What is the evidence that aggression and violence are biologically determined?

Tebartz van Elst, Woermann, Leemiex, Thompsam, and Trimble (2000) suggest aggressive and violent or antisocial behaviours are '*phenomenological and probably neurobiological heterogeneous'*. Substantial research emphasizes two types of aggression; predatory (PA) and defensive (DA) (Vitiello and Stoff, 1997;cited in Tebatz van Elst, et al., 2000). In addition, premeditated aggression, impulsive aggression, or violence due to illness, e.g. Attention Deficit Hyperactivity Disorder, have also been identified (Bradshaw and Mattingley, 1995). Furthermore, exhibited aggression/violence may be defined as physical or psychological (Smythies, 1970). Subsequently, their triggers may be conscious or unconscious and may be attributed to environmental/social factors, temperament, gender differences, genetic factors, and neurological determinants [including various brain structures and neurotransmitters] (Baron and Richardson, 1994; Wood, Wong and Chacherer, 1991; cited in Lau and Pihl, 1995;). Both excitatory and inhibitory (Carlson, 1998) brain structures are believed to mediate antisocial behaviours including; the temporal lobes, parts of the Limbic System (LS), Amygdala (AD), Periaqueductal Grey Matter (PAG), Hypothalamus (HT) and septal area), and the Orbitofrontal Cortex (OFC) (Marzuk, 1996). Furthermore, several excitatory and inhibitory neurotransmitters are implicated including; Serotonin (5-HT), dopamine and Norepinephrine (NE). Similarly, hormones have been suggested to play a role (Marzuk, 1996). In addition, genetic links have been suggested for Impulsive violence/Aggression (IA) (Brunner, Nelen, Breakefield and Ropers van Oost, 1993) with mutation of the monoamine oxidise A (MAO A) gene. This essay will principally concentrate on neurological influences, briefly outlining social, genetic, and hormonal contributors to aggression and violence. The essay will conclude by summarising, the illustrated factors believed to be involved in the mediation of aggression and violence, lastly, it will propose that a larger interconnective approach between biological determinants could be explored. However, it will firstly examine PA and IA in further detail.

PA is involved mainly in animal survival (Pinel, 1999). The author suggests such behaviour may be predetermined in man and be exhibited as a response to situations which were once a necessity for continued existence. Conversely, the primary objective of intentional aggression is to inflict harm and is more premeditated than PA (Smythies, 1970). IA may result from the release of feelings and frustration (Smythies, 1970) however; this is unlike opportunistic, premeditated (intentional) aggression, where an individual may profit from their behaviour (psychologically or materially) (Bradshaw and Mattingley, 1995). Thus, the difference between the degrees to which an aggressive act is consciously controlled may be divided into intentional aggression and unplanned aggression where the consequences have not been considered by the perpetrator (Smythies, 1970).

Environmental and social contributors to aggressive and violent behaviour have been suggested to start in the home (Smythies, 1970). Early findings suggest high correlations between antisocial behaviour and low socio-economic class (cited in Peters and McMahon, 1992). Furthermore, conformity to social norms has been associated with such behaviours (Hogg and Vaughan, 1998). However, these social considerations only describe triggers and emancipators; they do not identify how the response is derived. Antisocial behaviour derivatives are suggested therefore to stem from biochemical abnormalities and temperament (Peters and McMahon, 1993).

Male social aggression has been associated with **hormonal levels** (Nelson, 1995; Cited in Rosenzweig, Leimen, and Breedlove, 1996), namely testosterone. Ehrenkranz (1974) suggested a positive correlation between testosterone levels and social hostility (cited in Rosenzweig et al., 1996). Lloyd, (1971) similarly found androgen levels reduced after losing an aggressive encounter (cited in Rosenzweig et al., 1996). However, the causality is questionable as early studies on rats (Beeman, 1947; cited in Carlson, 1998) suggest increases in testosterone promote PA. Nethertheless, Albert, Walsh, and Jonik, (1993) indicated that social and DA are different, (cited in Pinel, 1999). The author suggests this may clarify the differences in causality between Beeman's and Ehrenkanz's findings. However, as these studies are correlational they do not imply causality. Additionally, hormones have been implicated in increased aggression in women, Floody (1983) reported that pre-menstrual women might show heightened levels of aggression. However, this has been contested, as individuals cope differently with the monthly changes (Carlson, 1998).

Gene encoding is important to consider as Chen, Rainnie, Greene, and Tonegawa (1994) found abnormalities in some psychiatric patients' genes heightens aggressiveness. However, though this suggests that genes are causal in the role of antisocial behaviour it is not believed to be entirely so, as there is a low concordance rate between monozygote twins (Atren and Curthoys, 1996). In addition, Monoamine Oxidisase genes (MAO) are important to consider as they are principal enzymes for eliminating all monoamine neurotransmitters, including, NE, epinephrine, dopamine and 5-HT (Maes and Coccaro, 1998). Brunner et al., (1993) found that in some individuals a deficiency in the enzyme MAO A exacerbates IA. The gene for MAO A is located on the X chromosome, thus the deficiency of this is more common in males, due to their possession of only one X chromosome. Though literature suggests male possession of an extra 'Y' chromosome may cause some males to express more aggression, prisoners possessing an extra 'Y' are not necessarily convicted for violent crimes (Rosenzweig et al., 1996). The author suggests that the evidence for genetic and hormonal influences is inconclusive and thus will proceed to explore neurological contributors. It is important to note that there are inhibitory and excitatory areas of the brain that influence aggressive behaviour (Schere, 1975). It is suggested that responses to stimulations are individual and environmental and the causality of brain stimulation and aggressiveness is open to various interpretations (Schere, 1975). Many structures and injuries to different brain areas have been reported to be responsible for different aggression types (Hill, 1998). Luria, (1980) proposes that aggressive and violent behaviour is regulated by salient stimulus cues or impulsive attentional responses. Under provocative conditions, Lau and Pihl, (1995) found damage to the frontal lobe (FL) increased aggression. This is supported by findings of increased impulsivity and violence (MacKinnon &Yudofsy, 1996; cited in Stoff & Cairns, 1996). It has further been suggested that there is a connection between impulsivity, frontal temporal lobe regions and a serotonin mechanism (Stoff & Cairns, 1996). Emotional expression is regulated through a linkage between the temporal lobes, LS, thalamus, HT, and midbrain (Levine, 1991; Maes and Coccaro, 1998). The LS is involved in the regulation of motivated behaviours including 'fleeing, feeding, fighting and sexual behaviour' (Pinel, 1999; Levine, 1991). Similarly, the AD is suggested to play a critical role in emotional behaviour, and particularly with the mediation of aggression (Schere, Abeles, and Fischer, 1975), as are the PAG, HT, and FL. The AD facilitates the received sensory information (for example threatening stimulus) by the temporal structures to the LS (Herbert, 1984; cited in Trimble, 1996). Reductions in social dominance, and recondition of aggressive facial expression have been found to occur in patients with AD lesions (Adolph, 1994; Trimble, 1996). Similarly, amygdalectomies are reported to produce docility (Lilly et al., 1983; cited in Atren and Curthoys, 1996). Thus, it is suggested

that AD lesions may have a helpful effect for controlling aggressiveness in some uncontrollable individuals (Ramamurth, 1988).
The AD projects out on to the OFC, which is believed to have an excitatory response to anger (Pietrini Guazzelli, Basso, and Grafman, 2000). Lesions to this region have conversely been found to heighten aggressiveness, disrupting inhibitory mechanisms and resulting in inappropriate behaviour, which is predominantly planned (Pietrini et al., 2000). The author suggests, therefore, that through the integration of the mentioned brain structures, antisocial behaviour may be exhibited. Furthermore, localised lesions to various brain areas may produce different types of responses. Examining the HT role in aggression and violence can further show this. The HT is involved in the endocrine and sympathetic systems (Smythies, 1970) using NE as its postganglionic transmitter (Backer, Barasi & Neal, 2000). These are inhibitory responses, which can be associated with 'flight or fight' response (Reber, 1995). This may be explained by considering the differences in aggression when the HT is lesioned in different places (Carlson, 1998). For example, Toates, (1992) reported medial HT lesion heightens DA, yet a lateral lesion reduces PA. This distinction in behaviour produced by lateral and medial lesions was also found in the septum (Atren and Curthoys, 1996). Furthermore, Shaikh, Schubert and Siegel, (1994) found that through stimulating the PAG in various places, DA and PA were produced, where the HT and the AD promote behaviour through inhibitory and excitatory connections to the PAG (Carlson, 1998). The author notes that this evidence focuses on the connection produced between the dorsal PAG and medial HT. Thus, it is proposed that LS structures may activate strong emotional responses (Kawashima Sugiuar, Takashi, Nakamura, Hatano, Ito, Fukuda, Kojima, and Nakamura, 1999).

In addition to the involvement of the AD, HT, and the PAG in aggressive and violent behaviour, other neurological components are also involved (Carlson, 1998). Earlier, the role of MAO in 5-HT distribution (Carlson, 1998) was discussed. As MAO depletes, it is assumed to increase 5-HT distribution, which would reduce the amount in the brain. Therefore, it could be assumed aggression should decrease. However, Brunner et al. (1993) conversely found aggression to increase. Subsequently, deficiencies in MAO A's low concentration levels of 5-hydoxyindole-3-acetic acid (5-HIAA), a 5-HT metabolite, in the Cerebrospinal Fluid (CSF) have been associated with premeditated and violent aggression (Coccaro, Siever, Klar, Maurer, Cochrane, Cooper, Mohs, Davis, 1989). Additionally, Linnoila,Virkkunen, Scheinin, Nuutila, Rimon, and Goodwin, (1983) found that, although CFS monoamine was found to be lower in impulsive aggressive behaviour, this was not so for premeditated violence. Hence, the author suggests that underlying variables may have existed in the two studies, which affected the different types of aggressive behaviour found, and that low CFS monoamine levels may have a role to play, but that it is inconclusive.

Serotonergic synapse activity is also thought to inhibit aggressiveness. This, however, is believed to reduce through drug administration, through the destruction of serotonergic axons (Carlson, 1998). Serotonergic activity increases due to poor reuptake of 5-HT at the terminal buttons. 5-HT then breaks down into 5-HIAA and enters the CSF (Carlson, 1998). Thus, levels of 5-HIAA are high. Subsequently, low production rates of 5-HT have been associated with an altered 5-HT metabolism (Valzelli, 1982; cited in Brunner, et al., 1993). Consequently, an inverse relationship between 5-HT turnover and IA has been reported (Coccaro, Kavoussi, Sheline, Lish, and Csernansky, 1996). However, it is noted that self IA (suicide), characteristically has lower MAO levels in addition to

low 5-HIAA CSF levels (Oreland, 1983; cited in Trimble, 1996). Conversely, Raleigh (1992) found a positive relationship between increased primate social assent (PA) and increased 5-HT levels (cited in Rosenzweig, et al., 1996). However, the causality is questionable, for it could be said that social assent arises due to increased 5-HT levels and not vice-versa. Mehlman (1995) supports this, by finding monkeys with low social proficiency have lower serotonergic activity levels (cited in Carlson, 1998). It could therefore be inferred that 5-HT pays an inhibitory role in aggression. While Raleigh (1992) emphasises PA, Oreland (1983), emphasises IA (cited in Rosenzweig, et al., 1996). The author therefore suggests that the conflicting findings of directional variance of serotonergic activity levels may be attributed to the various types of aggressive behaviour. However, 5-HT is not an aggression transmitter, but merely a facilitator. In reflection, Soubrie (1986) proposed that, through the integration of several neurotransmitters, such behaviours might result when single neurotransmitters alone are unlikely to produce an effect (cited in Stoff & Cairns, 1996).

This essay has presented aggression and violence as being predominantly initiated by environmental stimuli (Trimble, 1996) where responses are mediated through a collection of genetic, biochemical and neurophsiological acts. Biological determinants of aggression and violence are proposed to be mediated through various changes or abnormalities in testosterone levels, MAO genes, the temporal lobes, parts of the limbic system [HT, AD, PAG and septal area], the OFC and 5-HT and its metabolite 5-HIAA. However, the causal relationship between social or simulated triggers and aggression is open to interpretation. Furthermore, due to the various behaviour responses recorded by stimulating cortical areas or altering neurotransmitter functioning, it is apparent that aggression is a complex behaviour and non-pathological. It is proposed that a larger interconnective approach could be used to consolidate the biological determinants listed above which mediate aggressive and violent behaviour.

References

- Adolphs, R., Tranel. D., Damasio. H., and Damasio. A. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature*. **372**, 669-672
- Archer, J. (1991) The influence of testosterone on human aggression. *British Journal of Psychology*, **82**, 1-28
- Atren, D., and Curthoys, I., (1996) *The Neurosciences and Behaviour: An Introduction.* Harcourt Brace. Australia.
- Backer, R.A., Barasi, S., and Neal, M.J., (2000). *Neuroscience at a glance.* Blackwell. *Hong Kong*
- Bradshaw, J.L and Mattingley, J.B. (1995) *Clinical Neuropsychology: Behavioural and brain science.* Academic press. USA.
- Brunner, H.G, Nelen. X.H., Breskefield, H.H., and Ropers Van Oost, B.A. (1993) Abnormal Behaviour Associated with a point mutation in the structural gene for monoamine oxidase A: *Science.* ***262,*** 578-580
- Chen. C., Rainnie. D.G., Greene. R.W., and Tonegawa.S., (1994) Abnormal fear response and aggressive behaviour in mutant mice deficient for Alpha-calcium-calmodulin kinase II. *Science:* **266**, 291-294.
- Coccaro, E.F., Kavoussi, R.J., Sheline, Y. I., Lish, D.J., and Csernansky, J.G. (1996). Impulsive aggression in personality disorder correlates with tritiated paroxetine biding in the platelet. *Archives of General Psychiatry.* ***53*,** 531-536.
- Coccaro, E.F., Siever, L.J., Klar, H.M., Maurer, G., Cochrane, K., Cooper, T.B., Mohs, R.C., Davis. K.L. (1989) Serotonergic studies in patients with affective and personality disorder: correlates with suicide and impulsive aggressive behaviour. *Archives of General Pyschiatry.* **46,** 587-599.
- *Carlson .G.R (1998) Physiology of behaviour: Allyn and Bacon*
- Higley, J.D., Mehlman, P.T., Taub, D.M., Higley, S.B., Suomi, S.J., Vickes, J.H., and Linnoila, M. (1992) Cerebrospinal fluid monoamine and adrenal correlates of aggression in free ranging rhesus monkeys. *Archives of General Psychiatry*. **49,** 436-441
- Hogg, M. A., and Vaughan, G. M., (1998) *Social Psychology:* Prentice Hall. Glasgow.
- Kawashima, R., Sugiuar, M., Takashi, K., Nakamura, A., Hatano, K., Ito, K., Fukuda, H., Kojima, S., and Nakamura, K. (1999). The human amygdala plays an important role in gaze monitoring; A PET study. *Brain.* **122,** 779-783.
- Lau, M.A., and Pilhl, R.O., (1995) Provocation, acute alcohol intoxication, cognitive performance and aggression. *Journal of Abnormal Psychology:* **104** (1), 150-155

- Lau. M.A., Robert. O.P., and Perterson. L.B. (1995) Provocation, acute alcohol intoxication, cognitive performance, and aggression. *Journal of Abnormal Psychology.* **104** (1) 150-155
- *Levine, D.S., (1991) Introduction to neural and cognitive modelling. LEA Publishers. London.*
- Limson, L., Goldman, D., Roy, A., et al., (1991) Personality and cerebrospinal fluid monoamine metabolites in alcoholics and controls. *Archives of General Psychiatry.* **48**, 437-441.
- Linnoila, M., Virkkunen, M., Scheinin, M., Nuutila, A., Rimon, R., and Goodwin, F.K., (1983) Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from nonimpulsive violent behaviour. *Life Sciences*, **33** 2609-2614
- Maes, M., and Coccaro, E.F., (1998) *Neurobiology and clinical views on aggression and impulsivity:* Wiley, UK.
- Marzuk, P.M., (1996) Violence, crimes, and illness. How strong a link? *Archives of General Psychiatry*. **53,** 479-484
- Mednick et al., (1977) Cited in Rosenzweig, M.R., Leimen, A.L., and Breedlove, S.M., (1996) *Biological Psycholog*y: Sinauer Associates, Inc.
- Peters, R.D., and McMahon, R.J. (1992). *Aggression and violence throughout the lifespan. S*age Publisher. London.
- Pietrini, P., Guazzelli, M., Basso, G., and Grafman, J., (2000) Neural correlates of imaginal aggressive behaviour assessed by positron emission topography in health studies. *American Journal of Psychiatry,* **157 (11)** 1773-1781
- Pinel. J.P.J., (1999). *Biopsychology*. Allyn and Bacon. USA.
- Reber, A.S., (1995) *Dictionary of psychology*. Penguin. St Ives.
- Rosenzweig, M.R., Leimen, A.L., and Breedlove, S.M., (1996) *Biological psycholog*y: Sinauer Associates, Inc.
- Schere, K.R., Abeles, R.P., and Fischer, C.S, (1975) *Human aggression and conflict: Interdisciplinary perspectives.* Prentice Hall. New Jersey.
- Shaikh, M.B., Schubert, K.J., and Siegel, A. (1994) Basal amygdala facilitation of midbrain preiaqueductal gray elicited defensive rage behaviour in the cat is mediated through NMDA receptors. *Brain Research:* **635,** 187-195
- Smythies. J.R., (1970) *Brain Mechanisms and behaviour.* Blackwell. Oxford.
- Stoff, D.M., & Cairns, R.B., (1996) *Aggression and Violence: Genetic, neurobiological and biosocial perspectives*: Lawrence Erlbaum associates New Jersey.

- Tebartz van Elst, L., Woermann, F.G., Leemiex, L., Thompsam, P.J., and Trimble, M.R. (2000) Affective aggression in patients with temporal lobe epilepsy: A quantitative MRI study of the AD. *Brain*. **123,** 234-243
- Toates, F., (1992) *Biology: Brain and Behaviour. Book 5 control of behaviour*. Open university press. UK.
- Trimble, M.R. (1996). *Biological Psychiatry.* Wiley. New York.

Key:

AD	→ Amygdala	MAO	→ monoamine oxidise
CFS	→ Cerebrospinal Fluid	NE	→ Norepinephrine
DA	→ Defensive aggression	OFC	→ Orbitofrontal Cortex
GABA	→ Gamma-aminobutyric acid	PA	→ Predatory aggression
FL	→ Frontal Lobes	PAG	→ Periaqueductal Grey Matter
HT	→ Hypothalamus	5-HT	→ Serotonin
IA	→ Impulsive aggression	5-HIAA	→ 5-hydoxyindole-3-acetic acid
LS	→ Limbic system		