Critically discuss the current evidence for a biological component of a specific psychological process of your choice:

The role of the amygdala and prefrontal cortex in aggression

Abstract (word count: 231)

Aggression is hostile behaviour with the intention of inflicting damage on another individual and is evolutionarily important. Reactive aggression is highly emotional whereas predatory aggression is planned and goal orientated. Understanding the neural basis for aggression is important in order to gain insight into a number of psychiatric conditions, potentially help prevent deaths caused by violence and to further our knowledge of human behaviour. Evidence for the involvement of the amygdala and prefrontal cortex in aggression were evaluated.

Removal of the amygdala has been shown to decrease aggressive behaviour and structural studies have suggested that aggression is associated with a smaller amygdala volume, although longitudinal studies are needed to establish the direction of the relationship. Functional imaging techniques have suggested that individuals displaying reactive aggression have a hyperactive amygdala whereas those showing predatory aggression have a hypoactive amygdala.

The prefrontal cortex is considered to exert modulatory control over aggressive behaviours. Research suggests that reactive aggression is associated with diminished prefrontal functioning whereas that predatory aggression is associated with normal, or higher levels of functioning.

The evidence assessed has a number of limitations which future studies need to address, in particular by comparing predatory and reactive aggression directly.
Aggression is one of the most evolutionarily preserved psychological processes and although there are still unanswered questions about its underlying mechanisms, there is clear evidence that the amygdala and prefrontal cortex play key roles.

**Key words:**
- Aggression
- Amygdala
- Prefrontal Cortex
- Psychological process
- Neural basis

**Introduction**

Aggression refers to hostile behaviour with the intention of inflicting damage on another individual (1,2). It is found across a wide range of species and the evolutionary function of aggression has been well documented (3). It has been a subject of interest to researchers for many years, in the hope that understanding the neural and biological mechanisms behind it will help in our pursuit to acquire a deeper understanding of human behaviour and emotion (1). There are also a number of psychiatric disorders in which aggression is a key symptom (4) and so research into this area may provide an insight into the pathophysiology of these disorders and how to treat them. In addition, aggressive acts of violence currently result in 1.3 million deaths per year in the world, with many more people being non-fatally injured (5) and therefore unravelling the complex mechanism by which aggression occurs may, in the future, help to prevent some of these deaths from occurring.

Research so far has implicated a number of brain areas in the expression of aggression (including the prefrontal cortex, limbic system and temporal lobe) (1), however this essay will focus on the
evidence for the role of the amygdala and prefrontal cortex only and will also discuss possible avenues for future research.

Aggression can be classified into two categories: reactive aggression and predatory aggression (1). Reactive aggression (also known as impulsive or affective aggression) is characterised by high levels of autonomic arousal and aggressive behaviours are impulsive and emotional charged (6). On the other hand, predatory aggression (also known as instrumental or proactive aggression) is not necessarily associated with autonomic arousal and is a planned behaviour serving to achieve a goal in a controlled, purposeful manner (1,2). These two types of aggression are often not divided in the literature (7) and therefore a direct separation is not made in this essay. The problems caused by the lack of distinction, however, will be addressed.

**Amygdala lesions**

It is well known that the amygdala plays an important role in the processing of emotions (8,9), which has made it a key target when investigating aggressive behaviours (2). Early animal studies have shown that animals can be tamed by lesioning the amygdala bilaterally (10) and that electrically stimulating the amygdala evokes aggressive responses (11,12). More recent animal studies have also shown that damage or ablation of the amygdala results in a complex pattern of aggression dysregulation (13,14).

Early studies on humans have presented similar results, showing that removal of the amygdala decreases aggressive behaviours in patients (15,16) and this procedure has been used specifically for the treatment of inappropriate and pathological aggression (17).

**Amygdala reactivity**
Building on initial lesion studies, functional imaging techniques have been used to study the amygdala and research so far suggests that the reactivity of the amygdala may be different in aggressive individuals. It has been shown that in healthy individuals, amygdala reactivity is positively correlated with trait anger when viewing pictures of negative emotions (18,19) and neutral emotions (20), though violent subjects tend to perceive neutral faces as negative (21), implying these studies agree. Studies done in psychiatric populations have also shown that violent individuals demonstrate an exaggerated amygdala response when viewing angry (22,23) and threatening (4) faces. Moreover, in patients with intermittent explosive disorder (IED), in which the cardinal symptom is impulsive aggression, the degree of amygdala hyperactivity was positively correlated to the extent of previous aggressive behaviour (4).

In contrast to these results, there is a body of evidence that proposes that amygdala reactivity actually decreases with increasing aggression (24). This work has mainly been done in aggressive psychopaths, who show diminished amygdala activity during an emotion regulation task (25) and significantly lower amygdala activity during an aggression inducing task, compared to controls (26).

These inconsistencies surrounding amygdala activity can potentially be explained by acknowledging the fact that there are two subtypes of aggression (reactive and predatory (1)), that are rarely separated in studies (7). It has been suggested that the two subtypes may have a different neural basis (27,28) and amygdala reactivity may be able to distinguish between them (2). The proposed hypothesis is for a continuum, with amygdala hypoactivity and predatory aggression at one end, and amygdala hyperactivity and reactive aggression at the other extreme (20,29-31). The conflicting evidence presented so far fits with this emerging hypothesis because most of the studies showing increased activity were done in healthy volunteers (who mostly exhibit reactive aggression (32)) and patients with IED; whereas the research showing decreased activity has predominantly been done in psychopaths, who mainly demonstrate predatory aggression (24,33). Therefore, future studies
should clearly address the two subtypes of aggression separately, in order to delineate whether the continuum hypothesis is correct (34).

**Amygdala Volume**

Along with functional imaging studies, technology advances have also led to an increase in structural studies and results so far suggest smaller amygdala volumes may be correlated with increased levels of aggression (35). Matthies et al. found that females with high levels of trait aggression had 16-18% smaller amygdala volumes compared to those with low levels and that a significant negative correlation exists between lifetime aggression scores and amygdala volumes (36). Similarly, a study in 2014 found that in a sample of 56 men, lower amygdala volume was significantly associated with aggressive behaviour in childhood and adolescence (28) and further studies have suggested that it may be the grey matter in particular that is lacking in violent individuals (20). These studies confirm the amygdala’s involvement in aggression but because of their cross-sectional nature, causality cannot be assumed. Although it can be postulated that a smaller amygdala volume causes more aggressive behaviour, it is also unclear the effect that aggressive behaviour has on the structure of the brain. One study so far has used longitudinal methods and found that smaller amygdala volume was associated with an increased risk of committing violence and higher levels of aggression at three years follow up, even when prior levels of aggression were adjusted for (28). This study unfortunately has a number of limitations, including multiple testing, and therefore the results must be interpreted cautiously.

Whilst these studies provide compelling evidence that a small amygdala volume is correlated with higher levels of aggression, null findings have been reported (37-39). These studies used patients with anti-social personality disorder (associated with predatory aggression (2)), whereas the studies reporting positive findings were often related to reactive aggression (36). Therefore, drawing on the
evidence for amygdala reactivity, it could be hypothesised that reactive aggressive individuals have smaller amygdalae which are hyperactive and individuals displaying predatory aggression have normal size amygdalae that are less reactive. However, only a few studies explicitly stated which type of aggression they were studying (28) meaning firm conclusions cannot be drawn.

Prefrontal Cortex

The prefrontal cortex is known to be a key region involved in decision making which explains why it is considered to exert modulatory control over aggressive behaviours (1,2). Cases such as Phineas Gage (1) have shown that patients with orbitofrontal lesions become irritable, with inappropriate outbursts of anger and are more likely to use physical threats and intimidation in conflict situations (40).

Studies on healthy volunteers have shown that the prefrontal cortex is activated in response to anger-inducing script imagery (41,42) and angry faces (4), but in an unrestrained aggressive scenario, blood flow to the orbitofrontal cortex is reduced, suggesting that the control normally exerted by this area is released these situations (43). Thus, it has been proposed that diminished prefrontal functioning results in more aggressive behaviour (1,33). Research into this has shown that there is an inverse relationship between history of impulsive aggression and glucose metabolism in the orbitofrontal cortex in borderline personality disorder (44,45) and in violent psychiatric patients (46). Similarly, Raine et al. have consistently found murderers to have decreased glucose metabolism in the lateral and medial prefrontal cortices when compared to controls, with no differences in other cortical areas (47,48). Patients with IED have also been shown to have diminished orbitofrontal cortex activation when shown angry faces, but not to other negative emotions (4).

Just like amygdala reactivity, the link between the prefrontal cortex and aggression may be more complex than a simple inverse relationship. Raine et al. went on to compare reactive and predatory
murderers and found that reactive murderers showed the expected decreased prefrontal functioning, but predatory murderers show prefrontal functioning comparable to healthy controls (6). In relation to this, it is known that psychopaths show increased levels of prefrontal functioning (49) and that they exhibit predatory aggression (6). It has therefore been suggested that reactive and predatory aggression differ in terms of prefrontal functioning (6). The proposed hypothesis is that predatory aggression is associated with normal, or higher prefrontal functioning, which allows the subject to control their aggression in a way to manipulate others in order to achieve their goals; whereas reactive aggression is associated with decreased functioning leading to reduced modulation of impulses and an increase in deregulated impulsive aggression (1,6,24). However, this hypothesis has not been directly tested other than the study by Raine et al. and so requires further attention in future studies.

**Limitations of current research**

Whilst there is compelling evidence for the involvement of the amygdala and prefrontal cortex in aggressive behaviour, the studies conducted so far have a number of limitations (33); some of which have already been mentioned. Firstly, there is a lot of variation in the subjects used for research. Some studies use healthy individuals (36), whereas others use patients with a psychiatric illness (4), or incarcerated criminals (6). This makes the results difficult to collate and means some studies have many potential confounding factors.

Secondly, even though it has been demonstrated that the subtypes of aggression may have different neural correlates, the majority of studies do not separate them or explicitly state which they are measuring, which may explain inconsistencies in research findings so far (6,7,27,31,34).

Lastly, a range of methods for studying aggression have been used; for example some studies use an interview to measure lifetime levels of aggression (36), some use anger inducing tasks (26) and
others show people angry or threatening faces (4). This variety, again, makes it difficult to collate results and it is unclear whether some of the methods, such as viewing angry faces, are actually assessing aggression itself or the neural process of emotion recognition (50).

**Future studies**

Future research needs to study reactive and predatory aggression in contrast to each other so that more clear conclusions can be drawn about the neural correlates of each. In particular, studies need to discern whether amygdala volumes differ between aggression types and test the hypothesis that amygdala reactivity is a continuum (34). This should be done using techniques such as magnetoencephalography (MEG) alongside haemodynamic measures such as fMRI scans in order to achieve the best temporal and spatial resolution and therefore the most accurate results (33,51). Researchers should also consider investigating structural abnormalities and functional deficits in the same study as this would provide a global view of the brain in aggressive individuals, but does not seem to have been done so far.

Research also needs to be done to determine whether the hypothesis that only reactive aggression is associated with prefrontal dysfunction and not predatory aggression, is correct. This future research will ideally build on Raine et al.’s findings by using a larger sample size (they only had fifteen predatory murderers and nine reactive murderers) and techniques such as MEG.

There is also currently only one study that used longitudinal data to correlate lower amygdala volume with future acts of violence (28). This means the influence of brain abnormalities on the likelihood of future aggression is unknown and therefore future studies should use longitudinal methodology in order to address this issue.
In order to draw more definitive conclusions from studies in the future, researchers should also use validated aggression-inducing tasks rather than recording participants' history of aggression or viewing threatening faces.

**Conclusion**

In conclusion, aggression is one of the most evolutionarily preserved psychological processes (3) and has been the subject of neurological research for many years. There is clear evidence that the amygdala and prefrontal cortex both play a crucial role in aggression, although so far the results of research have been inconclusive, meaning the specific details of their roles remains unclear. Studies have shown that aggressive individuals can have smaller amygdalae and that both increased and decreased amygdala and prefrontal responses are associated with aggression. Some of these disparities may be explained by studying the subtypes of aggression separately but this has yet to be done. It is important to remember that whilst the abnormalities mentioned confirm a biological basis of aggression, they are by no means the only underlying mechanism and there are many other brain areas and chemical processes involved. Understanding more about the neural mechanisms behind human aggression will provide insights into how the brain deals with emotion, as well as furthering our knowledge of pathological aggression. So, whilst our knowledge has come a long way since Phineas Gage, there still remains a number of questions unanswered which means the research into aggressive behaviour will continue and should provide some exciting insights in the near future.

**Word count: 2196**

**References**


(34) Blair RJ. Neurocognitive models of aggression, the antisocial personality disorders, and psychopathy. J Neurol Neurosurg Psychiatry 2001 Dec;71(6):727-731.


