Human as opposed to medical conditions such as

Human behaviour is complex. One of the approaches used in the endeavour to understand its intricacies canbe explored using a biological or medical model. The biological approachexamines the roles of physiology- the brain and other organs' reactionsinfluenced by chemicals and the control of certain motor functions, ontogeny -or how a pattern of behaviour develops over a period because of genetics andexperiences, evolution – for instance, the reasons as to why certain functionsexist, even though their relevance in the here and now is questionable andfunctionality – or why these structures evolved the way they have. (Tinbergen1951).

Abnormal behaviour, asbroadly defined by the American Psychological Association is the occurrence ofa clinically significant pattern of behaviour that causes distress ordisability which has a greater risk in resulting in death, disability or theloss of one's freedom. The purpose of this essay is to attempt to explain the occurrence of abnormal behaviour in the context of two biologicalfactors; genes and the role of neurotransmitters. The question as to whethera set of behaviour has been inherited or is a result of environmental factors, or both, has been long debated over. For it to be determined that anindividual's genotype is indeed the sole reason for the resulting behaviour, itmust be proven that no other environmental contributor is evident as a causalfactor.

As referenced by Plomin etal. (1994) research conducted on comparative studies of identical and fraternaltwins, has indicated a greater genetic influence on behavioural disorders suchas schizophrenia, major affective disorders and antisocial personality disorderas opposed to medical conditions such as Parkinson's disease or RheumatoidArthritis. Further

studies conducted in behavioural genetic research alsoindicate that the findings pertaining to the influence of environmental factorsin adoptive sibling studies indicate that a shared environment has no bearingon the manifestation of psychological traits (Plomin et al – 1994: 1735 and McGue and Bouchard 1998: 5). There is no conclusive evidence to date, to suggest that genetics is the onlyconstant in resulting abnormal behaviour.

The reason for this could beattributed to the fact that the study of the approximately 30, 000 human genes, is still a work in progress. It has been said that once decoded one would beable to access the data of every aspect of what determines the development ofhuman beings (Human Genome Project 2001) Consider the currentresearch available on anti-social behaviour (ASB) for instance. There is no denying that genetics play a rolein the occurrence of such behaviour (Raine 2008: 323-324).

It has been determined that genetic influenceaccounts for more than 50% of resulting anti-social behaviour (Moffit 2005: 535). However, the variance at play here is attributed to environmental factors. Ofthe two methods used to determine the influence of environmental factors inresulting ASB, is the hypothesis that MZ twins' genetic construct being twiceas similar than that of DZ twins, should thus result in a greater similarity inthe resulting behaviour, if only genetics were considered. If this were not thecase, then it is reasonable to assume that that other factors influenced theincreased levels of similarity. This has been proven to be true with most behaviouralcharacteristics, the exception being ASB.

In a study conducted byKendler et al (2003) on the genetic and environmental risk involved in commonpsychiatric and substance use disorders, it was discovered that environmentalinfluence played a more significant role in resultant ASB as opposed toabnormal behaviour and experiences such as anxiety and substance abuse. Furtherstudies conducted to ascertain ASB in the form of aggression and rule-breakingby using behavioural genetics biometric modelling has indicated that bothgenetics and environment concurrently play a role in its occurrence and whilebetween ages 9 and 10 its effects are a 41% – 40% – 19% split; genetics toshared environment to non-shared environment, with the 14-15 age group this wasfound to shift to a 44% – 79%, environmental factors. It can be said that agetoo therefore, plays a role in determining ASB (Niv et al 2003). The Serotonin Transportergene is one of the most studied variants in psychiatry and neuroscience. It wasdiscovered that a polymorphism in the 5-HTTLPR region of the SerotoninTransporter gene effects the reabsorption of Serotonin after its release.

In astudy conducted among 847 participants, the existence of two variants, i. e. long and short, of this gene was discovered. When tested in concurrence withhighly stressful events experienced by participants aged 21 -26, it wasdiscovered that the individuals with two short forms of the gene had a greaterpropensity to develop depression when subjected to a highly stressful sequenceof events over time. The short gene by itself, didn’t cause the depression, butincreased the individuals’ susceptibility in getting it, when subjected tostressful events (Caspi et al 2003). Amore recent GEM study further revealed that a significant G X E interactionresulted in a greater

propensity for depression. It was also revealed that ageand gender played significant roles in resulting depression; in, that olderadolescent carriers of the short allele subjected to peer pressure over timeand females subjected to long term peer pressure, were more prone to bediagnosed with depression (Hankin et al 2015: 804-812).

The effect of the existenceof this polymorphism has also been observed in individuals with apredisposition to psychopathy. In a study carried out to determine the extentof the role played by the 5-HTT gene, in relation to impulse control; a commontrait of psychopathy, and, the 5-HTT gene in relation to a G X E context ofcallousness and narcissism, individuals carrying the genotype s/s rated high onthe APSD (Antisocial Process Screening Device) on impulsivity, whereas those withthe l/l genotype raised in low SES environments indicated a greater propensitytowards callousness and narcissism. The research also indicated that nocorrelation was seen to exist between low SES and the 5-HTT gene (Sadeh et al 2010: 604-608). The premise of the theoriesassociated with neurotransmitters is, that it influences chemical imbalancesthat result in abnormal behaviour (Sue et al 2010: 38).

More specifically, studies have revealed that many endogenous compounds such as acetylcholine(ACh), dopamine (DA), serotonin (5HT), gamma aminobutyric acid (GABA) andnorepinephrine (NE) – (among those more commonly referenced), are said toinfluence the occurrence of several abnormal conditions or psychiatric statessuch as schizophrenia, depression (Hanin 1978: 135-138), and more recently inaggressive behaviour (Narvaes et al 2014: 601-607) and psychopathy (Buckholtz etal 2010: 419-421).

Consider the complexities of schizophrenia forinstance. After over 1200 studies in trying to understand the reason for itsoccurrence it has been determined that no one gene but the existence of severalsusceptible genes code for varying molecular abnormalities that influenceinsufficient information processing that results in a genetic bias towardsschizophrenia (Stahl 2007 cited in Sue et al 2010: 375). More recent analysis of theinfluence of identifying specific genes, by way of involving treatments of PCPand clozapine in a pharmacogenomic mouse model coupled with genetic linkage andpost-mortem brain data uncovered the involvement of 14 genes responsible for acombination of neurotransmission and functional inconsistencies in resultingschizophrenia. Of these 14 genes, primarily featuring on the list were threegenes associated with the neurotransmission of GABA. This data could explainthe abnormal EEG gamma band activity detected in schizophrenic patients(Le-Niculescu et al 2007). Depression, has long been asubject of psychiatric research. The monoamine hypothesis of depression wasfirst observed as a result of the effects caused by two monoamine oxidaseinhibitors, namely iproniazid and imipramine, in non-psychiatric patients.

Thecompounds had a marked impact on the transmission of serotonin causingsignificant anti-depressant effects. Furthermore, it was observed thatReserpine caused depressive symptoms in others. To date, antidepressant agentsare designed to influence monoamine transmission by inhibiting either neuralreuptake or degradation.

Aggressive behaviour, atrait associated with psychopathy, which in turn results in criminal behaviour, has given cause to greater scientific research

as its social and economicburden is immense. Monetarily in the US alone this is indicative to exceed $1trillion (Buckholtz et al 2010). There is now, documented research confirmingthe roles of neurotransmitters such as GABA, dopamine and serotonin existent inresulting aggressive behaviour (Narvaes et al 2014). These findings will have aconsiderable impact on the perspective in which such behaviour is viewed, particularly from a legal clinical point of view. Further to this point, in astudy carried out to determine the hypersensitivity of the dopamine rewardsystem in individuals with psychopathic traits, it was discovered that dopaminewas a key determiner in antisocial personality traits with animpulsive-antisocial temperament (associated with aggression) indicatingincreased interaction with the mesolimbic dopamine system in response toreward. Furthermore, several studies conducted on rodents in preclinical testsindicate mesolimbic dopamine to be critical in the expression of aggression(Buckholtz et al 2010). In reviewing the geneticand neurochemical premises of abnormal behaviour, we find that neither one northe other can be designated as the sole cause of the resulting abnormal behaviour.

There is evidence in several resultant abnormal patterns of behaviour tosuggest that genetic predisposition plays a role in creating susceptibility inindividuals in relation to certain chemical functions. As referenced forinstance with schizophrenia the existence of three genes responsible for thetransmission of GABA was observed. With aggressive behaviour and resultantpsychopathy, we see a predisposition towards delinquent behaviour because of apolymorphism of the serotonin transporter gene and a heightened interaction ofthe mesolimbic dopamine system in response to

reward. With depression, theexistence of two short alleles of the serotonin transporter gene indicated agreater probability of encountering depression.

Although monoamine basedanti-depressants are the first course of therapeutic measures, certain genepolymorphisms significantly contribute towards a depressed individual’sefficacy in relation to anti-depressants (Uhr et al 2007). Furthermore, although notpreviously discussed in this essay, it should be noted that abnormal behaviour, can also be attributed to other factors. Evolution, for example.

Take theevolutionary predisposition to fear certain species such as snakes and spidersresulting in present day phobias of the same (Ohman and Mineka 2001). Abnormal behaviourcan also be the result of the formation or structure of the brain – consideranti-social behaviour for instance; there is mounting evidence to suggestimpairments to the brain’s prefrontal cortex in individuals exhibiting traitsthat constitute such behaviour (Raine and Yang 2006); in fact, it’s been foundthat murderers show reduced glucose metabolism in this area of the brain whenfaced with a task that’s meant to activate it (Raine 2008). With schizophrenia, according to the neurodevelopmental hypothesis, abnormalities in the brainstructure attributed to genetics, prenatal or postnatal development coupledwith environmental influences could aggravate its symptoms (Weinberger 1996). Among a group of 390 individuals with schizophrenia, it was discovered thatabnormalities were present in the left temporal and frontal areas of the cortexand that the thalamus was smaller than average among these individuals (Harmset al 2007). Environmental factors tooplay a key role.

For instance, although the susceptibility of depression isgreater among those who have the short serotonin transporter gene, theexistence of other genes that increase the probability of acquiring depressionis found among individuals who have been subjected to various forms of abuseand neglect in their upbringing (Bradley et al 2008 and Haeffel et al 2008). Some cases of depression have also been linked to viral infections such asBorna disease (Kalart 2010: 439) and yet some others could be the result ofgiving birth – statistics state that at least 20% (Hopkins et al 1984 cited inKalart 2010: 440) of women report some level of postpartum depression. Thus, the explanation ofthe complexities of abnormal behaviour must be approached from a broaderperspective, for it to be comprehended to some degree.