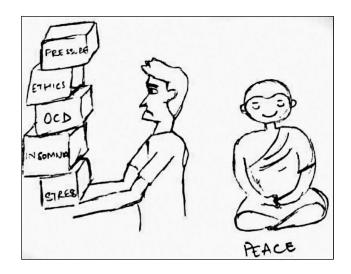
Neuropsychology of Aggression and Mental Illness

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Introduction

Examination of neuropsychological functioning, both in healthy populations and in individuals with brain injury, has provided important information with regard to lateralisation of cognitive function, gender differences in neuropsychological performance, functional differences associated with disconnection syndromes, and cognitive capacity at various developmental stages. To date a large body of research has focused not only on structural brain development, but also on the maturation of individual neuropsychological domains and the process by which these domains become integrated during



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development (Webb et al., 2001). Among cognitive domains, association between aggression and mental illness has been the focus of research due to the costs associated with anti-social personality disorders plaguing criminal justice system. Aggression in terms of physical or mental illness, often leads to behavioural problems both to the aggressor and victim. Aggressive tendencies at times closely relate to mental health challenges which an individual might be facing, if not properly diagnosed, might lead to abnormalities and harm.

In past two decades, developmental neuropsychology of aggression measured and predicted that human brain is plastic in nature and cortical specialisation is largely determined by interplay between brain activity and experience. Research has largely shown that the brain demonstrates remarkable plasticity during developmental stages, additional malleability of structure and function imbibes re-configuration during adulthood. We can conceptualise aggression as an integration of approaches at different levels wherein the brain operates in a plastic, self-organising fashion and, as such, is less constrained by predetermined boundaries than previously thought. Today, the notion of "nature versus nurture" has been set aside in favour of a newer, much integrated model in which processes and outcomes of development are viewed as products of bidirectional interchanges between biology and the environment. Bi-directionality implies that interactions with the environment have an impact on the course of biological development, which in turn affects behaviour and functioning, and these changes in behaviour further alter experiences.

Given this predicament of interaction between biological processes and environmental influences, unraveling mental illness among individuals with the neuropsychological approach proves to be a reliable technique. Studying neuropsychological processes may help our understanding of which brain regions are involved, and when and why they first show dysfunction. While anomalies of aggressive processes are good models that partly explain higher-level behavioural dysfunction, the link between aggression and mental illness will ultimately be critical to our understanding of the neuropsychological basis of neuropsychiatric disorder pertaining to violence. If neuropsychological models are to be able to inform treatment selection, and describe the mechanisms that underlie treatment response then integrating neuropsychological models with the significant clinical database which is organised around diagnostic entities will be required. Of course, eventually neuropsychological models may improve clinical diagnosis, by identifying which processes are unique to specific disorders (and their associated treatment responses), as well as those that are common across a range of disorders.

Over past three decades, since the publication of the influential article by psychiatrist George Engel (Engel, G. (1977), The Need for a New Medical Model: A Challenge for Biomedicine. Science, 196, 129–136, which was directed towards bridging the gap between psychiatrists and other medical professionals to enable "psychiatry to become better integrated with medical practice". The outcome of such concept is now known as "biopsychosocial model". The model is now used worldwide with a notion that each individual is a biological as well as a psychological being consisting of subjective feelings and is considered to be a part of diverse social groups. Despite such a serious attempt to find a unitary home for the previously disassembled humans, the model vividly distinguish between the biological, psychological and social aspects of "mental" illness. Philosophers still assume that human beings exist in two separate worlds, i.e., "physical" and "mental". The spectre of the proverbial Cartesian dualism of a res extensa, the world of matter, and the res cogitans, the world of the mind, still persists. The title "neuropsychology," connecting the "mental" and the "neurological" concepts, precisely defines the ideology that the mental world is clearly based on the neural circuits of the brain. Patients experiencing mental disorders show a high risk of developing aggressive behaviour throughout their lifetime (Volavka, 2013). Reducing the risk of violent and aggressive behaviour inpatients with schizophrenia remains a clinical priority. Hence, with this aim we provide a remarkable resource of modern advances in the field of aggressive behaviour and mental illness.

Assessing Aggression and Mental Illness: Methodological Considerations

Current section focuses on summarising the research conducted on the neuropsychology of aggression explaining methodological and procedural barriers that might arise during reading and interpreting or conducting research on aggression and its effect on well-being. One of the approaches in the field of neuropsychology involves the comparison of neuropsychological behaviours of individuals with a different diagnostic dimension. A prominent example of this approach is the difference seen in the individuals with a known brain damage or an individual with a disease without a specific defined etiology. As a result, this approach leads to a significant progress in identifying and explaining the relation between damaging of the brain along with behavioural and psychological functioning (Lezak, 1983). In addition to this approach the neuropsychological functioning of individuals with differentiated "aggressiveness" is also studied in the field of aggression. Aggression risk assessment is a process of identifying patients who are at greater risk of aggression in order to facilitate the timing and prioritisation of preventative interventions. Clinicians should base these

risk assessments on empirical knowledge and consideration of case-specific factors to inform appropriate management interventions to reduce the identified risk (All nut et al., 2013). An attempt to study "aggressiveness" as an independent variable was undertaken to understand criminal behaviour, however no significant results were observed as it was conducted on incarcerated offenders (Trasler, 1987). Studies focusing on offenders used numerous methods such as self-report inventories for measuring criminality, severity, chronicity, and recidivism.

Another approach is examining individuals who exhibit "antisocial" or "psychopathic" behaviour on the basis of difference in aggressiveness. Hare & Connolly (1987) have summarised the difficulties and issues encountered in operationally defining psychopathy. In a clear summary by Wolf (1987) he suggested that there is no universal meaning in the scientific community for the term antisocial. In spite of such difficulties, the research groups have shown significant advancement in studying "antisocial" and "psychopathic" behaviour, mainly focusing on analysing behavioural component (e.g., Hare & Connolly, 1987; Raine & Jones, 1987). However, the most stringent operational definitions of "antisocial" or "psychopathic" behaviour typically involve criminal behaviour with extreme severity. Such definition often creates difficulties for operationalising the criminality. Recently, researchers started use of self-reported inventories to study the individual differences with the context of family violence. However, the major concern in using these self-reported instruments involves the base rate in the general population for the aggressive behaviour being studied and the ability of such instruments in improving the identified base rate.

Stating further, one of the issues considered to be essential in comparing the groups which differ in aggressiveness are the variables which causes differences between the groups. Such cofounding variables includes: socioeconomic status, marital status, age, family size, and gender. Similarities in subjects found in these potential confounding variables eliminates the possibility of group differences due to these variables. Although, identifying such similarities among individuals on these dimensions is a tedious task, therefore, researchers have adopted more "loose" matching criteria, whereas others use more "tight" matching procedures. For example, trying to statistically equate two groups of subjects on several demographic dimensions is a relatively "loose" matching procedure, whereas comprising two groups of subjects in matched pairs that are equivalent on several demographic dimensions is a more "tight" matching procedure. Anecdotally, it is considered that the "tight" matching procedures can lead to further efficiency and confidence among the researchers for any observed group differences.

In the preceding section, aggressiveness was discussed as an independent variable reflecting individual differences. In contrast, studies have also focused on aggressiveness as a dependent variable. Instrumental aggression is typically defined as purposeful, in order to obtain some desired goal, whereas, hostile aggression is typically seen as rewarding in and of itself. Various methodologies have been developed to measure aggressiveness as a dependent variable (Kaplan, 1984 and Konecki, 1975). Pen-and-pencil based inventories are one of the methods which are widely used by the researchers (Buss & Durkee, 1957; Myer & Megargee, 1977). For instance, the Buss-Durkee Inventory includes items which are designed to assess seven types of aggressive and hostile responses. Megargee and his colleagues (Megargee & Bohn, 1979) have used Minnesota Multiphasic Personality Inventory (MMPI) to characterize convicted criminals based on their personality profile. Although both of these approaches are widely used, however, questions are raised in identifying the degree to which these types of self-report measures can be correlated with overt aggressive or violent behaviours (Edmunds & Kendrick, 1980). Another approach which can be used to assess aggressiveness involves the laboratory based tools that purportedly deliver electric shocks of varying magnitude to a subject who is actually a confederate (Konecki, 1975). Moffitt (1993) proposed that 'neurocognitive deficits present from an early age are a key mechanism underlying the expression of serious and persistent antisocial behaviour that emerges in childhood and continues throughout the life course.' Current literature evolved with measurements of autonomic and central nervous system activations and popularly utilising brain imaging techniques.

Autonomic Nervous System Activity and Aggression

In the last two decades, brain functioning of criminals has frequently been the subject of neuroscientific investigations (Raine & Young, 2006; Raine, 2013). There may be significant interest areas and researches associated with aggression in criminals or psychopaths concerning to the events in the autonomic nervous system (Hare, 1978; Schalling, 1978; Siddle, 1977; and Venables, 1987). Individuals who are prone to physically assaulting their children or are at risk for such assaultive behaviour is the second area of research interest to investigation the autonomic responses (Disbrow et al., 1977; Friedrich et al., 1985; Frodi & Lamb, 1980; Pruitt & Erickson, 1985; Stasiewicz & Lisman, 1989; Wolfe et al., 1983). Interestingly, the theories behind the above two research approaches are prominently different. Studies on criminogenic behaviour or psychopathic traits assume that individuals with psychopathic personality traits have low-arousal in terms of autonomic and/or central nervous system functioning (Zahn, 1986). Such low level of arousal is considered to be

a causal factor of individuals with psychopathic personality who manifest little reaction to punishment and appear erratic, and sometimes aggressive in stimulating acts. On the contrary, in response to stressful stimuli physical child abusers and those at risk for child abuse show an increased autonomic arousal which is an important precursor to more impulsive and aggressive responses.

A key concept which is essential in studies focusing on autonomic functioning is the discrimination between tonic and phasic activity. Often the distinction between the two terms is unclear; tonic activity measures the resting level of autonomic response, whereas phasic activity measures the momentary change in autonomic activity. Differences found in individuals between resting or tonic levels of autonomic response may be an indicator of differential arousal levels, or an indication that the subjects in the experiment were pre-occupied in different activities prior to the experiment. For example, smoking a cigarette, drinking a cup of coffee, or climbing a set of stairs prior to participation in a laboratory experiment can produce significant elevations in heart rate, particularly during the initial stages of an experiment.

Therefore, the experimenters should ensure that the participants must refrain from such activities prior to the experiment. In addition, the use of adaptation periods at the beginning of an experiment can decrease the potential contribution of such activities to group differences. Changes in phasic activity autonomic system occurs in response to a change in the condition of stimulus introduced by an experimenter. Studies focusing on psycho-physiological components aims to demonstrate differential changes in autonomic activity of the individuals who are prone to abuse or psychopaths as compared to more "normal" control subjects in response to specific types of stimuli. One of the major factors in evaluating phasic-autonomic changes is the habituation of autonomic activity during an experiment. Autonomic activity is typically higher during the initial stages of an experiment as compared to the later stage because of the novelty of the situation and the associated arousal of the subject. Using an initial adaptation period may obstruct some parts of the habituation, but it seldom eliminates entirely the general downward trend of psycho-physiological activity that occurs overtime in an experiment.

The magnitude of phasic changes in autonomic activity during the course of an experiment depends, in part, on the tonic level of activity upon which the phasic changes are superimposed. In general, many research scholars focusing on aggression and autonomic activity such as heart rate and electrodermal conductance reflects the general arousal level of the autonomic nervous system. However, it is not necessary that all components of the autonomic nervous system

will respond equally in stressful or arousing stimuli. (Lacey, 1959; Lacey & Lacey, 1958). Hence, there can been an increased level of heart rate with relatively small changes in electrodermal conductance in one subject during a stressful situation or vice versa. Such differential autonomic patterns in different subjects are referred to as individual response specificity (Andreassi, 1980), and it has been suggested that the measurement of a single type of autonomic arousal cannot represent the overall autonomic arousal in general terms. Therefore, researchers tend to measure more than one autonomic variable in order to prevent such issues. However, such measures lead to different results for one autonomic variable as compared to other variables. The problem then arises on the interpretation of statistical increase or decrease in only one autonomic variable and no other variables. Hence, the measurement of desired multiple autonomic variables can often lead to difficulties in examining the distinction between different autonomic arousal in subjects.

A variety of different concepts other than arousal can be used for examining autonomic variables associated with aggression. The concept of inter-stressor stereotopy of response (Ax, 1953; Engel, 1960, 1972; Lacey et al. 1963) suggests that differential patterns of autonomic response occur during different affective and cognitive stimulus situations. For instance, it has been found that decreased level of heart rate can be an indicator of increased attention to environmental events (Lacey & Lacey, 1970). Frodi (1978) and Frodi & Lamb (1980), citing research by Schachter (1957). Similarly, Geen et al. (1975), suggested that subsequent increase in diastolic blood pressure may reflect feelings of aversion, anger, or a disposition to aggress. Although, facial muscle tension is not an autonomic variable, however, few psycho-physiological studies are also focused on identifying differential patterns of facial muscle tension during specific affective states (Fridlund & Izard, 1983 and Cacioppo & Petty, 1981). Therefore, such psycho-physiological components of aggression can be a focus of researchers interested in this area of study.

Central Nervous System Activity and Aggression

Electroencephalography (EEG) and cerebral Event-related potential (ERP) are the two major approaches for measuring the electrical activity of the brain. Numerous research studies have attempted to examine differential cerebral functioning by means of EEG recordings in identifying the criminal behaviour. (Flor-Henry, 1976; Gorenstein, 1982; Mednick & Volavka, 1980; and Syndulko, 1978). Only few researchers have focused on evaluating the ERP correlates associated with criminal behaviour. There are various different sources for additional information related to these approaches (e.g., Donchin et al., 1986;

Hillyard & Hansen, 1986; johnson, 1980; Picton, 1980; Shagass, 1972). Most of the research that has been conducted on EEG activity and criminal behaviour has relied upon visual inspection of EEG recordings and subsequent classification of the activity as indicative of normal or abnormal cerebral function. The studies have found a relatively slow EEG activity particularly in alpha frequency band (8-13 Hz) in criminal populations, however, non-significant results have also been reported. (Driver et al., 1974; Gibbs et al., 1945). Volavka (1987) highlighted the significant difficulties in defining EEG "abnormality" and in ensuring that different judges utilise the same criteria in making such a diagnostic statement.

In quantifying EEG data, researchers are typically interested in the occurrence of particular frequency bands and the amplitude of the EEG wave forms within each band. The technique which is widely accepted in analysing EEG signals is spectral analysis. It is basically a statistical procedure which yields an estimate of the spectral power at various frequencies of the EEG signal. Spectral power determines a combination of the probability of occurrence of a particular frequency and the amplitude of the waveforms within that frequency. A statistical combination of frequency and amplitude into a composite measure has therefore resulted in the increased acceptance and popularity of spectral analysis. The popularity of spectral analysis is likely due to the statistical combination of frequency and amplitude into a composite measure. However, the unique and separate contributions of frequency and amplitude to the spectral power cannot be differentiated, once the composite measure of spectral power has been calculated.

The ERP literature on aggressiveness and/or criminality is comparatively highly popular than work on EEG correlates. The EEG recording and sampling is relatively at a fast rate (frequently as fast as once every millisecond) in ERP research. Typically, the sampling of the EEG recording is time locked to a brief (usually a few milliseconds) stimulus event. Time taken for sampling is not more than 1-2 seconds and is associated with the stimulus event. The stimuli are presented in large numbers and the subsequent samples associated with each stimulus are averaged together for the ERP recording. The "noise" inherent in the background EEG recording is eliminated through the averaging procedure, thus allowing the ERP signal to emerge. Since, the evoked potential generated are relatively small as compared to the noise in the background EEG, this inturn creates the possibility of the development of potential artifact. Eyes are considered to be one of the best known sources of non-cerebral artifact. Eye blinks and movements of the eyes are associated with shifts in the standing potential between the cornea and retina of the eye. Such movements and shifts

can create an artifact in the ERP data and as a result can affect the EEG recording. A variety of procedures have been suggested for detecting and eliminating this contamination (see Picton, 1980 and Donchin et al., 1977). Recently, a wide variety of imaging techniques have been developed to provide images of the brain (Nietzel & Bernstein, 1987). Such techniques include: computerized axial tomography (CAT scans), positive emission (trans-axial) tomography (PET scans), and magnetic resonance imaging (MRI). These techniques provide clear images of portions of the brain and indicate anatomical problems in cerebral tissue. In addition, alterations in brain functioning associated with variations in metabolic functioning can be reflected in PET techniques. These techniques can be used to assess the brain functioning of individuals with various kinds of psychopathology (Nietzel & Bernstein, 1987). The application of these approaches is useful in assessing aggression. However, it should be remembered that each of these techniques yield only a momentary "snapshot" of the brain at one point in time.

Traditional Neuropsychological Assessment and Aggressiveness

Lezak (1983) suggested that "neuropsychological assessment is a...method of examining the brain by studying its behavioral products" (p. 16). This statement highlights the behavioural element of neuropsychological assessment; that is, in conducting a neuropsychological evaluation the examiner measures the behaviour of the subject and then draws inferences about areas in the brain that may be dysfunctional based on the pattern of behaviooral data. The application of neuropsychological test to assess individual traits or abilities lies on the ideology that several disease or damage in discrete location in the brain is associated with specific behavioural changes. Studies have attempted to integrate existing neuropsychological assessment devices within a comprehensive theory of brain-behaviour relationships (e.g., Golden, 1981). One of the theories which have been widely used is given by Luria (1966, 1973). The theory defines that behavioural changes in patients with cortical damage can be described by the functioning of the cerebral cortex. The concept which determines that specific areas in the brain are associated with specific behavioural pattern is referred to as functional localization (Lezak, 1983, p. 83). Although this concept is an oversimplification of the complexity of cortical functioning, the notion that behavioural functioning is linked to cortical localisation is one that has been well established for many types of behaviours. There are two major neuropsychological test batteries, the Halstead-Reitan Neuropsychological Test Battery (Reitan & Davison, 1974) and the Luria Nebraska Neuropsychological Test Battery (Golden, 1981; Golden et al., 1980). In addition, the Wechsler scales of intelligence (Wechsler, 1958, 1974, 1981), although not originally intended to assess neuropsychological functioning, are frequently employed in neuropsychological assessment, with other more narrowly focused tests (Kendall & NortonFord, 1982).

Anti-Social Behaviour, Executive Dysfunction and Mental Illness: The Dark Triad in Brain

Impairment in neuropsychological functioning of an individual might be a key mediating process which interplays the pragmatic effects of genetic and psychosocial developmental trajectory on Anti-Social Behaviour (ASB) (Friedman et al. 2008; Raine and Yang 2006; Yang, Glenn, and Raine 2008). In recent decades, deficits in executive functions and response inhibition have been associated with brain dysfunction in relation to anti-social behaviour. Impairment in executive functions (EF) increases the probability of engaging in ASB and decreases behavioural inhibition, and as a result individual fails to anticipate behavioural consequences and assess punishment and reward, which in-turn affects the capability to generate socially appropriate behaviour in challenging contexts (Giancola 1995; Ishikawa and Raine 2003; Seguin 2008). Deficits in executive functions have been consistently linked with various associated factors of ASB such as criminality, delinquency, physical aggression, conduct disorder, psychopathy and antisocial personality disorder (Morgan and Lilienfeld 2000). Patients with frontal lobe dysfunction most commonly exhibit EF impairments; however, EF impairments are also evident among patients with damage to other brain regions. The frontal cortex, particularly the prefrontal cortex (PFC), plays a central role in mediating EF processes, although efforts to localise EF processes to discrete frontal areas have produced equivocal results (Ardila 2008; Collette et al. 2005; Duncan and Owen 2000; Stuss and Knight 2002; Tanji and Hoshi 2008). Recent research evidence indicates that optimal performance on EF tasks depends on the integrity of the whole brain (Collette et al. 2005; Funahashi 2001; Prabhakaran et al. 2000; Stuss and Alexander 2000). EF impairments have also been found in a wide range of neuropsychiatric and medical disorders, including schizophrenia, major depression, alcoholism, structural brain disease, diabetes mellitus and normal aging (Royall et al. 2002).

Studies in recent decades have also highlighted that general psychopathology is more strongly associated with EF impairments as compared to specific psychiatric illnesses (Stordal et al. 2005). EF measures are generally designed to capture clinically significant performance in experimental settings (Burgess et al. 2006; Chan et al. 2008; Chaytor, Schmitter-Edgecombe, and Burr 2006). Deficits in EF experienced by a large proportion of antisocial individuals are likely to be sub-clinical and representative of individual differences rather than

pathology in EF abilities. These individual differences in EF abilities associated with ASB may produce subtle impairments that impact on the regulation of everyday behaviour. However, it should also be noted that the existence of EF pathology in specific subgroups of antisocial individuals, including serious and persistent antisocial individuals can be initiated at a young age (Moffitt 1993). EF commonly comprises of a broad range of cognitive abilities and can be assessed by a limited range of tests. Since, a single measure cannot measure all the components of EF, therefore, a wide variety of batteries are used to assess EF in individuals. Examples of EF test batteries include the Behavioural Assessment of the Dys-executive Syndrome (BADS; Wilson et al. 1996), the Cambridge Neuropsychological Test Automated Battery (CANTAB; Robbins et al. 1998), and the Delis-Kaplan Executive Function System (D-KEFS Delis, Kaplan, and Kramer 2001).

Antisocial behaviour is a complex construct, as it encompasses a diverse range of socially unacceptable behaviours, therefore it cannot be conceptualised in a single theoretical framework (Rutter, 2003). Antisocial behaviours are categorised according to three major categories: clinical psychiatric diagnoses, the violation of legal or social norms and aggressive or violent behaviour. Clinical diagnostic categories most frequently associated with ASB are CD, Oppositional Defiant Disorder (ODD), Antisocial Personality Disorder (ASPD) and psychopathy. CD is defined as a pattern of persistent behaviour characterised by the violation of the rights of others or major age-appropriate norms and is usually diagnosed after the age of 9 years but not after 18 years (American Psychiatric Association, 2000). Examples of such behaviours include aggression, property destruction and theft. ODD is associated with persistent patterns of negativistic, hostile, defiant, provocative, and disruptive behaviour and is usually diagnosed after 9 years but not after 18 years (American Psychiatric Association, 2000). ASPD is characterised with a persistent pattern of behaviour characterised by a disregard and violation of the rights of others. The diagnosis of ASPD requires the diagnostic features of CD before 15 years of age and cannot be diagnosed before the age of 18 years (American Psychiatric Association, 2000). Psychopathy is characterised by a lack of empathy or insight for the effect of one's behaviour on others, callous, shallow and superficial traits, and behavioural characteristics including impulsiveness and poor behavioural control (Hare 1996).

Although these disorders often involve persistent deviant or criminal behaviour, they are not synonymous with crime (Rutter, Giller, and Hagell 1998). The ASB pertaining to physical aggression or violent behaviour most commonly refers to engagement in behavioural aggression directed towards others, including

bullying, initiating physical fights, using a weapon and causing serious physical harm. Similarities between the features of ASB and deficits in EF highlights the EF processes are considered to be an important factor in examining ASB. However, it must be noted that current evidences linking ASB and EF does not clearly support the conclusion that EF underlies ASB in a causal manner. The observation of EF impairments among antisocial individuals does not explain how such impairments develop over time and may lead to ASB. Morgan and Lilienfeld (2000) conducted a meta-analysis to quantify the association between ASB and EF. The findings suggested an association between ASB and EF that held across varying study methodologies. This meta-analytic review remains as the only systematic quantitative review of studies examining the relationship between ASB and EF, with narrative reviews being more common (e.g., Brower and Price 2001; Hawkins and Trobst 2000; Ishikawa and Raine 2003; Seguin 2008; Teichner and Golden 2000).

Concluding Remarks

The causal mechanism for aggressive behaviour is still a major concern in mental health domains. The identification of subtypes of aggressive behaviour has opened new vistas in unraveling mental illness among such individuals. Neuroimaging studies may further help elucidate the interrelationship between neuro-cognitive functioning, personality traits, and antisocial and violent behaviour. The association between ASB and EF impairments has implications for the treatment of ASB. EF abilities may be targeted to improve treatment effectiveness and reduce the likelihood of future ASB. Recent evidences have highlighted that poorer EF is associated with a range of negative treatment outcomes among offenders, including increased treatment drop out and increased disruptive behaviour during treatment (Fishbein and Sheppard, 2006). Treatment programs that aim to improve EF abilities may be useful in reducing the occurrence of ASB, particularly in children. There is evidence to suggest that cognitive enhancement programs can be effective in improving the development of EF abilities among preschool children (Diamond et al., 2007).

It is therefore important to examine the effectiveness of cognitive enhancement programs in preventing the development of ASB. This highlights the need of further research to examine specificity in impairments across types of antisocial individuals and measures of EF, factors that may moderate the association (ADHD and substance abuse), and the role of EF development in the expression of ASB. A concerted effort from researchers is needed in examining EF and ASB from theoretical frameworks to better specify the constructs. Despite the challenges, we should strive to find a way to give priority

to emotional and psychological support to patients alongside task-based medical care. Research in different areas appears to be converging on similar results and needs to be integrated to elucidate a comprehensive model of the effects of aggression, taking into account cognitive, emotional, and neurobiological development.

References

- 1. Allnutt, S.H. et al. 2013. Managing Aggression and Violence: The Clinician's Role in Contemporary Mental Healthcare. *Aust N Z J Psychiatry*; 47: 728-36.
- 2. American Psychiatric Association. 2000. *Diagnostic and Statistical Manual of Mental Disorders: Text Revision*. 4th ed. Washington, DC: American Psychiatric Publishing.
- 3. Andreassi, J.L. 1980. *Psychophysiology: Human Behavior and Physiological Response*. New York: Oxford University Press.
- 4. Ardila, Alfredo. 2008. On the Evolutionary Origins of Executive Functions. *Brain and Cognition* 68: 92-99.
- 5. Ax, A.R. 1953. The Physiological Differentiation between Fear and Anger in Humans. *Psychosomatic Medicine*, 15, 147–150.
- 6. Buss, A.H., & Durkee, A. 1957. An Inventory for Assessing Different Kinds of Hostility. *Journal of Consulting Psychology*, 21, 343–349.
- 7. Cacioppo, J.T., & Petty, R.E. (1981). Electromyograms as Measures of Extent and Affectivity of Information Processing. *American Psychologist*, 36, 441–456.
- 8. Collette, Fabienne. et al. 2005. Exploring the Unity and Diversity of the Neural Substrates of Executive Functioning. *Human Brain Mapping*, 25: 409-423.
- 9. Delis, Dean, Edith Kaplan, & Joel Kramer. 2001. *Delis-Kaplan Executive Function System*. Odessa, FL: Psychological Assessment Resources.
- 10. Disbrow, M.A., Doerr, H., & Caulfield, C. 1977. Measuring the Components of Parents' Potential for Child Abuse and Neglect. *Child Abuse & Neglect*, 1, 279–296.
- 11. Donchin, E., et al. 1986. In M.G.H. Coles, E. Donchin, & S.W. Porges (eds.), *Psychophysiology: Systems, Processes, and Applications*, New York: Guilford Press, 244–267.
- 12. Driver, M.V., West, L.R., & Faulk, M. 1974. Clinical and EEG Studies of Prisoners Charged with Murder. *British Journal of Psychiatry*, 125, 583–587.
- 13. Duncan, John, & Adrian M. Owen. (2000). Common Regions of the Human Frontal Lobe Recruited by Diverse Cognitive Demands. *Trends in Neuroscience*, 23 (10): 475-483.
- 14. Edmunds, G., & Kendrick, D.C. (1980). *The Measurement of Human Aggression*. Chicester, England: Ellis Horwood Limited.
- 15. Engel, B.T. 1960. Stimulus-response and Individual-response Specificity. *Archives of General Psychiatry*, 2, 305–313.
- 16. Engel, G. 1977. The Need for a New Medical Model: A Challenge for Biomedicine. *Science*, 196 (4286), 129-136. Retrieved from http://www.jstor.org/stable/1743658
- 17. Flor-Henry, P. 1976. Lateralized Temporal-limbic Dysfunction and Psychopathology. *Annals of the New York Academy of Sciences*, 280, 777–797.
- 18. Fridlund, A.J., & Izard, C.E. 1983. Electromyographic Studies of Facial Expressions of Emotions and Patterns of Emotions. In J. R. Cacioppo & R. E. Petty (eds.), *Social Psychophysiology: A Sourcebook*. New York: Guilford Press, 243–386.

- Friedman, Naomi P., et al. 2008. Individual Differences in Executive Functions are Almost Entirely Genetic in Origin. *Journal of Experimental Psychology: General*, 137 (2): 201-225.
- Friedrich, W.N., Tyler, J.D., & Clark, J.A. 1985. Personality and Psychophysiological Variables in Abusive, Neglectful, and Low-income Control Mothers. *Journal of Nervous* and Mental Disease, 173, 449–460.
- 21. Frodi, A.M. 1978. Experiential and Physiological Responses Associated with Anger and Aggression in Women and Men. *Journal of Research in Personality*, 12, 335–349.
- 22. Frodi, A.M., & Lamb, M.E. 1980. Child Abusers' Responses to Infant Smiles and Cries. *Child Development*, 51, 238–241.
- 23. Funahashi, Shintaro. (2001). Neuronal Mechanisms of Executive Control by the Prefrontal Cortex. *Neuroscience Research*, 39: 147-165.
- 24. Geen, R.G., Stonner, D., & Shope, G.L. 1975. The Facilitation of Aggression by Aggression: Evidence Against the Catharsis Hypothesis. *Journal of Personality and Social Psychology*, 31, 221–226.
- Giancola, Peter R. 1995. Evidence for Dorsolateral and Orbital Prefrontal Cortical Involvement in the Expression of Aggressive Behavior. Aggressive Behavior, 21: 431-450.
- Golden, C.J. 1981. A Standardized Version of Luria's Neuropsychological Tests: A
 Quantitative and Qualitative Approach to Neuropsychological Evaluation. In B.S. Filskov&
 T.J. Boll (eds.), Handbook of Clinical Neuropsychology. New York: John Wiley, 608–642.
- 27. Golden, C.J., Hammeke, T., & Purisch, A. 1980. *The Luria-Nebraska Neuropsychological Battery: Manual (Revised)*. Los Angeles: Western Psychological Services.
- 28. Gorenstein, E.E. (1982). Frontal Lobe Functions in Psychopaths. *Journal of Abnormal Psychology*, 91, 368–379.
- 29. Hare, R.D. 1978. Electrodermal and Cardiovascular Correlates of Psychopathy. In R.D. Hare & D. Schalling (Eds.), *Psychopathic Behaviour*, 107–143. New York: Wiley.
- 30. Hare, R.D., & Connolly, J.F. 1987. Perceptual Asymmetries and Information Processing in Psychopaths. In Madnick, S.A., Moffitt, T.E., and Stack, S.A. (eds.), *The Causes of Crime: New Biological Approaches*. Cambridge: Cambridge University Press, 218-238.
- 31. Ishikawa, Sharon S., & Adrian Raine. 2003. Prefrontal Deficits and Antisocial Behavior: A Causal Model. In *Causes of Conduct Disorder and Juvenile Delinquency*, edited by B.B. Lahey, T.E. Moffitt and A. Caspi. New York: The Guilford Press.
- 32. Kaplan, R.M. 1984. The measurement of Human Aggression. In R.M. Kaplan, V.J. Konecni & R.W. Novaco (Eds.), *Aggression in Children and Youth*. The Hague, The Netherlands: MartinusNijhoff, 44–72.
- 33. Kendall, P.C, & Norton-Ford, J. D. 1982. *Clinical psychology: Scientific and Professional Dimensions*. New York: John Wiley.
- 34. Konecki, V.J. 1975. The Mediation of Aggressive Behavior: Arousal Level vs. Anger and Cognitive Labeling. *Journal of Personality and Social Psychology*, 32, 706–712.
- 35. Lacey, J.I. 1959. Psychophysiological Approaches to the Evaluation of Psychotherapeutic Process and Outcome. In E.A. Rubinstein & M.B. Parloff (eds.), *Research in Psychotherapy*. Washington, DC: American Psychological Association, 160–208.
- 36. Lacey, J.I., & Lacey, B.C. 1958. Verification and Extension of the Principle of Autonomic Response Stereotypy. *American Journal of Psychology*, 71, 50–73.
- 37. Lacey, J.I., & Lacey, B.C. 1970. Some Autonomic-central Nervous System Interrelationships. In P. Black (ed.), *Physiological Correlates of Emotion*. New York: Academic Press, 205–227.

- 38. Lacey, J.I., Kagan, J., Lacey, B.C, & Moss, H.A. 1963. The Visceral Level: Situational Determinants and Behavioral Correlates of Autonomic Response Patterns. In P.H. Knapp (Ed.), *Expression of the Emotions in Man*. New York: International Universities Press, 161–196.
- 39. Lezak, M.D. 1982. The Problem of Assessing Executive Functions. *International Journal of Psychology, 17* (2-3), 281-297. http://dx.doi.org/10.1080/00207598208247445
- 40. Lezak, M.D. 1983. *Neuropsychological Assessment* (2nd Ed.). New York: Oxford University Press.
- 41. Luria, A.R. 1966. Higher Cortical Functions in Man. New York: Basic Books.
- 42. Luria, A.R. 1973. *The Working Brain: An Introduction to Neuropsychology*. New York: Basic Books.
- 43. Mednick, S.A., & Volavka, J. 1980. Biology and crime. In N. Morris & M. Tonry (Eds.), *Crime and Justice: An Annual Review of Research*. Vol. 2, Chicago: University of Chicago Press, 85–158.
- 44. Megargee, E.I., & Bohn, M.J. 1979. Classifying Criminal Offenders: A New System Based on the MMPI. Beverly Hills: Sage.
- 45. Moffitt, Terrie E. 1993. Adolescence-limited and Life-course-persistent Antisocial Behaviour: A Developmental Taxonomy. *Psychological Review*, 100 (4): 674-701.
- 46. Morgan, Alex B., and Scott O. Lilienfeld. 2000. A Meta-analytic Review of the Relation between Antisocial Behavior and Neuropsychological Measures of Executive Function. *Clinical Psychology Review*, 20 (1): 113-136.
- 47. Myer, J., & Megargee, E. I. 1977. A New Classification System for Criminal Offenders, II: Initial Development of the System. *Criminal Justice and Behavior*, 4, 115–124.
- 48. Nietzel, M.T., & Bernstein, D.A. 1987. *Introduction to Clinical Psychology* (2nd Ed.). Englewood Cliffs, NJ: Prentice-Hall.
- 49. Prabhakaran, Vivek, K. Narayanan, Z. Zhao, & J.D.E. Gabrieli. 2000. Integration of Diverse Information in Working Memory within the Frontal Lobe. *Nature Neuroscience*, 3 (1): 85-90.
- 50. Pruitt, D.L., & Erickson, M.R. 1985. The Child Abuse Potential Inventory: A Study of Concurrent Validity. *Journal of Clinical Psychology*, 41, 104–111.
- 51. Raine, A. 2013. *The Anatomy of Violence: The Biological Roots of Crime.* Vintage, New York.
- 52. Raine, A. & Yang, Y. 2006. Neural Foundations to Moral Reasoning and Antisocial Behavior. *Social Cognitive and Affective Neuroscience*, 1, 203-213.
- Raine, A., & Jones, F. 1987. Attention, Autonomic Arousal, and Personality in Behaviorally Disordered Children. *Journal of Abnormal Child Psychology*, 15, 583-599.
- 54. Raine, Adrian, & Yaling Yang. 2006. Neural Foundations to Moral Reasoning and Antisocial Behavior. *Social Cognitive and Affective Neuroscience*, 1: 203-213.
- 55. Reitan, R.A., & Davison, L.A. 1974. *Clinical Neuropsychology: Current Status and Applications*. New York: John Wiley.
- 56. Robbins, Trevor W. et al. 1998. A Study of Performance on Tests from the CANTAB Battery Sensitive to Frontal Lobe Dysfunction in a Large Sample of Normal Volunteers: Implications for Theories of Executive Functioning and Cognitive Aging. *Journal of the International Neuropsychological Society*, 4: 474-490.
- 57. Royall, Donald R. et al. 2002. Executive Control Function: A Review of its Promise and Challenges for Clinical Research. *Journal of Neuropsychiatry and Clinical Neuroscience*, 14 (4): 377-405.

- 58. Rutter, Michael. 2003. Commentary: Causal Processes Leading to Antisocial Behavior. *Developmental Psychology*, 39 (2): 372-378.
- 59. Schachter, J. 1957. Pain, Fear, and Anger in Hypertensives and Normotensives. *Psychosomatic Medicine*, 19, 71–79.
- 60. Schalling, D. 1978. Psychopathy-related Personality Variables and the Psychophysiology of Socialization. In R.D. Hare & D. Schalling (Eds.), *Psychopathic Behaviour*. New York: Wiley, 85–106.
- 61. Seguin, Jean R. 2008. The Frontal Lobe and Aggression. European Journal of Developmental Psychology, 1: 1-20.
- 62. Shagass, C. 1972. Electrical Activity of the Brain. In N.S. Greenfield & R.A. Sternbach (Eds.), *Handbook of Psychophysiology*. New York: Holt, Rinehart, & Winston, 263–328.
- 63. Siddle, D.A.T. 1977. Electrodermal Activity and Psychopathy. In S.A. Mednick & K.O. Christiansen (Eds.), *Biosocial Bases of Criminal Behaviour*. New York: Gardner Press, 199–211.
- 64. Stasiewicz, P.R., &Lisman, S.A. 1989. Effects of Infant Cries on Alcohol Consumption in College Males at Risk for Child Abuse. *Child Abuse & Neglect*, 13, 463–470.
- 65. Stordal, Kirsten I. at al. 2005. General Psychopathology is More Important for Executive Functioning than Diagnosis. *ACTA Psychiatrica Scandinavica*, 111: 22-28.
- 66. Stuss, Donald T., and Robert T. Knight, (eds.) 2002. *Principles of Frontal Lobe Function*. New York: Oxford University Press.
- 67. Stuss, Donlad T., and Micahel P. Alexander. 2000. Executive Functions and the Frontal Lobes: A Conceptual Review. *Psychological Research*, 63: 289-298.
- 68. Syndulko, K. 1978. Electrocortical Investigations of Sociopathy. In R.D. Hare & D. Schalling (eds.), *Psychopathic Behavior: Approaches to Research*. New York: John Wiley, 145–156.
- 69. Tanji, Jun., & Eiji Hoshi. 2008. Role of the Lateral and Prefrontal Cortex in Executive Behavioral Control. *Physiological Review*, 88: 37-57.
- Trasler, G. 1987. Some Cautions for the Biological Approach to Crime Causation. In Madnick, S.A., Moffitt, T.E., and Stack, S.A. (eds.). Sept. 20-24, 1982. *The Causes of Crime: New Biological Approaches*. Cambridge: Cambridge University Press. Proceedings NATO Conference, Skiathos Greece, 7-24.
- 71. Venables, P.H. 1987. Autonomic Nervous System Factors in Criminal Behavior. In S.A. Mednick, T.E. Moffitt, & S.A. Stack (Eds.), *The Causes of Crime: New Biological Approaches*. Cambridge: Cambridge University Press, 110–136.
- 72. Volavka J. 2013. Violence in schizophrenia and bipolardisorder. *Psychiatria Danubina*; 25: 24-33.
- Volavka, J. 1987. Electroencephalogram among criminals. In S.A. Mednick, T.E. Moffitt,
 S.A. Stack (Eds.), *The Causes of Crime: New Biological Approaches*. Cambridge: Cambridge University Press, 137–145.
- 74. Webb, S.J., Monk, C.S. & Nelson, C.A. 2001. Mechanisms of Postnatal Neurobiological Development: Implications for Human Development. *Developmental Neuropsychology*, 19(2), 147–171.
- 75. Wechsler, D. 1958. *Measurement and Appraisal of Adult Intelligence* (4th Ed.). Baltimore: Williams & Wilkins.
- 76. Wechsler, D. 1974. *Manual for the Wechsler Intelligence Scale for Children-Revised (WISC-R)*. New York: Psychological Corporation.

- 77. Wechsler, D. 1981. *Manual for the Wechsler Adult Intelligence Scale-Revised (WAIS-R)*. New York: Psychological Corporation.
- 78. Wilson, B., N. Alderman, P. Burgess, H. Emslie, & J. Evans. 1996. *Behavioural Assessment of the Dysexecutive Syndrome*. Bury St Edmonds, UK: Thames Valley Test Company.
- 79. Wolf, M.M., Braukmann, C.J., & Ramp, K.A. 1987. Serious delinquent behavior as part of a significantly handicapping condition: cures and supportive environments. *Journal of Applied Behavior Analysis*, 20 (4), 347–359. doi:10.1901/jaba.1987.20-347
- 80. Wolfe, D.A., Fairbank, J.A., Kelly, J.A., & Bradlyn, A.S. 1983. Child abusive parents' physiological responses to stressful and non-stressful behavior in children. *Behavioral Assessment*, 5, 363–371.
- 81. Yang, Yaling, Andrea L. Glenn, & Adrian Raine. 2008. Brain abnormalities in antisocial individuals: Implications for the law. *Behavioral Sciences and the Law*, 26: 65-83.
- 82. Zahn, T.P. 1986. Psychophysiological approaches to psychopathology. In M.G.H. Coles, E. Donchin, & S.W. Porges (eds.), *Psychophysiology: Systems, Processes, and Applications*. New York: Guilford Press, 508–610.