

Evolutionary Special Interest Group of the Royal College of Psychiatrists



Evolutionary Psychiatry Special Interest Group (EPSIG)

Newsletter No.17 November/December 2019

Editors:

Dr Paul St John-Smith (Consultant Psychiatrist Single point Of Access Team, Hertfordshire Partnership University foundation Trust);

Dr Annie Swanepoel (Clinical Director CAMHS, Consultant Child and Adolescent Psychiatrist, Potter's Bar Hospital, Elysium Healthcare).

Contact Emails: -

p.stjohnsmith@nhs.net or paulstjohnsmith@hotmail.com

EPSIG Chair: Riadh Abed (Retired Consultant Psychiatrist and Medical Director; currently Medical Member of the Mental Health Tribunal Service in England) abedrt@btinternet.com

World Psychiatric Association (WPA) Evolutionary psychiatry email group moderator: Mohammed Abbas mohdgum@hotmail.com

Contents

1. Notes from the editor
2. Future EPSIG meetings
3. Conference Report: Ethology, Psychology, Psychiatry: An Evolutionary Approach. Ettore Majorana Foundation and Centre for Scientific Culture, Erice (Sicily)- Italy, 22-27 October, 2019
4. Student Article on "Biological explanations of aggression and the medicolegal implications" by Lavin Assad
5. Resources and EPSIG Website

Evolutionary Special Interest Group of the Royal College of Psychiatrists

1. Notes from the editor

We now have a fifth edition of the newsletter this year, as lots of exciting things are happening. We have a report from the Italian conference some of us attended with many of the big names in evolutionary psychiatry; Riadh Abed has written a brief report on the presentations.

There are 2 EPSIG conferences in the pipeline (see below) and a scientific meeting and a May 2020 EPSIG scientific meeting at the college on the Evolutionary Perspective on Childhood Trauma

We now have 6 confirmed keynote speakers for our 4th International EP symposium on 16 October 2020. They are: Prof Edward Bullmore, Prof Jonathan Hill, Prof Paul Gilbert, Prof Randolph Nesse, Ms Gul Deniz Salali and Ms Daniela Sieff

We are also planning to hold an additional conference in 2021 with the theme of 'The Evolutionary Roots of Attachment Theory'. Given the centrality of the attachment to modern psychiatric thinking especially in child development and its clear evolutionary origins it seems to be a logical subject for us to try to promote evolutionary thinking among our colleagues.

This will be over and above our regular EP symposium in 2021 and may be planned for March of 2021. We have 4 confirmed keynote speakers for this (in principle): Prof Martin Brune, Prof Jeremy Holmes, Dr Annie Swanepoel and Prof Marinus Van Ijzendoorne.

We are now including a student section in future newsletters. This student section of the newsletter would be peer-reviewed with a lighter touch than other articles to encourage contributions. Remember it is a newsletter, so popular science is OK as long as it is science. Articles on evolution and psychology/psychiatry will be the mainstay, though we are open to related topics. Up to 2000 words is fine. Projects and preliminary findings would be suitable too. Any appropriate contribution on human behaviour with an evolutionary slant can be considered. If in doubt, email us:-

abedrt@btinternet.com , paulstjohnsmith@hotmail.com or annie.panzer@gmail.com

We also are involved in a new project with a possible student/trainee essay prize, an important piece of advice to all authors is for them to consider using the Tinbergen 4 questions (Quadrants) to structure the essay. Evolutionary explanations are about populations and vulnerability; individual behaviours in the present are not solely explained by evolutionary theories although there may be evolutionary angles to be explored. An assumption evolutionary theories attempt to explain individual situations is a fundamental misunderstanding. Individual behaviours may be influenced by development, learning and culture, mechanisms, and context. For instance, the human vulnerability to experience bereavement may have various evolutionary threads, and explanations, but clearly evolution alone will not explain why or how a given individual is experiencing bereavement (with a specific set of symptoms) in the here and now.

We have included an example from the Forensic faculty from Ms Lavin Assad who has included a number of evolutionary ideas and hypotheses. As mentioned above, the essay would have benefited greatly from the application of Tinbergen's causal system. Simply organising the causes of aggression into mechanisms, developmental, phylogenetic and functional (causes) can help elucidate the kind of contribution evolution could make in understanding human aggression/violence/criminality.

However congratulations to Ms Lavin for making such a good effort in an area where students, trainees and indeed many doctors are in unfamiliar territory!

We are also planning for the 1 May 2020 for another half day scientific meeting on 'Evolutionary Perspective on Childhood Trauma'

Evolutionary Special Interest Group of the Royal College of Psychiatrists

We also hope to work with the RSM on a joint meeting on an interesting question:

‘Has Natural Selection Ceased to Affect Humans?’

Also next year we are planning to set up a ‘Charles Darwin Essay Prize’ for non-consultant grades in UK. This will involve writing on a topic related to both Darwinian evolution and psychiatry. We look forward to receiving some ideas or titles / topics to put forward to entrants.

Finally there was an election for college officers; this is required every 4 years. EPSIG officers are changing. Hence, from June 2020 Riadh Abed will become the financial officer, taking over from Agnes Ayton, Paul St John-Smith will become the Chair and Annie Swanepoel will become the newsletter editor. Of course we greatly appreciate the help and support of all EPSIG members and supporters who have assisted in organising and running previous and forthcoming EPSIG events and activities .

There is a college meeting of the Chairs of SIGs later this year (SIG Officers Day- 27 November 2019) where Riadh Abed will be advocating on behalf of EPSIG and discussing a number of issues with the college. We are pleased to report the EPSIG membership at the end of the month of October 2019 stands at 1266 (Paul St John-Smith Editor)

2. Meetings

You may wish to note the following the 2 confirmed dates for next year:

1/5/2020 AGM and half day scientific meeting on th Evolutionary Perspective on Childhood Trauma at the Royal College of Psychiatrists, with Prof Vivette Glover and Prof Zanna Clay as confirmed speakers.

and

16/10/2020 4th International EPSIG symposium at the Royal College of Psychiatrists

3. Conference Report

Ethology, Psychology, Psychiatry: An Evolutionary Approach

Ettore Majorana Foundation and Centre for Scientific Culture, Erice (Sicily) - Italy, 22-27 October, 2019 by Riadh Abed

This was a major evolutionary event with more than 20 speakers over 6 sessions. The setting was the breathtakingly picturesque mountain-top medieval Sicilian town of Erice with its ancient, cobbled narrow alley ways and numerous small boutique cafes and restaurants.

The speaker line-up included many of the top opinion leaders, authors and researchers in evolutionary psychopathology and psychiatry. Also, given the number and breadth of topics covered it would be impossible for me to do justice to this meeting in a short review. I will therefore attempt to highlight some of the more salient subjects covered and offer my apologies in advance for any omissions or other inevitable shortcomings or misunderstandings.

As in all conferences, networking with other evolutionists during breaks and mealtimes was as fruitful as listening to the presentations and discussions during formal sessions. Also, as a single

Evolutionary Special Interest Group of the Royal College of Psychiatrists

stream event (a rarity in large conferences nowadays) it was easier to discuss and exchange ideas with other delegates as everyone attended the same sessions.

It was pleasing to see that alongside seasoned evolutionists (of various disciplines) there was a group of Italian high school students together with their biology teachers; hopefully the next generation of evolutionary scientists. Also, it was notable that the delegates came from a wide range of disciplines including psychiatry, psychology, anthropology, primatology, microbiology and others.

The keynote address was delivered by Randolph Nesse, the co-founder of the modern evolutionary medicine movement worldwide (together with the late George Williams). Prof Nesse gave a panoramic overview of the current state of evolutionary thinking in psychiatry and medicine summarising in broad outline the contents of his most recent book 'Good Reasons for Bad Feelings'. This set the scene for the rest of conference. Nesse's approach advocates a radical rethinking of the nature of mental disorder considering both evolutionary and proximate causes guided by Tinbergen's causal system which he helped elucidate over the years. Concepts such as the smoke detector principle that helps explain why anxiety is so common and the cliff-edge phenomenon could only be conceived if we take an evolutionary perspective. The cliff-edge model stipulates that whenever maximal fitness is near to the cliff-edge, even a slight overshoot can lead to a catastrophic state of dysfunction we identify as mental disorder. This is likely to explain our vulnerability to psychosis.

There were 3 presentations on inflammation/infection and mental disorder. Two of them discussed the possible role of *Toxoplasma Gondii* (TG) in schizophrenia. TG is a protozoan parasite that can infect a number of warm-blooded animals including humans, however, the definitive hosts are members of the cat family including domestic cats and intermediate hosts include rodents and birds. TG produces behavioural changes in rats and mice that render them fearless of cats which increase their risk of being killed and eaten by cats thus serving the purpose of the parasite in getting into its definitive host. The finding of a possible causal role of TG in schizophrenia raises interesting evolutionary questions regarding the influence of TG infection on behaviour in humans. Martin Brune discussed this issue reminding us that there are multiple evolutionary pathways that lead to mental disorder and that infection can be one of these while Paul Ewald discussed TG infection and schizophrenia highlighting the interaction between genes and germs. However, while there is undoubtedly an evolutionary angle to the adaptation of the *Toxoplasma* parasite to humans as a new and non-definitive host, the evolutionary aspect from the point of view of humans in the causation of schizophrenia is less readily apparent. Is the disturbance in thinking and behaviour in schizophrenia analogous to the changes in behaviour that occur in rodents? The 3rd presentation on infection and immune response focused on depression and this was delivered by Holly Ewald. This is a very topical issue in psychiatry today and is being actively researched in a number of major centres but again the evolutionary angle needs further elucidation.

Marco Del Giudice presented the latest iteration of his evolutionary framework for mental disorder based on Life History Theory. He summarised and updated the ideas fleshed out in great detail in his book 'Evolutionary Psychopathology' published in 2018. His system classifies mental disorders along 3 axes which are: fast spectrum disorders (e.g. borderline personality disorder, psychopathy and schizophrenia spectrum), slow spectrum disorders (e.g. ASD, anorexia nervosa) and defence activation disorders (e.g. avoidant personality disorder, anxiety disorders). He presented a critique of the current psychiatric classification systems contending that they do not represent natural kinds and discussed eating disorders in some depth as an exemplar.

Evolutionary Special Interest Group of the Royal College of Psychiatrists

Alfonso Troisi's presentation involved a theoretical and philosophical critique of Wakefield's concept of 'Harmful Dysfunction'. In Troisi's view the evolutionary definition of dysfunction must be based upon ultimate causation as this redefines dysfunction as a factual concept that is disanchored from cultural values. However, Troisi acknowledges that while such a redefinition is theoretically useful and valid it cannot be directly implemented in clinical practice. Clinicians must operate within existing socio-cultural norms and hence focus on the immediate interests of individual patients and strive to reduce distress and suffering rather than focus on biological fitness or adaptation. Nevertheless, evolutionary thinking, according to Troisi, can help guide clinical practice through giving less weight to symptoms (than has been hitherto the case), greater weight to context, greater weight to observed behaviour and much greater weight to defining and assessing the individual patient's functional capacities in achieving vital biosocial goals. In this sense, it seems to me that Troisi's ideas expand on and enrich Wakefield's work rather than refute it.

There were excellent presentations on attachment theory by Marinus Van Ijzendoorne deriving lessons from studies on chimpanzees and rodents, the importance of ethology for human psychology and psychiatry by Paola Palanza and the importance of early experiences and brain plasticity by Pier Francesco Ferrari and the Nature-Nurture effects on neuro-development by Marian Bakermans-Kranenburg.

Some of the important messages from these presentations included the fact that Attachment Theory was the first application of evolutionary theory to human development after Darwin. The relevance of attachment theory has now been firmly established in the development of a range of species including humans although more robust data is still needed especially with regard to measurement of environmental factors (including stress). Another important message was regarding the evolutionary foundation of the phenomenon of differential susceptibility. Differential susceptibility is the capacity of the organism to respond to early experiences through modification of its developmental trajectory that results in the development of different phenotypic characteristics. This evolutionarily-based concept has now superseded and supplanted the older stress-diathesis model. Hence, individuals vary in their biological sensitivity to context (through gene-environment interactions) and this can have beneficial as well as negative consequences. Hence, a better understanding of these interactions can have important implications for intervention.

In this regard, Bruce Ellis' presentation was of particular interest. Ellis introduced the concept of stress adaptation whereby the consequences of stress on the developmental trajectory can simultaneously have both negatively and positively valenced consequences. This has been demonstrated in a range of species including birds, rodents and humans. The concept of stress-adaptation (or stress-adapted individuals) moves away from the one-sided and potentially stigmatising conventional concept of stress-damaged individuals by recognising that stress can lead to both negative as well as positive (and highly adaptive) phenotypic traits. This can have significant implications for future understanding of the effects of early-life stress and contribute to constructing more appropriate interventions designed to help stress-adapted individuals.

Other presentations included a behavioural ecology view of facial displays by Carlos Crivelli. His research on facial expression advocates a move away from a simple relationship between facial expression and internal emotional states to a more nuanced view that recognises that facial expression is a means of communication that is highly influenced by social context. According to this view one would not expect a one to one relationship between facial expression and the emotional state of the individual. This has been supported by the findings of cross-cultural research.

Evolutionary Special Interest Group of the Royal College of Psychiatrists

Joseph Polimeni presented a hypothesis on the evolutionary origins of humour which is that jokes optimise social norms and laughter synchronises social attitudes. His presentation was not only interesting but was also the most entertaining of the whole conference.

Gul Deniz Salali discussed the lessons we can learn from hunter-gatherers to use in preventive medicine. She presented findings from anthropological fieldwork by their team from UCL. She highlighted areas where we can gain greater understanding of health problems in the modern environment through studying the lives and social organisation of hunter gatherers. She proposed that modern society conditions give rise to a range of mismatches and these have health consequences. Areas of particular interest include:

Whether learning from childrearing practices in hunter gatherers can help reduce ADHD, anxiety & depression.

Whether learning more about social networks in hunter gatherers can help reduce anxiety, depression and schizophrenia.

Whether present orientation can help reduce anxiety & depression.

Whether physical activity levels can help reduce ADHD, anxiety & depression.

Whether diet and the attention to the microbiome can reduce anxiety & depression, cognitive dysfunction.

Attending this conference was a great experience for me and I am grateful to the speakers and organisers for setting this up. I hope to see even greater interest among psychiatrists both in the UK and world-wide in such cross-disciplinary evolutionary events in the future.

4. Student Essay Forensic faculty medical student essay submission

Biological explanations of aggression and the medico legal implications

Lavin Assad

Introduction

Jeffery Landrigan was a difficult child. Adopted at birth, he threw temper tantrums as a toddler, progressing to substance abuse and burglary by the age of 11. At 20, he killed his first victim. Imprisoned, he repeatedly stabbed an inmate and later escaped, committing his second murder. He was then sentenced to death. An inmate noticed a striking resemblance between Landrigan and Darrel Hill, a man on death row in another state. Hill, it transpired, was the biological father of Landrigan – both murderers, sentenced to die. Hill's father and grandfather had also been criminals. Despite being adopted, Landrigan was a fourth-generation felon. Does this mean that we can inherit behaviours? (Glenn and Raine, 2014)

This essay covers the neural, hormonal, genetic, and evolutionary factors that may play a role in causing aggressive behaviour. Whilst there are flaws within the research, the theories are of some use in the real world. The implications of the discussed theories apply both medically and legally. From a clinician's perspective, how can we explain, prevent, and treat behaviours that are deemed undesirable and/or harmful? In terms of the law, can the reasons for violent behaviours justify leniency in convicting people if they have no control over their physiology?

Evolutionary Special Interest Group of the Royal College of Psychiatrists

Biological mechanisms linked to aggression

Neurotransmitters

Research into neural factors often highlights serotonin as a key player in aggressive behaviour. Low serotonin is associated with impulsive behaviour, aggression and violent suicide. The neurotransmitter inhibits responses to emotional stimuli that could result in aggressive responses. Studies can either look at the levels of serotonin in people who have a history of violence, compared to those who do not, or can adjust the levels using drugs to see how behaviour may change. Linnoila and Virkkunen (1992) found consistently low levels of serotonin in violent offenders. This suggests that serotonin is important in impulse control. Fenfluramine is a drug that reduces serotonin uptake in the brain and has been linked to increased hostility. (Cherek and Lane, 1999) Antidepressants, which primarily aim to raise serotonin levels, have been linked to reduced aggression and irritability. (Bond, 2005)

Dopamine has a less well-established link with aggressive behaviour than serotonin but is still worth a brief mention as it relates to the genetic links discussed later. Amphetamines, which raise dopamine levels, result in aggression (Lavine, 1997) whereas antipsychotics, which lower dopamine levels, are associated with reduced rates of violent crime. (Fazel, 2014)

Hormones

Testosterone and cortisol are two steroid hormones linked to aggression. A low positive correlation has been found between testosterone levels and aggression in meta-analyses (Archer, 1991; Book et al., 2001). However, the methodological problems and differences between the studies mean that the coefficients, which are already low, may need to be lower. For example, Archer et al. (2005) claim that the coefficient in the meta-analysis by Book et al. (2001) should have been 0.08, rather than 0.14. Whilst this is not a strong correlation, there is a breadth of studies that have linked testosterone to aggression, suggesting it plays some role. Further studies include Dabbs et al. (1987), who found higher salivary testosterone in people with a history of violent crime than those with a history of *non-violent* crime, and Lindman et al. (1987), who found that men who are more aggressive when drunk have higher testosterone levels. Testosterone also has a role to play in evolutionary explanations of aggression, discussed in further detail below. The Challenge Hypothesis, proposed by Wingfield et al. (1990), states that males of a monogamous species (which humans may be loosely classified under) should not have a rise in testosterone above a baseline unless challenged. This surge of testosterone causes aggression in response to a threat that is deemed detrimental to reproductive success, such as threats to status or a dispute over a female.

Cortisol is thought to interact with testosterone levels to mediate its effects. Cortisol increases anxiety and social withdrawal, potentially reducing opportunities to be aggressive. (Dabbs et al., 1991) Low cortisol levels in habitual offenders (Virkkunen, 1985) and violent children (Tennes and Kreye, 1985) suggests that low cortisol increases the *likelihood* of aggression, in conjunction with testosterone.

Genetics

Research into genetic links to aggressive behaviour relies mostly on twin and adoption studies, looking at differences between people with the same, or similar, genetic makeup. Twin studies rely on the premise that, if aggressive behaviour is inherited, monozygotic twins (with near 100% identical DNA) will both show the trait. Dizygotic twins (~50% identical genes) should show lower concordance rates than monozygotic twins if the trait is genetically linked. It is assumed that the twins will have similar environments to each other, so differences are accounted to their genes. Adoption studies focus on children who are raised by people other than their biological parents (and family). If these children show aggressive behaviours similar to their parents despite not being raised

Evolutionary Special Interest Group of the Royal College of Psychiatrists

by them, it is assumed that this aggression was caused by genetic factors, rather than through observing their parents, as proposed by some psychological explanations.

A large Danish study found that, in 14,000 adoptions, adopted boys that had a criminal conviction usually had parents (especially fathers) with a criminal conviction (Hutchings and Mednick, 1975). In a review of 24 twin and adoption studies, Miles and Carey (1997) suggested up to 50% of aggressive behaviour can be accounted for by genetic factors. These results were repeated by Rhee and Waldman (2002) in a meta-analysis of 51 studies. However, both studies agreed that, whilst genes can make a massive contribution to the development of aggressive behaviour, their influence is moderated by several variables, including the age of the participants and their rearing environment. Further research focuses on particular outcomes of genetic variation in relation to the monoamine oxidase A (MAOA) enzyme. Brunner et al. (1993) studied a Dutch family known for being violent, with many of the men convicted of rape and arson. They were found to have abnormally low levels of MAOA, attributed to a defective gene. A second study by Caspi et al. (2002) evaluated 500 male children. Those with a gene variant leading to low levels of MAOA were significantly more likely to exhibit antisocial behaviour later on in life but *only* if they were abused. Those with high MAOA levels that were maltreated and those with low levels that were *not* maltreated did not generally display antisocial behaviour. This highlights that aggressive behaviour relies on the interaction between genes and the environment.

With a plethora of research linking genes and their sequelae to aggressive behaviour, we could easily make the mistake of assuming that our genome can cause us to commit crime. However, the research claims that inherited temperamentality only places individuals at a higher risk of committing crimes, with a triggering contribution from the surrounding environment. The highest rates of crime in adopted children occur when both the biological and adoptive parents have a history of crime. (Rhee and Waldman, 2002)

Evolution

Though many of us like to separate ourselves from the rest of the animal kingdom, we all evolved from common ancestors and have a drive to survive as a species. Evolutionary explanations rely on the fact that retained characteristics are either beneficial or otherwise not harmful to survival or else they would have been erased through natural selection.

The first broad explanation discusses the causes of violence in interpersonal relationships. Daly and Wilson (1988) consider the various strategies used by males to prevent their female sexual partners from committing adultery. Sexual jealousy stems from the fact that males could never be sure if the offspring of their female partner is theirs. Evolutionarily, this would mean a male would be investing his resources in a child that did not carry his genetic code, therefore male sexual jealousy exists to minimise this risk by deterring a female sexual partner from having sex with other males. This can range from vigilance to outright violence. This includes 'direct guarding' by reducing the partner's autonomy, as well as 'negative inducements' such as threats and violence if a male perceives infidelity in their mate. Female victims of domestic abuse often cite jealousy as a key cause of violence directed at them (Dobash and Dobash, 1984). The rates of domestic abuse double if the female is pregnant and their partner suspects the child belongs to another male (Burch and Gallup, 2004). Male sexual jealousy accounts for 17% of murders in the UK (Dell, 1984), as well as being the most common cause of killings in domestic disputes in the US (Daly et al, 1982). In same-sex killings (where the third person is killed), 92% were male-male murders, suggesting that sexual jealousy is almost entirely exclusive to males. Murder of a sexual partner could be an unintended outcome of physical violence as the purpose of aggression in these cases is to gain control over the (potentially) adulterous partner.

Evolutionary Special Interest Group of the Royal College of Psychiatrists

Research into sexual jealousy suggests that it is caused by physiological processes. Imagined scenes of infidelity triggers activation in the amygdala and hypothalamus in men, but less so in women (Takahashi et al, 2006). However, there is little research that considers the role of moderating factors such as which person is perceived to be responsible for the adultery (i.e. whether or not the male rival initiated the sexual encounters – if they happened).

The second evolutionary theory explains how aggressive displays in groups are an adaptive response to evolutionary threats. Our ancestors used aggression to protect their group, as well as benefitting them.

Xenophobia is the fear and hatred of those different from us and is documented in virtually all species that are highly socially organised (Wilson, 1975). Although altruism towards each other is favoured by natural selection, there is an evolutionary advantage to intolerance of strangers. By rejecting strangers, our ancestors did not risk exposing themselves to attack or illness, sparing themselves and their offspring. Exaggerating negative stereotypes is less risky than *under*perceiving threat for those wanting to protect themselves (MacDonald, 1992). This may be one of the underlying causes of racism and other intolerances that are still prevalent in society.

Territory protection can also explain aggressive displays. Aggressive behaviour was more adaptive for our ancestors who would have had to protect their land and its associated resources that were key to survival. Being territorial is associated with higher testosterone levels, as found in football players in 'home' games where they carry the burden of defending their own 'land.' This surge of testosterone at 'home' games also makes a team more likely to win than if they were playing 'away' (Neave and Wolfson, 2003). The claim that this is due to spectator support has been disputed by Moore and Brylinsky (1993), who showed that teams perform better without spectators at 'home' games. A more extreme situation relating to aggression is war. Though risking your life may seem evolutionarily disadvantageous, it can provide status which then elevates their reproductive fitness, resulting in more chances to mate and produce offspring (Divale and Harris, 1976). The inordinate risk displayed by members of a group during warfare signals their commitment to their group. The most obvious sign of this is scars and mutilation. By being perceived as dedicated to their group, our ancestors would have been able to reap the benefits from anything won in war against another group (mates, food, and shelter) (Irons, 2004). Scars and piercings inflicted within the group would also minimise absconion to other groups and force a person to be committed to their group, increasing the group's chance of survival (Thorpe, 2003).

Limitations within the research

Inconsistency

Different biological explanations could be incompatible. Low levels of MAOA are associated with aggression (Brunner et al., 1993) but the enzyme also breaks down serotonin so a deficiency in MAOA would also correspond to a higher concentration of serotonin. However, *low* levels of serotonin are associated with aggression (Linnoila and Virkkunen, 1992). The research has yet to specify if these things exist in different areas in the brain or if they are linked within the same pathways. This shows that the mechanisms are not yet fully understood and can only offer ideas of factors that influence our behaviours, rather than concrete explanations.

Correlation

Though many factors are *associated* with aggressive behaviour, they have not been established as a cause of it. In fact, the relationships could be reversed or bidirectional. Couppis and Kennedy (2008) found that high levels of dopamine associated with aggression could be a consequence of the behaviour, rather than the cause. Reward pathways in the brains of mice become engaged in response

Evolutionary Special Interest Group of the Royal College of Psychiatrists

to aggressive events and dopamine positively reinforces this. This could explain why people intentionally seek out aggressive encounters as it can provide them with a rewarding sensation.

Sampling bias and data collection

Most research focuses on people with convictions of violent crime. This is only a minority of aggressive behaviour as not all aggression results in a conviction. This means the population sample may not be representative of the entire population of people who show aggressive behaviour. Often, the convictions may be for a one-time offence and may not necessarily be the most serious or persistent offenders. This could be why the evidence for the heritability of aggressive behaviour is sometimes weak.

Much of the collected data on behaviour relies on surveys and/or retrospective data. These are not the most objective methods and are subject to various issues, from forgetfulness to social desirability. People may lie to make themselves seem more or less aggressive, depending on how they feel they should answer, or how they want to be seen.

Non-human animal research

The results of experimentation on non-human animals corroborate the findings in research on humans. For example, Raleigh et al (1991) found that vervet monkeys with lower levels of serotonin displayed more aggression. Popova et al (1991) found an increase of serotonin concentrations over generations in species that had been domesticated and bred for their docile temperaments. The problem with this, aside from the ethical debate of using animals, is that it reduces a social behaviour that is infinitely complex in humans down to a simpler physiological process just because there is a well-established link in other species. Whilst we may not be massively superior to other species, it is fair to say that the explanations given by biologists are insufficient. After millennia of human civilisation, do we really have no control over evolutionary traits? Do we exist only to survive and procreate?

Warfare as a genetic trait

LeBlanc and Register (2004) have considered that war may not be an evolutionary trait, but a circumstance of the environment. When humans shifted to a nomadic setting, they were tied to their agricultural and fishing sites. Walking away from danger is easier when you are a hunter-gatherer, but when you have more to lose by leaving an area, you have more to fight for, and fighting was a rational response in this case. Rather than being compelled by nature, aggression may be a result of developing populations and diminishing food supplies.

Gender

Research into aggressive behaviour has focussed mainly on males, meaning that the current explanations may not apply to females (or people with intersex conditions). Although testosterone is strongly associated with aggression in males, Archer et al (2005) found that the association between testosterone levels and aggression was stronger for females than males. Baucom et al (1985) took a different approach and found that females with higher testosterone levels had a higher occupational status due to more assertive behaviour. Additionally, Eisenegger et al (2011) found that testosterone may make females 'nicer' to others, depending on the situation; testosterone increases status-seeking behaviours and aggression is only one type of this.

The evolutionary explanations fail to explain why women can also commit acts of violence such as abuse and murder. In fact, women are twice as likely to kill out of jealousy than men (Felson, 1997). In evolutionary terms, females have much less to gain reproductively, even in war, so the role of the 'woman warrior' is unclear. (Adams, 1983)

Individual differences

Evolutionary Special Interest Group of the Royal College of Psychiatrists

Beyond gender differences, evolution cannot explain why people react differently to the same situation. For example, whilst aggressive mate-retention methods are used by some, others will cope with sexual jealousy by drinking alcohol or other (less violent) strategies (Buss and Shackelford, 1997). Evolutionary explanations often divorce free-will from biology, assuming that we cannot override a built-in survival mechanism, when we manage it every day, and have managed it as a species for countless generations.

Heteronormative bias

The fundamental flaw in evolutionary explanations is that they only focus on heterosexual relationships. Evolution relies on reproduction which was not accessible to cisgender homosexual couples, but this does not mean that homosexual relationships did not exist. Regardless of personal feelings on the topic, gay people exist and have relationships. Like any heterosexual couple, these can vary from healthy to abusive. Evolution cannot explain why sexual jealousy, in either males or females could result in violence, rendering this explanation somewhat futile beyond explaining why men abuse women.

Polyamory

Around the world, romantic-sexual relationships exist beyond two people. This can be cultural or individual and can include marriage (polygyny and polyandry). As with heteronormative biases, evolution cannot explain why violence does not consistently occur in these relationships despite the ample opportunity for jealousy.

Application beyond research

Although the limitations of the research are many, there is still some usefulness to the theories.

The Challenge Hypothesis (hormonal explanations)

Klinesmith et al (2006) found that males who were given a gun were more aggressive to each other than those who were given a child's toy in the study. They also had raised salivary testosterone. This supports the challenge hypothesis and has important implications in gun and knife crimes. If these are more readily available, they are more likely to be used. By having tighter controls on weapons and who can access them, we decrease the chance of their involvement in crime. However, this can easily mislead people into blaming the gun for shooting someone, when the person using the gun must also be held accountable for their actions. After all, not everyone who picks up a knife in the kitchen will go on to stab another person.

Xenophobia (evolutionary explanations)

Understanding xenophobia and its role in prejudice can help to educate populations that are privy to racism. For example, schemes such as 'Boys Against Bigotry' and 'Football unites, racism divides' have been deployed to tackle the racism that is rife in some football fans. Whilst no one should be prevented from loving and supporting their team, we all must work to make sure we do not allow prejudices to stop other people from also enjoying the sport.

Mate-retention (evolutionary explanations)

Raising awareness of the strategies used by romantic and/or sexual partners to keep their partner from cheating can help people alert their family and friends before violence even occurs. By seeking or being offered help earlier on, we may be able to reduce the amount of physical harm that people may suffer at the hands of their partners. This is especially important in the domestic abuse of men as they are less likely to report it or even recognise it (ONS BCS, 2016).

Implications for the Clinical Setting

Evolutionary Special Interest Group of the Royal College of Psychiatrists

Claiming that aggressive behaviour is a result of biological mechanisms has many implications, clinically and legally. It is important to treat this topic with sensitivity as it deals with cases such as rape and murder and may be interpreted as removing the blame, either wholly or partially, from the perpetrator.

It is important to look at the possible causes of something if we wish to treat or prevent it. Though aggression is not a medical condition, it features in various illnesses and is a symptom that many patients and their families want to reduce. Some medical causes of aggressive behaviour are: autism spectrum disorder (ASD), attention-deficit hyperactivity disorder (ADHD), bipolar disorder, schizophrenia, and brain damage (stroke, head injury) that limits your ability to control aggression (Healthline, 2016).

Sometimes, the underlying reason for behaving aggressively can be due to stress or frustration in communicating feelings, especially in children. These can often be reduced by acknowledging the underlying reason and making accommodations, such as using different ways of expressing oneself. This is particularly central in conditions where barriers to communication already exist, such as ASD. Beyond advice and psychological therapies to understand the reasons for someone's aggressiveness, clinicians can also prescribe medications that help. Mood stabilisers can be prescribed in bipolar disorder or schizophrenia that may help to regulate emotions, reducing aggression in those who present with it (Healthline, 2016).

When aggression is caused by a medical condition, a doctor can try to treat the condition to alleviate the symptom of aggression. However, if aggression continues, or exists outside of a medical diagnosis, how can we treat it? If low levels of serotonin are considered the cause of a person's aggression, treatment may be as simple as a course of antidepressants. But what other options exist? If a genetic predisposition is identified, should we screen for it and explore gene therapies? Should we consider rejecting or modifying embryos with identified genes? The answers to these questions are beyond the scope of this essay but may someday be pivotal to people's lives if these explanations are accepted as medically significant. Treatments built on biological theories will unleash many ethical debates as we are interfering with people's nature. The next section on legal implications will discuss this in further detail.

Currently, treating medical conditions and offering psychosocial therapies are the main remedies. It is important to consider the societal and environmental factors that trigger predispositions. Psychology can help us tackle these and teach people how to better regulate their emotions. However, the evidence on the effectiveness of these interventions is limited for some populations so other methods may need to be explored (Ali et al., 2015). Gene therapy for specific genes is developing constantly though may not be available in the near future. To replace a 'faulty' gene with one that we think functions better could be used in the reduction of aggression, but ethical debates must be considered, as well as the various logistical issues (Thome et al., 2011). Removing genes we deem 'faulty' from the genepool is effectively eugenics. When the genes relate to socially undesirable behaviours, we are tampering with the uniqueness of human nature, submitting ourselves to a dystopian world in which we are all expected to fit a mould of how to be human.

Implications for the Legal System

Glenn and Raine (2014) discuss three aspects of the legal implication of this research: punishment, prediction, and prevention. Prevention has been discussed above so will not be included in this section.

Punishment

Perhaps one of the most far-reaching questions in this area, can someone with a biological reason for committing a violent crime be held responsible for their actions? Glenn and Raine (2014) highlight the case of a schoolteacher who molested his stepdaughter. This was attributed to a brain tumour

Evolutionary Special Interest Group of the Royal College of Psychiatrists

which was resected. His behaviour returned to normal, but months later, his wife found child pornography on his computer. When this happened, it was found that the tumour had regrown. Its removal returned his behaviour to normal once again. Can this man be blamed when his actions were likely a direct result of a biological factor that he could not control?

In Thailand, a schoolteacher battered his wife to death after he found out she had met with a former boyfriend. This illustrates aggression due to sexual jealousy. The court gave him a suspended sentence of two years, sparking outrage from feminist groups (Head, 2002). If it is 'in a man's nature' to assault his partner for suspected infidelity, should he face legal action?

The degree of responsibility needs to be carefully assessed in every situation to determine if a person is blameworthy. Glenn and Raine (2014) suggest that criminals are "assessed on a continuum using measures that include neurobiological variables."

"Although a sensible dividing line needs to be drawn for practical reasons, in theory one can conceive of a set of multiple neurobiological and genetic influences that, combined with social influences, diminish responsibility to varying degrees. To the extent that neuroscience provides reliable methods to document these influences objectively, and assuming that methodologies become less expensive and quicker and easier to implement than hitherto, we anticipate that responsibility will eventually be conceptualized more broadly than it is today."

Prediction

Even if the relationships between biological factors is only correlational, there may be value in predicting the likelihood of a released prisoner reoffending (Raine, 2014). However, though we may be able to tell which people are *more likely* to be aggressive, we cannot precisely say whether they will go on to commit misdemeanours or more serious crimes, even if their history gives us some idea. It is easier to collect data on someone's behavioural, social, and psychological circumstances than their biology but if we can do this, should we? In the UK, police can take DNA samples of anyone they arrest, without the person's consent. Whilst some may argue that it is unethical to use offender's DNA, some may say we have a moral obligation to use it to enhance probation and parole decisions. Beyond this, there are far-reaching consequences of applying predictions to non-offenders. Labelling someone as predisposed to aggression can create stigma. Between this and self-fulfilling prophecies, people may face increasing psychological pressures that trigger the pathways resulting in aggression. Sometimes ignorance is bliss. In addition to this, false positive results would put those at lower risk of offending in a position where they are told they almost definitely commit crimes.

Furthermore, by saying our violent behaviours are out of our own control, will people be less inclined to inhibit themselves to follow norms, relying on their biology for mitigation?

An insidious implication of predicting someone's behaviour relates to eugenics. In a horribly dystopian world, those with less desirable biological characteristics may be forcefully sterilised in an attempt to eradicate aggression. More subtly, those with predisposing factors may be encouraged or feel that they are expected to be more careful if they are planning on having children. Some bright spark may even decide, as our proficiency in altering biology advances, that we should genetically engineer embryos so that they are less likely to be criminals.

Conclusions

Whilst this essay is by no means a comprehensive overview of biological mechanisms behind aggression, it gives us an insight into some of them. Despite limitations to the data, there is still some value in the findings. For a clinician, it can be useful to know what biological processes to tackle to reduce aggression. Often, treating underlying disease processes will be the solution but a myriad of pharmacological and psychological interventions could be explored, if necessary.

Evolutionary Special Interest Group of the Royal College of Psychiatrists

It is unlikely that a huge change to the legal system will occur in the near future because of this research, but there is potential for influence, both in deciding how to punish someone, and predicting rates of reoffending.

There is plethora of other mechanisms to be explored. This includes neuroanatomy, epigenetics, and perinatal mechanisms. These can help further influence our practices. Collating these different explanations must also be prioritised. The most useful part is to understand how the environment interacts with these to trigger or worsen behaviours. Though we should be aiming to reduce poverty and abuse anyway, this research may help to promote the need to improve the psychological wellbeing of those who are exposed to more triggers.

Therapeutically, the brain is the biggest target. Even if genes are linked to aggressive behaviour, there is an intermediate process in the brain before any behaviour is acted out. This means that it may be more beneficial to target the brain, rather than use gene therapy (if available). This could be through drugs, supplements or psychological interventions. Omega-3 supplementation is shown in randomised controlled trials to reduce up to 34-36% of serious offending in young people (Zaalberg et al., 2010 cited in Glenn and Raine, 2014; Gesch et al., 2002 cited in Glenn and Raine, 2014) whilst mindfulness has been shown to reduce aggressive behaviour in offenders (Himmelstein, 2010 cited in Glenn and Raine, 2014; Wupperman et al., 2012 cited in Glenn and Raine, 2014). Behaviour cannot be entirely divorced from biology but there are cognitive pathways we do not understand that may be tackled more effectively with psychological techniques.

Some recommendations from this research are:

- Reducing access to weapons (though the topic of further gun control in some countries is still controversial).
- Tackling prejudices in vulnerable groups, as shown in the sporting world.
- Minimising environmental risk factors that trigger aggressive behaviours (such as poverty or domestic abuse).

Regardless of where the research takes us, it is important to have thorough discussions about how to ethically implement any changes, or if they should be implemented at all. This is especially imperative for permanent treatments – another era of lobotomies is not the most desirable outcome, for the scientific community or the patient. At all stages, people should have as much autonomy over these decisions as possible, regardless of their criminal status. Many of the proposed questions here are unanswered and may remain unanswered for a long time but working together to understand and reduce aggressive behaviour in a thoughtful manner may help us achieve something that resembles more of a utopia than a dystopia.

References

- Adams, DB., 1983. Why there are so few women warriors. *Behaviour Science Research*, 18(3), pp.196-212.
- Ali, A., Hall, I., Blickwedel, J., Hassiotis, A., 2015. Behavioural and cognitive-behavioural interventions for outwardly-directed aggressive behaviour in people with intellectual disabilities. *Cochrane Database of Systematic Reviews*, issue 4.
- Archer, J., 1991. The influence of testosterone on human aggression. *British Journal of Psychology*, 82(1).
- Archer, J., Graham-Kevan, N., Davis, M., 2005. Testosterone and aggression: A reanalysis of Book, Starzyk, and Quinsey's (2001) study. *Aggression and Violent Behavior*, 10(2), pp.241-261.
- Baucum, DH., Besch, PK., & Callahan, S., 1985. Relation between testosterone concentration, sex role identity, and personality among females. *Journal of Personality and Social Psychology*, 48(5), 1218-1226.

Evolutionary Special Interest Group of the Royal College of Psychiatrists

- Bond, A.J., 2005. Antidepressant treatments and human aggression. *European Journal of Pharmacology*, 526(1-3), pp.218-225.
- Book, A.S., Starzyk, K.B., & Quinsey, V.L., 2001. The relationship between testosterone and aggression: A meta-analysis. *Aggression and Violent Behavior*, 6(6), 579-599.
- Brunner, H.G., Nelen, M., Breakefield, X.O., Ropers, H.H., van Oost, B.A., 1993. Abnormal behaviour associated with a point mutation in the structural gene for monoamine oxidase A. *Science*, 262(5133), pp.578-580.
- Burch, R.L., Gallup, G.G., 2004. Pregnancy as a stimulus for domestic violence. *Journal of Family Violence*, 19(4), pp.243-247.
- Buss, D.M., Shackelford, T.K., 1997. From vigilance to violence: Mate retention tactics in married couples. *Journal of Personality and Social Psychology*, 72(2), pp.346-361.
- Caspi, A., McClay, J., Moffitt, T.E., Mill, J., Martin, J., Craig, I.W., Taylor, A., Poulton, R., 2002. Role of genotype in the cycle of violence in maltreated children. *Science*, 297(5582), pp.851-854.
- Cherek, D.R., Lane, S.D., 1999. Effects of d,l-fenfluramine on aggressive and impulsive responding in adult males with a history of conduct disorder. *Psychopharmacology*, 146(4), pp.473-481.
- Couppis, M.H., Kennedy, C.H., 2008. The rewarding effect of aggression is reduced by nucleus accumbens dopamine receptor antagonism in mice. *Psychopharmacology*, 197(3), pp.449-456.
- Dabbs, J.M., Frady, R.L., Carr, T.S., Besch, N.F., 1987. Saliva testosterone and criminal violence in young adult prison inmates. *Psychosomatic medicine*, 49(2), pp.174-182.
- Dabbs, J.M., Jurkovic, G.J., Frady, R.L., 1991. Salivary testosterone and cortisol among late adolescent male offenders. *Journal of Abnormal Child Psychology*, 19(4), pp.469-478.
- Daly, M., Wilson, M., Weghorst, S.J., 1982. Male sexual jealousy. *Ethology and Sociobiology*, 3(1), pp.11-27.
- Daly, M., Wilson, M., 1988. Evolutionary social psychology and family homicide. *Science*, 242(4878), pp.519-524.
- Dell, S., 1984. *Murder into Manslaughter*. Oxford: Oxford University Press.
- Divale, W.T., Harris, M., 1976. Population, warfare, and the male supremacist complex. *American Anthropologist*, 78(3), pp.521-538.
- Dobash, R.E., Dobash, R.P., 1984. The nature and antecedents of violent events. *The British Journal of Criminology*, 24(3), pp.269-288.
- Eisenegger, C., Haushofer, J., Fehr, E., 2011. The role of testosterone in social interaction. *Trends in Cognitive Sciences*, 15(6), pp.263-271.
- Fazel, S., Zetterqvist, J., Larsson, H., Långström, N., Lichtenstein, P., 2014. Antipsychotics, mood stabilisers, and risk of violent crime. *Lancet*, 384(9949), pp.1206-1214.
- Felson, R.B., 1997. Anger, aggression, and violence in love triangles. *Violence and Victims*, 12(4), pp.345-362.
- Gesch, S.M., Hampson, S.E., Eves, A., Crowder, M.J., 2002. Influence of supplemental vitamins, minerals and essential fatty acids on the antisocial behaviour of young adult prisoners. *British Journal of Psychiatry*, 181, pp.22-28.
- Glenn, A.L., Raine, A., 2014. Neurocriminology: implications for the punishment, prediction and prevention of criminal behaviour. *Nature Reviews Neuroscience*, 15, pp.54-63.
- Head, J., 2002. Thailand frees wife-killer. *BBC*, [online]. Available at: <<http://news.bbc.co.uk/1/hi/world/asia-pacific/2138210.stm>> [Accessed on 14/09/2018]
- Healthline, 2016. Aggressive behaviour. [online] Available at: <<https://www.healthline.com/health/aggressive-behavior>> [Accessed on 12/09/2018]
- Himmelstein, S., 2010. Meditation research: the state of the art in correctional settings. *International Journal of Offender Therapy and Comparative Criminology*, 55, pp.646-661.
- Hutchings, B., Mednick, S.A., 1975. Registered criminality in the adoptive and biological parents of registered male criminal adoptees. *Proceedings of the Annual Meeting of the American Psychopathological Association*, (63), pp.106-116.

Evolutionary Special Interest Group of the Royal College of Psychiatrists

- Irons, W., 2004. An evolutionary critique of the created co-creator concept. *Zygon® Journal of Religion and Science*, 39(4), pp.773-790.
- Klinesmith, J., Kasser, T., McAndrew, FT., 2006. Guns, testosterone, and aggression: and experimental test of a mediational hypothesis. *Psychological Science*, 17(7), pp.568-571.
- Lavine, R., 1997. Psychopharmacological treatment of aggression and violence in the substance using population. *Journal of Psychoactive Drugs*, 29(4), pp.321-329.
- LeBlanc, SA., Register, KE., 2004. *Constant Battles: Why We Fight*. New York: St Martin's Press.
- Lindman, R., Järvinen, P., Vidjeskog, J., 1987. Verbal interactions of aggressively and nonaggressively predisposed males in a drinking situation. *Aggressive Behaviour*, 13(4).
- Linnoila, VM., Virkkunen, M., 1992. Aggression, suicidality, and serotonin. *The Journal of Clinical Psychiatry*, 53(10), pp.46-51.
- MacDonald, K., 1992. Evolution, the five factor model, and levels of personality. *Journal of Personality*, 63, pp.525-567.
- Miles, SR., Carey, G., 1997. Genetic and environmental architecture of human aggression. *Journal of Personality and Social Psychology*, 72(1), pp.207-217.
- Moore, JC., & Brylinsky, JA., 1993. Spectator effect on team performance in college basketball. *Journal of Sport Behavior*, 16(2), pp.77-84.
- Neave, N., Wolfson, S., 2003. Testosterone, territoriality, and the 'home advantage'. *Physiology and Behavior*, 78(2), pp.269-275.
- ONS BCS, 2016. *Focus on Violent Crime and Sexual Offences 2014/15*. [spreadsheet] Appendix table 4.28. Available at:
<<https://www.ons.gov.uk/peoplepopulationandcommunity/crimeandjustice/compendium/focusonviolentcrimeandsexualoffences/yearendingmarch2015/bulletintablesfocusonviolentcrimeandsexualoffences/yearendingmarch2015>> [Accessed on 14/09/2018]
- Popova, NK., Voitenko, NN., Kulikov, AV., Avgustinovich, DF., 1991. Evidence for the involvement of central serotonin mechanism of domestication of silver foxes. *Pharmacology, Biochemistry, and Behaviour*, 40(4), pp.751-756.
- Raleigh, MJ., McGuire, MT., Brammer, GL., Pollack, DB., Yuwiler, A., 1991. Serotonergic mechanisms promote dominance acquisition in adult male vervet monkeys. *Brain Research*, 559(2), pp.181-190.
- Rhee, SH., Waldman, ID., 2002. Genetic and environmental influences on antisocial behaviour: a meta-analysis of twin and adoption studies. *Psychological Bulletin*, 128(3), pp.490-529.
- Takahashi, H., Matsuura, M., Yahata, N., Koeda, M., Suhara, T., Okubo, Y., 2006. Men and women show distinct brain activations during imagery of sexual and emotional infidelity. *NeuroImage*, 32(3), pp.1299-1307.
- Tennes, K., Kreye, M., 1985. Children's adrenocortical responses to classroom activities and tests in elementary school. *Psychosomatic medicine*, 47(5), pp.451-460.
- Thome, J., Hässler, F., Zachariou, V., 2011. Gene therapy for psychiatric disorders. *World Journal of Biological Psychiatry*, 12(1), pp.16-18.
- Thorpe, IJN., 2003. Anthropology, archaeology, and the origin of warfare. *World Archaeology*, 35(1), pp.145-165.
- Virkkunen, M., 1985. Urinary free cortisol secretion in habitually violent offenders. *Acta Psychiatrica Scandinavica*, 72(1).
- Wilson, EO., 1975. *Sociobiology: The New Synthesis*. Cambridge: Harvard University Press.
- Wingfield, JC., Hegner, RE., Dufty, AM., Ball, GF., 1990. The "Challenge Hypothesis": Theoretical implications for patterns of testosterone secretion, mating systems, and breeding strategies. *The American Naturalist*, 136(6), pp.829-846.
- Wupperman, P., Marlatt, GA., Cunningham, A., Bowen, S., Berking, M., Mulvihill-Rivera, N., Easton, C., 2012. Mindfulness and modification therapy for behavioral dysregulation: results from a

Evolutionary Special Interest Group of the Royal College of Psychiatrists

pilot study targeting alcohol use and aggression in women. *Journal of Clinical Psychology*, 68(1), pp.50-66.

Zaalberg, A., Nijman, H., Bulten, E., Stroosma, L., van der Staak, C., 2010. Effects of nutritional supplements on aggression, rule-breaking, and psychopathology among young adult prisoners. *Aggressive Behavior*, 36, pp.117-126.

5. **Articles for the newsletter** We welcome submissions for future newsletters in the form of articles, reviews and interviews. Correspondence: Replies, suggestions and clarifications on articles are welcomed and may be printed/included in our next newsletter. Also, we welcome brief reviews of seminal articles where there is an evolutionary or other relevant conceptual angle (please include the weblink if the article is open access).

Please send any submissions to me at: - paulstjohnsmith@hotmail.com