups depending on anxiety level is incorrect. Features of neurohumoral status of persons with high and low anxiety levels may not be revealed in total group of participants.

The results of our study indicate that it is very important to take into account an anxiety level of participants in research of aggression development mechanisms.

Serotonin and noradrenalin are the most important mediators involved in the anxiety development. Rats with low anxiety behavior have the increased serotoninergic neurotransmission as compared with high anxiety behavior rats [17]. These results are consistent with our previous data regarding the content of serotonin in submissive and dominant rats. In our study submissive animals correspond to rats with high anxiety; aggressive (dominant) animals correspond to rats with low anxiety [13]. In the first group the serotonin content in hippocampus and frontal cortex is lowered, in the second group it is elevated. Noradrenalin content in both brain regions is higher in submissive animals compared to dominant ones [12,13]. As serotonin facilitates the conditioned anxiety [7-9] and provides inhibitory control of aggression [18], in individuals with low anxiety the interrelation between anxiety and aggression is negative. As noradrenalin initiates unconditioned fear, corticotrophin- releasing factor release [7], the increased noradrenalin and decreased serotonin levels provides the positive correlation between anxiety and aggression in the individuals with high anxiety.

Hypothalamic pituitary adrenal axis is also involved in the development of depression and anxiety [19]. Although some authors suggest that corticotrophin-releasing factor is a result of fear/anxiety, rather than a cause, that is corticotrophin- releasing factor could be responsible for stress responses to cope with dangerous situations but not for fear/anxiety itself [20], most of studies support the participation of glucocorticoids in anxiety development. Prolonged, excessive glucocorticoid exposure has potent effects on the architecture of neuronal connectivity in diverse regions of the brain [21]. Chronic stress reduces hippocampal neurogenesis [22]. It should be noted that corticosterone concentrations show U-shaped dose-response interrelation with granule cell death speed: both adrenalectomy and elevated corticosterone concentrations lead to cell loss [23]. The hippocampus is critical in mediation of glucocorticoid-dependent negative feedback and controls the response duration to stress [24]. The strong relationship between depression and the hippocampus atrophy is found [25]. Different animal models of depression are associated with reducing hippocampal neurogenesis [26,27]. An excess of circulating corticosteroids can shift the metabolism of tryptophan from serotonin to kynurenine production by increasing activity of liver tryptophan-pyrrolase (tryptophan-2,3-dioxygenase) [28]. Rate-limiting enzyme of kynurenine pathway in brain is indoleamine 2,3-dioxygenase, activated by proinflammatory cytokines [28]. Several findings suggest that microglia, activated by repeated stress, are involved in emotional and cognitive changes as a source of inflammation-related molecules [29]. Not only conventional neurotransmitters (monoamines, gamma-amino-butyric acid and glutamate), but also many other modulators, including adenosine, cannabinoids, numerous neuropeptides, hormones, neurotrophins, cytokines and several cellular mediators, are involved in the induction and inhibition of anxious states [15]. It is believed that the increased expression of the genes producing inflammatory cytokines would determine a genetic predisposition to develop depression by up-regulating the indoleamine 2,3-dioxygenase activity, while environmental stressors would activate tryptophan-2,3-dioxygenase via hormonal activation [29]. Activation of kynurenine pathway of tryptophan metabolism is accompanied by reducing serotonin content and kynurenine pathway metabolites accumulation. Last ones have many neurotropic effects. Both consequences of kynurenine pathway activation can play a role in the development of anxiety, psychotic symptoms and cognitive impairment associated with depression [29]. According to previous our data, hypothalamic pituitary adrenal axis hyperactivity is observed in submissive rats [13].

In our study we revealed the existence of valid moderate relationship between physical, verbal and indirect aggressions in total group of young men and young men with moderate anxiety, no correlations between these components in adolescents. In young men with high and low anxiety correlations between these parameters were changed. In young men with high anxiety correlations between indirect aggression and physical or verbal aggressions were stronger, but between physical and verbal aggression it was insignificant. In young men with low anxiety only relationship between verbal and indirect aggressions was significant. These changes may be due to imbalance of neuromediators in aggression-controlling brain centers.

Absence of correlations between these components, between anxiety and aggression index in male adolescents may be explained by the brain maturation process. Although tubulinogenesis, axonogenesis, and synaptogenesis may be accomplished during prenatal and immediate postnatal life, myelinogenesis remains active during adolescence [30]. Glutamatergic neurotransmission predominates,

whereas gamma-aminobutyric acid transmission is in formation process. The insufficiency of inhibitory processes may be responsible for impulsive behavior in adolescence [30].

So the results of our study show the existence of strong positive correlation between anxiety and aggression index in persons with high anxiety and tendency to negative correlation between these parameters in individuals with low anxiety. This interrelation may be taken into account in anxiety treatment and in the prevention of impulsive aggression manifestation.

Obtained data indicate the necessity of participants division depending on anxiety level and using groups with limited age ranges to study the mechanisms of aggression development.

Conclusion

1. In individuals with high anxiety the aggression index positively correlates with anxiety.
2. In persons with low anxiety the tendency to negative correlation between anxiety and aggression index exists.
3. The absence of correlation between aggression index and anxiety in total group of participants indicates the necessity of participants division depending on anxiety level to correctly interpret the obtained results.
4. The features of interrelations between anxiety and aggression index and its components in adolescents and a disappearance of these features after combining of young men and adolescents groups show the necessity of using groups with limited age ranges to obtain valid results.

References

1. Neumann ID, Veenema AH, Beiderbeck DI (2010) Aggression and anxiety: social context and neurobiological links. Front Behav Neurosci 4: 12.
2. van der Vegt BJ, Lieuwes N, Cremers TI, de Boer SF, Koolhaas JM (2003) Cerebrospinal fluid monoamine and metabolite concentrations and aggression in rats. Horm Behav 44: 199-208.
3. Ressler KJ, Nemeroff CB (2000) Role of serotonergic and noradrenergic systems in the pathophysiology of depression and anxiety disorders. Depress Anxiety 12: 1: 2-19.
4. Cambon K, Dos-Santos CR, Groc L, Carbon A, Weissmann D, et al. (2010) Aggressive behavior during social interaction in mice is controlled by the modulation of tyrosine hydroxylase expression in the prefrontal cortex. Neuroscience 171: 840-851.
5. Witte AV, Flöel A, Stein P, Savli M, Mien LK, et al. (2009) Aggression is related to frontal serotonin-1A receptor distribution as revealed by PET in healthy subjects. Hum Brain Mapp 30: 2558-2570.
6. de Boer SF, Koolhaas JM (2005) 5-HT1A and 5-HT1B receptor agonists and aggression: A pharmacological challenge of the serotonin deficiency hypothesis. Eur J Pharmacol 526: 125-139.
7. Graeff FG, Guimarães FS, De Andrade TG, Deakin JF (1996) Role of 5-HT in stress, anxiety and depression. Pharmacol Biochem Behav 54: 129-141.
8. Nunes-de-Souza V, Nunes-de-Souza R, Rodgers RJ, Canto-de-Souza A (2011) Blockade of 5-HT(2) receptors in the periaqueductal grey matter (PAG) abolishes the anxiolytic-like effect of 5-HT(1A) receptor antagonism in the median raphe nucleus in mice. Behav Brain Res 225: 547-553.
9. Graeff FG (2004) Serotonin, the periaqueductal gray and panic. Neurosci Biobehav Rev 28: 239-259.
10. Stone EA, Quartermain D, Lin Y, Lehmann ML (2007) Central alpha1-adrenergic system in behavioral activity and depression. Biochem Pharmacol 73: 1063-1075.
11. Thompson BL, Stanwood GD (2009) Pleiotropic effects of neurotransmission during development: Modulators of modularity. J Autism Dev Disord 39: 260-268.
12. Popova LD, Vasylyeva IM (2014) Roles of central monoaminergic systems in the formation of different types of aggressiveness in rats. Neurophysiology 46: 263-266.
13. Popova L, Vasylyeva I (2014) Neurohumoral status and aggression. Lap Lambert Academic Publishing, Germany.
14. Steimer T (2002) The biology of fear- and anxiety-related behaviors. Dialogues Clin Neurosci 4: 231-249.
15. Millan MJ (2003) The neurobiology and control of anxious states. Prog Neurobiol 70: 83-244.
16. Barden N (2004) Implication of the hypothalamic-pituitary-adrenal axis in the physiopathology of depression. J Psychiatry Neurosci 29: 185-193.
17. Keck ME, Sartori SB, Welt T, Müller MB, Ohl F, et al. (2005) Differences in serotonergic neurotransmission between rats displaying high or low anxiety/depression-like behaviour: Effects of chronic paroxetine treatment. J Neurochem 92: 1170-1179.
18. Witte AV, Flöel A, Stein P, Savli M, Mien LK, et al. (2009) Aggression is related to frontal serotonin-1A receptor distribution as revealed by PET in healthy subjects. Hum Brain Mapp 30: 2558-2570.
19. Reul JM, Holsboer F (2002) Corticotropin-releasing factor receptors 1 and 2 in anxiety and depression. Curr Opin Pharmacol 2: 23-33.
20. Ohmura Y, Yoshioka M (2009) The roles of corticotropin releasing factor (CRF) in responses to emotional stress: Is CRF release a cause or result of fear/anxiety? CNS Neurol Disord Drug Targets 8: 459-469.
21. Liston C, Gan WB (2011) Glucocorticoids are critical regulators of dendritic spine development and plasticity *in vivo*. Proc Natl Acad Sci U S A 108: 16074-16079.
22. Lee KJ, Kim SJ, Kim SW, Choi SH, Shin YC, et al. (2006) Chronic mild stress decreases survival, but not proliferation, of new-born cells in adult rat hippocampus. Exp Mol Med 38: 44-54.
23. Hanson ND, Owens MJ, Nemeroff CB (2011) Depression, antidepressants and neurogenesis: A critical reappraisal. Neuropsychopharmacology 36: 2589-2602.
24. Herman JP, Mueller NK (2006) Role of the ventral subiculum in stress integration. Behav Brain Res 174: 215-224.
25. Posdeyeva EA (2007) Hypothesis affective disorders based on neuroplasticity. A new view at the theory of depression. Psychiatry and Psychopharmacology 43: 49-52.
26. Lee KJ, Kim SJ, Kim SW, Choi SH, Shin YC, et al. (2006) Chronic mild stress decreases survival, but not proliferation, of new-born cells in adult rat hippocampus. Exp Mol Med 38: 44-54.
27. Surget A, Saxe M, Leman S, Ibarguen-Vargas Y, Chalon S, et al. (2008) Drug-dependent requirement of hippocampal neurogenesis in a model of depression and of antidepressant reversal. Biol Psychiatry 64: 293-301.
28. Marazziti D, Baroni S, Picchetti M, Piccinni A, Silvestri S, et al. (2013) New developments on the serotonin hypothesis of depression: Shunt of tryptophan. Riv Psichiatr 48: 23-34.
29. Furuyashiki T (2012) Roles of dopamine and inflammation-related molecules in behavioral alterations caused by repeated stress. J Pharmacol Sci 120: 63-69.
30. Arain M, Haque M, Johal L, Mathur P, Nel W, et al. (2013) Maturation of the adolescent brain. Neuropsychiatr Dis Treat 9: 449-461.