

# Serotonin and it's Influence on Aggression and Determination of Social Status

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**Abstract:- Serotonin, or 5-hydroxytryptamine, is a biogenic amine best known for its functioning as a neurotransmitter. In many species of animals, including humans, serotonin, social status, and aggressiveness seems to be connected. The connections are complicated, and specifics about how the amine affects behaviour are mostly unknown. This review paper studies the nature between serotonin and aggression and serotonin and social status.**

## I. INTRODUCTION

Neurotransmitters are chemical messengers that enable neural cells in the central and peripheral nervous systems to communicate with one another. Neurotransmitters can either excite or inhibit downstream neurons from "firing." This aspect of neurotransmitter activity in the functional brain aids in the regulation of information transmission throughout complex neuronal networks linked to emotional, cognitive, and behavioural experiences. Serotonin is a chemical that has a variety of effects on both the central nervous system and the peripheral nervous system. It is found in all animals and functions as a hormone, neurotransmitter, and mitogen. In 1948, Maurice Rapport and Irvine Page isolated and described serotonin, also known as 5-hydroxytryptamine or 5-HT (Rapport et al., 1948a–c). After decades of research, scientists were finally able to isolate serotonin, a vasoconstrictor chemical thought to be present in platelets (Janeway et al., 1918; Reid & Bick, 1942; Zucker, 1944). Serotonin is derived from the Latin term serum and the Greek word tonic. Vittorio Erspamer, an Italian scientist, isolated a chemical from enterochromaffin cells in the gastrointestinal tract that caused smooth muscle contractions and termed it enteramine (Erspamer & Asero, 1952). Serotonin (5-hydroxytryptamine, 5-HT) is a neurotransmitter that plays a vital role in the central nervous system (CNS). The CNS contains roughly 1–2% of the total quantity of 5-HT in the body (Cooper JR et al., 1996). A small but significant number of serotonergic cells reside in the 5HT system in the central nervous system. Cell bodies (soma) are primarily found in the midbrain and hindbrain (TÖRK, I., 1990) and both rostral and caudal parts of the brain are served by serotonergic neurons (Jacobs, B. & E. Azmitzia., 1992). The rostral projections, in particular, are assumed to play a significant role in the activation of the serotonergic system in the pathophysiology of numerous psychiatric diseases. The serotonergic system is complicated, and a slew of new discoveries over the last decade have radically altered the fundamental paradigm of the neuron–neurotransmitter–receptor axis. Within the serotonin receptor family, 14 distinct serotonin receptors may currently be distinguished: 5-HT1A, 5-HT1B, 5-HT1D, 5-HT1E, 5-HT1F, 5-HT2A, 5-HT2B, 5-HT2C, 5-HT3, 5-HT4, 5-HT5A, 5-HT5B, 5-HT6, and 5-HT7. The numerous receptors

are neuroanatomically located at diverse locations across the CNS (O. MENGOD et al., 1990).

The relationship between serotonin, social rank, and aggressive behavior in animals and humans is the subject of a broad and often inconsistent literature. Much of the ambiguity stems from the limited metrics employed to describe serotonergic activity, as well as the numerous diverse methods in which researchers quantify social status and aggression. Despite this ambiguity, the majority of researchers agree that serotonin has a role in aggression as well as the establishment and sustaining of rank. Several broad generalisations have arisen as a result: First, reduced serotonergic function is linked to increased aggressiveness and low social status in mammals, whereas higher serotonergic function is linked to decreased aggression, affiliative behaviour, and superior social skills, all of which contribute to higher status (Sheard MH., 1983; Raleigh MJ., 1991; Coccaro., 1992). Second, in fish, inferior animals with improved serotonergic activity had less fighting behaviour (Winberg S., 1993; Winbera Y et al., 1997). Third, increasing aggression and dominating position in crustaceans appear to be connected with higher serotonergic activity, which is the polar opposite of what is seen in most vertebrates. Serotonin was recognised as a vital molecule in the vertebrate physiology early on, and studies in the domain has exploded since its revelation. We will discuss what is presently learned regarding the effects of serotonin on social status and violence in this review.

## II. SEROTONIN AND AGGRESSION

Although the initial documented clinical study directly addressing the relationship between serotonin activity and human aggressiveness (Brown G.L. et al., 1979) is less than 20 years old, the range of studies in this field has expanded significantly. Previous reviews of the research concluded that there is significant data to substantiate the notion that serotonin neurotransmitter activity plays a major role in human aggression (Linnoila et al., 1992; Coccaro, 1992; van Praag et al., 1990). Furthermore, suggesting that 5HT activity plays a significant role in human aggression may be premature for a variety of reasons. To begin with, there have been relatively few research that have looked at the link between 5-HT level and actual aggressive behaviours. To support the concept that 5-HT modulates violent behaviour, many research have used measurements of emotions, attitudes, or personality traits (e.g., Coccaro, 1992; Linnoila & Virkkunen, 1992). A vast majority of research rely on hostility or anger measures. For two reasons, these researches cannot be used to investigate the effects of serotonin on aggression. First, anger and hostility are theoretically separate from aggressiveness (Biaggio & Maiuro, 1985).

Second, assessments of rage and hostility may understate the link between aggressiveness and biological characteristics. (Archer,1991).

### III. NEUROCHEMICALS AND AGGRESSION

The concentration of 5-HIAA (5-hydroxyindoleacetic acid, a key metabolite of 5-HT) in CSF collected from the lumbar sack has been utilized as a measure of central 5-HT activity. Nerve cells in the spinal column create 5-HIAA in the CSF, and levels of this metabolite correspond with 5HIAA in the frontal cortex of the brain. As a result, CSF 5-HIAA is regarded as one of the more reliable neurotransmitter activity indicators. CSF 5-HIAA levels, on the other hand, reveal little about neuronal activity in specific brain regions.

In an experiment covering 26 individuals detained to a military forensics facility, Brown and associates were among the first to discover an inversely proportional relationship among CSF 5-HIAA and aggressive conduct in humans (Brown et al., 1979). All of the subjects were diagnosed with at least one personality disorder in the DSM-II, but no other significant psychopathology. Documentation from hospital records and psychiatric interviews was used to construct lifetime evaluations of various aggressive and antisocial behaviours. The complete life of aggression score was calculated by adding the rating scores (Brown- Goodwin Aggression scale: BGA). CSF 5-HIAA levels were also inversely correlated to a measure of "behavioural impulsivity." Males diagnosed with antisocial, violent, juvenile, or histrionic personality disorder exhibited lower CSF 5-HIAA levels than males diagnosed with passive-aggressive, schizoid, or obsessive compulsive personality disorder. A follow-up study of 12 military men diagnosed with DSM-III borderline personality disorder found less confirmation for the serotonin hypothesis of violent conduct (Brown et al., 1982).

Brown studies had a number of flaws, one of which was the lack of comparison groups. As a result, the investigations don't reveal anything concerning the link between CSF 5-HIAA and aggressive behavior in people who haven't been diagnosed with anything. Another disadvantage has to do with the BGA's content. Both aggressive and antisocial behaviours are included in the BGA. Aggressiveness may or may not be linked to antisocial actions. As a result, it's unclear if low CSF 5-HIAA is linked to aggressive behaviour in particular.

The Brown study's findings have been replicated, although with statistical connections of lesser magnitude. On a sample of 57 alcoholics and 15 healthy subjects, Limsonn et al. (1991) discovered a small but substantial negative connection ( $r = -.31$ ) among CSF 5-HIAA and aggressiveness. A BGA modification was employed.

In contradiction to these positive observations, one research reported that men found guilty of murder had CSF 5-HIAA levels that were much higher than healthy counterparts (Lidberg et al.,1985). Furthermore, exploratory analysis found that certain subgroups of aggressive men have

decreased CSF 5-HIAA. Men who murdered a sexual partner had reduced CSF 5-HIAA concentrations than men who murdered a nonintimate acquaintance, and non-alcoholic violent men had lower CSF 5-HIAA levels than alcoholic violent men.

Children's trials are largely non-existent owing to the invasive nature of the procedure employed to gather CSF. Kruesi, et al. (1990) investigated the relationship between (ZSF 5-HIAA and aggressive behaviors in children with disruptive behaviour disorders (i.e., conduct disorder, attention deficit hyperactivity disorder [ADHD], oppositional defiant disorder;  $n = 33$ ) as well as children with obsessive compulsive disorder ( $n = 43$ ). Age-corrected CSF 5HIAA levels were inversely linked with assessments of aggressive conduct, as anticipated, but only in the disruptive behaviour disorder group. Only three of the 32 associations between CSF 5-HIAA and impulsive behaviour, adverse effects, and violence were statistically noteworthy. The number of statistical tests performed was not adjusted, making it more difficult to make sense of the data.

Simeon et al. (1992) contrasted those who had previously self-mutilated ( $n = 26$ ) to people who had never self-mutilated ( $n = 26$ ). The groups were similar in terms of core demographic factors as well as the percentages of particular major mental disorders and personality disorders detected within each group, with major depression and borderline personality disorder being the most prominent classifications in both. Findings from a semi-structured interview was used to quantify a person's history of aggressive behaviour. Self-mutilators scored significantly higher on aggressive behavior than non-self-mutilators, however there were no differences in CSF-5-HIAA levels or peripheral 5HT functioning indexes between the two groups. Furthermore, no significant link was discovered between 5HT functional indexes and aggressive behaviour in either group.

One research group's series of CSF 5HIAA investigations gives some backing for the concept that low 5-HT is connected with an "impulsive" subtype of aggressive conduct (e.g., Linnoila et al., 1983; Virkkunen et al., 1987;

Rawlings et al., 1994). Impulsively hostile males were shown to have decreased CSF 5-HIAA concentrations than non-impulsively hostile men in a widely regarded research of 36 Finnish men who murdered or tried to commit homicide (Linnoila et al., 1983). Men were classified as impulsively violent if no explanation could be found for their actions (i.e., they were not predetermined) and the perpetrator was not connected to his target, according to clinical judgement. The authors concluded, that "low 5-HIAA concentration in the CSF of violent offenders" is more "a marker of impulsivity than violence per se" (p. 2610). However, the hypothesis that impaired 5-HT functioning is primarily related with an impulsive subtype of violent behaviour was not firmly confirmed for a multitude of reasons. There was no evidence that the approach employed to identify perpetrators as impulsive or non-impulsive was reliable or valid. It might also be claimed that impulsive, spontaneous violence is more likely to occur in close relationships (e.g., interfamily

aggression) than in casual relationships or when the aggressor is unaware of the victim. Indeed, the findings of Linnoila et al. appear to contradict those of Lidberg et al. (1985), who found that homicide amongst intimates was linked to lower CSF 5HT<sub>1A</sub> levels. More importantly, Linnoila et al. (1983) did not acknowledge the question about whether a record of aggressive conduct in general is linked to CSF 5-HIAA deficiency.

#### IV. IS SEROTONIN INHIBITORY IN AGGRESSION?

The major assumption in the link between serotonin and aggressiveness is that 5-HT decreases aggression, based on research in which neurotoxicity drugs like pCPA or 5,7-DHT, which decrease serotonin from serotonergic cells, reduced serotonin levels in the brain. Although assessments of 5-HT activities in people were predicated on CSF levels of the major metabolite of serotonin, 5-HIAA, such an inverse association between 5-HT and aggressiveness has been established in both humans and animals. Despite harsh critiques, for many years it was the sole criteria in humans that (implicitly) reflected the functioning state of the 5-HT system. 5-HT and its metabolite 5-HIAA can be detected readily in the brain of animals, therefore the negative link between functional serotonergic activities and aggressiveness should be straightforward to substantiate. However, there have been various contradicting findings, including claims of a favourable link between 5-HT and aggressiveness. Aggression is linked to suicidal behaviour in humans, and both appear to be linked to reduced serotonergic activity, while it's likely that the two manifestations are regulated separately (Mann & J.J., 2003). The levels of 5-HT and 5-HIAA in postmortem neural tissue, as well as the turnover rate calculated from these two parameters, were first reported to be lower in aggressive mice compared to nonaggressive animals (Giacalone, E., 1968). Analyzing CSF data in humans repeatedly confirmed this serotonergic hypofunction (Brown et al., 1979; Linnoila et al., 1983; Kruesi et al., 1990). However, instead of violence, this 5-HT hypofunction or deficient feature has lately been linked to impulsive and danger behaviour (Mann & J.J., 2003). Static measures of 5-HT or 5-HIAA in brain tissue or CSF-fluid cannot be used to infer a causal relationship between 5-HT activity and aggressiveness or impulsivity. Although considerable gain has been achieved utilising *in vivo* microdialysis techniques in freely moving (aggressive) animals, a functional role of serotonergic neurons in the start, execution, and termination of aggression (Coccaro & E.F., 1989; Miczek et al., 2002) has yet to be demonstrated.

Prior, during, and afterwards a 10-minute aggressive contact with a male conspecific, Van Erp and Miczek (2000) assessed extracellular serotonin (and dopamine) production in the nucleus accumbens (Nac) and prefrontal cortex of rats in 10-minute samples. The NAc showed no significant difference in 5-HT release during the agonistic engagement, whereas 5-HT levels in the prefrontal cortex had already been reduced during fighting. After the confrontation, 5-HT levels in the PFC persisted lower for at least 1 hour (relative to the pre-confrontation benchmark), although 5-HT

levels in the NAc were unaffected. After (but not during) the agonistic encounter, dopamine levels increased in both brain locations. 5-HT levels, on the other hand, were shown to be lower in the NAc of rats who had been trained to fight at a set time every day over a 10-day period (Ferrari et al., 2003). Heart rate and dopamine release were assessed simultaneously in the latter studies, and both were boosted in anticipation of the fight. Presumably, the actual conduct of aggressiveness can be distinguished from the expectation of a fight, with dopamine playing a key role in the physiological and behavioural consequences of aggression performance and anticipation, while serotonin appears to be especially linked to aggression discontinuation.

Analyzing the distinctions amongst high-aggressive and low-aggressive people, as Koolhaas' group has recently undertaken, is a contemporary approach to unravelling the function of the 5-HT system in aggressive behavior (2003). Their thesis was founded on the idea that a rat's distinct level of aggressiveness (offensive aggression) is a part of the animal's particular coping mechanisms and thus a good predictor of a trait-like physiological and behavioural responses. Serotonin was explored in depth in their research on the endophenotypes of aggressive and non-aggressive mice. In contradiction to the previous hypothesis of an inverse link between 5-HT activity and aggressiveness, researchers discovered a positive correlation between trait-like aggression (high or low) and basal CSF 5-HT and 5-HIAA levels (Cremers, et al., 2003.) Furthermore, after microdialysis, concentrations of 5-HT and 5-HIAA in the frontal cortex did not vary amongst endophenotypes. Normal offensive aggression appears to be favourably connected to serotonergic neural activity, but 5-HT activity appears to be inversely related to impulse-like violent aggression. (Coccaro & E.F. 1989).

#### V. SEROTONIN AND SOCIAL STATUS

Raleigh and colleagues looked at the impact of changing amine neuron function on aggressiveness and social status in vervet monkey social circles in another series of highly controlled tests. The monkeys were accommodated in 12 social groups of three males, at least three females, and their young in outdoor facilities. The trial was split into five 8-week interims: first, a standard dominance relationship had been formed; second, the dominant male monkey was excluded from the group, and one of the other males monkey (medicated male) was injected with an agent that either amplified (tryptophan or fluoxetine) or lowered (fenfluramine or cyproheptadine) serotonin neuron activity for half of the treatment phase. The initial dominant male was reintroduced to the group, whereas the third male was treated with vehicle. fourth, the dominant male was separated from the group once more, and the treated males were given compounds that had the reverse result as the first; fifth, the group's original dominating male was reintroduced. During the intervention periods, the findings were evident and constant. In the uncertain circumstance (second and fourth phases), animals whose treatments improved serotonergic activity were dominant due to improved affective behaviour and social abilities, engaging females to defend their status.

Animals with impaired serotonergic functioning, on the other hand, exhibited elevated levels of instigating aggression and lower social rank. When the initial untreated dominant males were restored to their social circle, they all reestablished their social standing (the third and fifth intervals). "Serotonergic systems promote the acquisition of male dominance under unstable social situations," Raleigh et al. conclude cautiously. These findings corroborate those of other researchers, emphasising the contrast amongst aggressiveness and domination.

There are temporal changes in regional serotonergic function between dominating and submissive males of the lizard *Anolis carolinensis*. While both dominant and subordinate lizards' serotonergic reactions seem to be mediated in the same brain areas, the timing of the reaction is substantially different. (Summers, Cliff et al., 1998) Celerity-dependent neuroendocrine activities tend to regulate social status formation. Serotonergic activation in the medial amygdala, a region involved with adrenal axis stress hormonal activation (Dunn JD & Whitener J., 1986; Herman JP & Cullinan WE., 1997), is raised within an hour of the start of combat in dominant males. However, until 24 hours after agonistic behaviour begins, submissive males do not show increased serotonergic function in the medial amygdala. It takes a week of cohabitation for submissive males to attain peak levels of serotonergic activity. The skin colour of this lizard reflects variations in central neurochemical status between dominating and submissive males. *Anolis carolinensis* exhibits a distinctive visual sign of social status: the degree of postorbital skin darkening (eyespot). The skin directly above the eyes darkens (as it does with any stress) throughout the development of dominant-subordinate interactions, and it darkens more rapidly in dominant males (Summers CH, Greenberg N., 1994) The rapid darkening of the eyespot is a social indicator trigger that reduces aggression considerably (Korzan WJ et al., 2000; Summers TR et al., 2000) Sympathetic activity causes eyespot darkening via endocrine stimulation of  $\beta_2$  adrenergic receptors (Hadley ME & Goldman JM, 1969). As a result, a major neurochemical feature that identifies dominant male status tends to be faster serotonergic and autonomic activation. It was suggested that the length of serotonergic response played a significant role in defining social status considering neuroendocrine reactivity seemed to be rapid and short in dominant males. The focus of this research was to use a selective serotonin reuptake inhibitor to imitate chronically enhanced serotonergic function in submissive males in order to impact social behaviour. Sertraline (Zoloft; Pfizer) was specifically thought to diminish aggression, delay eyespot darkening, and enhance submissive conduct.

Lorenzi, V. et al. (2009) explored the influence of serotonin in the control of sex change in the bluebanded goby *Lythrypnus dalli* in an experiment. Female *L. dalli*, as the submissive animals, would have more serotonergic activity than males, and this increased serotonergic activity would prevent them from changing sex, according to the authors. The experiment's findings did not support this assumption, but rather revealed a trend in the opposite direction: females

had lower levels of 5-HT, 5-HIAA, and 5-HIAA/5-HT than males, despite the fact that the variation was not significant.

Stress-induced activities increases in brain serotonin (5-hydroxytryptamine, 5-HT) turnover are observed in low-dominance fish, as evidenced by enhanced brain levels of 5-hydroxyindoleacetic acid (5-HIAA, the primary 5-HT metabolite) and 5-HIAA/5-HT ratios. (Winberg, S., & G. E. Nilsson., 1993). Socially subordinate fish often show prominent behavioural inhibition, which could be facilitated by brain 5-HT and portray a passive way of coping in subordinate animals, reducing the regularity of agonistic interplay with dominant individuals and thus potentially stress reduction in subordinates. (Davis, M. N, 1980; Soubrie', P., 1986).

## VI. CONCLUSION

Many, if not all, other neurotransmitter networks in the brain engage with the serotonergic network in the CNS. Monoaminergic activity is frequently altered as a function of social behaviour. Locomotion, nutrition, asymmetries of alcohol effects, depression, stress, and aggression are all linked to serotonin (5-HT). Despite decades of investigation on the serotonergic system's possible role in aggressiveness and social status, no clear picture surfaces. At least in certain populations, finding demonstrates that 5-HT function is linked to aggressive behaviour and social rank determination. This evidence, however, appears to be less persuasive than is widely supposed. Given the absence of scientific proof, it's still questionable whether 5-HT functioning is causally linked to aggression and determining social status in humans, or simply linked with both. The impact of serotonin on social status in human populations has received very little attention. In such intricate social behaviours, HT status is expected to play only a minor influence. As a result, it's critical that future studies minimize the multiple flaws that plague this field of research. In conclusion, there is some evidence that 5-HT anomalies are linked to dominant-submissive behaviours and aggressive behaviour, although it is far from conclusive. Future comprehensive study on possibly relevant biologic variables could reveal critical details about how individual differences in behaviour emerge, as well as how biological and psychological factors interact to evoke or prevent such behaviour. To gain a complete knowledge of the role of serotonin in human behaviour, arguments for the serotonin theory of aggression and social status must be combined with such data.

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