The role of emotional intelligence and a functional polymorphism in the MAO-A *gene on aggression in humans*

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Dissertation submitted in fulfilment of the requirements for the degree ***Magister Scientiae*** (Behavioural Genetics) in the Faculty of Natural and Agricultural Sciences (Department of Genetics) at the University of the Free State.

May 2012

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LIST OF ABBREVIATIONS

α – Cronbach's correlation coefficient

ACE model – model attributing total phenotypic variance of a trait to the effects of A (additive genetic influences), C (shared environmental influences) and E (non-shared environmental influences).

ANOVA – Analysis of variance

b – Regression coefficient

B – Unstandardized regression coefficients

Beta – Standardized regression coefficients

BIDR – Balanced inventory of desirable responding

bp – Base pair

Cov – Covariance

df – Degrees of freedom

DNA – Deoxyribonucleic acid

DRD1 – Dopamine receptor D1 gene

DRD2 – Dopamine receptor D2 gene

DRD3 – Dopamine receptor D3 gene

DRD4 – Dopamine receptor D4 gene

DRD5 – Dopamine receptor D5 gene

DSM-IV – Diagnostic and statistical manual of mental disorders four

EDTA – Ethylenediamine tetraacetic acid

F – Test statistic for analysis of variance

g – Relative centrifuge force

GABA – γ-aminobutyric acid

HTR1β – Human serotonin receptor 1B gene

5HT – 5-hydroxytryptamine (serotonin)

5-HT2 – Serotonin 2 receptor gene

5HTT – Serotonin transporter gene

IQ – Intelligence quotient

MAO-A – Monoamine oxidase A enzyme

MAO-A – Monoamine oxidase A gene

MAO-A-H – high expression (3.5 and 4 repeat) alleles of the *MAO-A* gene

MAO-A-L – low expression (3-repeat) allele of the *MAO-A* gene

MAO-A-uVNTR – Monoamine oxidase A upstream variable number of tandem repeats polymorphism

MEGA – Molecular evolutionary genetics analysis

µl – Microliter

min – Minutes

ml – Millilitre

MS – Mean of squares

ng – Nanogram

ºC – Degrees Celsius

p – Cohen's effect size

PCR – Polymerase chain reaction

PTSD – Posttraumatic stress disorder

r – Pearson product moment correlation coefficient

R (statistics) – Multiple correlation coefficient

R – repeats

R^2 – Coefficient of multiple determination

RNA – Ribonucleic acid

RPQ – Reactive proactive aggression questionnaire

s – Seconds

SD – Standard deviation

SDB – Social desirability bias

SDR – Socially-desirable responding

Sig. – Level of statistical significance, also (p)

SLESQ – Stressful life events screening questionnaire

SS – Sum of squares

SSRI – Selective serotonin reuptake inhibitors

Std. Error – Standard error

t – t-test for independent samples

TE buffer – Tris ethylenediamine tetraacetic acid buffer

TEIQue – Trait emotional intelligence questionnaire

TEIQue-SF – Trait emotional intelligence questionnaire-short form

UV – Ultraviolet

Var – Variance

Var(A) – Variance due to additive genetic influences

Var(C) – Variance due to shared environmental influences

Var(E) – Variance due to nonshared environmental influences

Var(P) – Total phenotypic variance

Z – Fisher's *r- to z-* transformation score

ACKNOWLEDGEMENTS

- First and foremost I want to thank my Heavenly Father for giving me the passion, the ability and the courage to complete this dissertation.
- Thank you to my supervisor, Prof Johan Spies for all his help and patience throughout the past year and a half.
- Thank you to Zurika Odendaal, Sue-Rica Schneider and Paula Spies for assisting me in the laboratories and editing the dissertation.
- Thank you to the Department of Genetics and the University of the Free State for providing the facilities necessary for the completion of this study.
- Special thanks to the National Research Foundation for providing financial assistance for completion of the study.
- Lastly, I want to thank my wonderful parents and brother, as well as all my amazing friends for all their love and support throughout this period. You are my life. Without you I am nothing.

PREFACE

This dissertation was written in article format, with each chapter representing an article. Chapter 1 consists of a short introduction to the study. In Chapter 2 the literature concerning all the variables studied is reviewed. This chapter has already been published in the journal *Philosophical Transactions in Genetics* 1: 42-66 (2011). The questionnaires chosen for use in this study are reviewed in Chapter 3. Chapter 4 is an analysis of the influence of possible environmental factors on aggression scores, whilst chapter 5 shortly describes why social desirability bias was not controlled for in Chapter 4. Chapter 6 consists of a family study testing the variable influences of heredity and environment on aggression. A genetic association study testing the influence of the *MAO-A* gene on aggression comprises Chapter 7. Chapter 8 presents a short summary of the whole dissertation, whilst Chapter 9 provides the list of references used in the dissertation. Finally, the final part of the dissertation consists of appendices for the various questionnaires used, as well as for the molecular sequencing data. Chapters 4 to 7 are all in the process of being submitted to various journals for publication.

The names mentioned on the cover pages of each chapter, namely S. Schneider and Z. Odendaal, are those of unofficial co-supervisors. They played no other role in the writing of the various articles making up this dissertation.

CHAPTER 1

INTRODUCTION

The genetic and environmental influences on aggression

Abstract

Aggression is a complex trait, with both genetic and environmental factors important in its aetiology. It is a universal problem, for which better solutions are needed. This study will focus mainly on the influence of genetic and environmental factors on two subtypes of aggression, namely reactive- and proactive aggression. The moderate heritability values of these subtypes make them ideal candidates for such a study. The genetic components of aggression include the upper-limit heritability estimates for the subtypes by means of correlations between first-degree relatives. Thereafter the role of variants of one specific gene, the *MAO-A* gene will be examined. The role of emotional intelligence as a specific environmental factor influencing aggression is discussed. Very few studies have been done on the possible influence of emotional intelligence on aggression. Traumatic event exposure will also be studied as a possible secondary influencing factor. Since self-report measures are used, the effect of social desirability bias will be determined. In this chapter each of the variables under study is briefly described, followed by the most salient motivations why these specific variables were seen as the most suitable for this investigation. In addition, the specific aims of the dissertation are briefly outlined.

Keywords: Aggression, emotional intelligence, serotonin, socially desirable responding, trauma

Behavioural genetic research aims to determine the influence of an individual's genetic makeup on his/her behaviour. However, behavioural traits are also extremely sensitive to variation in the environment. For this reason, the science of behavioural genetics also aims to determine the influence of environmental factors on behaviour (Anholt & Mackay, 2010). This dissertation consists of both genetically informative studies and studies of environmental factors possibly influencing aggression.

Aggression is a highly prevalent and costly problem in societies throughout the world (Miczek *et al.*, 2002; Weinshenker & Siegel, 2002; Buckholtz & Meyer-Lindenberg, 2008). Anthropologists, psychologists and sociologists have examined the societal implications of aggressiveness in people (Lesch & Merschdorf, 2000). These studies have revealed that each antisocial individual costs society up to 10 times more than non-aggressive individuals in healthcare and social services costs (Scott *et al.*, 2001; Buckholtz & Meyer-Lindenberg, 2008). With a lifetime prevalence of 12.3% for adult antisocial behaviour (Compton *et al.*, 2005; Buckholtz & Meyer-Lindenberg, 2008), better ways need to be found to combat aggression and its myriad of negative consequences. Behavioural geneticists are striving to better understand the aetiology of aggression and other behavioural problems. The rationale behind this is that understanding the cause of a behaviour problem may lead to better treatment of the problem. This is in contrast to just trying to understand and treat the overt symptoms.

Different theorists have proposed many different definitions for aggression, but the main feature of all these definitions seems to be behaviours that are intended to hurt or harm others (Weinshenker & Siegel, 2002; Archer & Coyne, 2005; Ligthart *et al.*, 2005). Lesch and Merschdorf (2000) noted that systematic studies on the inheritance of aggression have led scientists to the conclusion that aggression is a complex trait, influenced by many genes in combination with environmental factors. Although various subtypes of aggression have been defined, the subtypes chosen as most applicable for study in this dissertation are reactive aggression and proactive aggression. Reactive aggression occurs in the context of provocation, and is described by Tuvblad *et al.* (2009) as an angry

response to a real or perceived threat. It is important to note that with reactive aggression the emotional response of anger is involved. In contrast, proactive aggression is a more instrumental form of aggression which does not involve provocation or anger. Instead, it is goal-oriented, including such goals as obtaining goods or ascertaining power (Tuvblad *et al.*, 2009).

These two forms of aggression were chosen for the studies undertaken in this dissertation for two main reasons. Firstly, they are the most frequently defined and most widely studied subtypes of aggression in the literature (Dodge & Coie, 1987; Geen, 2001; Brendgen *et al.*, 2006; Baker *et al.*, 2008). Secondly, reactive and proactive aggressions have both shown moderate heritability values, ranging from 26% to 39% for reactive aggression, and from 32% to 41% for proactive aggression in studies of twins in the United States of America (Brendgen *et al.*, 2006; Tuvblad *et al.*, 2009). The moderate heritability values make these forms of aggression ideal for studying the possible influences of specific genes (*MAO-A*) and specific environmental factors. In addition, heritability estimates for aggression are not known in South African populations.

A good starting point for finding putative genes influencing aggression is to look at genes that control brain neurotransmitter activity. The reason for this is that a number of studies have found that neurotransmitter systems play a key role in the aetiology of aggression, and that these systems can be seen as behavioural regulators in the brain (Rhee & Waldman, 1997; Schmidt *et al.*, 2002; Popova, 2006; Eisenberger *et al.*, 2007). It thus makes sense to assume that genes affecting neurotransmitter systems probably play a key role in behaviour. Regarding aggressive behaviour, the main neurotransmitter systems implicated include the serotonin, dopamine, and γ-aminobutyric acid (GABA) systems in the brain (Miczek *et al.*, 2002). Genes influencing these systems include among others precursor genes (e.g. *tyrosine hydroxylase* gene for dopamine), receptor genes (*DRD1, D2, D3, D4* and *D5* for dopamine, and *HTR1β* for serotonin), transporter genes (e.g. *5HTT* for serotonin) and metabolite genes (e.g. *MAO-A* for dopamine, norepinephrine and serotonin) (Rhee & Waldman, 1997).

The *MAO-A* gene has drawn particular attention for its possible role in aggressive behaviour. This gene encodes for the monoamine oxidase A (MAO-A)

enzyme. The main function of this enzyme is the degradation of the neurotransmitters dopamine, norepinephrine, and most importantly, serotonin. In the process the availability of these neurotransmitters are terminated. The *MAO-A* gene in humans has been mapped to the short arm of chromosome X (Xp-11.23) (Lesch & Merschdorf, 2000; Popova, 2006; Eisenberger *et al.*, 2007; Reif *et al.*, 2007). Of the three named neurotransmitters, serotonin has been found to especially influence aggression; although whether it inhibits or facilitates aggression is still uncertain (Carillo *et al.*, 2009). Thus, it would be logical to assume that a gene influencing serotonin activity will also influence aggressive behaviour [see Popova (2006) for a review of possible mechanisms describing the influence of the serotonergic system on aggression].

The possible importance of the *MAO-A* gene in aggressive behaviour first became apparent after Brunner *et al.* (1993) reported a missense mutation in the *MAO-A* gene in the male members of a large Dutch family. This mutation resulted in a functional *MAO-A* knockout in the hemizygous males. All of the males with this mutation were characterized by a very specific phenotype, which included persistent and extreme reactive aggressive behaviours. These findings have been replicated in mice models, with mice deficient in the MAO-A enzyme showing hyperaggressiveness (Lesch & Merschdorf, 2000).

Subsequent studies have also found a possible gene-environment interaction in the effects of the *MAO-A* gene on aggression. Genes and environment not only both contribute separately to the variance in behavioural traits, but also frequently interact in influencing the behavioural trait. A gene-environment interaction occurs when genes mediate the impact of environmental factors (Nuffield Council on Bioethics, 2002; Anholt & Mackay, 2010). The studies on aggression have shown that individuals carrying a certain allelic variant of the *MAO-A* gene and who have also been exposed to childhood trauma are more likely to engage in antisocial behaviour (Caspi *et al.*, 2002; Reif *et al.*, 2007; Siever, 2008). This association is in need of further investigation. For this reason, a trauma questionnaire is also included in this dissertation.

Taking into account the evidence presented above, the *MAO-A* gene was chosen as the candidate gene for aggression to be investigated in this study.

This does not imply that other genes affecting neurotransmitter systems are less important in aggressive behaviour. These genes will just not be considered in this dissertation, but may be studied in the future.

As mentioned, behavioural genetic researchers are not only interested in the influence of genes on behavioural traits, but also in the influence of the environment. In behavioural genetics, the term "environment" is used to describe all other influences on behaviour, apart from the influence of a specific gene (Nuffield Council on Bioethics, 2002). Environment in this sense has a much broader meaning than just an individual's external environment. As with all behaviour traits studied thus far, environmental factors play an important role in shaping levels of aggression (Rhee & Waldman, 1997; Kempes *et al.*, 2005; Baker *et al.*, 2006; Blair *et al.*, 2006; Brendgen *et al.*, 2006; Tuvblad *et al.*, 2009). Environmental and genetic factors also frequently interact in the determination of behavioural traits.

One environmental factor (environment *sensu lato*) that can possibly influence aggression is emotional intelligence. Malterer *et al.* (2008) described emotional intelligence as the ability to "recognize and regulate emotions in ourselves and in others". Authors frequently distinguish between two types of emotional intelligence, namely trait emotional intelligence and ability emotional intelligence (Mavroveli *et al.*, 2008). The main difference between these subtypes is the way in which they are measured. Trait emotional intelligence is measured with self-report measures, and ability emotional intelligence with maximal performance measures (Petrides & Furnham, 2003; Petrides *et al.*, 2006; Mavroveli *et al.*, 2008). Various authors have argued against the use of maximal performance measures for measuring emotional intelligence. The arguments are mainly that emotional intelligence is a subjective construct and that there is no such thing as "incorrect feelings" (Mavroveli *et al.*, 2008; Petrides *et al.*, 2006). For this reason, trait emotional intelligence was chosen for use in this dissertation, instead of ability emotional intelligence.

Emotional intelligence was chosen as a possible environmental factor for this dissertation mainly because very few studies have been done on the influence of a person's emotional intelligence on his/her levels of aggression. If empathy is

considered as an important aspect of emotional intelligence, as was done by Ali *et al.* (2009), a direct link can be drawn between low levels of emotional intelligence and aggression. Lauterbach and Hosser (2007) mentioned that empathic concern and being able to take another person's perspective inhibit aggression. This assumption seems valid, since being able to take another person's perspective should entail being able to share in that person's emotional distress. If an aggressor can thus experience the emotions of the person they aggress against vicariously, this should lead to a reduction in their aggressive actions in order to reduce their own distress. Thus, it would be expected that low levels of emotional intelligence are negatively correlated with aggression. This then provides a theoretical basis for choosing to study emotional intelligence with aggression.

As mentioned, trauma will be included as a variable in the molecular genetic study. Participants will thus complete a trauma questionnaire along with the emotional intelligence questionnaire to also look at the possible influence that trauma may play in aggressive behaviour. This is seen as feasible since trauma, especially repeated trauma, has been found to lead to aggression (Jakupcak & Tull, 2005; Dyer *et al.*, 2009; Vandenberg & Marsh, 2009). It must be noted, however, that the main focus of this literature review will be on emotional intelligence, and not on traumatic event exposure.

In addition, since self-report measures are used in this dissertation, socially desirable responding is also measured. Social desirability is defined as "the need for social approval and acceptance and the belief that it can be attained by means of culturally acceptable and appropriate behaviours" (Crowne & Marlow, as cited in Podsakoff *et al.*, 2003). Especially self-report measures of socially undesirable behaviours like aggression are subject to influence from socially desirable responding (Saunders, 1991). For this reason, a measure of social desirability is also included in this dissertation to ensure that this response bias is accounted for.

In light of the above discussion, the aims of this study are to determine:

1. among young adults from Central South Africa, whether:

1.1. different levels of emotional intelligence and/or aggression occur;

1.2. a correlation exists between reactive and/or proactive aggression and emotional intelligence in a sample consisting mostly of university students;

1.3. a correlation exists between trauma and aggression;

1.4. social desirability has a meaningful impact on scores of aggression;

1.5. different variants of the *MAO-A* gene occur;

1.6. different variants of the *MAO-A* gene, if present, lead to differing levels of aggression;

2. and to determine

2.1. upper-limit heritability estimates for aggression by studying a sample of first degree relatives of the participants.

This will be done in order to improve our understanding of aggressive behaviour and the factors that influence it. If the *MAO-A* gene can be shown to influence aggression, further studies can possibly lead to the development of psychopharmacological treatments targeting the functioning of this gene or the enzyme it encodes. Likewise, if emotional intelligence is correlated with reduced aggression, programs can be developed to enhance the degree of emotional intelligence in people. In both instances, this may lead to a reduction in aggressive behaviour and all of its negative consequences.

CHAPTER 2

A review of the influence of some neurotransmitter genes and emotional intelligence on aggression

Paper published in *Philos. Trans. Genet.* 1: 42-66 (2011) as "A review of the influence of some neurotransmitter genes and emotional intelligence on aggression" by Laubscher, N., Odendaal, Z., Schneider, S. & Spies, J.J.

Abstract

Aggression is a highly prevalent and costly problem in societies throughout the world. Treatment options are available, but needs to be improved or adjusted to really be able to curb the problem of aggression. The paper aims to highlight key points in the aggression literature in order to improve researchers' understanding of the construct of aggression and possible causes underlying aggression, as well as factors that may exacerbate aggression. Therefore, this paper reviews the literature on aggression in humans, and covers aspects relating to the definition of aggression, the various subtypes of aggression, evidence for variable environmental and heritable influences on aggression, as well as looking at specific genes and hormones influencing aggression. Specifically, the influence on aggression of the monoamine oxidase A enzyme, the gene that encodes it (*MAO-A*), and the neurotransmitters that it metabolizes (serotonin and norepinephrine) are looked at. In addition, emotional intelligence as a possible influencing factor on the occurrence of aggression is also covered. This is done to provide a starting point for research aiming to develop treatments for aggression, whether they are psychotherapeutic programmes aimed at improving emotional intelligence or psychopharmaceutic drugs aimed at the genetic and hormonal mechanisms underlying aggression.

Keywords: Monoamine oxidase A, proactive aggression, reactive aggression, serotonin, trait emotional intelligence

Introduction

Anderson and Carnagey (2004) makes it clear that violence, and thus aggression, in humans is not a new phenomenon. Indeed, archaeological and historical evidence prove that violence and aggression go as far back as 25,000 years ago, when it was prevalent among our hunter/gatherer predecessors.

Aggressive behaviour in humans ranges from benign behaviours aimed at establishing hierarchies and dominance, to more harmful forms of behaviour such as antisocial behaviour and delinquency. The societal implications of aggressiveness have been examined by many types of professionals, including anthropologists, psychologists and sociologists (Lesch & Merschdorf, 2000). Most researchers agree that human aggression and violence is a major public health concern around the world (Weinshenker & Siegel, 2002; Miczek *et al.*, 2002; Buckholtz & Meyer-Lindenberg, 2008). Compton *et al.* (2005) noted that adult antisocial behaviour has a lifetime prevalence of 12.3%. In a study by Scott *et al.* (2001) it was estimated that aggressive individuals cost society up to 10 times more than non-aggressive individuals in healthcare and social services costs. The problem, however, is that the available treatment options are not currently sufficient to curb the problem of aggression in societies (Volavka, 2002; Miczek *et al.*, 2002).

One possible solution might come from looking at the biological architecture of aggression. Since family and twin studies have proven convincingly that genetics play a critical role in aggression, it might be possible to treat or even prevent aggressive behaviour by understanding precisely how specific genes lead to aggressive behaviours. Before this can be done, however, it is of critical importance to first establish the specific genes involved in aggressive behaviour (Baker *et al.*, 2006). To do this, in turn, it is important to understand what is meant by the term “aggression”. Our understanding of aggression and the genes affecting aggression can be greatly enhanced by firstly adequately defining aggression, and secondly differentiating between subtypes of aggression (Bandura, 1973; Waschbusch *et al.*, 1998).

Different theorists have proposed many different definitions for aggression, but the main feature of all these definitions seems to be behaviours that are intended

to hurt or harm others (Weinshenker & Siegel, 2002; Archer & Coyne, 2005; Ligthart *et al.*, 2005). Geen (2001) agreed with this, but added that the motivation of the victim at whom the aggressive behaviour is aimed, also needs to be taken into consideration when defining aggression. As Geen (2001) pointed out, there are instances in which the "victim" might willingly seek out punishment in order to atone for guilt. In these instances, the so called victim might not necessarily refer to the behaviour of the supposed aggressor as "aggressive behaviour". Anderson and Carnagey (2004) added to this that not only must the victim be motivated to avoid the harm, but the aggressor must also believe that the victim is motivated to avoid the aggressive behaviour. Therefore, a comprehensive definition of aggression should include that the victim of the aggressive act is truly a "victim" in that this person is motivated to escape from or avoid the aggressive stimulus. In accordance, Geen (2001) defined aggression as follows: "Aggression is the delivery of an aversive stimulus from one person to another, with intent to harm and with an expectation of causing such harm, when the other person is motivated to escape or avoid the stimulus".

Apart from identifying genes affecting aggressive behaviour, looking at other factors that might influence aggression can also lead to clues that might help us find ways to treat aggression. One such possible factor is emotional intelligence. Low emotional intelligence scores have been shown to lead to rejection by peers in children, and as a result to antisocial conduct and delinquency later on in life (Petrides *et al.*, 2006). Emotional intelligence is also equated closely with empathy, and since empathy entails the ability to feel what other people are feeling, low empathy can possibly lead to increased aggression since the person is unable to perceive the influence their behaviour is having on other people (Joliffe & Farrington, 2004, 2007; Lauterbach & Hosser, 2007).

The aim of this paper is firstly to review the literature on the different subtypes of aggression, and then to examine the most important genes that has been found to influence aggressive behaviour. Finally, the literature on emotional intelligence and its possible involvement in aggression will be reviewed. The ultimate aim is to try to understand the mechanisms underlying and influencing aggression, in order to eventually find a possible treatment for this highly prevalent social problem.

2.1 Aggression

2.1.1 Types of aggression

When most people hear the word “aggression”, they immediately equate it with the physical force or violence seen in a fist fight or a loud verbal retort between two people in conflict with each other (Geen, 2001; Ligthart *et al.*, 2005). The concept of aggression, however, is much more complex and encompasses many more behaviours than only the use of physical or verbal force. As stated by Geen (2001), any behaviour that intents to harm another person against that person’s wishes, can be labelled as an aggressive act. In accordance with this idea, many different subtypes of aggression have been identified by researchers. For instance, relational aggression is defined by Archer and Coyne (2005) as behaviours that are intended to hurt others by manipulating or disrupting relationships. In addition to relational aggression, various definitions are also given for verbal aggression, physical aggression, direct aggression and indirect aggression, as well as intermale, fear-induced and irritable aggression (Archer & Coyne, 2005; Ligthart *et al.*, 2005; Popova, 2006).

The most commonly distinguished, and also the most frequently studied subtypes of aggression in the literature, however, are the constructs of reactive versus proactive aggression (Dodge & Coie, 1987; Geen, 2001; Brendgen *et al.*, 2006; Baker *et al.*, 2008). Geen (2001) referred to affective and instrumental aggression instead of reactive and proactive aggression. These terms are equivalent to the concepts of reactive and proactive aggression respectively.

2.1.1.1 Proactive and reactive aggression

Brendgen *et al.* (2006) noted that proactive aggression has been described as instrumental, offensive, and cold-blooded, whereas reactive aggression has been described as affective, defensive, and hot-blooded. According to Tuvblad *et al.* (2009) reactive aggression can be conceptualized as angry or frustrated responses to a real or perceived threat. Geen (2001) defined reactive aggression as “aggressive behaviour that is enacted in response to provocation, such as an attack or an insult, and it is manifested in both self-defence and angry actions”. On the other hand, Tuvblad *et al.* (2009) stated that proactive aggression can be conceptualized as a more instrumental form of aggression. This type of aggres-

sion does not require provocation or anger, but is rather motivated by such goals as asserting power, obtaining goods and assuring the approval of reference groups (Geen, 2001; Brendgen *et al.*, 2006). Simply put, the most important distinction between proactive and reactive aggression is that proactive aggression is goal-oriented, whereas reactive aggression is retaliatory (Hubbard *et al.*, 2010).

There has been much debate in the literature on whether proactive and reactive aggressions are really two distinct forms of aggression (Brendgen *et al.*, 2006; Baker *et al.*, 2008). These two forms of aggression have many overlapping features in their aetiologies, from both environmental and genetic perspectives. Proactive and reactive aggression also often co-occur in the same person, lending more support for their unification into one factor instead of two separate factors.

On the other hand, the associations of the different forms of aggression with different constructs lend support to the argument that reactive and proactive aggressions are two distinct forms of aggression. One such example is impulse control. Although impulsivity and aggression are two distinct psychological constructs, impulsive behaviour patterns can predispose people to anger and aggression. However, reactive forms of aggression are associated with a lack of impulse control, but not proactive forms of aggression (Strüber *et al.*, 2008). Emotional distress leads to the deterioration of impulse control and self-regulation, with the result being people acting on their angry impulses (Tice *et al.*, 2001). As previously mentioned, reactive forms of aggression are associated with anger and emotional distress, but not proactive forms of aggression. In addition, various studies have found that reactive and proactive aggression are factorially distinct, and most authors thus agree that reactive and proactive aggression are two distinct forms of aggression with a few overlapping features (Brendgen *et al.*, 2006; Baker *et al.*, 2008). In this review, reactive and proactive aggression will thus be treated as two distinct forms of aggression.

2.1.1.2 Heritable and environmental influences on proactive and reactive aggression

Multiple studies have examined the variable influences of environment and heredity on the occurrence of both proactive and reactive aggression in humans

(Brendgen *et al.*, 2006; Baker *et al.*, 2008; Tuvblad *et al.*, 2009). Tuvblad *et al.* (2009) found that heritability explained 26% of the total variance in reactive aggressive behaviour, and 32% of the total variance in proactive aggressive behaviour, with shared environmental factors explaining about a quarter of the variance in both proactive and reactive aggressive behaviour. Brendgen *et al.* (2006) found 39% and 41% heritability in reactive and proactive aggression respectively. In contrast to Tuvblad *et al.* (2009), they attributed the rest of the variance in both forms of aggression to nonshared environmental factors. Thus both reactive and proactive aggressions have environmental and heritable influences in their aetiology.

2.1.1.3 Evidence for the role of heritable factors in reactive aggression

Looking at the theoretical evidence, it is easy to understand why some scholars argue for the existence of genetic influences on the expression of reactive and proactive aggression in humans. Reactive aggression has been associated with a specific physiological phenotype (Geen, 2001; Hubbard *et al.*, 2002; Brendgen *et al.*, 2006). Geen (2001) reported a distinctive activation of the central and autonomic nervous systems in people who display reactive aggression, that amongst other things are characterized by increased blood flow to the muscles, elevations in blood pressure and pulse rate, dilation of the pupils of the eye, and a decrease in blood flow to the viscera. Brendgen *et al.* (2006) also reported on specific physiological correlates that have been found for reactive aggression, of which one is that only reactive, and not proactive, aggressive children show elevated levels of skin conductance during stress.

Other evidence for a genetic link to reactive aggression is the fact that reactive/affective aggression is the most common form of aggressive behaviour found in animals (Meloy, 1988; Baker *et al.*, 2008). This form of aggression serves as a self-defence mechanism that is critical to the survival of all animals, including primates and non-primates. Seeing that the neural circuitry underlying reactive aggression in animals is well-known by researchers, and seeing that brain circuitry is highly heritable, it is easy to come to the conclusion that there must be genetic underpinnings to reactive aggression in both humans and animals (Baker *et al.*, 2008).

Neurotransmitters have also been found to play a role in reactive aggression. In particular, it has been found that serotonin metabolites in the cerebrospinal fluid of people who exhibit reactive forms of aggression, tend to be lower than in non-impulsive-aggressive individuals. Low levels of serotonergic activity in people have consistently been linked to behavioural stimulation, which in turn leads to impulsive-aggressive behaviour, also known as reactive aggression (Kempes *et al.*, 2005; Baker *et al.*, 2006).

2.1.1.4 Evidence for the role of heritable factors in proactive aggression

The main evidence that proactive aggression might have a heritable basis comes from studies reporting a definite link between proactive aggression and psychopathic personality traits (Kempes *et al.*, 2005; Blair *et al.*, 2006; Brendgen *et al.*, 2006). According to Blair *et al.* (2006) psychopathy has at its core a tendency to make excessive use of proactive aggression. Unlike many other behavioural disorders, people with psychopathic tendencies are a quite homogenous group of people, which makes them easily identifiable. These individuals show a rather consistent array of symptoms, both emotionally and behaviourally (Blair *et al.*, 2006). Marsee *et al.* (2005) described psychopathic adults as frequently engaging in irresponsible and impulsive behaviour, being arrogant, callous and unemotional, and lacking in empathy and guilt. These psychopathic personality traits have been found to be highly heritable (Tuvblad *et al.*, 2009).

The connection between these psychopathic tendencies and an underlying genetic mechanism is related to findings that psychopathic individuals, and thus people who frequently display proactive forms of aggression, tend to have certain biologically based characteristics. Mostly, psychopathic individuals show underreactivity in their sympathetic nervous systems, as expressed by lower resting heart rates and lower electro-dermal responses. These factors have been shown to be at least partly heritable. The lower autonomic arousal shown by these individuals predispose them to be less sensitive to aversive or upsetting environmental stimuli, and also less sensitive to punishment cues (Kempes *et al.*, 2005; Baker *et al.*, 2006; Blair *et al.*, 2006; Brendgen *et al.*, 2006).

2.1.1.5 Environmental factors in reactive and proactive aggression

Although genes play a role in the aetiology of both reactive and proactive ag-

gressive behaviour, researchers are united in their view that environmental factors also play a considerable role in the development of both reactive and proactive aggressive behaviours (Rhee & Waldman, 1997; Kempes *et al.*, 2005; Baker *et al.*, 2006; Blair *et al.*, 2006; Brendgen *et al.*, 2006; Tuvblad *et al.*, 2009). Geen (2001) cautioned researchers not to view any genetic or environmental factor as a direct antecedent to reactive or proactive aggressive behaviour. Instead, the author proposed that environmental factors and genetic factors interact to create a level of potential for aggressive behaviours, but are not direct antecedents of the aggressive behaviours. Consequently, it is highly unlikely that a singular cause for aggression will ever be identified, whether environmental or genetic.

There is considerable evidence that environmental factors play a role in the development of reactive aggression. Specifically, the frustration-aggression model proposed by Berkowitz (1963) is used to explain the development of reactive aggressive behaviours. According to this model, aggressive behaviour is viewed as a learned response to frustration. Exposure to chronic life threatening danger is seen as a predisposing factor to the hyper vigilance associated with reactive aggressive behaviour. For example, children who display chronic reactive aggressive behaviours have been found to come from harsher family environments than children who display proactive or no aggressive behaviours. In accordance to this, reactive aggression (but not proactive aggression) has also consistently been linked to childhood abuse, as well as other early aversive experiences (Kempes *et al.*, 2005; Blair *et al.*, 2006; Brendgen *et al.*, 2006; Baker *et al.*, 2008; Tuvblad *et al.*, 2009).

In contrast to the frustration-aggression model used to explain reactive aggressive behaviour, proactive aggressive behaviour is mainly explained by the social learning theory. According to this theory, first proposed by Albert Bandura, proactive aggressive behaviours are first acquired through observational learning, but are then maintained by reinforcement of the behaviours. Thus, according to this theory, proactive aggressive behaviour is a learned response which is then reinforced by its positive consequences (Geen, 2001; Baker *et al.*, 2008). Baker *et al.* (2008) also specifically mentioned that coercive family processes play an important role in the development of proactive aggression. Brendgen *et al.* (2006) added to this that, in contrast to reactive aggression, people who tend to

display proactive aggressive behaviours usually come from an overly lenient family environment in which the use of aggression as a means of achieving one's goals is fostered. In addition, positive socialization experiences with peers are also characteristic of proactive forms of aggression.

A clear distinction can thus be drawn between the family environments and socialization experiences of people who display proactive aggressive behaviours and those who display reactive aggressive behaviours. This adds further weight to the general conclusion that proactive and reactive aggressions are two distinct forms of aggressive behaviour in people.

2.2 Genes and hormones involved in reactive and proactive aggression

Based on the above mentioned evidence that aggression is heritable, the next logical step for researchers is to locate specific genes underlying the aggressive traits seen in humans. Lesch and Merschdorf (2000) noted that systematic studies on the inheritance of aggression have led scientists to the conclusion that aggression is a complex trait, influenced by many genes in combination with environmental factors. Identifying a specific gene that plays a role in aggressive behaviour must thus not be interpreted as the discovery of the singular cause of aggression in humans.

Popova (2006) noted that genes cannot affect behaviour directly. Instead, candidate genes that might influence behaviour are selected by the possible involvement of their gene products in the aetiology of the particular behavioural disorder (Rhee & Waldman, 1997).

According to a neurobiological model of aggression proposed by Siever (2008), aggression emerges "when the drive of limbic-mediated affective prefrontal response to anger producing or provocative stimuli is insufficiently constrained by inhibition and is channelled into violent behaviour". Neurotransmitters have been found to facilitate prefrontal and subcortical inhibition, and thus inadequate neurotransmitter activity can play a key role in aggressive behaviour (Siever, 2008). However, as will be discussed later, it is not always clear whether inadequate or a surplus of a certain neurotransmitter leads to aggressive behaviour, especially when considering the role of serotonin on aggression.

Confirming the above association, various studies focusing on brain neuro-

transmitter systems have found that these systems play a key role in the aetiology of aggression (Rhee & Waldman, 1997; Schmidt *et al.*, 2002; Popova, 2006; Eisenberger *et al.*, 2007). According to Popova (2006) brain neurotransmitters can be seen as behavioural regulators occurring in the brain. According to this author, various neurophysiological, neurochemical and neuropharmacological studies have demonstrated that neurotransmitters in the brain play a central role in the regulation of behaviour, and that genes affecting behaviour probably act through brain neurotransmitters.

Popova (2006) stated that it is important to remember that the classic brain neurotransmitters are not proteins. For genes to play a role in the neurotransmitter systems, however, they need to act on the various neurotransmitters via proteins in accordance with the central genetic dogma of DNA-RNA-protein (Popova, 2006). Thus, candidate genes that play a role in the functioning of neurotransmitter systems may include:

precursor genes – code for proteins that affect the rate at which neurotransmitters are produced;

receptor genes – code for proteins that are involved in receiving neurotransmitter signals;

transporter genes – encode proteins involved in the reuptake of neurotransmitters back into the presynaptic terminal;

metabolite genes – code for proteins involved in the degradation of neurotransmitters; and

conversion genes – code for proteins that are involved in the conversion of one neurotransmitter into another (Rhee & Waldman, 1997; Hennig *et al.*, 2005; Popova, 2006).

In addition, researchers are also starting to examine transcription and translation control sequences as possible candidates in the expression of aggressive behaviour (Lesch & Merschdorf, 2000).

The neurotransmitter systems mostly thought to be involved in the regulation of aggressive behaviour include the serotonin (*5-HT* genes), dopamine, and γ-aminobutyric acid (GABA) systems in the brain, and most current pharmacother-

apeutic interventions make use of substances that target these systems (Miczek *et al.*, 2002). Most studies have focused on the role that the serotonergic neurotransmitter system plays in predisposing people to aggressive behaviour, and specifically reactive (impulsive) aggressive behaviour (Lesch & Merschdorf, 2000; Reif *et al.*, 2007; Zepf *et al.*, 2008). According to Lesch and Merschdorf (2000), an overwhelming amount of evidence have accumulated through research that especially the serotonin neurotransmitter system serves as a major modulator of emotional behaviour, including impulsivity and aggression. It thus follows naturally from this that genes influencing the serotonergic system probably play a role in aggressive behaviour.

2.2.1 Serotonin and aggression

In a meta-analytic review by Carrillo *et al.* (2009), the authors stated that there is currently no consensus regarding the role of serotonin in aggressive behaviour, with some studies proclaiming that serotonin inhibits aggression, whereas other studies have shown that increased activity of serotonin leads to an increase in aggressive tendencies. According to Siever (2008), serotonin plays an important role in the prefrontal cortical regions, such as the orbital frontal cortex and anterior cingulate cortex, where it modulates and often suppresses the emergence of aggressive behaviours. This is done primarily by the innervations of *5-HT2* receptors in these regions by serotonin. From this it follows that a shortage of serotonin in these regions will result in the promotion of aggressive behaviour. Thus, less serotonin equals more aggression.

Studies using SSRIs (selective serotonin reuptake inhibitors) are important tools to determine the link between serotonin and aggression. SSRIs block the reuptake of serotonin from the synaptic cleft by binding with the presynaptic 5-HT transporter. This results in an increase in the extracellular levels of serotonin in the synaptic cleft, and a resulting increase in the neurotransmission of serotonin (Carrillo *et al.*, 2009). If the observation made by Siever (2008) stated above is taken into account, it would be expected that SSRIs would lead to a decrease in aggressive tendencies since it leads to an increase in the neurotransmission of serotonin. Carrillo *et al.* (2009), however, stated that there is evidence supporting not only an inhibitory effect of SSRIs on aggression, but also facilitatory and null effects. Establishing a clear relationship between the serotonergic system and

aggression is thus not as straightforward as it may appear.

2.2.2 Role of Monoamine Oxidase A (MAO-A) and its genetic variations on aggression

An enzyme known as monoamine oxidase A (MAO-A) and the gene encoding it have been implicated in aggressive behaviour. MAO-A is an enzyme that degrades dopamine, norepinephrine, and most importantly, serotonin, and in the process terminates the availability of these neurotransmitters. The gene responsible for the production of MAO-A in humans is located on the short arm of chromosome X (Xp-11.23) (Lesch & Merschdorf, 2000; Popova, 2006; Eisenberger *et al.*, 2007; Reif *et al.*, 2007).

MAO-A is a mitochondrial enzyme, located on the outer mitochondrial membrane of mitochondria found in the presynaptic terminal of monoamine projection neurons. MAO-A is also found in astrocytes, which are located outside of neuronal cells. In these positions, MAO-A is perfectly located for its function of governing the availability of monoamine neurotransmitters for presynaptic packaging into vesicles, and also for inactivating the neurotransmitters in the synaptic cleft following their release (Buckholtz & Meyer-Lindenberg, 2008).

The *MAO-A* gene is mostly associated with reactive/impulsive aggression (Popova, 2006; Eisenberger *et al.*, 2007). Evidence for the role of the *MAO-A* gene in aggression comes from both human and animal studies. In a large Dutch family Brunner *et al.* (1993) found a missense mutation (C936T) resulting in a premature stop codon in the eighth exon of the *MAO-A* gene in some male members in the family. This mutation resulted in a functional *MAO-A* knockout in the hemizygous males. All of the individuals with this mutation showed a phenotype characterized by persistent and extreme reactive aggressive behaviours, which included assault, attempted murder, arson and exhibitionism. Thus, low levels of the MAO-A enzyme in the brain may result in an enhancement of especially impulsive aggression.

The findings on MAO-A has been replicated in animal models, in particular in studies of mice. Male mice deficient in the MAO-A enzyme were found to be hyperaggressive and also showed heightened fear responses. In addition, studies found that mice deficient in the MAO-A enzyme had elevated brain levels of sero-

tonin, norepinephrine, and to a lesser extent dopamine, and also showed increased reactivity to stress and violent motions during sleep (Lesch & Merschdorf, 2000; Eisenberger *et al.*, 2007; Buckholtz, & Meyer-Lindenberg, 2008).

Reif *et al.* (2007) reported on the description of a repeat length polymorphism (*MAO-A*-uVNTR) in the promoter region of the *MAO-A* gene. This polymorphism consists of a 30-bp repeat element with 3, 3.5, 4, 5 or 6 copies of the repeat sequence. The 3.5- and 4-repeat alleles result in two to ten times faster transcription of the *MAO-A* gene in comparison with the 3-repeat allele. Consensus has not as yet been reached on the transcriptional efficiency of the 5- and 6-repeat alleles (Huang *et al.*, 2004; Brummett *et al.*, 2008). Eisenberger *et al.* (2007) dichotomized the alleles into low expression (3-repeats) (*MAO-A*-L) and high expression (3.5- and 4-repeats) (*MAO-A*-H) alleles.

The presence of the low expression allele (*MAO-A*-L) in both males and females has been associated with significantly greater levels of reactive aggression (Eisenberger *et al.*, 2007). Thus, lower levels of the MAO-A enzyme are associated with increased reactive aggression. In addition to increased aggression, Siever (2008) noted that people with low activity of the *MAO-A* gene also display significant volume reductions in the bilateral amygdala, anterior cingulate cortex, and subgenual anterior cingulate cortex. A notable finding in the literature on *MAO-A* genes and aggression is that there might possibly be a gene-environment interaction in the association between *MAO-A*-L individuals and aggression. It seems that individuals who have been exposed to childhood maltreatment are at increased risk of developing aggression related traits if they carry the *MAO-A*-L alleles, whereas carrying the *MAO-A*-H alleles protects maltreated children from developing excessive aggression. This association needs further investigation (Caspi *et al.*, 2002; Reif *et al.*, 2007; Siever, 2008).

2.2.3 Norepinephrine, dopamine and aggression

Since the findings on the role of levels of serotonin on aggression is so contradictory, it is necessary to investigate the possible roles on aggression of the other two neurotransmitters metabolized by the MAO-A enzyme, namely norepinephrine and dopamine. According to Buckholtz and Meyer-Lindberg (2008) knockout mice for the *MAO-A* gene displays greatly increased levels of serotonin and

norepinephrine, but very small increases in the levels of dopamine. For this reason the MAO-A enzyme is not seen as important in regulating the levels of dopamine as it is in regulating serotonin and norepinephrine levels. Dopamine will thus not be considered as part of the equation.

Gridley and Hoff (2007) tried to figure out the seemingly paradoxical puzzle of serotonin activity and aggression. They suggested that even if high levels of serotonin decreases aggression, the opposite effect found for low levels of MAO-A resulting in an increase in aggression, can possibly be explained by the other neurotransmitter affected by low levels of MAO-A, namely norepinephrine. Norepinephrine plays a role in the fight or flight response initiated by the body when threatened (Gridley & Hoff, 2007). Specifically, norepinephrine is associated with the "fight" response when a person perceives a stressor as challenging his/her control of a particular situation, and several authors therefore refer to norepinephrine as the "fight hormone" (Maglione-Garves *et al.*, 2005; Bayazit *et al.*, 2009).

According to Haller *et al.* (1998), even if norepinephrine has no effect on mechanisms directly involved in aggression, its indirect effects on other mechanisms during the fight or flight response would result in it having a profound effect on aggressive behaviour. These authors explained the effects of increased levels of norepinephrine on the fight or flight response as follows: increased central and peripheral noradrenergic activity leads to energy mobilization, other somatic effects (increased heart rate, temperature etc.), increased vigilance, decreased pain perception, enhanced olfaction and enhanced memory. All of these reactions combine to prepare the animal for aggression. According to Gridley and Hoff (2007), an abnormal accumulation of this neurotransmitter, as happens when MAO-A enzyme levels are diminished, results in these fight or flight responses staying in high gear all the time, and in effect lowering the individual's thresholds for reactive aggressive responses.

Even if it thus proves to be true that high levels of serotonin leads to a reduction in aggression, the effect of the *MAO-A* gene on aggression can still be explained by the effect of enhanced levels of norepinephrine overriding the inhibiting effect of serotonin, and resulting in an enhanced tendency for reactive aggression. Gridley and Hoff (2007) cautioned however that the workings of serotonin are still poorly understood, and that the effect of the *MAO-A* gene on aggression may be

much more complicated than merely assuming that “high” or “low” levels of a neurotransmitter leads to aggression.

2.3 Emotional intelligence

According to Petrides and Furnham (2003), the construct of emotional intelligence has been under intense examination. They offered the following description of the construct of emotional intelligence: “Broadly speaking, the construct of emotional intelligence posits that individuals differ in the extent to which they attend to, process, and utilize affect-laden information of an intrapersonal (e.g. managing one’s own emotions) or interpersonal (e.g. managing others’ emotions) nature”. Malterer *et al.* (2008) formulate it more clearly by stating that emotional intelligence refers to the ability to “recognize and regulate emotions in ourselves and in others”.

Mavroveli *et al.* (2008) suggested that emotional intelligence can primarily be divided into trait emotional intelligence (or trait emotional self-efficacy) and ability emotional intelligence (or cognitive-emotional ability). The fundamental difference between these two forms of emotional intelligence lies in the measurement of the constructs. Trait emotional intelligence is measured via self-report measures, whereas ability emotional intelligence is measured via maximal performance measures (i.e. tests in which the items have correct and incorrect answers) (Petrides & Furnham, 2003; Petrides *et al.*, 2006; Mavroveli *et al.*, 2008).

Mavroveli *et al.* (2008) argued against the use of maximal-performance measures for measuring emotional intelligence by noting that emotional experience is inherently subjective, and can therefore not be measured by the same types of tests used to measure, for instance, IQ. Petrides *et al.* (2006) supported this argument by stating that it would be impossible to make a distinction between “correct feelings” that normal people should be feeling, and “incorrect feelings” that normal people should try to suppress. Since the arguments posited by these authors are completely valid, the following description, and this study in general, will focus on trait emotional intelligence, rather than the more controversial construct of ability emotional intelligence.

Vernon *et al.* (2008a) defined trait emotional intelligence as “a constellation of emotional-related self-perceptions and dispositions located at the lower levels of

personality hierarchies". People high in trait emotional intelligence tend to be good at handling stress, and also tend to function well psychosocially (Ali *et al.*, 2009). Various authors argued that trait emotional intelligence is a personality trait, rather than a cognitive ability (Vernon *et al.*, 2008a, 2008b; Mavroveli *et al.*, 2008), and there is abundant evidence supporting this claim. Studies have especially focused on the correlations between the Big Five personality traits and trait emotional intelligence (Vernon *et al.*, 2008b).

The Big Five or five-factor approach to personality is seen as one of the most widely accepted models of personality structure (Jang *et al.*, 1996), and, according to Ekehammar *et al.* (2010) this theory provides a "distinctive outline of normal personality". The Big Five theory of personality posits that there are five basic dimensions that need to be identified in order to summarize individual differences in adult personality. These dimensions are: Neuroticism, Extraversion, Openness to Experience, Agreeableness, and Conscientiousness (Vernon *et al.*, 2008b).

Vernon *et al.* (2008b) stated that a comprehensive inventory of trait emotional intelligence would be expected to show roughly a 70% overlap with a measure measuring the Big Five, and that the greatest overlap would be with the dimensions of Neuroticism and Extraversion. Thus, it is believed that emotional intelligence forms part of the personality hierarchy, and encompasses the emotion-related aspects of personality (Vernon *et al.*, 2008b).

Further evidence supporting the theory that emotional intelligence is a personality trait comes from studies revealing a zero correlation between measures of trait emotional intelligence and ability emotional intelligence, and also low to zero correlations with measures of cognitive ability (Mavroveli *et al.*, 2008). Clearly, trait emotional intelligence cannot be seen as a cognitive ability, but much rather as a personality trait.

2.3.1 Heritable and environmental influences on emotional intelligence

Vernon *et al.* (2008a; 2008b) noted that in almost all the studies done on personality traits, the individual differences in these traits have been found to be primarily due to a combination of genetic and non-shared environmental factors, with the shared environment playing a very minor to no role. Since, as described

previously, emotional intelligence is seen as a personality trait, the same results would be expected as those shown for the other personality traits. In a family study done by Vernon *et al.* (2008a), it was found that roughly a third of the variance in trait emotional intelligence can be attributed to additive genetic effects. As expected, the familial aggregation estimates for trait emotional intelligence was the same as those reported for the Big Five dimensions of personality. In a second study done by the same authors using a twin design, it was found that the variance in emotional intelligence is almost entirely due to genetic and non-shared environmental influences, with shared environmental influences contributing only negligibly. These results thus strengthen the notion that emotional intelligence forms part of the personality hierarchy.

Vernon *et al.* (2009) stated that 40% of the variance in global trait emotional intelligence scores can be attributed to genetic factors, whereas 60% of the variance can be seen as due to factors in the non-shared environment. Further strengthening the connection between personality and emotional intelligence, is the results of a study done by Jang *et al.* (1996). In this study heritability estimates for Extraversion and Neuroticism, the two main personality factors overlapping with trait emotional intelligence, was found to be 41% and 53% respectively, which is quite close to the 40% estimate found for emotional intelligence.

2.3.2 A biological basis for emotional intelligence

Zeidner *et al.* (2003) viewed temperament as forming the biological basis for the development of emotional intelligence. According to Rothbart *et al.* (2000) temperament influences and is influenced by individual experiences, and, importantly, stated that one of its outcomes is the adult personality. So once again, personality and emotional intelligence is equated, with temperament presumably forming the biological underpinnings of both.

Rothbart and Derryberry (1981) defined temperament as “individual differences in reactivity and self-regulation assumed to have a constitutional basis”. *Constitutional* is defined by the same authors as “the relatively enduring biological makeup of the organism, influenced over time by heredity, maturation, and experience”. Finally, *reactivity* refers to the “excitability or arousability of the behavioural and physiological systems of the organism” (Rothbart *et al.*, 2000). Ac-

cording to Saudino (2005) the behavioural dimensions seen as being temperamental in origin include not only reactivity, but also emotionality, activity level, attention/persistence and sociability. A great deal of individual differences in these behavioural dimensions is present soon after birth.

From the above it should be clear that temperament refers, at least in part, to a biologically based disposition in organisms. Zeidner *et al.* (2003) further strengthened this argument by noting that temperament is probably linked to brain systems controlling emotion, motivation and attention. The amygdala, orbitofrontal cortex and cingulate cortex are mentioned as playing a possible role in this regard.

Rothbart and her colleagues (as cited in Sigelman & Rider, 2009) identified three dimensions of temperament, namely:

> **Surgency/Extraversion** – "the tendency to actively and energetically approach new experiences in an emotionally positive way (rather than being inhibited and withdrawn)".
>
> **Negative affectivity** – "the tendency to be sad, fearful, easily frustrated, and irritable (as opposed to laid back and adaptable)".
>
> **Effortful control** – "the ability to sustain attention, control one's behaviour, and regulate one's emotions (as opposed to an inability to regulate one's arousal and stay calm and focused)".

It is important to note, however, that a child's temperament alone does not determine emotional intelligence. Rather, it is the interaction between that child's temperament and the environment, especially interactions between the child and his/her caregivers that play the biggest role in determining the child's level of emotional intelligence. The child's temperament may lead to parental interaction that may either limit or enhance the acquisition of emotional skills. In short, the child's biology interacts with environmental experiences and in the process leads to the development of emotionally intelligent behaviours (Zeidner *et al.*, 2003).

2.3.3 Environmental influences on emotional intelligence

As mentioned above, genes alone do not determine a person's degree of emotional intelligence. Instead, both the environment and the biology of the person

contribute to the development of emotional intelligence, and usually it is an interaction between these two systems that has the greatest influence. Zeidner *et al.* (2003), for example, mentioned that emotional interactions between a child and his/her caretaker may play a role in the growth of neural circuits that influence emotional awareness and regulation. If the emotional interaction between caretaker and child is not of sufficient quality, or if it is otherwise abnormal, the neural circuits involved in emotion regulation may not develop sufficiently.

Evidence for environmental influences on emotional intelligence also comes from studies that show that emotional intelligence can be taught and improved by tailor-made programmes (Humphrey *et al.*, 2007; Ulutas, & Ömeroglu, 2007). According to Humphrey *et al.* (2007), understanding how emotional intelligence can be taught requires an understanding of the neuroanatomical framework of emotional intelligence. According to this framework, emotional intelligence refers to the ability of higher brain centres, such as the frontal lobes, to monitor and direct primitive emotional signals from the older brain structures, such as the amygdala, in such a way that these signals can be used constructively by the individual, rather than destructively. Education to enhance emotional intelligence would thus entail teaching the higher brain centres new or different patterns of behaviour, and in effect also leading to the acceptance by the deeper cerebral structures of this new way of responding. The aim would then be to teach individuals to perceive their emotional states using higher cortical centres as an "observer" of internal state, and then to use this knowledge to direct and control these internal states to better suit the external world (Humphrey *et al.*, 2007).

If the above can be done, it would provide proof of the influence of the environment on emotional intelligence. Various authors agree that it can be done (Humphrey *et al.*, 2007; Ulutas & Ömeroglu, 2007). In a study done by Ulutas and Ömeroglu (2007), a sample of 40 six-year old children attended a 12-week emotional intelligence program. After the twelve week program there was a significant improvement in the children's emotional intelligence levels as measured by the Sullivan Emotional Intelligence Scale.

Humphrey *et al.* (2007) also reported on intervention programmes specifically designed to promote emotional intelligence, and state that these programs led to significant improvements in vocabulary and fluency in discussing emotional expe-

riences, in management of emotions, and in emotional understanding. Note that here once again it is not just the environment or just the biology that influence emotional intelligence, but an interaction between the two (brain structures and mechanisms as well as teaching programs).

2.4 Aggression and emotional intelligence

From the literature, a few links between emotional intelligence and aggression can be identified, with the main findings being that emotional intelligence and aggression are negatively correlated. In a study done by Petrides *et al.* (2006), it was found that trait emotional intelligence directly impacts children's social relationships with friends, with children who perceive themselves as emotionally adept being more desirable to friends, than their emotionally cold and withdrawn counterparts. According to the same authors, peer rejection in turn leads to aggression. Thus, the link can be drawn as follows: low emotional intelligence leads to inadequate social relationships and rejection by peers, with rejection by peers in turn leading to aggression.

A more direct link between emotional intelligence and aggression can be drawn if the construct of empathy is considered instead of emotional intelligence. According to Ali *et al.* (2009), empathy is a very important aspect of emotional intelligence. There are many different definitions that attempt to describe the concept of empathy, but, according to Jolliffe and Farrington (2004), the following definition is one of the most current and inclusive: empathy is seen as "the ability to understand and share in another's emotional state or context". Considering this definition, it is clear why empathy is seen as a part of emotional intelligence.

Lauterbach and Hosser (2007) mentioned that empathic concern and being able to take another person's perspective inhibit aggression. These authors refer specifically to the case of provocation, and therefore, as per the earlier mentioned definitions of aggressive subtypes, to reactive forms of aggression. According to these authors, when a person is able to take another's perspective and share in that person's emotional distress, a reduction in their aggressive actions should occur in order to reduce the aggressor's own vicariously experienced distress and concern.

Adding to the above, emotionally intelligent people should, in the case of provo-

cation and an immediate emotional reaction of anger, have the ability to step outside the emotion and act as judges of it. In this way, aggression should be reduced. If this cannot be done, the person will not be able to suppress the desire to act on this emotion, and the consequences will be, and often are, disastrous (Pizarro & Salovey, 2002).

The preceding discussion mostly refers to reactive forms of aggression, but links, albeit more indirect links, can also be drawn between proactive aggression and emotional intelligence. As mentioned earlier, psychopathic individuals often display proactive forms of aggression, with limited emotional arousal and no need for provocation. According to Ali *et al.* (2009), an important aspect of psychopathy is a reduced ability to respond empathically to victims. Psychopaths have the ability to repeatedly cause serious harm to others, which is a direct indicator of a disturbance in empathically understanding another person's pain. Thus, the conclusion can be drawn that psychopathic individuals have low levels of empathy, and in effect low levels of an important aspect of emotional intelligence. In this way a link can be drawn between proactive forms of aggression (which is the aggression mostly displayed by psychopaths) and low levels of emotional intelligence.

Summary

From this review of the literature it is clear that both emotional intelligence and aggression are influenced by environmental and genetic factors. Aggression is a very prevalent and costly problem in our society, which, frequently leads to criminal and delinquent acts and the incarceration of individuals. At the moment, however, very limited treatment options exist for treating or preventing aggressive behaviours.

If the specific roles of genes underlying aggression can be described by researchers, this information can be used to examine the exact physiological pathways involved in aggressive behaviour, and from there pharmacological treatments can possibly be developed. Candidate genes such as the *MAO-A* gene that has been shown to have a definite influence on the brain neurotransmitters that play a role in aggression (serotonin and norepinephrine) is a good point of departure in the search for the myriad variety of susceptibility genes influencing

aggression. As discussed in this review, the *MAO-A*-uVNTR has repeatedly been shown to influence not only the expression of the *MAO-A* gene, but also behavioural traits like aggression, and in particular reactive aggression. If this association can be confirmed by further research on this polymorphism, such data could be very valuable in the quest to find treatments for aggressive and anti-social behaviour. It should be noted, however, that aggression is a complex trait, and many more genes than *MAO-A* alone will play a role in its aetiology. Plomin *et al.* (2008) confirmed this by stating that any of the thousands of genes expressed in the brain could possibly be studied as candidate genes for behavioural traits.

In addition to genetic factors, this review shows that factors like emotional intelligence have an important influence on aggression, albeit a more indirect influence. It seems that emotional intelligence, and in particular empathy, is negatively correlated with aggression. However, limited research on the influence of emotional intelligence on aggression has been published, and this field is in need of further investigation. Once again, if the association between emotional intelligence and aggression can be confirmed by research, and since it has been proven that emotional intelligence can be improved through tailor-made programmes, this information can lead to the development of programmes specifically aimed at improving those aspects of emotional intelligence that influence aggression. In this way aggressive tendencies can possibly be controlled.

In summary, this review underscores the importance of finding genes and genotypes that influence aggressive behaviour, but also the importance of looking at other factors that might possibly have an influence on aggression. This information can then be used as a starting point for the development of treatments for aggressive behaviours and tendencies.

CHAPTER 3

An evaluation of possible questionnaires for testing emotional intelligence, traumatic event exposure and aggression

Manuscript submitted to *Philos. Trans. Genet.* as part of “An evaluation of questionnaires for testing emotional intelligence, traumatic event exposure and aggression and the effects of social desirability bias on aggression scores” by Laubscher, N., Odendaal, Z., Schneider, S. & Spies, J.J.

Abstract

A wide variety of questionnaires are available for measuring aggression, emotional intelligence, and traumatic event exposure. These questionnaires differ in their applicability, depending on the particular study that is to be conducted, as well as factors like their reliability and validity coefficients in particular populations. The aim of this paper is to review a possible test battery of questionnaires for use in behavioural genetics studies on aggression, emotional intelligence and traumatic event exposure. The influences of socially desirable responding on self-report measures are discussed, and possible remedies for this problem are provided. The questionnaires that are discussed are: The Reactive-Proactive Aggression Questionnaire (RPQ); The Balanced Inventory of Desirable Responding (BIDR); The Trait Emotional Intelligence Questionnaire-Short Form (TEIQue-SF); The Stressful Life Events Screening Questionnaire (SLESQ). A brief description of each questionnaire is provided; whereafter the reliability and validity coefficients of each of the questionnaires are reviewed. In addition, administration and scoring instructions for each of the questionnaires are provided. Finally, a brief motivation for using each of the above mentioned questionnaires are presented.

Keywords: Behavioural genetics, questionnaires, reliability, socially desirable responding, validity

Introduction

Behavioural research tends to rely increasingly on self-report measures. This trend is mainly because of the high cost of behavioural observation and physiological measurements (Saunders, 1991). Self-report measures of personality and psychopathology are highly regarded for use in both clinical and research settings, and especially for use with children (Graybill & Blackwood, 1996). Self-report measures also have a role to play in behavioural genetics research. It is used frequently by researchers to determine heritability estimates of a trait in family, twin and adoption studies (Rhee & Waldman, 2002; Maes *et al*., 2007; Vernon *et al*., 2008a). In addition, some of the most important findings in behavioural genetics concern the influence of the environment. Heritability of behavioural traits are rarely, if ever, above 50%. This means that at least 50% of the variance in behavioural traits is due to environmental factors (Plomin, 1990; Plomin *et al*., 2008). Thus, self-report measures are also useful to determine main effects of environmental influences on behaviour (e.g. Hessl *et al*., 2001).

In addition, researchers in behavioural genetics are proving more and more frequently that gene-environment interactions have an important role to play in determining behaviour. Self-report measures are also frequently employed to assess the environmental components of these gene-environment interactions (Moffitt, 2005; Frazzetto *et al*., 2007; Kinnally *et al*., 2009).

The use of self-report measures incurs a different type of cost, however. That is, the validity of the measure is compromised when it makes use of self-report. This is due to contamination by response bias (Saunders, 1991). The most studied form of response bias is a concept known as social-desirability bias (SDB) (Paulhus, 1991; Fisher & Katz, 2000), also termed socially-desirable responding (SDR) (Mills & Kroner, 2005). Crowne and Marlowe (as cited in Podsakoff *et al*., 2003) defined social desirability as “the need for social approval and acceptance and the belief that it can be attained by means of culturally acceptable and appropriate behaviours”. SDB is exhibited when participants in a study give answers that they think will be socially approved, or that will make them look good (Paulhus, 1991; Saunders, 1991). The SDB construct can be adequately represented by two factors, namely *self-deception* and *impression management*

(Paulhus, 1984; Mills & Kroner, 2005). According to Mills and Kroner (2005), *self-deception* occurs when "a person believes a statement to be true of him- or herself even though it is not true". In contrast, *impression management* occurs when a person deliberately misrepresents the truth to avoid negative evaluation. Impression management is also referred to as *other-deception*.

Although SDB has been found to affect the measurement of personality variables, attitudes and self-reported behaviours (Fisher & Katz, 2000), its effect is especially salient in measures of behaviours or emotions that are socially disapproved (Saunders, 1991). Thus, measures of emotional intelligence will not be greatly influenced by SDB, but, importantly, measures of aggression have a high likelihood of being adversely influenced by this form of response bias. Social desirability can hide the true relationship between variables, produce false relationships between variables, or serve as a moderator variable that impacts the nature of the relationships between variables (Ganster *et al*., 1983; Podsakoff *et al.,* 2003).

Other types of response bias can also occur, but the "faking-good" SDB is seen as the most likely type of response bias to influence measures of socially disapproved behaviours such as aggression and violence (Saunders, 1991). Helfritz *et al.* (2006) cautioned that impression management may compromise the validity of self-report data on violence and aggression to such an extent that the use of self-report in the assessment of these behaviours is highly questionable. As previously noted, however, direct behavioural and physiological measurements are expensive. Thus, self-report is often the only way that research on behaviours like aggression and violence can be carried out cost effectively.

Helfritz *et al.* (2006) did a study on the usefulness of self-report measures in men accused of domestic violence. In this study the authors found that both perpetrators and innocent fathers used impression management to portray them in the most favourable light possible. Importantly, however, actual perpetrators of domestic violence still tended to score higher on measures of aggression than non-perpetrators. This result occurred despite their attempts to minimize their aggressive tendencies in the measures. The authors hypothesized that these individuals might be unable to estimate "normal" levels of aggression since they

have no insight into their own problematic behaviour. This resulted in their inability to minimize their true aggressive tendencies in the questionnaires (Helfritz *et al.*, 2006).

In spite of the above findings, Helfritz *et al.* (2006) still cautioned that any information obtained from people with a motive to answer dishonestly should be interpreted carefully. Various techniques are available for controlling SDB. On the one hand, the researcher can make use of procedural remedies. This includes, among other remedies, protecting the respondents' anonymity. If respondents know that their responses to questions concerning socially undesirable behaviours like aggression is completely anonymous, they should be less likely to make use of socially desirable responding. The researcher can also make sure that respondents know that there are no right or wrong answers to the questions, and that they should answer as honestly as possible. This technique should reduce evaluation apprehension and thus SDB (Podsakoff *et al.*, 2003).

Apart from procedural techniques, various statistical remedies are also available for controlling for SDB (Saunders, 1991; Podsakoff *et al.*, 2003). One of the most reliable techniques is to add an additional scale of SDB to the test battery. Care should be taken when choosing the items of the SDB measure. There should not be a too high correlation between items on the SDB scale and the construct being adjusted. In a correlation design, the SDB scale can be used as the control variable in regression analyses. Self-report scores for each participant can then be adjusted by two computational steps. First, the unstandardized regression coefficient in predicting the unadjusted score from the SDB scale is derived. The formula for simple regression is used: $Y = a + bX$, where X is the score on the measure of SDB and b is the unstandardized regression coefficient. Importantly, the regression coefficient needs to be determined separately for each sample being studied. Secondly, the unstandardized regression coefficient becomes the correction factor. The adjusted score can then be derived by making use of the following formula: $Y' = Y - b(\text{SDB score})$. In this instance, Y' represents the adjusted score and Y is the unadjusted score. In this formula, the regression coefficient is multiplied by the SDB score, and then subtracted from the unadjusted score (Saunders, 1991).

By making use of any of the above techniques to control for SDB, self-report questionnaires can be used for measuring "socially undesirable" constructs like aggression.

A wide variety of self-report questionnaires are available for measuring aggression, emotional intelligence, traumatic event exposure, and social desirability bias. In this article a possible test-battery for measuring these constructs was described. In addition, reasons were given for why the chosen questionnaires were seen as most suitable for use in this study.

The Reactive-Proactive Aggression Questionnaire (RPQ) (Appendix A) (Raine *et al.*, 2006)

In the past teacher rating scales and observational measures were mostly used to measure reactive and proactive aggression, but a self-report measure such as the RPQ has an important advantage over these measures. Intrinsic motivation is one of the important factors distinguishing proactive from reactive aggression. Intrinsic motivation is not always clear to an outside observer of an aggressive act, such as a teacher reporting on the teacher rating scale, but it is very salient to the person committing the act. Thus, a self-report measure is much more accurate in discerning whether intrinsic motivation was present or not when committing an aggressive act, and in effect is much more sensitive in discerning proactive from reactive forms of aggression (Raine *et al.*, 2006).

The RPQ in its final form is a 23-item self-report measure designed to measure proactive and reactive aggression in children and adolescents from the age of eight, and is also appropriate for use with adults (Baker *et al.*, 2008; Tuvblad *et al.*, 2009).

Reliability of the RPQ

The reliability of a measure is determined by how consistent the measure is (Wolfaardt & Roodt, 2005). Wolfaardt and Roodt (2005) proposed the following definition for reliability: "The reliability of a measure refers to the consistency with which it measures whatever it measures".

Various forms of reliability are used by researchers. The most frequently used, and also the most important, is internal consistency reliability. *Internal consistency reliability* is used to determine whether the items on a particular test are consistent with one another. For the items to be consistent, they all need to represent the same dimension, construct or interest area. A coefficient developed by Cronbach (1951), known as Cronbach's alpha, was specifically designed to measure internal consistency in tests for which there are no right or wrong answers to the items (as in the case of the RPQ) (Wolfaardt & Roodt, 2005; Salkind, 2008). The higher the consistency with which individual item scores varies from the total score of the measure, the higher the value of Cronbach's alpha (Salkind, 2008). In turn, the higher the value of alpha, the higher the internal consistency of the test, and the more reliable it is.

The adequacy of the reliability coefficient for a particular test depends on the use of the test (various authors, as cited in Wolfaardt & Roodt, 2005). Generally, standardized personality and interest measures should have reliability coefficients of 0.80 to 0.85. Measures used for making decisions about individuals should have a reliability coefficient of at least 0.85 (Wolfaardt & Roodt, 2005). General guidelines are also available for the interpretation of reliability coefficients. A coefficient of 0.9 or greater is seen as excellent; 0.8 to 0.89 is seen as good; 0.7 to 0.79 is adequate; and below 0.7 is seen as having limited applicability (Bhandari & Joensson, 2009). In contrast, Nunnally and Bernstein (1994) deemed an alpha coefficient of 0.60 or above as adequate for non-cognitive measures. In a study done by Raine *et al.* (2006) on 334 school boys from the Pittsburgh Youth Study, all three scales of the RPQ showed internal consistencies of 0.83 or above. Likewise, Baker *et al.* (2008) reported acceptable internal reliability scores for the RPQ in school-aged twins from Los Angeles, with Cronbach's alpha scores ranging from 0.74 to 0.94.

Another frequently used form of reliability is test-retest reliability. *Test-retest reliability* is used to examine whether the scores on a test is reliable over time (Salkind, 2008). This is done by administering the test twice to the same group of people at different times. Tuvblad *et al.* (2009) found adequate test-retest reliabilities for both measures of the RPQ, with the scores as follows: reactive ag-

gression r = 0.81 and proactive aggression r = 0.79. This sample was drawn from the University of Southern California Twin Study of Risk Factors for Antisocial Behaviour.

Validity of the RPQ

Wolfaardt and Roodt (2005) defined validity as a construct that concerns what a particular test measures, and how well it does so. In simple terms validity can be determined by the following question: "How well does this test measure what it says it measures?"

Raine *et al.* (2006) assessed four forms of validity of the RPQ. Firstly, construct validity was assessed. *Construct validity* of a measure is "the extent to which it measures the theoretical construct or trait it is supposed to measure" (Wolfaardt & Roodt, 2005). One way to measure construct validity is to determine whether there is a correlation between the measure and a similar measure. Raine *et al.* (2006) determined the construct validity of the RPQ by comparing the scores of the test with the scores of five self-report personality measures (psychopathy, schizotypy, impulsivity, stimulation-seeking, anxiety), as well as with the scores of hyperactivity and social/family measures. Overall the RPQ showed good construct validity. Secondly, convergent validity was assessed. A measure shows *convergent validity* "when it correlates highly with other variables with which it should theoretically correlate" (Wolfaardt & Roodt, 2005). Raine *et al.* (2006) assessed convergent validity by correlating the scores from the RPQ with both self-report and parent-rated aggression scores at age 16. The proactive, reactive and total aggression scales correlated significantly and positively with mothers' ratings of aggression and delinquency at that age. The mothers made use of the Child Behaviour Checklist to rate their children's aggression and delinquency.

Thirdly, criterion validity was assessed. *Criterion-related validity* can be described as a quantitative procedure in which a correlation coefficient is determined between a predictor and a criterion (Wolfaardt & Roodt, 2005). The psychological measure, in this case the RPQ is seen as the predictor, whereas the criterion is seen as a variable against which scores on the psychological measure are compared. For instance, the scores from a measure measuring job perfor-

mance (the predictor) would be compared to the actual job performance of the person (the criterion). Raine *et al.* (2006) assessed criterion validity by looking at age seven aggression measures as well as age 16 delinquency/violence classifications in relation to scores from the RPQ. Boys using violent-strong-arm tactics at age seven had significantly higher raw proactive, but not reactive aggression scores at age 16 when compared to controls. Boys initiating fights at age seven had significantly higher raw proactive, raw reactive and total aggression scores at age 16 in comparison with controls. Concerning delinquency, a seriously delinquent group of boys at age 16 had significantly higher raw proactive, reactive, and total aggression scores in comparison to all other groups (less seriously delinquent and non-delinquent).

Fourthly, discriminant validity was assessed. A measure demonstrates *discriminant validity* if it correlates minimally with variables from which it should theoretically differ (Wolfaardt & Roodt, 2005). Discriminant validity was assessed by Raine *et al.* (2006) by comparing the scores from the RPQ with scores from scales of a measure (the Child Behaviour Checklist) which differs conceptually from aggression (scales of withdrawal, somatic complaints, thought problems, and social problems). There were no significant correlations between scores on the RPQ aggression scales and any of the Child Behaviour Checklist scales. Discriminant validity is thus shown by the scales of the RPQ.

Therefore, the individual scales of the RPQ, as well as the total scale, are valid and reliable measures for measuring reactive and proactive aggression.

Administration and scoring

Each item from this questionnaire is coded on a three-point Likert scale (0 = never, 1 = sometimes, 2 = often). The questionnaire consists of 11 items measuring proactive aggression and 12 items measuring reactive aggression. Three scores are provided by the questionnaire, namely a score for proactive aggression, a score for reactive aggression, and a score for total aggression (Baker *et al.*, 2008; Tuvblad *et al.*, 2009).

The proactive aggression items of the RPQ are items 2, 4, 6, 9, 10, 12, 15, 17, 18, 20, 21 and 23 (Appendix A). The reactive aggression items are items 1, 3, 5,

7, 8, 11, 13, 14, 16, 19 and 22 (Appendix A). The scores on each of the subscales are summated. For a total aggression score, the two subscale scores are combined (Raine *et al.*, 2006).

For the purposes of this study, the RPQ and all the other questionnaires in the proposed test battery have been translated into Afrikaans. This was done in order to make completion of the questionnaires easier for Afrikaans speaking participants.

The Balanced Inventory of Desirable Responding (BIDR) (Appendix B) (Paulhus, 1991)

The BIDR consists of 40 items and is a self-report measure developed to measure people's tendency to give socially desirable responses in self-reports (Mills & Kroner, 2005). The BIDR is designed to measure two constructs, namely self-deceptive positivity and impression management (Paulhus, 1991). Self-deceptive positivity is defined by Paulhus (1991) as "the tendency to give self-reports that are honest but positively biased". Impression management is defined by the same author as "deliberate self-presentation to an audience".

The self-deception items in the scale place emphasis on and measure a person's over-confidence in their own judgements and rationality. The impression management items were developed from the rational standpoint that some people tend to over report their tendency to engage in desirable behaviours, while underreporting their propensity for engaging in undesirable behaviours. Since the item-content involves outwardly visible behaviours (e.g. I have said something bad about a friend behind his or her back), the person is seen as consciously lying if they do not answer truthfully. This is in contrast to a person deceiving himself/herself subconsciously (Paulhus, 1991).

Reliability of the BIDR

Paulhus (1991) reported on a number of studies in which the BIDR was used. The samples consisted of 884 religious adults, 433 college students, 100 college students and 48 members of alcoholics anonymous respectively. For these samples, values of internal consistency for the self-deception scale of the BIDR

ranged from α = 0.68 to α = 0.8. Values of internal consistency for the impression management scale ranged from 0.75 to 0.86. Summing of all 40 items led to an alpha value of 0.83.

In a study done by Paulhus, test-retest correlations over a 5-week period yielded scores of 0.69 and 0.65 for the self-deception and impression-management scales respectively (as cited in Paulhus, 1991).

Validity of the BIDR

Good concurrent validity is shown by the BIDR when all 40 items are summed for a total score of SDB. This total score has been shown to correlate with a coefficient of 0.71 with the Marlowe-Crowne scale, and 0.8 with the Multidimensional Social Desirability Inventory (Paulhus, 1991). Discriminant validity is shown by the self-deception and impression management scales in that they form separate factors in factor analysis (Paulhus, 1991). Lanyon and Carle (2007) found satisfactory concurrent validity for both the impression management and self-deception scales in a sample of 519 forensic clients and college undergraduates.

Administration and scoring

Respondents are asked to rate their agreement or disagreement with the 40 items of the BIDR on a seven-point Likert scale. The 40 items are stated as propositions. The scale is scored by firstly reversing the negatively keyed items, and then adding one point for each extreme response (6 or 7). This results in total scores of between 0 and 20 for each of the scales. In this way, the scoring key ensures that the highest scores will be obtained by respondents who give exaggeratedly desirable responses. The scorer also has the option of summing the scores on all 40 items of the BIDR. In this way an overall score on SDB can be obtained (Paulhus, 1991; Baumgartner & Steenkamp, 2006). As is the case with the RPQ, the BIDR will also be administered under strictly the same conditions for all participants in a particular sample. Participants in different samples may however differ in the way they complete the questionnaire.

Trait Emotional Intelligence Questionnaire (TEIQue) and Trait Emotional Intelligence Questionnaire-Short Form (TEIQue-SF) (Appendix C) (Furnham & Petrides, 2003)

The TEIQue measures trait emotional intelligence. It consists of 153 items which are rated on a 7-point Likert scale ranging from 1 (strongly disagree) to 7 (strongly agree). The measure encompasses 15 subscales, namely: Adaptability, Assertiveness, Emotion expression, Emotion management, Emotion perception, Emotion regulation, Empathy, Happiness, Impulsiveness, Optimism, Relationship skills, Self-esteem, Self-motivation, Social competence and Stress management. The subscales are organized under four factors, namely: Well-being, Self-control, Emotionality and Sociability (Petrides & Furnham, 2003; Mikolajczak *et al.*, 2007). The TEIQue was developed on the basis of the trait emotional intelligence theory and model. This model postulates that emotional intelligence is a personality trait, which can be located at the lower levels of personality hierarchies (Pérez *et al.*, 2005; Mikolajczak *et al.*, 2007).

Reliability of the TEIQue

In a sample of 120 first year psychology students from a London university, Petrides and Furnham (2003) found a full-scale internal consistency score for the TEIQue of 0.86. In the same sample, the subscale alpha values were as follows: Adaptability = 0.78; Assertiveness = 0.83; Emotion expression = 0.89; Emotion management = 0.61; Emotion perception = 0.81; Emotion regulation = 0.67; Empathy = 0.71; Happiness = 0.92; Impulsiveness = 0.61; Optimism = 0.86; Relationship skills = 0.66; Self-esteem = 0.91; Self-motivation = 0.67; Social competence = 0.80; and Stress management = 0.78. Although the internal consistencies for some of the subscales are slightly low, the full scale internal consistency score is adequate.

Validity of the TEIQue

Mikolajczak *et al*. (2007) found preliminary evidence for adequate convergent/discriminant validity, criterion validity and incremental validity for the TEIQue in a sample of 740 French people. In a recent study by Gardner and Qualter (2010), different validities were compared across three forms of trait emotional

intelligence questionnaires, which included the TEIQue. In this study the authors found that the TEIQue outperformed the other two measures for both concurrent and incremental validity.

It can thus be safely assumed that the TEIQue is a reliable and valid measure of trait emotional intelligence (full scale $\alpha = 0.86$; all subscales: $\alpha > 0.60$).

TEIQue-short form

The TEIQue-SF is a 30 item questionnaire designed to measure global trait emotional intelligence (Furnham & Petrides, 2003). This questionnaire is based on the full form of the TEIQue (Petrides & Furnham, 2003), as described above. As already described, the full form of the TEIQue provides comprehensive coverage of the trait emotional intelligence domains. To ensure adequate internal consistency, two items from each of the 15 subscales of the TEIQue full form were selected for inclusion in the TEIQue-SF. This technique also ensured broad coverage of the sampling domain of the emotional intelligence construct (Petrides, 2011). According to Furnham and Petrides (2003), the TEIQue-SF can be trusted to provide highly reliable global trait emotional intelligence scores, which correlate to a meaningful extent with a range of criteria. These criteria include coping styles, life satisfaction, personality disorders, perceived job control, and job satisfaction.

Administration and scoring of the TEIQue-SF

For the purposes of the above mentioned studies, only the TEIQue-SF will be completed by participants. Therefore, only the scoring key of the TEIQue-SF will be discussed here, and not that of the original TEIQue. The TEIQue-SF is a self-report measure that should not take more than seven minutes to complete. As in the TEIQue full form, items in the TEIQue-SF are also responded to on a 7-point Likert scale. This questionnaire is scored firstly by reverse scoring the items listed below and secondly by obtaining the sum of all the responses (Petrides, 2011).

Items to be reverse scored (Petrides, 2011) (Appendix C):

Item nr. 2: I often find it difficult to see things from another person's viewpoint.
Item nr. 4: I usually find it difficult to regulate my emotions.
Item nr. 5: I generally don't find life enjoyable.
Item nr. 7: I tend to change my mind frequently.
Item nr. 8: Many times, I can't figure out what emotion I'm feeling.
Item nr. 10: I normally find it difficult to stand up for my rights.
Item nr. 12: On the whole, I have a gloomy perspective on most things.
Item nr. 13: Those close to me often complain that I don't treat them right.
Item nr. 14: I often find it difficult to adjust my life according to the circumstances.
Item nr. 16: I often find it difficult to show my affection to those close to me.
Item nr. 18: I normally find it difficult to keep myself motivated.
Item nr. 22: I tend to get involved in things I later wish I could get out of.
Item nr. 25: I tend to "back down" even if I know I'm right.
Item nr. 26: I don't seem to have any power at all over other people's feelings.
Item nr. 28: I find it difficult to bond well even with those close to me.

The Stressful Life Events Screening Questionnaire (SLESQ) (Appendix D) (Goodman *et al.*, 1998)

Traumatic event exposure is usually measured in terms of the ramifications of the traumatic events within populations of survivors. Most measures, however, do not address the exposure to traumatic events independently. Since it has been found that some people may be exposed to multiple traumatic events in their lives, and since exposure to previous traumatic events may influence reactions to subsequent exposures, it is imperative to be able to measure lifetime exposure to a variety of traumatic events (Goodman *et al.*, 1998). This need for a measure of lifetime traumatic event exposure led Goodman and colleagues to develop the Stressful Life Events Screening Questionnaire (Goodman *et al.*, 1998).

The SLESQ is a self-report measure designed to measure past exposure to traumatic events. The traumatic events assessed consist of 13 DSM–IV (Ameri-

can psychiatric association, 2000) posttraumatic stress disorder Criterion A1 traumatic events. These are traumatic events that involve serious injury or death, or the threat thereof (Goodman *et al.*, 1998; Schnider *et al.*, 2007). The measure does not address Criterion A2 which consists of the subjective reactions to an event (Goodman *et al*, 1998).

Reliability of the SLESQ

Goodman *et al.* (1998) determined reliability scores for the SLESQ by making use of a sample of 202 male and female summer school students at a large eastern university in United States of America. In this study the SLESQ appeared to have good test-retest reliability, with Goodman *et al.* (1998) reporting an overall correlation of 0.89 between the numbers of events reported at time one versus time two screening. Convergent reliability was established through correlating the number of events reported on the screening measure with the number of events reported in an interview two weeks later. A good correlation coefficient of 0.77 was obtained (Goodman *et al.*, 1998). In a sample of 123 students at a Midwestern U.S.A. state university, Schnider *et al.* (2007) reported an internal consistency coefficient for the SLESQ of $\alpha = 0.59$. This may seem low, but according to the authors this was expected, since exposure to trauma is not a unitary construct.

Validity of the SLESQ

In the sample of 202 male and female summer school students, Goodman *et al.* (1998) found that the SLESQ displayed good concurrent validity, with prevalence rates obtained by the SLESQ mostly consistent with or higher than those found in a number of large prevalence studies. Convergent validity was also adequate, with a correlation of 0.77 between the total number of events reported at time one and time two.

Administration and scoring

Participants are asked to endorse or not endorse 13 traumatic events. For each traumatic event endorsed by the participant, the questionnaire asks a series of additional questions about the nature of their exposure to this event. These addi-

tional questions differ from item to item and consist of questions such as the participant's age at the time of the event, and possible injuries they may have received (Orsillo, 2001). Apart from the 13 traumatic events, the questionnaire also contains two final items of which the first asks participants whether multiple items refer to the same event. The final item of the questionnaire asks participants whether an event happened more than once, and if this is the case, they are asked to describe the additional episodes of the event (Orsillo, 2001).

The SLESQ takes an average of 10 minutes to complete for most respondents (Orsillo, 2001). The scale can be scored in a number of ways. For example, the scorer can count the total number of endorsed events, or count the number of endorsed events in a specific category. Schnider *et al.* (2007) for instance, changed the scaling of the SLESQ by querying event frequency instead of querying the presence/absence of an event. Last named authors then summed the items for a total score of trauma frequency.

Conclusion

In order to conduct a comprehensive study of aggression, emotional intelligence, and traumatic event exposure, measurements of these constructs need to be chosen with careful consideration. Although multiple questionnaires exist for the measurement of a specific psychological construct, not all questionnaires are equally appropriate for use in a particular study.

Various questionnaires are available for measuring the construct of aggression. Among the more frequently used are the Brief Anger-Aggression Questionnaire (Maiuro *et al.*, 1987), the Brown-Goodwin Aggression Inventory (Brown & Goodwin, 1986) and the Buss-Perry Aggression Questionnaire (Buss & Perry, 1992). Aggression might, however, be better understood if it is not seen as a unitary construct (Weinshenker & Siegel, 2002). The most commonly distinguished, and also the most frequently studied subtypes of aggression in the literature, are the constructs of reactive versus proactive aggression (Dodge & Coie, 1987; Geen, 2001; Brendgen *et al.*, 2006; Baker *et al.*, 2008). Although the above mentioned questionnaires are reliable and valid measures of aggression, they do not specifically measure reactive versus proactive aggression. Since it is important to view

aggression as a multidimensional construct (Weinshenker & Siegel, 2002), and since reactive and proactive aggression are the most frequently defined subtypes of aggression, it would make sense to use a measurement of aggression that specifically identify these two subtypes.

Raine *et al.* (2006) and Fossati *et al.* (2009) noted that there is a lack of time-efficient self-report measures specifically measuring reactive and proactive aggression. For this reason, Raine *et al.* (2006) developed the Reactive-Proactive Aggression Questionnaire (RPQ). If taking all of the above into account, it should be clear why the RPQ was chosen for use in this study.

Socially desirable responding can have a negative influence on psychological research. This is especially true for socially undesirable constructs like aggression. For this reason a social desirability measure was included in the battery of questionnaires chosen above specifically to statistically control for socially desirable responding. Baumgartner and Steenkamp (2006) noted that the two most popular measures of SDR are the Marlowe-Crowne Social Desirability Scale and the Balanced Inventory of Desirable Responding (BIDR). From these two measures the BIDR was chosen mainly because it is a newer measure of social desirability, developed in 1991. The Marlowe-Crowne Social Desirability Scale, in contrast, was developed in 1960 (Baumgartner & Steenkamp, 2006).

Emotional intelligence is mainly divided into trait-emotional intelligence and ability-emotional intelligence. Trait-emotional intelligence is measured via self-report measures, whereas ability-emotional intelligence is measured via maximum performance measures (Petrides & Furnham, 2003; Petrides *et al.*, 2006; Mavroveli *et al.*, 2008). More success has been achieved with the measurement of trait emotional intelligence, rather than ability emotional intelligence (Pérez *et al.*, 2005). For this reason only trait-emotional intelligence measures were considered for this test-battery.

Apart from the TEIQue described in this chapter, various other forms of trait-emotional intelligence measures are available. These include, among others, the Trait Meta-Mood Scale (Salovey *et al.*, 1995), the Schutte Emotional Intelligence Scale (Schutte *et al.*, 1998), and the Emotional Quotient Inventory (Bar-On,

1997). The TEIQue was chosen for its good psychometric properties in comparison with other trait-emotional intelligence measures, as summarized in Pérez *et al.* (2005). The TEIQue was also chosen for the easy availability of the short-form version (TEIQue-SF; Furham & Petrides, 2003) which is the version that will be used in the above mentioned studies.

It has been found that exposure to trauma, and especially the resultant development of symptoms of posttraumatic stress disorder, can increase aggression (Jakupcak & Tull, 2005). In order to statistically control for the influence of trauma exposure on aggressive tendencies, a trauma questionnaire was included in the test battery. Apart from the SLESQ described in this chapter, a number of other measures have been developed that aim to assess lifespan traumatic event exposure (Goodman *et al.*, 1998). These include, among others, the Traumatic Stress Schedule (Norris, 1990), the Traumatic Events Questionnaire (Vrana & Lauterbach, 1994) and the Traumatic Life Events Questionnaire (Kubany, 2000). The SLESQ was chosen for the test battery since it is a general traumatic event screening questionnaire which was specifically developed for use in community samples (non-treatment seeking samples). Furthermore, it was also chosen because of the rigorous examination of its psychometric properties carried out by Goodman and colleagues and the easy availability of this data (Goodman *et al.*, 1998).

Each of the above questionnaires was adapted by changing some of the item wording so that it would be more applicable to the South African context. Care was taken to ensure equivalence of meanings. Since none of the above mentioned questionnaires were developed specifically for use with South African populations, the adaptations were deemed necessary.

CHAPTER 4

Putative environmental influences on aggression: The effects of emotional intelligence and traumatic event exposure

Shortened manuscript submitted to *S. Afr. J. Psychol.* as “The effects of emotional intelligence and traumatic event exposure on aggression” by Laubscher, N., Odendaal, Z., Schneider, S. & Spies, J.J.

Abstract

The purpose of this study was to test the following hypotheses: 1) Male and female aggression scores differ significantly; 2) Emotional intelligence and reactive/proactive/total aggression are negatively correlated; 3) Trauma exposure and reactive/proactive/total aggression are positively correlated; 4) Emotional intelligence and traumatic event exposure combined account for a significant percentage of the variation in total aggression scores. The sample for this study consisted of young adults (male and female) from the central region of South Africa, as well as some of their friends and family members (N=182). The average age of the participants was 27.38 years. Questionnaires measuring reactive-, proactive- and total aggression, emotional intelligence and traumatic event exposure were administered. The findings showed that no significant differences for aggression were observed between males and females. Emotional intelligence was, as expected, significantly negatively correlated with all three types of aggression, although the practical significance of these correlations was small. Traumatic event exposure was, also as expected, positively correlated with all three types of aggression, with small (proactive aggression) to medium practical significance (reactive- and total aggression). Traumatic event exposure and emotional intelligence combined accounted for 17.3% of the variance in total aggression scores (significant at the 99% level). In conclusion, environmental effects do account for a significant proportion of the variance in aggressive behaviours, with both traumatic event exposure and emotional intelligence significantly correlated with aggression.

Keywords: Aggression, Emotional intelligence, Environmental factors, Multiple regression, Trauma

Introduction

Geen (2001) provided the following comprehensive definition for aggression: "Aggression is the delivery of an aversive stimulus from one person to another, with intent to harm and with an expectation of causing such harm, when the other person is motivated to escape or avoid the stimulus". Human aggression and violence have become a major public health concern around the world (Miczek *et al.*, 2002; Weinshenker & Siegel, 2002; Buckholtz & Meyer-Lindenberg, 2008), with each antisocial individual costing society up to 10 times more than their non-aggressive counterparts in healthcare and social services costs (Scott *et al.*, 2001; Buckholtz & Meyer-Lindenberg, 2008). Few treatment options have proven to be effective in combating this universal problem (Miczek *et al.*, 2002; Volavka, 2002).

One way of combating aggression and all of its negative ramifications is to gain a better understanding of the construct. Bandura (1973) proposed that differentiating between subtypes of aggression might be a way of achieving just that (Waschbusch *et al.*, 1998). The subtypes of reactive aggression and proactive aggression are the most commonly distinguished subtypes of aggression in the literature (Dodge & Coie, 1987; Geen, 2001; Brendgen *et al.*, 2006; Baker *et al.*, 2008). Dodge and Coie (1987) defined reactive aggression as "a defensive reaction to a perceived threatening stimulus". Proactive aggression is defined by the same authors as "aggression without immediate provocation or instigation…used as a viable means of reaching some specific outcome". To put it more clearly, reactive aggression is an angry and impulsive form of aggression which occurs in response to perceived provocation. Proactive aggression, on the other hand, is not associated with anger, but is rather a reward-centred form of aggression (Dodge, 1991; Waschbusch *et al.*, 1998).

Distinguishing between subtypes of aggression is only the first step to gain a better understanding of the construct. An even better understanding and possible solutions may come from trying to identify etiological factors influencing aggression. Research in the field of behavioural genetics has shown that one of the main factors influencing aggression is an individual's genetic constitution (Brendgen *et al.*, 2006; Tuvblad *et al.*, 2009). In this regard, Tuvblad *et al.*

(2009) found that heritability explained up to 26% of the total variance in reactive aggressive behaviour, and up to 32% of the total variance in proactive aggressive behaviour.

Contrary to what the name suggests, the field of behavioural genetics does not only aim to provide information about the genetic influences on behaviour, but also about the environmental influences (Plomin, 1990). Likewise, both genes *and* environment are expected to influence individual levels of aggression. As mentioned, genetic factors only account for 26% of the variance in reactive aggression, and 32% of the variance in proactive aggression. The rest of the variance is accounted for by environmental factors (Tuvblad *et al.*, 2009).

One such possible environmental factor is emotional intelligence. Malterer *et al.* (2008) defined emotional intelligence as the ability to "recognize and regulate emotions in ourselves and in others". Furthermore, emotional intelligence is mainly divided into two types, namely trait emotional intelligence and ability emotional intelligence (Petrides & Furnham, 2003; Mavroveli *et al.*, 2008). The primary difference between these two types of emotional intelligence is the way in which they are measured. Trait emotional intelligence is mainly measured through making use of self-report measures. Ability emotional intelligence on the other hand is measured by maximal-performance measures (Petrides & Furnham, 2003; Petrides *et al.*, 2006; Mavroveli *et al.*, 2008). Emotional intelligence in this paper refers to the construct of trait emotional intelligence.

In the search for possible links between emotional intelligence and aggression, it has been found that low scores in emotional intelligence lead to the rejection of children by their peers, and may result in antisocial conduct and delinquency later in life (Petrides *et al.*, 2006). Furthermore, one of the components of emotional intelligence is empathy. Empathy, in a nutshell, entails the ability to feel what other people are feeling. Low empathy can thus possibly facilitate aggression, since the non-empathic person is unable to experience vicariously what their aggressive actions are doing to other people (Joliffe & Farrington, 2004, 2007; Lauterbach & Hosser, 2007). Theoretically then, emotional intelligence and aggression should be negatively correlated, with higher emotional intelligence scores leading to less aggression, and *vice versa*.

Apart from emotional intelligence, traumatic event exposure may also influence levels of anger and aggression (Jakupcak &Tull, 2005; Dyer *et al.*, 2009; Vandenberg & Marsh, 2009). Theoretically, people exposed to trauma may develop cognitive distortions that result in neutral events being interpreted as threatening. These individuals consequently experience excessive arousal in the face of an actually "non-threatening" event. This excessive arousal impairs the individual's ability to choose an appropriate response to cope with the "threatening" event, frequently resulting in the individual choosing aggressive responses (Chemtob *et al.*, 1997; Kivisto *et al.*, 2009). What happens in effect is that the person previously exposed to trauma rapidly shifts into "survival mode", notwithstanding the actual validity of the perceived threat. The activation of this "survival mode" in people has at its core the activation of anger structures (Chemtob *et al.*, 1997).

Since an event "provokes" the aggressive response in these individuals, and since anger is involved, it is clear that this theory points to reactive aggressive behaviours rather than proactive aggression. Research confirms this association, with studies consistently finding links between reactive forms of aggression and childhood abuse, as well as other aversive experiences. Proactive aggression has not been associated with these traumatic experiences (Kempes *et al.*, 2005; Blair *et al.*, 2006; Brendgen *et al.*, 2006; Baker *et al.*, 2008; Tuvblad *et al.*, 2009).

Dyer *et al.* (2009) reported that the vast amount of studies exploring aggression in people previously exposed to trauma, have mainly focused on male war veterans. Chemtob *et al.* (1997) cited a number of case studies involving male war veterans, each with a considerable amount of inappropriate and function-limiting anger and aggression. In order to generalize these results, Kivisto *et al.* (2009) did a study on the influence of symptoms of posttraumatic stress disorder (PTSD) on aggressive responses in undergraduate students. PTSD is a DSM-IV-TR (American Psychiatric Association, 2000) described anxiety disorder caused by exposure to a traumatic event. Individuals with this disorder tend to be chronically over aroused and quick to anger (American Psychiatric Association, 2000; Barlow & Durand, 2009). In line with the results on war veterans,

Kivisto *et al.* (2009) reported that PTSD symptomology led to an increase in aggressive responding in the undergraduate students. Since PTSD develops after exposure to trauma, direct links can be drawn between traumatic event exposure and hyperactive aggressive responses.

In accordance with the above discussion, the main aims of this study were firstly to determine whether there are correlations between trait emotional intelligence and reactive/proactive and total aggression scores. Secondly, possible correlations between traumatic event exposure and reactive/proactive and total aggression scores were also investigated. This study also aimed to determine the percentage of variance in total aggression that can be accounted for by traumatic event exposure and emotional intelligence. Finally, the next chapters of this dissertation will focus on the behavioural genetics of aggression. This study will thus be followed up by a family-study and a genetic association study to determine the genetic influences on aggression. Since gender may play a potentially important role in genetic studies, it was decided to also investigate the effects of gender on average aggression scores, and on the various correlation coefficients.

Research hypotheses

The following hypotheses were tested:

Hypothesis 1: There is a significant difference between male and female average aggression scores.

Hypothesis 2: There is a negative correlation between emotional intelligence and reactive/proactive and total aggression.

Hypothesis 3: There is a positive correlation between trauma exposure and reactive/proactive and total aggression.

Hypothesis 4: The combination of emotional intelligence and traumatic event exposure account for a significant proportion of the variance in total aggression.

METHODS

Research Design and Sample

A cross-sectional correlation design was used in order to test the various research hypotheses. Convenience and snowball sampling methods were used to recruit participants. Questionnaires were given to young adults from the Central regions of South Africa, who were asked in turn to hand the questionnaires to their friends and relatives[1]. The questionnaires contained a covering letter explaining the objectives of the study, and that participation in the study is completely voluntary. Participants were also informed in this letter that they were free to withdraw from the study at any time. Furthermore, participants were assured that any identifying particulars would be treated in the strictest confidence by the researchers. A total of 182 questionnaires were received back for analysis.

The final sample comprised of 63.2% female respondents and 36.8% male respondents, with a mean age of 27.38 years. Demographic information was also collected for race, home language and level of education (Table 4.1). It should be noted that the study sample was too small to be representative of the various South African population groups. The diversity of the South African population necessitates the recruitment of much larger sample groups in order to truly achieve representativeness.

Measuring instruments

Self-compiled Biographical Questionnaire.

The Reactive-Proactive Aggression Questionnaire (RPQ; Raine et al*., 2006)* is a 23-item self-report measure designed to measure proactive and reactive aggression in children, adolescents and adults. The questionnaire comprises two subscales assessing the reactive and proactive components of aggression. The subscale scores can also be summed to obtain a total aggression score (Baker *et al.*, 2008; Tuvblad *et al.*, 2009). All three subscales of the RPQ have been

[1] Ethical clearance for this study was obtained from the Medical Ethics Research Committee of the University of the Free State (ECUFS 152/2011)

shown to have good internal consistency scores, with α = 0.83 or above in a sample of 334 adolescent boys from the Pittsburgh Youth Study (Raine *et al.*, 2006). Good construct, convergent, criterion and discriminant validity have been quoted for the RPQ (Raine *et al.*, 2006). Items are responded to on a 3-point Likert scale, ranging from 0 (never) to 2 (often).

Table 4.1 Demographic information of the respondents

Item	Category	Number	Percentage
Gender	Male	67	36.8
	Female	115	63.2
Race	Black	57	31.3
	White	118	64.8
	Coloured	7	3.8
	Other	0	0
Home Language	Afrikaans	97	53.3
	English	29	15.9
	Sesotho (southern and northern)	24	13.2
	Siswali	2	1.1
	Setswana	6	3.3
	Tshivenda	1	0.5
	Isixhosa	8	4.4
	Isizulu	5	2.7
	Other	2	1.1
	Missing data	8	4.4
Education level	Grade 9 – 11	4	2.2
	Grade 12	101	55.5
	Grade 12 + diploma	12	6.6
	Grade 12 + degree	38	20.9
	Grade 12 + postgraduate degree/diploma	26	14.3
	Missing data	1	0.005

The Trait Emotional Intelligence Questionnaire – Short Form (TEIQue-SF; Furnham & Petrides, 2003) consists of 30 items and is designed to measure

global trait emotional intelligence (Furnham & Petrides, 2003). This questionnaire is based on the full form of the TEIQue (Petrides & Furnham, 2003). The full form provides comprehensive coverage of the trait emotional intelligence construct. Petrides and Furnham (2003) found a full-scale internal consistency score for the TEIQue as α = 0.86 in a sample of 120 first year students in London. Preliminary evidence was also found for convergent/discriminant validity, criterion validity and incremental validity for the full form of the TEIQue in a sample of 740 French speaking people (Mikolajczak *et al.*, 2007). In order to ensure similar reliability and validity scores for the TEIQue-SF, this measure was developed by choosing two items from each of the 15 subscales of the TEIQue full form (Petrides, 2011). Consequently, the TEIQue-SF can be trusted to provide highly reliable global trait emotional intelligence scores which correlate meaningfully with a range of criteria. Items are responded to on a 7-point Likert-scale, ranging from 1 (completely disagree) to 7 (completely agree).

The Stressful Life Events Screening Questionnaire (SLESQ; Goodman et al., 1998) is a 13 item self-report measure designed to measure past exposure to traumatic events. The traumatic events assessed encompasses post-traumatic stress disorder Criterion A1 traumatic events. These events involve serious injury or death, or the threat thereof (Goodman *et al.*, 1998; Schnider *et al.*, 2007). This measure does not assess subjective reactions to traumatic events (Goodman *et al.*, 1998). In a sample of 123 students at a Midwestern university in the United States, internal consistency for the SLESQ was reported as α = 0.59. Although this score may seem low, exposure to trauma is not a unitary construct, and this score is in the expected range (Schnider *et al.*, 2007). Goodman *et al.* (1998) found good concurrent and adequate convergent validity for the SLESQ in a sample of 202 male and female summer school students. Participants are asked to answer either “yes” or “no” to a series of questions. In addition, the questionnaire includes short descriptive questions under each main question.

Translation and adjustment of questionnaires

All questionnaires were translated from English into Afrikaans by a bilingual translator, and translated back into English by the researcher in order to ensure

equivalent meanings. The translations were deemed necessary since the population consisted of both Afrikaans and English speaking participants. The participants with African home-languages mostly speak English, and thus preferred completing English questionnaires.

The wording of the questions or statements in each of the questionnaires (RPQ, TEIQue-SF, SLESQ) was modified in order to make it easier to understand for the participants. Great care was taken, however, to ensure that the meaning of the questions/statements remained the same. The wording of the main items of the SLESQ was also modified to some extent in order to better suit the population under study (See Appendix D). The descriptive questions under each main item were removed in order to prevent possible re-traumatisation of participants. Thus, participants only answered "yes" or "no" to the main items. Apart from these modifications, one additional question was added to the SLESQ, namely: "Other than the experiences mentioned in item 7, have you ever been emotionally abused by a romantic partner, date, sibling, family member, stranger or someone else?". This question was added to ensure that emotional abuse was also covered by the questionnaire.

Statistical analysis

The Microsoft Excel software program was used for statistical analysis. In addition, the OpenStat4 (2011) program for Windows available freely from (http://www.statpages.org/Miller/openstat) was used. Since the questionnaires were modified and translated, it was deemed necessary to calculate Cronbach's alpha coefficients for both language versions of the questionnaires. Descriptive statistics were employed to summarize the data from the questionnaires. In order to test whether there was a significant difference between male and female average aggression scores (hypothesis 1), the *t*-test for independent groups was used. Since the sample sizes for the two groups differed (males = 67 and females = 115), it was possible that the variances of the two groups were not equal (a violation of one of the assumptions for a *t*-test). In this instance, the *"Welch-Satterthwaite approximation"* should be used instead of the *t*-test (Howell, 2010). In this study the variances did not differ, and thus only the *t*-test was used.

To test for possible correlations between emotional intelligence and aggression (hypothesis 2), as well as between trauma and aggression (hypothesis 3), Pearson's product moment correlation coefficients were calculated. In order to determine whether the results held any practical significance, effect sizes were determined. According to Cohen (as cited in Van der Westhuizen, 2008) the best way to test for effect sizes in linear correlations is by making use of the correlation coefficient itself (*p).* In accordance the following guidelines for interpreting effect sizes are proposed: $p = 0.1$ small effect, $p = 0.3$ medium effect and $p = 0.5$ large effect. In order to test whether the combination of emotional intelligence and traumatic event exposure account for a significant proportion of the variance in total aggression (hypothesis 4), stepwise multiple regression analysis was used.

The 99% level of statistical significance was employed throughout the study.

Results and Discussion

Reliability coefficients and descriptive statistics of questionnaires

The Cronbach's alpha (α) reliability coefficients for the Afrikaans and English versions of the TEIQue-SF, RPQ and SLESQ were calculated. Cronbach's alpha values were approximately 0.7, with the lowest alpha value being 0.6 (Table 4.2). This is deemed acceptable by Nunnally and Bernstein (1994), who proposed a minimum alpha coefficient of 0.6 for non-cognitive measures. Thus, both the Afrikaans and English questionnaires, as well as the various subscales of each, can be seen as reliable measures of the constructs under study.

Apart from Cronbach's alpha, the mean, standard deviation and range of the questionnaires were determined. Notably, participants showed considerably higher reactive aggression scores than proactive aggression scores. It thus seems that reactive aggression is much more prevalent in this sample (Table 4.3). Raine *et al.* (2006) also found that reactive aggression was considerably more prevalent than proactive aggression in a sample of adolescents from the Pittsburgh Youth Study.

Table 4.2 Cronbach's alpha coefficients for TEIQue-SF, RPQ and SLESQ.

Questionnaire	Subscales and Totals	Afrikaans α	English α	Afrikaans and English α
TEIQue-SF modified		0.84	0.83	0.84
RPQ	Reactive aggression	0.78	0.77	0.78
	Proactive aggression	0.74	0.60	0.68
	Total aggression	0.84	0.81	0.83
SLESQ		0.75	0.67	0.72

[α: Cronbach's correlation coefficient]

Table 4.3 Descriptive statistics for TEIQue-SF, RPQ and SLESQ.

		Male	Female		Total group				
		Mean	Mean		Mean	SD	Minimum	Maximum	Range
TEIQue-SF		152.36	153.11		152.84	18.47	97	192	95
RPQ	Reactive	7.42	7.14		7.24	3.45	0	19	19
	Proactive	1.67	1.22		1.39	1.86	0	10	10
	Total	9.09	8.36		8.63	4.78	0	28	28
SLESQ		3.28	2.83		3.00	2.52	0	10	10

[SD: Standard Deviation]

Comparisons of average aggression scores for the two genders

T-tests for independent groups were calculated to determine whether a significant difference exists between the mean aggression scores (reactive-, proactive-, and total aggression) of the two genders.

No statistically significant differences could be detected in the mean aggression scores of males and females (Table 4.4). This finding held true for reactive-, proactive- and total aggression scores ($p > 0.01$). Although not statistically

significant, it was also clear that the means for men for all three aggression types were slightly higher (Table 4.4). Since no statistically significant results were obtained, effect sizes were not calculated. The results concur with the conclusions drawn from a meta-analysis performed by Björkqvist (1994). Here the author concluded that it is "nonsensical" to claim that gender differences exist for aggression. The results are also in line with a more recent study done by Connor *et al.* (2003) in which it was concluded that no significant gender differences exist in reactive and proactive aggression.

Table 4.4 T-tests for comparison of the mean scores of the two genders

Variable	Gender						t	Sig (2-tailed)(p)
	Male			Female				
	N	Mean	SD	N	Mean	SD		
Reactive aggression	67	7.418	3.115	115	7.139	3.634	-0.526	0.600
Proactive aggression	67	1.672	2.055	115	1.217	1.721	-1.600	0.111
Total aggression	67	9.090	4.515	115	8.357	4.931	-0.997	0.320

[N: number of participants; SD: Standard Deviation; t: t-test for independent samples; Sig(2-tailed)(p): level of statistical significance]

Correlations between emotional intelligence and aggression

In order to test hypothesis 2, Pearson's product moment correlation coefficients were calculated. It is clear that statistically significant negative correlations existed between emotional intelligence and reactive, proactive, and total aggression (Table 4.5). All three correlation coefficients were smaller than the $p = 0.3$ level proposed by Cohen for a medium effect size. Thus, as expected and stated in hypothesis 2, emotional intelligence did correlate negatively with aggression, with the highest negative correlation being between total aggression and emotional intelligence ($r = 0.272$). Although the correlations with reactive-, proactive- and total aggression were statistically significant at the 99% level of significance, the practical significance of the correlations was very small. Surprisingly, very few studies have been performed on the relationship between emotional intelligence and aggression. However, the results are consistent with

a correlation of -0.15 ($p < 0.10$) found by Petrides *et al.* (2006) between trait emotional intelligence and aggression in 160 school age children. A possible reason for the low correlations found in both these studies could be the use of a global trait emotional intelligence score. According to Petrides *et al.* (2006), trait emotional intelligence comprises of several sub-domains, with well-being, self-control, emotionality and sociability being just a few. It could be that some of the sub-domains, if measured for specifically, might correlate higher with the re-active-, proactive-, and total aggression constructs. It is also possible, however, that the low correlations reflect the true nature of the relationship between emotional intelligence and aggression, with emotional intelligence having little effect on aggressive behaviour.

Table 4.5 Correlation coefficients between emotional intelligence and aggression for the total group as well as for the genders separately (one-tailed test).

Aggression	Emotional Intelligence				
	Total group		Male	Female	Z+
	r		*r*	*r*	
Reactive aggression	-0.250*		0.015	-0.359*	2.494
Proactive aggression	-0.235*		-0.132	-0.299*	1.121
Total aggression	-0.272*		-0.050	-0.369*	2.152

[*r:* pearson product moment correlation coefficient; Z: Fisher's *r- to z-* transformation score]

*$p \leq 0.01$

+ $p \leq 0.01$ (z required for significance for two-sided test: ±2.576)

Although not practically significant, the statistically significant negative correlations still indicate that the higher a person's emotional intelligence, the lower his/her levels of aggression. This holds true for scores on the proactive-, reactive-, and total aggression scales.

When looking at the different genders an interesting trend could be noticed. Females had statistically significant negative correlations between emotional intelligence and aggression for all three types of aggression. Medium effect sizes occurred, pointing to moderate practical significance of the correlations. For males, in contrast, none of the correlations were statistically significant.

While all the other correlations were negative, reactive aggression was slightly positively correlated with emotional intelligence.

In order to test the significance of these differences in the correlation coefficients of males and females, Fisher's *r- to z*-transformation was carried out. To determine whether a statistically significant (99% level) difference in correlations for males and females for all three types of aggression was present, the Z_{z1-z2}-value was calculated. None of the differences between the correlation coefficients for males and females were statistically significant at the 99% level (Table 4.5). Although the differences were not statistically significant, it does seem that the correlations between all three types of aggression and emotional intelligence are higher for females than for males. No studies could be found on the mitigating effect of gender on this correlation. This observation is thus in need of further investigation.

Correlations between trauma and aggression

Similarly Pearson's product moment correlation coefficient was used to determine whether a positive relationship exists between trauma and aggression, thus testing hypothesis 3.

Table 4.6 Correlation coefficients calculated between traumatic event exposure and aggression for the total group as well as for the genders separately (one-tailed test)

Aggression	Traumatic event exposure			
	Total group	Male	Female	Z+
	r	*r*	*r*	
Reactive aggression	0.340*	0.237	0.396*	-1.132
Proactive aggression	0.255*	0.145	0.325*	-1.220
Total aggression	0.344*	0.229	0.405*	-1.254

* $p \leq 0.01$

\+ $p \leq 0.01$ (z required for significance for two-sided test: ±2.576)

The results indicated positive correlations between exposure to traumatic events and aggression (Table 4.6). All three correlation coefficients (reactive-, proactive- and total aggression) were significant at the 99% level of significance.

In accordance with Cohen's guidelines for effect sizes, the correlations for both reactive aggression and total aggression had medium effect sizes ($0.3 < p < 0.5$). The correlation between proactive aggression and traumatic event exposure had only a small effect size ($p < 0.3$). These effect sizes indicated that the correlations between traumatic event exposure and reactive/total aggression were of moderate practical value. The correlation for proactive aggression, however, was of small practical value. The largest positive correlation occurred between total aggression scores and traumatic event exposure ($r = 0.344$). These results were in line with the literature reporting high levels of reactive aggression, but not proactive aggression, in people exposed to traumatic experiences (Kempes *et al.*, 2005; Blair *et al.*, 2006; Brendgen *et al.*, 2006; Baker *et al.*, 2008; Tuvblad *et al.*, 2009). This finding also supported the theory proposed by Chemtob *et al.* (1997), where exposure to traumatic events leads to quick and unnecessary activation of a "survival mode". This "survival mode" is characterized by hyper-arousal and anger, two of the cornerstones of reactive aggression. It should be noted, however, that only a correlation study was performed. This type of study does not indicate causation.

These statistically significant positive correlations indicated that the higher the number of traumatic events a person is exposed to, the more aggressive that person tends to be.

Regarding the different genders, males showed no statistically significant correlations (99% level) for either type of aggression. Non-significant positive correlations between traumatic event exposure could however be observed. As was the case with emotional intelligence, females showed statistically significant positive correlations (99% level) for all three types of aggression. These correlations all had medium effect sizes ($0.3 < p < 0.5$), indicating moderate practical significance.

The significance of the differences between the male and female correlations was examined by using Fisher's r to z transformation, as described for the emotional intelligence/aggression correlations. From the results it can be seen that none of the differences between the correlations for males and females reached statistical significance at the 99% level. If disregarding statistical significance, it

did seem that females tended to have higher correlations than males between the different types of aggression and traumatic event exposure (Table 4.6). One possible explanation for this is that higher rates of PTSD have been reported for female than for male trauma victims (Stein *et al.*, 1997; Holbrook *et al.*, 2002). Since, as already mentioned, increased aggression is part of the symptomology of PTSD, it is plausible that higher rates of PTSD in females would lead to higher rates of aggression. This would explain the higher positive correlation between trauma and aggression observed for females.

Multiple regression analysis of variables

In order to determine the percentage of variance in total aggression scores that can be explained by emotional intelligence and traumatic event exposure, a stepwise multiple regression analysis was carried out. Total aggression served as the dependent variable, while emotional intelligence and traumatic event exposure served as the independent variables. Tests of significance were carried out at the 99% level. Traumatic event exposure was entered into the regression model first, followed by emotional intelligence.

Table 4.7 Step 1 of stepwise multiple regression analysis

Independent Variable: Traumatic event exposure **Dependent Variable: RPQ total**					
Source	**df**	**SS**	**MS**	**F**	**Sig.**
Regression	1	493.503	493.503	24.357	0.000
Residual	180	3647.090	20.262		
Total	181	4140.593			

R	**R^2**	**F**	**Sig.**
0.345	0.119	24.357	0.000

[df: degrees of freedom; SS: sum of squares; MS: mean of squares; F: test statistic for ANOVA; Sig.: level of statistical significance; R: multiple correlation coefficient; R^2: coefficient of multiple determination]

From these results the following deductions could be made: In step 1 of the procedure, traumatic event exposure was entered into the regression equation first. R^2 = 0.119, thus 11.9% of the variance in total aggression could be at-

tributed to traumatic event exposure. This percentage was significant at the 99% level of statistical significance ($p < 0.01$) (Table 4.7).

Table 4.8 Step 2 of stepwise multiple regression analysis

Independent variables: Traumatic event exposure, Emotional intelligence **Dependent variable: RPQ total**					
Source	**df**	**SS**	**MS**	**F**	**Sig.**
Regression	2	714.424	357.212	18.663	0.000
Residual	179	3426.169	19.141		
Total	181	4140.593			

R	**R^2**	**F**	**Sig.**
0.415	0.173	18.663	0.000

Variable	**Beta**	**B**	**Std. Error**	**t**	**Sig.**
Traumatic event exposure	0.317	0.600	0.130	4.623	0.000
Emotional intelligence	-0.233	-0.060	0.018	-3.397	0.001

[Beta: standardized regression coefficients; B: unstandardized regression coefficients; Std. Error: standard error]

In step 2 of the procedure, emotional intelligence was entered into the regression equation as a second independent variable. The R^2 value increased to 0.173, thus 17.3% of the variance in total aggression could be explained by the *combined effects* of traumatic event exposure and emotional intelligence. This percentage is also significant at the 99% level of statistical significance (Table 4.8). Both traumatic event exposure and emotional intelligence made significant unique contributions to the variance in total aggression (Table 4.8; $p < 0.01$). From the Beta values could be deducted that traumatic event exposure made the largest contribution to the variance in total aggression, and can thus be seen as a better predictor of aggression than emotional intelligence. This result was expected, since traumatic event exposure was correlated higher with total aggression. Although 17.3% of the variance in total aggression could be explained by the two independent variables, this percentage was still relatively small. The residual variance in this case was equal to 0.827 ($1 – R^2$), thus 82.7% of the variance in total aggression was still left unexplained. In light of

the behavioural genetics literature, this result was expected. Behavioural traits are influenced by a wide variety of environmental factors in combination with genetic factors (Plomin, 1990). Thus, many more independent variables would need to be entered into the regression equation to account for all the variance observed in aggressive behaviour.

With this said, according to Tuvblad *et al.* (2009) genetic influences account for up to 32% of the variance in aggressive behaviours. Thus, 68% in the variance is due to environmental factors. Taking this into account, emotional intelligence and aggression account for up to 25% of the variance in total aggression that is due to environmental influences. This is quite an impressive percentage.

Conclusion

In conclusion, this study once again proved that environmental factors play an important role in the aetiology of behavioural traits. This is in line with the general assumption in behavioural genetics that *both* environmental and genetic factors are important in determining behaviour.

Especially traumatic event exposure was shown to significantly influence aggressive behaviour. The results do however also show that one or two environmental factors alone cannot account for all the variance observed for a behavioural trait. Looking just at variance due to environmental influence, much room is still left open by this study for the examination of other possible specific environmental influences on aggression. Future research can build on this study by adding many more plausible environmental variables to the regression equations.

CHAPTER 5

The influence of social desirability bias on aggression scores

Manuscript submitted to *Philos. Trans. Genet.* as part of “An evaluation of questionnaires for testing emotional intelligence, traumatic event exposure and aggression and the effects of social desirability bias on aggression scores” by Laubscher, N., Odendaal, Z., Schneider, S. & Spies, J.J.

Abstract

This chapter sought to explain why social desirability bias (SDB) was not included as a control variable in Chapter 4. Firstly, the authors tested whether the conditions necessary for controlling for SDB were met. The first condition was that there must be significant differences between the scores of the "high SDB scorers" and "low SDB scorers" on the self-report measure under question. The second condition was that the researcher must be able to verify that the "high SDB scorers" were dishonest in answering the self-report measure under question. Secondly, the authors tested whether controlling for SDB would make a difference in the correlations between the three types of aggression, emotional intelligence and traumatic event exposure. Results indicated that significant differences did exist between "high SDB scorers" and "low SDB scorers" for all three types of aggression scores (reactive-, proactive-, and total aggression; $p < 0.01$). Due to time constraints an objective evaluation of the truthfulness of the responses of "high SDB scorers" could not be included in the study. Thus, only the first criterion for controlling for SDB was met. To explore the possible impact of SDB on the correlation coefficients obtained in chapter 4, the correlation coefficients before and after controlling for SDB were compared. SDB made no statistically significant differences for either the correlations between emotional intelligence and aggression ($Z < \pm 2.576$), or the correlations between trauma and aggression ($Z < \pm 2.576$).

Keywords: Aggression, Emotional intelligence, Response bias, Social desirability, Trauma

Introduction

In psychological research the methods of behavioural observation and physiological measurements have become so expensive that researchers had to start looking at alternative ways to obtain answers to their questions (Saunders, 1991). One research method that has become popular is the use of self-report measures (Saunders, 1991; Graybill & Blackwood, 1996), which is also the method employed in Chapter 4.

The use of self-report measures are, however, not infallible. These measures are unfortunately amenable to contamination by response bias (Saunders, 1991; Fisher, 1993). The main form of response bias is known as social-desirability bias (SDB) (Paulhus, 1991; Fisher & Katz, 2000). This form of response bias occurs when individuals give answers to questions that they think will be socially approved, rather than being honest in their responses (Saunders, 1991). The SDB construct can be seen as comprising of two factors, namely *self-deception* and *impression management*. A person who engages in self-deception believes that answers they are giving are true of themselves, while in fact it is actually not true. A person engaging in impression-management in contrast, purposely lies when responding to questions in order to avoid negative evaluation (Paulhus, 1984; Mills & Kroner, 2005).

The negative effects of SDB are most salient when socially unacceptable behaviours are being measured (Saunders, 1991). Thus, importantly, measures of emotional intelligence will not be greatly influenced by SDB. Measures of aggression on the other hand are susceptible to influence by this type of response bias.

The presence and possible negative influence of SDB should not just be assumed (Paulhus, 2002; Mills & Kroner, 2005). Instead, before controlling for SDB, it should be proven that it has a significant negative influence on the self-report measure under question. This can be done by adding a separate measure of SDB to the study (Paulhus, 1991). Once SDB is measured separately, Mills and Kroner (2005) proposed that two criteria must be met before deciding to control for SDB. First, the scores of individuals with high scores of SDB must

differ on the self-report measure from those of their low scoring SDB counterparts. Only if this criterion holds true can the second criterion be considered. The second criterion states that the researcher must be certain that the respondents are not reflecting reality in their answers on the self-report measure under question. Taking the above into account, a measure of SDB was included for the sample described in Chapter 4. The aim of this chapter is to explain why SDB was not taken into account and adjusted for in Chapter 4.

Methods

Research design and sample

The above mentioned criteria were tested by using the same sample of 182 participants described in Chapter 4. The participants were given a measure of SDB along with the measures of emotional intelligence, aggression and traumatic event exposure. Only the aggression questionnaire and the measure of SDB were analysed in this study in order to determine the influence of SDB on responses to a socially undesirable construct (aggression). The two criteria necessary for controlling for SDB were then considered.

Measuring instruments

The Balanced Inventory of Desirable Responding (BIDR; Paulhus, 1991*).* The BIDR is a 40 item self-report measure designed to measure people's tendency to give socially desirable responses in self-report questionnaires (Mills & Kroner, 2005). When all the items in the scale are summed, a total score of SDB is obtained (Paulhus, 1991). In a review of a number of studies by Paulhus (1991), the internal consistency score for the sum of all 40 items was $\alpha = 0.83$. The samples cited by Paulhus (1991) included, among others, 433 college students. Good concurrent validity was shown for the total SDB scale when all 40 items were summed and compared with the Marlowe-Crowne scale and the Multidimensional Social Desirability Inventory (Paulhus, 1991). Items are responded to on a 7 point Likert-scale, ranging from 1 (Not true) to 7 (Very true). For the purposes of this study, the items in the BIDR were rephrased in order to better suit the sample. Care was taken, however, not to alter the meaning of the items. Since the sample under study included both Afrikaans and English

speaking participants, the questionnaire was also translated into Afrikaans by a translator, and then translated back into English by the researchers to ensure equivalent meanings.

The Reactive-Proactive Aggression Questionnaire (RPQ; Raine *et al*., 2006*).* This questionnaire was described fully in Chapter 4. As with the BIDR, items on the RPQ were also rephrased in order to enhance clarity. Care was once again taken to ensure that the meanings stayed the same. The RPQ was also translated into Afrikaans by a translator, and translated back into English by the researchers to ensure equivalent meanings.

Cronbach's alpha coefficients for questionnaires

Since the questionnaires were adapted and translated, it was deemed necessary to calculate Cronbach's alpha coefficients for the sample under study (Table 5.1).

Table 5.1 Cronbach's alpha coefficients for the different translations of the RPQ and BIDR questionnaires.

Questionnaire	Subscales and Totals	Afrikaans α	English α	Afrikaans and English α
RPQ	Reactive aggression	0.78	0.77	0.78
	Proactive aggression	0.74	0.60	0.68
	Total aggression	0.84	0.81	0.83
BIDR	Total	0.85	0.75	0.86

Alpha values for the two questionnaires ranged from 0.60 to 0.86 (Table 5.1). Nunnally and Bernstein (1994) proposed that alpha values of 0.60 or higher are acceptable for non-cognitive measures. The above values are thus seen as sufficient. The adapted and translated forms of the questionnaires can thus be seen as reliable measures of the constructs under question.

Results and Discussion

Consideration of criterion 1

In order to determine whether individuals scoring high on SDB differ from their low scoring counterparts on the self-report instrument, the average aggression scores for "high SDB scorers" were compared to the average aggression scores for "low SDB scorers". This was done by making use of the t-test for independent samples. "High SDB scorers" were defined as individuals scoring 20 points or higher on the total BIDR scale. "Low SDB scorers" were defined as individuals scoring lower than 20 points on the total BIDR scale (Table 5.2).

Table 5.2 T-test for independent samples for estimating the differences in mean aggression scores between "high" and "low" BIDR scorers.

Average	High BIDR scorers	Low BIDR scorers	t score	Sig.
Reactive aggression	5.03	7.62	-3.994*	0.000
Proactive aggression	0.56	1.56	-2.820*	0.005
Total aggression	5.59	9.18	-4.006*	0.000

* $p \leq 0.01$

It is clear that statistically significant differences (99% level) occurred between the average aggression scores (reactive-, proactive-, and total aggression) for high and low scorers on the SDB scale (Table 5.2). Individuals scoring high on SDB tend to have lower aggression scores than individuals scoring low on SDB. Thus, the first criterion necessary for controlling for social desirability was met.

Consideration of criterion 2

Once the first criterion is met, Mills and Kroner (2005) proposed that a second criterion must also be met before SDB is controlled for. The researcher has to be sure that individuals with high scores on SDB did, in fact, make use of deception when responding to questions on the self-report measure under question. The authors propose that this can be done by making use of an objective criterion. In effect, what has to be proven is that an individual's actual behaviour differs from what they stated in the self-report measure. A possible way to do

this is to ask individuals who are close to each respondent to verify the respondents' answers. Since this would be very time consuming, such a process was not employed in the studies to follow. Thus, the second criterion could not be tested.

Correlation coefficients after controlling for SDB

Although both criteria could not be met, the statistically significant differences in aggression scores for the "high" and "low" SDB scorers prompted the authors to investigate whether controlling for SDB would result in significant differences in the correlations examined in Chapter 4. Consequently, "high SDB scorers" (as defined before) were deleted from the dataset, and new Pearson product moment correlation coefficients were calculated for the remaining respondents. These correlations were then compared with the correlations obtained in Chapter 4 for the full dataset (Tables 5.3 & 5.4).

Table 5.3 Differences in correlations coefficients between emotional intelligence and aggression for SDB controlled and for SDB not controlled

Aggression	**Emotional Intelligence**		
	Group including "high SDB scorers"	**Group excluding "high SDB scorers"**	**Z+**
	r	***r***	
Reactive aggression	-0.250	-0.157	-0.876
Proactive aggression	-0.235	-0.193	-0.397
Total aggression	-0.272	-0.190	-0.782

+ $p \leq 0.01$ (Z required for significance for two-sided test: ±2.576)

The results show that controlling for SDB lowers the correlation between emotional intelligence and all three types of aggression (reactive, proactive and total) (Table 5.3). In order to test the significance of these differences in the correlation coefficients, Fisher's r- to z- transformation was carried out by calculating $Z_{z1\text{-}z2}$ – values (Table 5.3).

From the Fisher's r- to z-transformation no significant differences between the correlations for the total group and the correlations for the SDB controlled group could be detected (99% level of statistical significance). These results were applicable to all three types of aggression (reactive, proactive and total).

Table 5.4 Differences in correlations coefficients between traumatic event exposure and aggression for SDB controlled and for SDB not controlled

Aggression	Traumatic event exposure		
	Group including "high SDB scorers"	Group excluding "high SDB scorers"	Z+
	r	*r*	
Reactive aggression	0.340	0.316	0.243
Proactive aggression	0.255	0.199	0.533
Total aggression	0.344	0.306	0.383

+ $p \leq 0.01$ (Z required for significance for two-sided test: ±2.576)

The correlations between trauma and aggression, after controlling for SDB, were slightly lower than that for the total group. Once again, this trend was evident for reactive-, proactive- and total aggression. In order to test whether these differences were statistically significant, Fisher's r-to z- transformation was once again utilized (Table 5.4).

Once again no statistically significant differences could be detected between the correlations of traumatic event exposure and aggression (reactive, proactive, and total) of the total group and that of the SDB controlled group (99% level of significance).

Conclusion

The aim of this chapter was to explain why SDB was not controlled for in Chapter 4. It is clear from the above discussion that only one of the necessary criteria for controlling for SDB could be met. There was a slight significant difference between the aggression scores of "SDB high scorers" and "SDB low scorers". The second criterion, namely verifying the truthfulness of the responses of the "SDB high scorers" to the aggression questionnaires, could not

be tested. It was deemed too time consuming to verify each respondent's answers. This alone provided enough reason for why SDB was not controlled for in Chapter 4. However, since the first criterion was met, the authors decided to test whether controlling for SDB would have made a significant difference in the correlations observed in Chapter 4. Considering first the correlation between emotional intelligence and aggression, no significant differences were found for the correlations of the total group and the correlations of the group in which SDB was controlled for. The same result held true for the correlations between traumatic event exposure and aggression. Thus the decision was taken not to control for SDB in Chapter 4.

CHAPTER 6

A family study of reactive and proactive aggression in a South African population

Manuscript submitted to *Afr. J. Psychiatr.* as “A family study of reactive and pro-active aggression in a South African population” by Laubscher, N., Odendaal, Z., Schneider, S. & Spies, J.J.

Abstract

The objective of this study was to determine upper-limit heritability estimates, as well as the influence of the non-shared environment on reactive- and proactive aggression in a sample of South African families. In addition the possible influence of assortative mating was determined. Convenience and snowball sampling methods were used to obtain participants. The final sample consisted of 88 participants from 23 nuclear families. From the 88 participants, 79 parent-offspring pairs were obtained. Participants completed an aggression questionnaire. Upper-limit heritability estimates were determined by means of two different statistics. Firstly, upper-limit heritability was determined by calculating single parent-offspring correlation coefficients, and then doubling these coefficients. Secondly, upper-limit heritability was determined by calculating midparent-offspring regression coefficients. Latter named statistic controls for assortative mating, and by comparing the results of the two types of statistics, the possible role of assortative mating was determined. The findings showed that 41.2% to 42% of the variance in reactive aggression in this sample can be attributed to the joined effects of additive genetic and non-shared environmental influences (upper-limit heritability). For proactive aggression, the percentages dropped to between 6% and 12.9%. Conversely, the influence of the non-shared environment accounted for 58% to 58.8% of the variance in reactive aggression, and for 87.1% to 94% of the variance in proactive aggression. Assortative mating played no role in this sample. In conclusion, the influence of genetic and shared environmental factors on reactive aggression seemed much more profound than on proactive aggression. The non-shared environment is important in influencing aggressive behaviour.

Keywords: Non-shared environment, Proactive aggression, Reactive aggression, Shared environment, Upper-limit heritability

Introduction

Aggression in humans is not a new phenomenon. In fact, archaeological and historical evidence prove that violence, and therefore aggression, already occurred some 25,000 years ago, when it was prevalent among our hunter/gatherer predecessors (Anderson & Carnagey, 2004). Researchers in various different professional fields agree that aggression is a major public health concern around the world (Lesch & Merschdorf, 2000; Weinshenker & Siegel, 2002; Miczek *et al.*, 2002; Buckholtz & Meyer-Lindenberg, 2008), with each antisocial individual costing society up to ten times more that non-aggressive individuals in healthcare and social services costs (Scott *et al.*, 2001; Buckholtz & Meyer-Lindenberg, 2008).

Curbing this universal problem is clearly important. Before a problem can be sufficiently solved, however, it is important to truly understand the nature of the problem. Truly understanding the construct of aggression requires adequately defining the construct. Geen (2001) provided the following comprehensive definition of aggression: "Aggression is the delivery of an aversive stimulus from one person to another, with intent to harm and with an expectation of causing such harm, when the other person is motivated to escape or avoid the stimulus". Various authors have provided definitions differing from the above (Weinshenker & Siegel, 2002; Archer & Coyne, 2005) but the main feature of all these definitions tends to coincide with the main feature of the definition by Geen (2001), namely that aggression is any behaviour intending to hurt or harm others.

Since aggression is a multidimensional construct, only providing a broad definition is not sufficient for truly comprehending the construct. Definitions of the most frequently defined subtypes of aggression are also required. Many different subtypes of aggression have been defined by researchers, including verbal aggression, physical aggression, direct- and indirect aggression, as well as intermale-, fear-induced- and irritable aggression (Archer & Coyne, 2005; Ligthart *et al.*, 2005; Popova, 2006). The most frequently studied subtypes of aggression are the constructs of reactive aggression versus proactive aggression (Dodge & Coie, 1987; Geen, 2001; Brengen *et al.*, 2006; Baker *et al.*, 2008).

Reactive aggression is defined by Geen (2001) as "aggressive behaviours that are enacted in response to provocation, such as an attack or an insult, and are manifested in both self-defence and angry actions". In contrast, proactive aggression can be seen as a more instrumental form of aggression, not requiring provocation or anger. Rather, it is motivated by such goals as asserting power, obtaining goods and assuring approval of reference groups (Geen, 2001; Brendgen *et al.*, 2006; Tuvblad *et al.*, 2009). Proactive aggression can be seen as goal-oriented, whereas reactive aggression can be seen as retaliatory (Hubbard *et al.*, 2010).

Properly defining the construct of aggression is only the first step in trying to understand the construct. The next important step is trying to understand the aetiological influences on the defined constructs. The age-old nature-nurture debate is of relevance here. Regarding the heritability component of this debate, clear evidence exist that both reactive aggression and proactive aggression are subjected to genetic influence. The most convincing evidence regarding the influence of a heritable component in reactive aggression is that this is the most common form of aggression found in animals, serving as a self-defence mechanism in both primates and non-primates (Meloy, 1988; Baker *et al.*, 2008). The neural circuitry underlying reactive aggression in animals is well-known, and since brain circuitry is highly heritable, it is easy to come to the conclusion that heritable factors form an important part of the aetiology of reactive aggression (Baker *et al.*, 2008).

The evidence for a genetic component to proactive aggression comes mainly from studies reporting a link between proactive aggression and psychopathic personality traits (Kempes *et al.*, 2005; Blair *et al.*, 2006; Brendgen *et al.*, 2006). Blair *et al.* (2006) noted that a substantial component of psychopathy is the tendency to make excessive use of proactive aggressive behaviours. People with psychopathic tendencies show a consistent array of symptoms, including frequently engaging in irresponsible and impulsive behaviour, being arrogant, callous and unemotional, and lacking in empathy and guilt (Hare, 1999; Marsee *et al.*, 2005; Blair *et al.*, 2006). With proactive aggression as a core component, these psychopathic personality traits have been found to be highly heritable

(Viding *et al.*, 2005; Tuvblad *et al.*, 2009). Thus it follows naturally that proactive aggressive behaviours should at least be partly heritable.

It must be noted that despite the evidence for the influence of genetic factors, environmental factors also play an important role in all behavioural traits, including aggression. Environmental influences have been found to be at least as important as genetic influences in the aetiology of behavioural dimensions, with heritability estimates seldom exceeding 0.50 (Plomin *et al.*, 1994, Plomin *et al*., 2008). Consequently, at least 50% of the variance in most behavioural traits can be explained by the influence of the environment.

Family studies

Before discussing family studies, it is important to explain what is meant by the term "heritability". Heritability is broadly defined as "the proportion of phenotypic variance that can be accounted for by genetic differences among individuals" (Plomin *et al*., 2008). In addition to this broad definition, two types of heritability estimates are described. Broad sense heritability is defined by Anholt and Mackay (2010) as "the extent to which variation in phenotypes in a population is due to variation in genotypic values, including non-additive genetic variation". Narrow-sense heritability is defined by the same authors as "the portion of additive genetic variance that contributes to the total phenotypic variation and represents the extent to which variation in phenotypes is determined by variation in effects of alleles transmitted by parents to offspring". Additive genetic variance is variance in phenotypes caused by independent effects of genes or loci. In contrast, non-additive genetic variance refers to variance caused by the interaction of genes or loci (dominance or epistasis) (Plomin *et al*., 2008). Therefore, narrow-sense heritability refers only to the influence of additive genetic variance, whereas broad-sense heritability refers to the influences of both additive and non-additive genetic variance. A final important note on heritability is that the term refers to the *population* and not to an individual (Plomin, 1990). Therefore, if the heritability for a trait is quoted as 0.30, it means that 30% of the variance in the trait in the population can be attributed to the genetic variance in the population.

One of the methods available for assessing the influence of heritable factors on aggression is family studies (Plomin, 1990; Baird & Bergeman, 2011). The family study design is not as powerful as the other two designs commonly employed in behaviour genetic research, namely twin- and adoption designs. The reason for this is that family members, and in particular first-degree relatives, are very likely to share an environment. Thus, correlations between first-degree relatives are not only indicative of the effects of shared genetic factors, but also of shared environmental factors (Plomin, 1990; Vernon *et al.*, 2008a).

Despite these limitations, studies of first degree relatives can still be useful. First-degree relatives (siblings, parents and offspring) share 50% of additive genetic influences, in addition to a shared environment. Thus, doubling correlations between first degree relatives should provide an estimate of the upper limits of heritability (narrow-sense). Upper-limit heritability refers to a statistic that takes into account both shared genes and shared environmental factors (Plomin, 1990). Upper-limit heritability estimates can also be calculated by determining the slope of the regression line for offspring and midparent scores. This value is also known as the regression coefficient (Hamilton, 2009).

Since upper-limit heritability estimates account for the effects of both shared genes and shared environmental factors, heritability estimates are unlikely to be higher. It can, however, be considerably lower (Plomin, 1990; Vernon *et al.*, 2008a; Baird & Bergeman, 2011). Thus, family studies are useful for determining upper-limit heritability estimates.

In addition, family studies are also useful for determining the influence of the non-shared environment. Brendgen *et al.* (2006) and Baker *et al.* (2008) made use of the ACE model to explain the variable influences of genetic and environmental factors on aggression. According to this model, Var(P) = Var(A) + Var(C) + Var(E), where Var(P) represents the total phenotypic variance, Var(A) represents the influence on the phenotype due to the variance in additive genetic factors, and Var(C) and Var(E) represent the influence due to the variance in shared- and nonshared environmental factors respectively (Plomin *et al.*, 2008). Thus, if upper-limit heritability represents the variance explained by the combination of additive genetic factors (A) and the shared environment (C), the influ-

ence of the non-shared environment (E) can be calculated from the upper-limit heritability estimate.

Assortative mating

Anholt and Mackay (2010) provided the following definition for assortative mating: assortative mating is "mating between individuals who share more traits in common than would be expected from random mating in the population". This form of assortative mating is known as positive assortative mating (Plomin, 1990). Vandenberg already noted in 1972 that people tend to marry individuals who are similar to them in age, socioeconomic status and religious background (Vandenberg, 1972). Regarding aggressive behaviour, Krueger *et al.* (1998) found that positive assortative mating was present between couples. Notably, correlations between couples for specific antisocial actions were as high as 0.54.

Assortative mating is important in behavioural genetic research for a number of reasons. The most important of these are firstly that assortative mating affects the quality of the marital relationship, which in turn affects the *environmental* conditions in which the children are raised. Secondly, assortative mating tends to inflate heritability estimates, since it increases genetic resemblance between first-degree relatives (Vandenberg, 1972; Plomin, 1990). Importantly, when upper-limit heritabilities are estimated by doubling the single parent-offspring correlation, a high correlation between spouses will inflate the heritability estimates (Vernon *et al.*, 2008a). It is therefore important to determine whether assortative mating is present before interpreting upper-limit heritability estimates for a trait.

In light of the above discussion, this study aimed firstly to determine the upper-limit heritability estimates for reactive- and proactive aggression scores in a South African sample. Secondly, these upper-limit heritability estimates were calculated by two different types of statistics, in order to determine whether assortative mating played a role. Finally, this study also aimed to determine the influence of non-shared environmental factors on reactive- and proactive aggression scores.

Methods

Research design and sample

A nuclear family-study design was used in this study in order to obtain upper-limit heritability estimates for reactive- and proactive aggression. Convenience and snowball sampling methods were used to obtain participants. Young adults from the central region of South Africa were asked to complete a self-report questionnaire measuring reactive and proactive aggression respectively (see measuring instruments). Each young adult was given more of the above questionnaires for their first-degree relatives (parents and siblings) to complete.

The final sample consisted of a total of 88 participants from 23 nuclear families. Of the family members, 39 were male, while 49 were female. From these families 79 parent-offspring pairs were obtained.

Measuring instruments

Participants were asked to complete the Reactive-Proactive Aggression Questionnaire (RPQ; Raine et al., 2006), fully described in Chapter 4. In addition, participants were asked to complete a self-compiled biographical questionnaire.

Statistical analysis

Statistical analysis was carried out by making use of the MS Excel software program, as well as the OpenStat4 (2011) program for Windows available freely from (http://www.statpages.org/Miller/openstat). Two types of statistical analysis were performed to estimate upper-limit heritability for reactive- and proactive aggression. Firstly, single parent-offspring correlations were determined by making use of Pearson's product moment correlation coefficient. As already mentioned, upper-limit heritability values for a trait can be obtained by doubling the correlations between first-degree relatives (parents and offspring).

Secondly, upper-limit heritability was also calculated by determining the regression coefficients for midparent-offspring scores. The regression coefficients can be calculated by dividing the covariance between midparent and offspring values with the variance of midparent values (Hamilton, 2009), and is indicated

by the symbol *b*:

$$b = \frac{\text{Cov (midparent;offspring)}}{\text{Var (midparent)}}.$$

The midparent-offspring regression coefficient controls for assortative mating, since the mean score of the two parents does not carry any information about the father-mother correlations (Vernon *et al.*, 2008a). Consequently, if lower heritability values are obtained from the regression statistic than from the correlation statistic, assortative mating is present.

In addition the influence of the nonshared environment (E) was estimated by: Var(E) = Var(P) – upper-limit heritability estimate. Since Var(P) represents the total phenotypic variance, it can be replaced by 1. Thus, Var(E) = 1 – upper-limit heritability estimate.

Results

It was clear from the parent-offspring correlation results that very low positive correlations exist between aggression scores for parents and offspring. This result held true for both reactive- and proactive aggression scores (Table 6.1).

Table 6.1 Correlation coefficients between parents and offspring for reactive- and proactive aggression scores.

Aggression	Parent-offspring correlation *r*	Standard Deviation
Reactive	0.206**	3.300
Proactive	0.030	1.749

* $p \leq 0.01$
** $p \leq 0.05$

The only statistically significant correlation (95% level) occurred between parent and offspring reactive aggression scores (r = 0.206).

In order to obtain upper-limit heritability estimates, the parent-offspring correlations were doubled, as suggested by Plomin (1990) (Table 6.2). Upper-limit heritability estimates were 0.060 and 0.412 for proactive and reactive aggression respectively (Table 6.2).

Table 6.2 Upper-limit heritability estimates for reactive- and proactive aggression (obtained by doubling parent-offspring correlations).

Aggression	Upper-limit heritability estimates
Reactive	0.412
Proactive	0.060

For reactive aggression, 41.2% of the variance in the population could be attributed to additive genetic variance in combination with environmental factors shared by family members. For proactive aggression this percentage dropped to 6%. Thus, only 6% of the population variance in proactive aggression could be attributed to additive genetic and shared environmental factors combined.

Upper-limit heritability estimates were also determined by calculating midparent-offspring regression coefficients. These estimates were slightly higher than those obtained by doubling midparent-offspring correlation coefficients (Table 6.3).

Table 6.3 Upper-limit heritability estimates for reactive- and proactive aggression (obtained by calculating midparent-offspring regression coefficients).

	Midparent-offspring regression coefficient	Standard Deviation
Reactive aggression	0.420	3.12
Proactive aggression	0.129	1.71

Once again proactive aggression showed the lowest upper-limit heritability, with 12.9% of the variance being ascribed to additive genetic and shared environmental influences. This is in contrast to an upper-limit heritability estimate of 0.42 (42%) for reactive aggression.

Finally, the influence of the non-shared environment on reactive- and proactive aggression scores were determined. Non-shared environmental influences explained 58% to 58.8% of the variance in reactive aggression, and 87.1% to 94% of the variance in proactive aggression (Table 6.4).

Table 6.4 Non-shared environmental influences on reactive- and proactive aggression scores, calculated from upper-limit heritability estimates determined by parent-offspring correlations and midparent-offspring regression coefficients.

	Non-shared environmental influence (1 – upper-limit heritability)	
	Parent-offspring	Midparent-offspring
Reactive aggression	0.588	0.580
Proactive aggression	0.940	0.871

Discussion

This study aimed to determine upper-limit heritability estimates, as well as the role of non-shared environmental influences, for reactive- and proactive aggression scores. In addition, the possible presence of assortative mating was also investigated.

The upper-limit heritability estimates obtained from the midparent-offspring regression coefficients were lower for both types of aggression than the estimates obtained from doubling the single parent-offspring correlations. Assortative mating therefore did not play a role in this sample.

From the results, it is clear that a substantial amount of the population variance in reactive aggression can be attributed to the combined influence of additive genetic factors and shared environmental factors. Upper-limit heritability estimates ranged from 0.412 (obtained by doubling parent-offspring correlation coefficients) to 0.420 (obtained from calculating midparent-offspring regression coefficients). These estimates are in line with results from studies performed by Brendgen *et al.* (2006) and Baker *et al.* (2008). Brendgen *et al.* (2006) found that additive genetic variance accounted for 39% of the variance in reactive aggression scores. This study was conducted on 172 six-year-old twin pairs from the greater Montreal area. In the latter study the influence of the shared environment was found to be negligible. Baker *et al.* (2008) found that additive genetic influences in combination with shared environmental influences accounted

for 36% to 63% of the variance in reactive aggression, across different informants and for different genders. This study was conducted on 605 families of nine-to-ten-year-old twins and triplets in Los Angeles.

Concerning proactive aggression, the results from this study contrast sharply with results from the Brendgen *et al.* (2006) study. They reported that 41% of the variance in proactive aggression can be accounted for by additive genetic influences, with the influence of the shared environment once again being negligible. This estimate is considerably higher than the upper-limit heritability estimates of 6% and 12.9% obtained in the current study. In the Baker *et al.* (2008) study, additive genetic and shared environmental influences combined accounted for 14% to 59% of the variance in proactive aggression; with 14% being for girls' self-report of proactive aggression. This estimate is much closer to what was observed in the current study. One shortcoming in the current study was the small range of proactive aggression scores in the population. Only ten points separated the highest and lowest proactive aggression scores (see Chapter 4). Since heritability depends on phenotypic *variation* in a population, heritability for any trait with low phenotypic variance will be very low (Anholt & Mackay, 2010). This does not mean that genetic influences play no role in the trait. To explain this, Anholt and Mackay (2010) made use of the analogy of eye colour in Siamese cats. Eye colour is determined solely by genetic influences, but since nearly all Siamese cats have blue eyes, and no phenotypic variation can be observed, heritability estimates are nearly zero. Consequently the low phenotypic variation for proactive aggression was a crucial shortcoming in this study.

It should still be noted that heritability estimates are only specific to a particular population at a particular time. Different populations at different time points may have differing values for heritability (Plomin *et al.*, 2008). The reason for this is that heritability estimates will change if gene frequencies change, and also if environmental influences change (Anholt & Mackay, 2010). Thus it is not impossible for heritability values to vary across different samples, as has happened for proactive aggression in this instance. The finding in the current study that genetic factors influence reactive aggression to a greater extent than proac-

tive aggression is also plausible, since reactive aggression is the form of aggression most frequently observed in animals (Meloy, 1988; Baker *et al.*, 2008). It can therefore be viewed as a more "natural" form of aggression which is not as much shaped by environmental factors. It is also plausible that proactive aggression is more influenced by environmental factors than genetic factors. Bandura (as cited in Baker *et al.*, 2008) argued that proactive aggression is a response learned in overly lenient family environments, and is then maintained by positive consequences of the behaviours. Proactive aggression is thus viewed here as more a product of the environment than as the result of genetic factors. Finally, it should be noted that the Brendgen et al. (2006) and Baker et al. (2008) studies made use of much larger sample sizes. Anholt and Mackay (2010) made it clear that greater accuracy is achieved with larger samples.

Concerning the influence of the non-shared environment, for reactive aggression the estimates of 58% and 58.8% observed in this study are very similar to that found by Brendgen *et al.* (2006) (61%) and Baker *et al.* (2008) (37% to 64%). Regarding proactive aggression, Brendgen *et al.* (2006) reported that the non-shared environment accounted for 59% of the total phenotypic variance. This is considerably lower than our estimates, which ranges from 87.1% to 94%. In the Baker *et al.* (2008) study, non-shared environmental influences ranged from 41% to 50% for teacher report (both sexes), mother report (both sexes) and boys' self-report. Once again this is considerably lower than our estimates. Interestingly, for girls' self-report, latter authors found that the non-shared environment explained 86% of the variance in proactive aggression. This is very close to our estimates of 87.1% and 94%. Since the influence of the non-shared environment is calculated simply by subtracting the upper-limit heritability estimates from the total phenotypic variance (therefore from 1), the same possible explanations given for the differences in upper-limit heritability estimates can be applied here.

Conclusion

In conclusion this study again underlined that both genetic and environmental factors play a role in reactive- and proactive aggressive behaviour. Importantly, the results suggested that the aetiological factors influencing the two types of

aggression differ in the present sample. In reactive aggression, the combination of additive genetic and shared environmental influences (upper-limit heritability) seemed to influence the phenotype more or less to the same degree as non-shared environmental influences. The non-shared environment still played the bigger role, but the differences were slight.

In contrast, variation in proactive aggression was almost entirely due to the non-shared environment, with upper-limit heritability estimates not even surpassing 13%. Since the environment is more easily amenable than genetic factors, this result would mean that proactive aggressive behaviours can be reduced significantly or even prevented by adjusting the environment. Importantly, latter result could also be due to the low phenotypic variance observed for proactive aggression in this sample. It is thus imperative that more studies of this kind be carried out in the South African population. The samples for future studies should however be much larger, and include individuals with a much wider range of proactive aggression scores. If the results from this study are replicated in such a population, this could mean that reactive aggression should be the focus of molecular genetic studies, instead of proactive aggression.

CHAPTER 7

The effect of the *MAO-A* gene on aggression in a South African population

Manuscript submitted to *Aggressive Behavior* as "The effect of the *MAO-A* gene on aggression in a South African population" by Laubscher, N., Odendaal, Z., Schneider, S. & Spies, J.J.

Abstract

The aims of this study was to: 1) Determine whether the *MAO-A*-uVNTR 30 bp repeat polymorphism is present in a South African population; 2) Whether the polymorphism had an effect on reactive-, proactive- or total aggression, and to 3) Determine whether any gene-environment interaction effects could be detected between the *MAO-A*-uVNTR and traumatic event exposure in their influence on reactive-, proactive- and total aggression. In total, 20 young adults from the central regions of South Africa were genotyped for the 30 bp functional VNTR in the promoter region of the *MAO-A* gene. In addition, each individual was given a questionnaire measuring reactive-, proactive- and total aggression, along with a trauma questionnaire to complete. Genotyping results indicated 3.5, 4.5 and 5.5 repeat sequences, in contrast to the 3, 4 and 5 repeat sequences reported by most authors. We concluded that these are in actual fact equivalent repeat sequences, since the published Genbank sequence (Genbank accession number M89636) was wrongly interpreted as having 4 repeats, instead of the actual 4.5 repeats. Results showed no main effect of the *MAO-A*-uVNTR on reactive-, proactive-, or total aggression. In addition, no statistically significant interaction effects were detected between the *MAO-A*-uVNTR and traumatic event exposure. A non-significant effect where the 3.5 repeat allele and high trauma exposure combined led to enhanced reactive aggression was however detected. We conclude that the *MAO-A*-uVNTR by itself does not significantly influence aggression, but that a gene-environment interaction effect is possible. Our sample size was too small to significantly prove the latter.

Keywords: MAOA-uVNTR, proactive aggression, reactive aggression, South African population, trauma

Introduction

Findings from Chapter 6 clearly indicated that aggression, and in particular reactive aggression, is at least partly influenced by genetic factors. The next logical step to take is to try to find specific genes influencing aggressive behaviours, and particularly reactive aggressive behaviours. For quite some time this search for genes seemed futile since researchers failed to find any genes or sets of genes influencing aggression (Buckholtz & Myer-Lindenberg, 2008). This changed when a landmark study performed by Brunner *et al.* (1993) found greatly increased impulsive aggression in the males of a large Dutch family. The researchers subsequently found a complete deficiency in the activity of an enzyme known as monoamine oxidase A (MAO-A) in all affected family members. The deficiency was the result of a point mutation in the gene encoding MAO-A (*MAO-A*-gene*)*, producing a functional knockout of this gene. Since this finding, the *MAO-A* gene has become the subject of numerous research projects into the aetiology of aggressive behaviour (eg. Cases *et al.*, 1995; Caspi *et al.*, 2002; Kinnally *et al.*, 2009), and is seen as the most compelling candidate gene for aggression (Buckholtz & Meyer-Lindenberg, 2008).

MAO-A is a catabolic enzyme responsible for the degradation of biogenic amines in the brain and peripheral tissues. Among the substrates of MAO-A are the neurotransmitters serotonin, norepinephrine and dopamine. MAO-A terminates the availability and transmission of these neurotransmitters, serving as a critical regulator of their functioning in the brain (Shih & Thompson, 1999; Buckholtz & Meyer-Lindenberg, 2008). The influence of MAO-A on neurotransmitter functioning makes this enzyme and the gene coding for it attractive candidates when studying behaviour, since brain neurotransmitters can be seen as the behavioural regulators in the brain (Shih & Thompson, 1999; Popova, 2006). Underlining the importance of neurotransmitters, Popova (2006) stated that genes influencing behaviour probably act through brain neurotransmitter systems.

The MAO-A enzyme is located on the outer membrane of mitochondria in the presynaptic terminals of monoamine projection neurons (Westlund *et al.*, 1993; Alia-Klein *et al.*, 2008). This enzyme has also been found in astrocytes (Westlund *et al.*, 1988; Buckholtz & Meyer-Lindenberg, 2008). This localization

of MAO-A enables it to degrade above mentioned neurotransmitters presynaptically (by the mitochondrially bound MAO-A) or extrasynaptically (by astrocyte bound MAO-A) (Buckholtz & Meyer-Lindenberg, 2008). The gene responsible for the production of the MAO-A enzyme (*MAO-A* gene) has been mapped to the short arm of the X chromosome, at position Xp11.23 (Lan *et al.*, 1989; Shih *et al.*, 1999), and comprises of 15 exons (Shih *et al.*, 1999; Buckholtz & Meyer-Lindenberg, 2008).

The findings regarding MAO-A and aggression by Brunner *et al.* (1993) were replicated by Cases *et al.* (1995) in an animal model. Latter authors found that mice with a functional knockout of the *MAO-A* gene, and a resultant deficiency in MAO-A, displayed a distinct behavioural syndrome, which included enhanced aggression in males. More recently strength was added to these findings by a study reporting an inverse relationship between MAO-A activity in cortical and subcortical areas and self-reported aggression in a sample of healthy men. Lower MAO-A activity resulted in higher self-reported aggression (Alia-Klein *et al.*, 2008).

Importantly, the functional knockout of the *MAO-A* gene examined by Brunner *et al.* (1993) is exceedingly rare and has not been found outside the Dutch family (Frazzetto *et al.*, 2007). Much more common is a variable number of tandem repeats (VNTR) polymorphism in the promoter region of the *MAO-A* gene, first described by Sabol *et al.* (1998). This polymorphism is located 1.2 kb upstream of the MAO-A coding sequences, and consists of a 30 base pair sequence repeated a variable number of times. This polymorphism is commonly referred to as the *MAO-A*-uVNTR (Sabol *et al.*, 1998; Deckert *et al.*, 1999; Kinnally *et al.*, 2009).

The repeat sequence, which is commonly present in 2, 3, 3.5, 4 or 5 copies, has been found to influence transcription and expression of the *MAO-A* gene (Sabol *et al.*, 1998; Deckert *et al.*, 1999, Guo *et al.*, 2008). Sabol *et al.* (1998) found that 3.5 or 4 copies of the repeat sequence resulted in two to ten times faster transcription of the gene, when compared with alleles carrying 3 or 5 copies of the repeat. Deckert *et al.* (1999) replicated these results, except for finding that the 5 repeat allele also resulted in higher transcriptional activity, along

with the 3.5 and 4 repeat alleles. Accordingly, Deckert *et al.* (1999) grouped the alleles into two groups, with the short allele group (*s*) containing alleles with 3 repeats, whereas the long allele group (*l*) contained the 3.5, 4 and 5 repeat alleles. Guo *et al.* (2008) reported on a rare 2 repeat variant, and found that this variant exhibited even lower promoter activity than the 3 repeat allele.

With the findings that lower MAO-A activity resulted in aggression, the finding of the functional *MAO-A*-uVNTR polymorphism led to multiple studies into a possible link between the low expression alleles and aggression (Koller *et al.*, 2003; Guo *et al.*, 2008; Tikkanen *et al.*, 2009; Reti *et al.*, 2011). The findings for a main effect of the *MAO-A*-uVNTR polymorphism on aggression have been contradictory. Some studies found a correlation between the shorter alleles of the polymorphism and aggression (Guo *et al.*, 2008; Reti *et al.*, 2011), whereas other studies found no association between the *MAO-A*-uVNTR and aggression (Koller *et al.*, 2003; Tikkanen *et al.*, 2009).

In contrast to the inconsistencies in the above findings, a number of studies have found that the *MAO-A*-uVNTR moderated the effects of childhood traumatic experiences on aggression. Specifically, individuals who were exposed to childhood traumatic experiences were more likely to exhibit aggressive behaviour as adults, but only if they carried the low expressing *MAO-A* genotypes. Individuals carrying the high expression genotypes who were exposed to childhood trauma did not display increased aggression as adults. Likewise, individuals who carried the low expression variants who were not exposed to childhood trauma also did not display an increased risk for aggressive behaviour as adults (Caspi *et al.*, 2002; Kim-Cohen *et al.*, 2006; Widom & Brzustowicz, 2006; Frazzetto *et al.*, 2007).

These findings might explain why only some studies found a main effect for the relationship between *MAO-A*-uVNTR genotype and aggression. In addition, the study in Chapter 4 of this dissertation indicated a relatively low positive correlation between traumatic event exposure and aggression. Thus, only some individuals exposed to trauma had correspondingly high aggression scores. As also noted by Caspi *et al.* (2002), these findings of a gene-environment interac-

tion might also explain why only some individuals exposed to trauma show enhanced aggressive tendencies.

The localization of the *MAO-A* gene on the X chromosome has made studies in males easier than in females. Since males carry only one X chromosome, they are straightforward to genotype for the *MAO-A* gene, and can easily be characterized as “high activity” or “low activity” depending on which single allele they carry. Females in contrast carry two X chromosomes, and in addition to being homozygotes for low and high activity alleles, they can also be heterozygotes and carry one low activity and one high activity allele (Caspi *et al.*, 2002). The findings concerning *MAO-A* X inactivation have been conflicting (Carrel & Willard, 2005; Kim-Cohen *et al*., 2006; Nordquist & Oreland, 2006; Stabellini *et al*., 2009). In addition, if X-inactivation occurs, it is not possible to determine which of the two alleles in heterozygote females is inactivated (Caspi et al., 2002). Thus characterizing heterozygote females as high expressing or low expressing is difficult at this stage. For these reasons, very few studies thus far have focused on the influence of the *MAO-A*-uVNTR on aggression in females.

Should X-inactivation occur, heterozygote females carrying one high and one low expressing allele would be expected to be mosaics for the high and low expressing *MAO-A*-uVNTR genotypes, and would most likely have intermediate enzymatic activity (Frazzetto *et al.*, 2007). Homozygous females should display enzymatic activity equivalent to hemizygous males carrying the same allele. Meyer-Lindenberg *et al.* (2006) provided proof of this by showing that female heterozygotes display patterns of neural activity intermediate to high and low expressing male hemizygotes. Female homozygotes displayed neural activity equivalent to male hemizygotes carrying the same allele. Accordingly, Frazzetto *et al.* (2007) divided females into three genotypic groups: homozygous subjects carrying two high activity alleles, homozygous subjects carrying two low activity alleles, and heterozygous subjects carrying one low and one high activity allele.

In light of the above discussion the main aims of this study were to: 1) Determine whether the different alleles of the *MAO-A*-uVNTR polymorphism occur in a South African population; 2) Whether the different alleles had effects on reac-

tive-, proactive-, or total aggression scores in males and females, and 3) Determine whether any gene-environment interaction effects could be detected between traumatic event exposure and the *MAO-A*-uVNTR in their influence on reactive-, proactive-, and total aggression scores in males and females.

MATERIALS AND METHODS

Research design and sample

The sample for this study consisted of a total of 20 young male and female adults, from the central region of South Africa, obtained by means of convenience sampling methods. Each participant was given an aggression questionnaire measuring reactive and proactive aggression, in addition to a trauma questionnaire (see measuring instruments). Participants also received a covering letter (Appendix A) explaining the aims of the study, as well as the methods used to obtain DNA samples. Participants were informed in the covering letter that participation in the study is completely voluntary, that they may withdraw from the study at any time and that all their personal information will be treated in the strictest confidence. Finally, the participants signed an informed consent form (Appendix A). Permission to conduct the study was obtained from the Medical Ethics Research Committee of the University of the Free State (ECUFS 152/2011).

DNA extraction and genotyping

DNA was obtained from participants by means of swirling 10 ml of Listerine mouthwash in their mouths for 20 seconds, at least one hour after eating or drinking. Samples were centrifuged (14 000 *g* for 5 min) in order to obtain a pellet of buccal cells. DNA was extracted from the buccal cells by following the 'AquaGenomic^{TM} DNA extraction kit' protocol. DNA was stored in 100 µl TE buffer at -21°C.

Primers were obtained from Whitehead Scientific: *MAO-A* forward primer (5'-ACAGCCTGACCGTGGAGAAG-3') and *MAO-A* reverse primer (5'-GAACGGACGCTCCATTCGGA-3') (Sabol *et al.*, 1998). Reactions were performed in a total volume of 20 µl containing 100 ng genomic DNA, 10 µl Dream-

taq Mastermix (Fermentas Life Sciences), 0.5 µl of each primer at a concentration of 10 µM and 7 µl nucleic acid free water (Fermentas Life Sciences). PCR was carried out using the following conditions: an initial 3 min denaturing step at 95ºC, followed by 35 cycles at 95ºC for 1 min, 64ºC for 1 min and 72ºC for 1 min, followed by a final extension phase at 72ºC for 5 min.

PCR products were separated electrophoretically on a 2% agarose gel, intercallated with GelRed and visualized under UV light. Homozygote samples were sequenced by means of an ABI 3130 Automated Capillary Sequencer. The pre-sequencing PCR was performed in separate forward and reverse total reaction volumes of 10 µl, containing 1 µl PCR product, 4.3 µl nucleic acid free water (Fermentas Life Sciences), 0.5 µl Ready Reaction Premix (Applied Biosystems Big Dye^{R} Sequencing Kit), 1 µl Big Dye^{R} 5 X Sequencing Buffer (Applied Biosystems Big Dye^{R} Sequencing Kit) and 3.2 µl of each primer at a concentration of 10 µM (Fermentas Life Sciences). Pre-sequencing PCR was carried out using the following conditions: an initial 3 min denaturing step at 94ºC, followed by 25 cycles at 94ºC for 10 s, 50ºC for 5 s and 60ºC for 4 min, followed by a final extension phase at 72ºC for 5 min. An EDTA/Ethanol precipitation protocol was followed for the sequencing clean-up. Sequencing reaction volume was adjusted to 20 µl and transferred to a 1.5 ml Eppendorf tube containing 5 µl 125 mM EDTA. Hereafter, 60 µl absolute ethanol was added, and the samples were vortexed for 5 sec, and precipitated at room temperature for 15 min. Centrifugation was carried out at 4ºC for 15 min at 20 000*g*, where after the supernatant was completely aspirated. After centrifugation 60 µl 70% ethanol was added to the tubes, followed again by centrifugation at 4ºC for 5 min at 20 000*g.* The supernatant was once again completely aspirated, and tubes were placed in a heating block for 5 min at 65ºC. Finally, samples were stored in the dark at 4ºC.

Sequencing results were edited by comparing forward and reverse sequences by making use of the Geneious software program (Drummond *et al*., 2011), Geneious V.5.4.; available from (http://www.geneious.com/). Hereafter results were aligned by making use of the MEGA software program (Tamura *et al*., 2011); available freely from (http://www.megasoftware.net/). The number of repeats of the *MAO-A*-uVNTR was determined by counting the number of times

the following 30 bp sequence was repeated for each sample: ACCGGCAC-CGGCACCAGTACCCGCACCAGT.

Heterozygote samples were genotyped by comparing their fragment sizes with those of sequenced homozygote samples on the agarose gel.

Measuring instruments

Assessment of reactive and proactive aggression

Reactive and proactive aggression was assessed by means of the Reactive-Proactive Aggression Questionnaire (RPQ; Raine *et al.*, 2006) (fully described in Chapter 4).

Assessment of traumatic life events

Traumatic life events were assessed by means of the Stressful Life Events Screening Questionnaire (SLESQ; Goodman *et al.*, 1998) (fully described in Chapter 4). The descriptive items in the SLESQ were left out, and participants were only required to indicate yes/no to whether they have experienced a particular traumatic event. The total number of traumatic events experienced by each participant was then tallied.

Statistical analysis

The OpenStat4 (2011) program for Windows available freely from (http://www.statpages.org/Miller/openstat), as well as the Microsoft Excell software program was used for statistical analysis. The precise analysis used for each step of the study is fully described under the Results and Discussion section.

Results and Discussion

Through sequencing, three different repeats of the 30 bp *MAO-A*-uVNTR were identified: 3.5, 4.5 and 5.5 repeats (see Appendix E). Thus, this genomic region is polymorphic in the South African population. As stated, heterozygotes were genotyped by comparing their fragment sizes with those of sequenced homozygotes or hemizygotes (Figure 7.1)

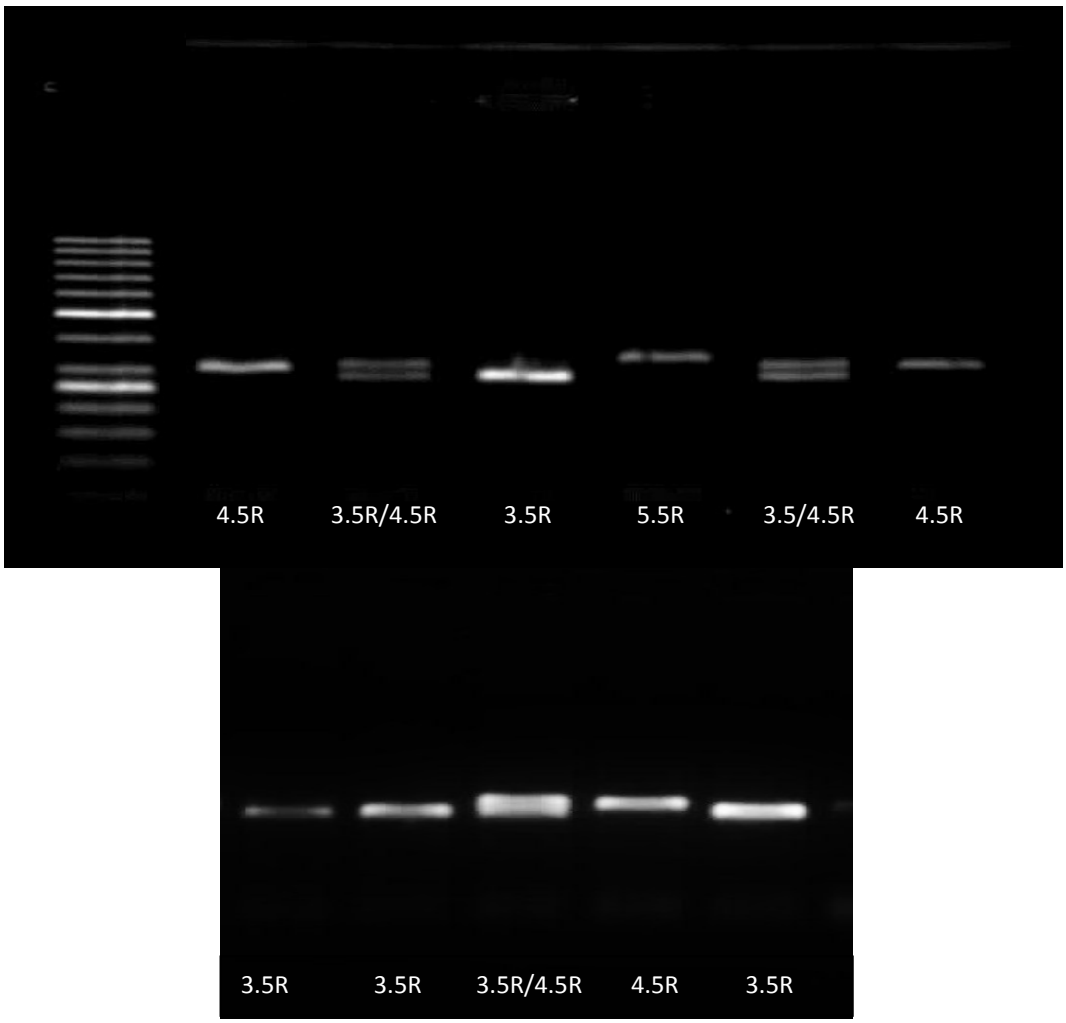

Figure 7.1 2% agarose gels with sequenced homozygote/hemizygote fragments for identification and genotyping of heterozygotes

Allelic frequencies

During this study the 4.5 repeat allele was observed most frequently (frequency = 0.58), followed by the 3.5 repeat allele (frequency = 0.39) (Table 7.1). We did not observe the 2.5 repeat or 4 repeat allele (2 repeats and 3.5 repeats respectively according to Sabol *et al.*, 1998) observed by other authors (Sabol *et al.*, 1998; Deckert *et al.*, 1999; Guo *et al.*, 2008). The lowest frequency allele in our sample was the 5.5 repeat allele (frequency = 0.032). These results correspond with previous studies reporting that the 3.5 repeat allele (3 repeats according to these authors) and 4.5 repeat allele (4 repeats according to these authors) are the most commonly observed alleles in the human population (Sabol *et al.*, 1998; Deckert *et al.*, 1999; Jönsson *et al.*, 2000; Manor *et al.*, 2002; Lawson *et al.*, 2003; Ito *et al.*, 2003; Yu *et al.*, 2005).

Table 7.1. Allelic variation of the *MAO-A*-uVNTR in a sample of males and females from central South Africa

Gender	Number of chromosomes	MAO-A-uVNTR allele frequencies (R = Number of repeats)		
		3.5R	4.5R	5.5R
Male	9	0.11	0.78	0.11
Female	22	0.5	0.5	0
Total	31	0.39	0.58	0.032

Sequence variations

The most salient feature of our results was that instead of the 3, 3.5, 4, and 5 repeats of the 30 bp repeat sequence found by all previously named authors (eg. Sabol *et al.*, 1998; Guo *et al.*, 2008), we found repeat lengths of 3.5, 4.5 and 5.5 repeats (Figure 7.2). Further investigation revealed that only two other studies reported the same repeat lengths. The first of these was done by Jorm *et al.* (2000) in an Australian sample and found 2.5, 3.5, 4, 4.5, and 5.5 repeats of the 30 bp repeat motive. The second study was done by Das *et al.* (2006) in an Indian sample, and found 2.5, 3.5, 4.5 and 5.5 repeats of the repeat motive. Only latter authors commented on the discrepancy. They noted that Sabol *et al.* (1998) wrongly stated that the sequence available with Genbank (*MAO-A,* Genbank M89636) (Zhu *et al.*, 1992) contained 4 repeats of the repeat motive, while in actual fact it consisted of 4.5 repeats of the 30 bp repeat sequence.

Investigation for the present study revealed that Das *et al.* (2006) came to the correct conclusion. Sabol *et al.* (1998) did indeed state that the allele they found to be identical to the published sequence by Zhu *et al.* (1992) (GenBank accession number M89636), consisted of 4 repeats of the 30 bp repeat sequence, while in actual fact it consisted of 4.5 repeats. Apart from the two mentioned studies, all studies done after the Sabol *et al.* (1998) study followed the norm established by Sabol *et al.* (1998) and consequently reported the repeat numbers incorrectly. Therefore, as was done by Das *et al.* (2006), we drew the

conclusion that the 3.5, 4.5 and 5.5 repeats found in our sample correspond respectively to the 3, 4 and 5 repeats described by Sabol *et al.* (1998).

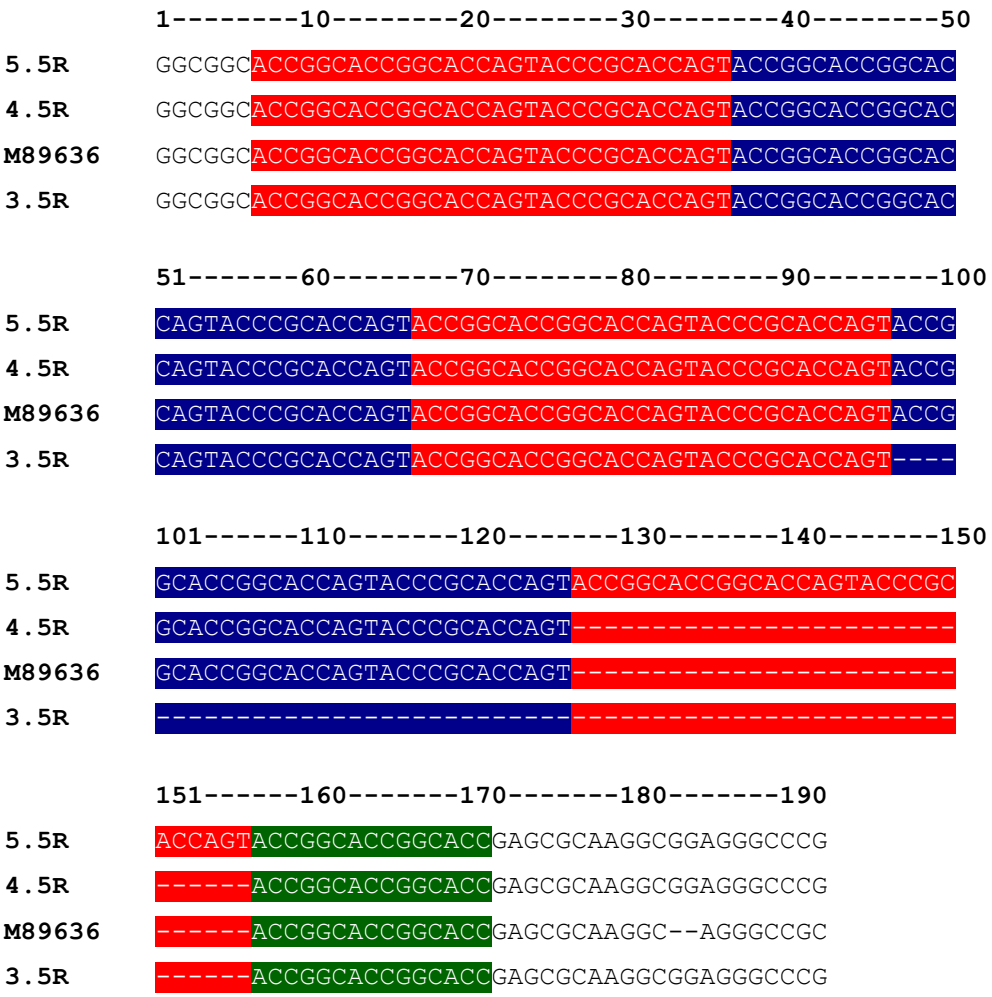

Figure 7.2. Alignment of the 4.5 repeat *MAO-A*-uVNTR allele from Zhu *et al.* (1992) stored in Genbank (Assession number M89636) with different lengths observed during this study. The 'full' repeat regions are highlighted consecutively in red and blue and the half repeat is indicated in green.

Apart from the differing allocation of number of repeats, one deletion (at positions 183-184 in Figure. 7.2) and two nucleotide substitutions (G>C and C>G) (at the last two positions in Figure. 7.2) was also noted in the Zhu *et al.* (1992) sequence compared to the one obtained in the present study. The exact same substitutions were noted by Das *et al.* (2006).

Main effects of the *MAO-A*-uVNTR on aggression

In order to determine whether the *MAO-A*-uVNTR had any main effects on aggressive behaviour, average aggression scores for the different allele carriers

were compared by means of a one way analysis of variance (ANOVA) (Tables 7.2 & 3). Due to very low frequency, the 5.5 repeat allele was not taken into consideration in the analysis. Analysis were carried out separately for reactive-, proactive-, and total aggression. Individuals were divided into three groups: 3.5 repeat allele carriers, 4.5 repeat allele carriers and 3.5/4.5 heterozygotes. Apart from the heterozygotes, women and men were grouped together since the latest evidence (Stabellini *et al*., 2009) suggests that the *MAO-A* gene does undergo X-inactivation, rendering the homozygote females equivalent to the hemizygote males. Since it is unknown which X chromosome is inactivated, it was deemed appropriate to group the 3.5/4.5 heterozygotes separately.

Table 7.2 Mean reactive-, proactive- and total aggression scores for different *MAO-A*-uVNTR allele carrier groups

Groups	**N**	**Reactive aggression**		**Proactive aggression**		**Total aggression**	
		Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
3.5 allele group	5	6.60	5.98	1.20	1.30	7.80	7.26
4.5 allele group	11	7.27	3.20	2.00	2.37	9.27	4.92
3.5/4.5 allele group	3	7.67	4.16	1.33	2.31	9.00	5.29

Table 7.3 Summary of one way ANOVA results for comparison of reactive, proactive and total aggression mean scores compared for the different *MAO-A*-uVNTR genotype carrier groups

ANOVA	F	Sig. (p)
Reactive aggression	0.071	0.932
Proactive aggression	0.287	0.754
Total aggression	0.119	0.889

No significant differences between the mean reactive aggression scores of the different *MAO-A*-uVNTR allele carrier groups could be detected on either the

99% ($p > 0.01$) or the 95% ($p > 0.05$) levels of statistical significance. The same result held true for both mean proactive aggression and mean total aggression scores for the different *MAO-A*-uVNTR allele carrier groups. Thus, in the present study no main effect for the *MAO-A*-uVNTR on reactive-, proactive- or total aggression could be detected. These results are in accordance with results from a number of different authors, all reporting that the *MAO-A*-uVNTR has no main effect on various types of aggression and antisocial behaviour (Koller *et al.*, 2003; Widom & Brzustowicz, 2006; Frazzetto *et al.*, 2007; Tikkanen *et al.*, 2009).

As mentioned, some authors did find a main effect for the *MAO-A*-uVNTR on aggression (Guo *et al.*, 2008; Reti *et al.*, 2011). Since this finding is observed infrequently, it is possible, and even probable, that these authors randomly and inadvertently sampled individuals who were homogenous for factors like trauma, which interacted with *MAO-A*-uVNTR, and thus resulted in a false main effect for this polymorphism. The findings from the present study clearly exclude the *MAO-A*-uVNTR as the only contributing factor to high aggression scores.

MAO-A-uVNTR as a mediating factor in the effect of traumatic event exposure on aggression

In order to determine the possible influence of a gene-environment interaction effect between traumatic event exposure and the *MAO-A*-uVNTR in causing aggressive behaviour, a one way ANOVA model was used (Tables 7.4 & 5). Once again analyses were carried out separately for reactive-, proactive-, and total aggression. Traumatic event exposure was grouped as follows: exposure to 0 or 1 traumatic event = low trauma, and exposure to 2 to 6 traumatic events = high trauma. In accordance with this grouping, individuals were grouped into 6 separate groups as follows: 4.5 repeats/low trauma; 4.5 repeats/high trauma; 3.5 repeats/low trauma; 3.5 repeats/high trauma; heterozygotes/low trauma; heterozygotes high trauma.

No statistically significant differences (95% or 99% level of statistical significance) could be detected between the aggression means of the six different *MAO-A*-uVNTR/traumatic event exposure groups for reactive-, proactive- or to-

tal aggression ($p > 0.05$) (Table 7.5). This is in contrast to results by various authors who found a significant gene-environment interaction between the *MAO-A*-uVNTR genotype, traumatic event exposure and aggression. They found that those individuals carrying the short repeat alleles (in our study the 3.5 repeat) and who were exposed to significant trauma, showed significantly higher levels of aggression (Caspi *et al.*, 2002; Kim-Cohen *et al.*, 2006; Widom & Brzustowicz, 2006; Frazzetto *et al.*, 2007). When not considering statistical significance, the present results showed that heterozygote individuals with high trauma had the highest reactive and total aggression scores. Since only three heterozygotes were included in our study, this result is in need of replication with a much larger sample of heterozygotes, and does not warrant further discussion in this article.

Table 7.4 Mean aggression scores of individuals grouped according to number of *MAO-A*-uVNTR repeats/ trauma exposure

***MAO-A*-uVNTR repeats/trauma groups**	**Reactive aggression**	**Proactive aggression**	**Total aggression**
	Mean	**Mean**	**Mean**
3.5 repeats/low trauma	5	1	6
3.5 repeats/high trauma	9	2	11
4.5 repeats/low trauma	5	2	7
4.5 repeats/high trauma	7	2	9
Heterozygote/low trauma	3	0	3
Heterozygote/high trauma	10	2	12

Table 7.5 Summary of one way ANOVA results for comparison of mean reactive-, proactive-, and total aggression scores compared for the six different *MAO-A*-uVNTR/trauma groups

ANOVA	F	Sig. (p)
Reactive aggression	1.052	0.429
Proactive aggression	0.266	0.924
Total aggression	0.763	0.592

Disregarding statistical significance and the heterozygote groups, the present results also did show that individuals carrying the 3.5 repeat allele and who had high traumatic event exposure, showed the highest reactive and total aggression scores of all the groups. Thus, our failure to replicate the statistically significant results of the other studies may only be because of our much smaller sample size (e.g. N=20 in our study, versus N = 235 in Frazetto *et al*., 2007 study).

Conclusions

In conclusion, the present study showed discrepancies between the number of repeats of the *MAO-A*-uVNTR reported on previously, and our results. We concluded that a mistake was made by previous authors by wrongfully assuming that the published Genbank sequence (*MAO-A,* Genbank M89636) (Zhu *et al.*, 1992) contained 4 repeats of the 30 bp sequence, while it actually contained 4.5 repeats. Consequently, we found that the previously stated 3, 4 and 5 repeats of the 30 bp repeat motive are in actual fact 3.5, 4.5, and 5.5 repeats respectively.

We found no main effect for the *MAO-A*-uVNTR on reactive-, proactive- or total aggression scores. In contrast to previous studies, no statically significant gene-environment interaction effects on aggression were observed between the different *MAO-A*-uVNTR alleles and traumatic event exposure. We did find that, when excluding the heterozygotes, individuals carrying the short 3.5 repeat allele, who were also exposed to two or more traumatic events, showed the highest reactive and total aggression scores. Although statistical significance was not reached with this finding, we concluded that this was the result of our small sample size. It is thus imperative that this study be repeated with a much larger sample size in order to see whether previous results of a statistically significant gene-environment interaction effect on aggression can be replicated in a South African sample.

CHAPTER 8

Summary

The aim of this dissertation was to identify putative genetic and environmental influences on reactive, proactive, and total aggression. The environmental influences chosen for this study were emotional intelligence and traumatic event exposure. Since self-report measures were made use of to measure these environmental factors, there was a risk for people providing dishonest answers by responding in a socially desirable manner. For this reason, a measure of socially desirable responding was included. Genetic influences were first examined by means of a family study, followed up by a genetic association study. Only one gene was studied, namely the Monoamine Oxidase A (*MAO-A*) gene. A 30 bp repeat polymorphism in the promoter region of the *MAO-A* gene (*MAO-A*-u-VNTR) was studied for its possible influence on aggressive behaviour. In addition, a possible gene-environment interaction between the *MAO-A*-u-VNTR and traumatic event exposure in influencing aggression was examined.

Social desirability bias did not have significant influence on the various correlation coefficients calculated, and was henceforth excluded from the study.

Results indicated statistically significant negative correlations between emotional intelligence and reactive-, proactive- and total aggression. Effect sizes for these correlations were, however, small, indicating low practical significance. Statistically significant positive correlations occurred between traumatic event exposure and all three types of aggression. Practical significance ranged from small (proactive aggression) to medium (reactive- and total aggression). Results from a regression analysis indicated that both traumatic event exposure and emotional intelligence made significant unique contributions to the variance in total aggression. Of the two, traumatic event exposure made the largest unique contribution. Traumatic event exposure and emotional intelligence combined accounted for 17.3% of the variance in total aggression scores.

Results from the family study indicated upper-limit heritability values ranging from 41.2% to 42% for reactive aggression. Proactive aggression showed much lower upper-limit heritability values, with values ranging from 6% to 12.9%. Latter result could be due to the low range of proactive aggression scores obtained from the sample under study. Consequently, the non-shared

environment was shown to exercise the greatest influence on both reactive- and proactive aggression, compared to the combined effects of additive genetic and shared environmental influences (upper-limit heritability). Assortative mating for aggression was not found to play a role in the family study.

In the genetic association study the number of 30 bp repeats of the *MAO-A*-u-VNTR varied from those previously reported. Instead of the 3, 4, and 5 repeats noted by most authors, we found 3.5, 4.5 and 5.5 repeat sequences. The conclusion was drawn that an error was made in the first study reporting on the repeats, which established the norm followed by later studies. Consequently our 3.5, 4.5 and 5.5 repeats were respectively equivalent to the 3, 4, and 5 repeats previously reported on. Results showed no main effects for the *MAO-A*-u-VNTR on reactive-, proactive- or total aggression scores. A small gene-environment interaction effect was observed, with individuals carrying the short 3.5 repeat allele with high traumatic event exposure showing higher reactive aggression scores. This result was not statistically significant.

Taken overall, traumatic event exposure was shown to have the greatest influence on reactive-, proactive-, and total aggression scores, when compared with emotional intelligence and the influence of the *MAOA*-u-VNTR. In line with behavioural genetic research up to date, both the environment and genetic factors appeared to play a role in aggressive behaviour. The finding that the *MAOA*-u-VNTR had no main effect on aggression made it clear that future research should not focus on the effect of single genes alone, but rather on the interaction between multiple genes and environmental factors. In addition, future studies should include much larger sample sizes.

Keywords: Reactive aggression, proactive aggression, serotonin, emotional intelligence, trauma, monoamine oxidase A, promoter polymorphism, young adult population, heritability, non-shared environmental factors

Opsomming

Die doel van die verhandeling was om die invloed van moontlike genetiese en omgewingsfaktore op reaktiewe-, proaktiewe- en totale aggressie te ondersoek. Twee omgewingsfaktore, naamlik emosionele intelligensie en blootstelling aan traumatiese gebeurtenisse, is vir die ondersoek gekies. Die omgewingsfaktore is deur middel van eie-verslag vraelyste gemeet. Gevolglik was daar 'n moontlikheid dat deelnemers sosiaal aanvaarbare antwoorde sou verskaf. Daarom is 'n vraelys wat "sosiaal aanvaarbare beantwoording" meet, ingesluit in die studie. Die moontlike effek van genetiese invloede is eerstens deur 'n familiestudie gemeet.

Dit is gevolg deur 'n genetiese assosiasie studie om die effek van een geen, naamlik die Monoamien Oksidase A (*MAO-A*) geen, op aggressie te bepaal. Die invloed van 'n 30 basispaar herhalingspolimorfisme in die promotor area van die *MAO-A* geen (*MAOA*-u-VNTR) is bestudeer. Die moontlike invloed op aggressie van 'n geen-omgewing interaksie tussen die *MAO-A*-uVNTR en blootstelling aan traumatiese gebeurtenisse is ondersoek.

"Sosiaal aanvaarbare beantwoording" het geen noemenswaardige effek op die verskeie korrelasie koeffsiënte gehad nie. Gevolglik is besluit om hierdie faktor nie verder te gebruik nie.

Statisties beduidende negatiewe korrelasies is tussen emosionele intelligensie en reaktiewe-, proaktiewe- en totale aggressie gevind. Effekgroottes vir hierdie korrelasies was egter klein, wat dui op lae praktiese beduidendheid. Statisties beduidende positiewe korrelasies is tussen trauma blootstelling en al drie tipes aggressie gevind. Praktiese beduidenheid van die korrelasies het gewissel van klein (proaktiewe aggressie) tot middelmatig (reaktiewe- en totale aggressie). 'n Regressie analise het getoon dat beide trauma blootstelling en emosionele intelligensie betekenisvolle individuele bydraes tot die variasie in totale aggressie lewer. Trauma blootstelling het egter die grootste unieke bydrae gemaak. 'n Deel (17.3%) van die variasie in totale aggressie tellings word deur die gekombineerde effek van trauma blootstelling en emosionele intelligensie verklaar.

Familiestudie dui op boonste-limiet erflikheidstellings van 41.2% tot 42% vir reaktiewe aggressie, teenoor proaktiewe aggressie se laer waardes van 6% tot 12.9%. Hierdie resultaat kan die gevolg wees van die lae spanwydte van proaktiewe aggressie tellings in die steekproef. In vergelyking met die gekombineerde effek van additief genetiese en gedeelde omgewingsinvloede (boonste-limiet erflikheid), is gevolglik bevind dat die nie-gedeelde omgewing die grootste rol speel in beide reaktiewe- en proaktiewe aggressie tellings. Voorkeurparing het geen rol in die familiestudie gespeel nie.

In die genetiese assosiasie studie is gevind dat die aantal 30 basispaar herhalings van die *MAO-A*-uVNTR verskil van gepubliseerde data. In die huidige studie is 3.5, 4.5 en 5.5 herhalings gevind. Meeste vorige outeurs beskryf egter 3, 4, en 5 herhalings. Kontrolering van data dui op ‘n fout wat gemaak is deur een van die eerste outeurs wat die herhalings beskryf het. Latere outeurs het die norm wat so gestel is gevolg. Gevolglik is die 3.5, 4.5, en 5.5 herhalings van hierdie studie, ekwivalent aan die 3, 4 en 5 herhalings respektiewelik. Die resultate het getoon dat die *MAO-A*-u-VNTR geen hoofeffek op reaktiewe-, proaktiewe- of totale aggressie het nie. ‘n Klein geen-omgewing interaksie effek is bespeur deurdat individue wat die kort 3.5 herhaling alleel dra en ook hoë trauma blootstelling toon, hoër reaktiewe aggressie tellings getoon het. Hierdie resultaat was egter nie statisties beduidend nie.

Trauma blootstelling was, in vergelyking met emosionele intelligensie en die *MAO-A*-u-VNTR, die grootste bydraende faktor tot reaktiewe-, proaktiewe- en totale aggressietellings. In ooreenstemming met gedragsgenetiese navorsing tot op datum, is gevind dat beide die omgewing en genetiese faktore ‘n rol speel in aggressiewe gedrag. Die bevinding dat die *MAOA-u*-VNTR nie die belangrikste enkele effek op aggressie het nie behoort ‘n aanduiding te wees dat toekomstige navorsing nie slegs na die effek van een geen moet kyk nie, maar eerder kyk na die interaksie tussen ‘n aantal gene en omgewingsfaktore. Groter steekproewe moet ook bekom word vir toekomstige studies.

Sleutelwoorde: Reaktiewe aggressie, proaktiewe aggressie, serotonien; emosionele intelligensie, trauma, monoamien oksidase A, promoter po-

limorfisme, jong volwasse populasie, erfbaarheid, nie-gedeelde omgewings-faktore

CHAPTER 9

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APPENDICES

Appendix A1: Cover letter for questionnaire, including informed consent

Appendix A2: Afrikaans translation of Appendix A1

Appendix A3: Self-compiled biographical questionnaire – English and Afrikaans translation

Appendix A4: The Reactive-Proactive Aggression Questionnaire (RPQ)

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Appendix B1: The Balanced Inventory of Desirable Responding (BIDR)

Appendix B2: Afrikaans translation of Appendix B1

Appendix C1: The Trait Emotional Intelligence Questionnaire-Short Form (TEIQue-SF)

Appendix C2: Afrikaans translation of Appendix C1

Appendix D1: The Stressful Life Events Screening Questionnaire (SLESQ)

Appendix D2: Afrikaans translation of Appendix D1

Appendix E: Data from sequencing reactions

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Appendix A1: Information leaflet for participants

Dear participant

The aim of this study is to find out whether there are certain genes that can be linked to aggression in people, and also whether there is a link between emotional intelligence and aggression.

You will only be asked to fill in 4 quick questionnaires, and a DNA sample will be taken from you by means of a cheek swab or a blood sample. You can take as long as you need to fill out the questionnaires. Furthermore, the information we collect from you will only be used by us to research aggression and emotional intelligence, and we will **NOT** give it to any other people. We will **NOT** use your DNA for **ANY OTHER REASON** than to try and find the genes that affect aggression! However, the results of this study may be published in a scientific journal, but we assure you that no one will be given your name or any other personal information about you.

You will **NOT** be hurt physically or emotionally if you take part in this study. However, you are free to stop participating in this study at any time, and doing so will have **NO** negative effect on you in any way. Participating in this study will not cost you any money. Similarly, you will also not receive any money or any other compensation if you participate in this study.

Researcher: Nadia Laubscher

Supervisor: Prof. J.J. Spies

I, _____(Name and surname), hereby **AGREE** to participate in the research described above. I understand that my participation in the study is completely voluntary, that participating or choosing to withdraw from the study will not hold any negative consequences for myself, or cost anything. I further understand that the data gathered in this study may be published in a scientific journal, but if this should happen, you will remain completely anonymous.

_____

Signature Date

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Appendix A2: Afrikaans translation of Appendix A1

Inligtingsbladsy vir deelnemers

Beste deelnemer

Die doel van hierdie studie is om te bepaal of daar sekere gene is wat moontlik aggressie in mense kan veroorsaak. Die studie sal ook gebruik word om te bepaal of daar 'n verwantskap is tussen emosionele intelligensie en aggressie.

U sal slegs gevra word om 4 kort vraelyste in te vul, en 'n DNA monster sal by u geneem word deur middel van 'n wangskraping of 'n bloedmonster . U kan solank as wat u nodig het neem om die vraelyste in te vul. Verder sal die inligting en DNA wat ons by u gaan kry slegs deur ons gebruik word om aggressie en emosionele intelligensie na te vors . Ons sal **NIE** u DNA vir **ENIGE ANDER REDE** gebruik as om gene vir aggressie te probeer kry nie! Ons sal NIE u inligting vir enige iemand anders gee nie. Die resultate van hierdie studie kan moontlik in 'n wetenskaplike joernaal gepubliseer word, maar in hierdie geval verseker ons u dat **NIE u naam** of enige persoonlike inligting van u bekend gemaak sal word **NIE**.

Om aan hierdie studie deel te neem sal u **NIE** fisies of emosioneel seermaak nie. Dit staan u egter vry om in enige stadium van die studie te onttrek en deur dit te doen, sal geen negatiewe gevolge vir u inhou nie. Om deel te neem aan die studie sal u geen geld kos nie. Terselfdertyd sal u geen geld of ander vergoeding ontvang vir u deelname aan die studie nie.

Navorser: Nadia Laubscher

Studieleier: Prof. J.J. Spies

Ek, _____(Naam en van), **STEM HIERMEE IN** om aan die studie wat hierbo beskryf is deel te neem. Ek verstaan dat my deelname aan die studie geheel en al vrywillig is en dat die besluit om aan die studie te onttrek geen negatiewe gevolge vir my sal inhou of my enige iets sal kos nie. Verder verstaan ek dat die data wat in hierdie ondersoek versamel word in 'n wetenskaplike tydskrif gepubliseer kan word, maar dat geen persoonlike inligting van myself in hierdie geval bekend gemaak sal word nie.

_____

Handtekening Datum

Appendix A3: Self-compiled biographical questionnaire – English and Afrikaans translation

INSTRUCTIONS: Please mark the applicable response with an X.					
1. Gender:	Male			Female	
2. Age: _____					
3. Highest education level achieved: _____					
4. Home language: _____					
5. Race (For statistical purposes only):	White	Black	Colored	Indian	Other
6. Are you currently taking any medication?	Yes		No	Please specify _____ _____ _____	

AFDELING A **INSTRUKSIES:** Merk asb. die gepaste reaksie met 'n kruisie.					
1. Geslag:	Manlik			Vroulik	
2. Ouderdom: _____					
3. Hoogste opvoedkundige vlak behaal: _____					
4. Huistaal: _____					
5. Rassegroep **a. (Slegs vir statistiese doeleindes):**	Wit	Swart	Kleurling	Indiër	Ander
6. Gebruik u tans enige medikasie?	Ja	Nee	Spesifiseer asb. _____ _____		

Appendix A4: The Reactive-Proactive Aggression Questionnaire (RPQ)

INSTRUCTIONS:

There are times when most of us feel angry, or have done things we should not have done. Rate each of the items below by making use of the following response categories:
0 = never
1 = sometimes (less than once a week)
2 = often (more than once a week)
Do not spend a lot of time thinking about the items – just give your first response.

How often have you……				
1.	Yelled at others when they have irritated you or made you cross.	0 Never	1 Sometimes	2 Often
2.	Had fights with others to show who is the leader.	0 Never	1 Sometimes	2 Often
3.	Reacted angrily when pushed or irritated by others.	0 Never	1 Sometimes	2 Often
4.	Taken things from other people with the aim of hurting them.	0 Never	1 Sometimes	2 Often
5.	Gotten angry when frustrated.	0 Never	1 Sometimes	2 Often
6.	Vandalized something for fun.	0 Never	1 Sometimes	2 Often
7.	Had temper outbursts or lost your temper	0 Never	1 Sometimes	2 Often
8.	Damaged things because you felt mad.	0 Never	1 Sometimes	2 Often
9.	Had a fight to be cool.	0 Never	1 Sometimes	2 Often
10.	Hurt others to win a game.	0 Never	1 Sometimes	2 Often
11.	Become angry or mad when you don't get your way.	0 Never	1 Sometimes	2 Often
12.	Used physical force to get others to do what you want.	0 Never	1 Sometimes	2 Often
13.	Gotten angry or mad when you lost a game.	0 Never	1 Sometimes	2 Often
14.	Gotten angry when others threatened you.	0 Never	1 Sometimes	2 Often
15.	Used force to obtain money or things from others.	0 Never	1 Sometimes	2 Often
16.	Felt better after hitting or yelling at someone.	0 Never	1 Sometimes	2 Often

How often have you……				
17.	Threatened and bullied someone.	0 Never	1 Sometimes	2 Often
18.	Made obscene phone calls for fun	0 Never	1 Sometimes	2 Often
19.	Hit others to defend yourself.	0 Never	1 Sometimes	2 Often
20.	Gotten others to gang up on someone else.	0 Never	1 Sometimes	2 Often
21.	Carried a weapon to use in a fight.	0 Never	1 Sometimes	2 Often
22.	Gotten angry or mad or hit others when teased or made fun of.	0 Never	1 Sometimes	2 Often
23.	Yelled at others so they would do things for you.	0 Never	1 Sometimes	2 Often

Appendix A5: Afrikaans translation of Appendix A4

INSTRUKSIES:

Daar kom tye wanneer die meeste van ons kwaad voel, of iets doen wat ons nie behoort te doen nie. Merk elkeen van die onderstaande items deur gebruik te maak van die volgende reaksie kategorieë:
0 = nooit
1 = soms (minder as een keer per week)
2 = dikwels (meer as een keer per week)
Moenie te lank oor die items dink nie – gee eerder net u eerste reaksie.

Hoe dikwels het u...				
1.	Geskree op ander wanneer hulle u geïrriteer of kwaad gemaak het.	0 Nooit	1 Soms	2 Dikwels
2.	In gevegte met ander betrokke geraak om te wys wie die leier is.	0 Nooit	1 Soms	2 Dikwels
3.	Kwaad geword as ander u druk of irriteer.	0 Nooit	1 Soms	2 Dikwels
4.	Goed van ander mense gevat met die doel om hulle te na te kom.	0 Nooit	1 Soms	2 Dikwels
5.	Kwaad geword wanneer u gefrustreerd was.	0 Nooit	1 Soms	2 Dikwels
6.	Iets verniel (gevandaliseer) vir die pret.	0 Nooit	1 Soms	2 Dikwels
7.	Woede-uitbarstings gehad of u humeur verloor.	0 Nooit	1 Soms	2 Dikwels
8.	Items beskadig omdat u kwaad gevoel het.	0 Nooit	1 Soms	2 Dikwels
9.	In ‘n bakleiery betrokke geraak om “cool” te wees.	0 Nooit	1 Soms	2 Dikwels
10.	Ander seergemaak om ‘n speletjie te wen.	0 Nooit	1 Soms	2 Dikwels
11.	Kwaad geraak indien u nie u sin kry nie.	0 Nooit	1 Soms	2 Dikwels
12.	Fisiese geweld gebruik om ander sover te kry om te doen wat jý wil.	0 Nooit	1 Soms	2 Dikwels
13.	Kwaad geword wanneer u ‘n speletjie verloor het.	0 Nooit	1 Soms	2 Dikwels
14.	Kwaad geword wanneer ander u gedreig het.	0 Nooit	1 Soms	2 Dikwels

Maak gebruik van die volgende reaksiekategorieë: 0 = nooit 1 = soms (minder as een keer per week) 2 = dikwels (meer as een keer per week)				
Hoe dikwels het u...				
15.	Geweld gebruik om geld of items by ander te kry.	0 Nooit	1 Soms	2 Dikwels
16.	Beter gevoel nadat u op iemand geskree of iemand geslaan het.	0 Nooit	1 Soms	2 Dikwels
17.	Iemand gedreig en afgeknou.	0 Nooit	1 Soms	2 Dikwels
18.	Afskuwelike telefoonoproepe gemaak vir die pret	0 Nooit	1 Soms	2 Dikwels
19.	Ander geslaan om uself te verdedig.	0 Nooit	1 Soms	2 Dikwels
20.	Ander gekry om saam met u teen iemand te baklei.	0 Nooit	1 Soms	2 Dikwels
21.	‘n Wapen gedra om in ‘n bakleiery te gebruik.	0 Nooit	1 Soms	2 Dikwels
22.	Kwaad geword of ander geslaan wanneer hulle u geterg of gespot het.	0 Nooit	1 Soms	2 Dikwels
23.	Op ander geskree sodat hulle goed vir u sal doen.	0 Nooit	1 Soms	2 Dikwels

Appendix B1: The Balanced Inventory of Desirable Responding (BIDR)

INSTRUCTIONS

For each statement listed below, please indicate to what extent you agree or disagree with it by using the scale below as a guide.

1 ----------2----------3----------4----------5----------6----------7
NOT TRUE — SOMEWHAT TRUE — VERY TRUE

1.	My first impressions of people usually turn out to be right.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
2.	It is hard for me to break my bad habits.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
3.	I don't care what other people think of me.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
4.	I am not always honest with myself.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
5.	I always know why I like things.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
6.	When my emotions are aroused, it biases my thinking.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
7.	Once I've made up my mind, other people can seldom change my opinion.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
8.	I am not a safe driver when I exceed the speed limit.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
9.	I am fully in control of my own fate.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
10.	It's hard for me to shut off a disturbing thought.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
11.	I never regret my decisions.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
12.	I sometimes lose out on things because I can't make up my mind soon enough.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True

13.	I vote because I feel my vote can make a difference.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
14.	My parents were not always fair when they punished me.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
15.	I am a completely rational person.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
16.	I rarely appreciate criticism.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
17.	I am very confident of my judgments.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
18.	I have sometimes doubted my ability as a lover.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
19.	It's all right with me if some people don't like me.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
20.	I don't always know the reasons why I do the things I do.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
21.	I sometimes tell lies if I have to.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
22.	I never cover up my mistakes.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
23.	I have taken advantage of someone before.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
24.	I never swear.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
25.	I sometimes try to get even rather than forgive and forget.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True

26.	I always obey laws, even if I'm unlikely to get caught.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
27.	I have said something bad about a friend behind his or her back.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
28.	When I hear people talking privately, I avoid listening.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
29.	I have received too much change from a salesperson without telling him or her.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
30.	I always declare everything at customs.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
31.	When I was young I sometimes stole things.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
32.	I have never dropped litter on the streets.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
33.	I sometimes drive faster than the speed limit.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
34.	I never read sexy books or magazines.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
35.	I have done things that I don't tell other people about.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
36.	I never take things that don't belong to me	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
37.	I have taken sick-leave from work or school even though I wasn't really sick.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
38.	I have never damaged a library book or store merchandise without reporting it.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
39.	I have some pretty awful hab-its.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True
40.	I don't gossip about other people's business.	1 Not True	2	3	4 Somewhat true	5	6	7 Very True

Appendix B2: Afrikaans translation of Appendix B1

INSTRUKSIES

Dui asb. vir elke onderstaande stelling die mate aan waartoe u verskil of saamstem met die stelling. Gebruik die volgende skaal as riglyn:

1…….2…….3…….4…….5…….6…….7
NIE WAAR NIE — TOT 'N MATE WAAR — BAIE WAAR

1.	My eerste indrukke van mense is gewoonlik reg.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
2.	Dit is vir my moeilik om af te sien van my slegte ge-woontes.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
3.	Ek gee nie om wat ander mense van my dink nie.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
4.	Ek is nie altyd eerlik met my-self nie.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
5.	Ek weet altyd hoekom ek van iets hou.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
6.	Wanneer ek emosioneel raak bevooroordeel dit my denke.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
7.	Wanneer ek op iets besluit het, kan ander mense my selde van opinie laat ve-rander.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
8.	Ek is nie 'n veilige bestuurder wanneer ek die spoedgrens oorskry nie.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
9.	Ek is ten volle in beheer van my eie lot.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
10.	Dit is vir my moeilik om 'n ontstellende gedagte uit my denke te sny.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar

11.	Ek berou nooit my besluite nie.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
12.	Ek mis soms uit op sekere dinge omdat ek nie vinnig genoeg ‘n besluit kan neem nie.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
13.	Ek stem in ‘n verskiesing omdat ek voel dat my stem ‘n verskil kan maak.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
14.	My ouers was nie altyd regverdig wanneer hulle my gestraf het nie.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
15.	Ek is geheel en al ‘n rasionele mens.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
16.	Ek waardeer selde kritiek.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
17.	Ek het baie vertroue in my beoordelings.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
18.	Ek het al soms my vermoëns as ‘n minnaar/minnares be-twyfel.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
19.	Dit pla my nie as party mense nie van my hou nie.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
20.	Ek weet nie altyd wat die redes is vir hoekom ek dinge doen nie.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
21.	Ek vertel soms leuens as ek voel ek moet.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
22.	Ek steek nooit my foute weg nie.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
23.	Ek het al voorheen iemand anders tot my eie voordeel misbruik.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar
24.	Ek vloek nooit nie.	1 Nie waar nie	2	3	4 Tot ‘n ma-te waar	5	6	7 Baie waar

25.	Ek probeer soms wraak neem, eerder as om te vergewe en te vergeet.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
26.	Ek gehoorsaam altyd wette, selfs al is dit onwaarskynlik dat ek uitgevang sal word.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
27.	Ek het al iets slegs van 'n vriend/vriendin gesê agter sy/haar rug.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
28.	Wanneer ek mense privaat hoor praat, weerhou ek my-self daarvan om te luister.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
29.	Ek het al te veel kleingeld van 'n verkoopspersoon ontvang sonder dat ek hom/haar daarvan gesê het.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
30.	Ek verklaar altyd alles by doeane.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
31.	Ek het soms goed gesteel toe ek jonk was.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
32.	Ek het nog nooit rommel in die straat gestrooi nie.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
33.	Ek oorskry soms die spoed-grens wanneer ek bestuur.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
34.	Ek lees nooit "sexy" boeke of tydskrifte nie.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
35.	Ek het al goed gedoen wat ek nie vir ander mense van ver-tel nie.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
36.	Ek vat nooit goed wat nie aan my behoort nie.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
37.	Ek het al siekeverlof van die werk of skool geneem sonder dat ek werklik siek was.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
38.	Ek het nog nooit 'n biblio-teekboek of winkelitem beskadig sonder dat ek dit aangemeld het nie.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
39.	Ek het 'n paar aaklige ge-woontes.	1 Nie waar nie	2	3	4 Tot 'n ma-te waar	5	6	7 Baie waar
40.	Ek skinder nie oor ander	1	2	3	4	5	6	7

	mense se besigheid nie.	Nie waar nie			Tot ‘n ma-te waar			Baie waar

Appendix C1: The Trait Emotional Intelligence Questionnaire-Short Form (TEIQue-SF)

INSTRUCTIONS:

Please answer each statement below by putting a circle around the number that best reflects your degree of agreement or disagreement with that statement. Do not think too long about the exact meaning of the statements. There are no right or wrong answers. There are seven possible responses to each statement ranging from 'Completely Disagree' (number 1) to 'Completely Agree' (number 7).

		1……..2……..3……..4……..5……..6……..7						
		Completely Disagree						**Completely Agree**
1.	It is easy for me to express my feelings in words.	1.	2.	3.	4.	5.	6.	7.
2.	I find it difficult to see things from another person's viewpoint.	1.	2.	3.	4.	5.	6.	7.
3.	I am a highly motivated person.	1.	2.	3.	4.	5.	6.	7.
4.	It is difficult for me to regulate my feelings.	1.	2.	3.	4.	5.	6.	7.
5.	I don't enjoy life.	1.	2.	3.	4.	5.	6.	7.
6.	I can handle people well.	1.	2.	3.	4.	5.	6.	7.
7.	I change my mind a lot.	1.	2.	3.	4.	5.	6.	7.
8.	I often can't figure out what I'm feeling.	1.	2.	3.	4.	5.	6.	7.
9.	I feel that I have a number of good qualities.	1.	2.	3.	4.	5.	6.	7.
10.	It is difficult for me to stand up for my rights.	1.	2.	3.	4.	5.	6.	7.
11.	I can influence the way other people feel.	1.	2.	3.	4.	5.	6.	7.

Please use the following response categories: **1……..2……..3……..4……..5……..6……..7** **Completely Disagree** — **Completely Agree**								
12.	I have a negative outlook on most things.	1.	2.	3.	4.	5.	6.	7.
13.	Those close to me often complain that I don't treat them right.	1.	2.	3.	4.	5.	6.	7.
14.	It is difficult for me to adjust my life according to the circumstances.	1.	2.	3.	4.	5.	6.	7.
15.	I can deal with stress.	1.	2.	3.	4.	5.	6.	7.
16.	It is difficult for me to show those close to me that I care.	1.	2.	3.	4.	5.	6.	7.
17.	I can experience what other people are feeling.	1.	2.	3.	4.	5.	6.	7.
18.	It is difficult for me to keep myself motivated.	1.	2.	3.	4.	5.	6.	7.
19.	I can control my feelings when I want to.	1.	2.	3.	4.	5.	6.	7.
20.	I'm generally pleased with my life.	1.	2.	3.	4.	5.	6.	7.
21.	I am a good negotiator (I can convince people to do things my way).	1.	2.	3.	4.	5.	6.	7.
22.	I tend to get involved in things that I later wish I could get out of.	1.	2.	3.	4.	5.	6.	7.

Please use the following response categories: **1……..2……..3……..4……..5……..6……..7** **Completely Disagree**								**Completely Agree**
23.	I often pause and think about my feelings.	1.	2.	3.	4.	5.	6.	7.
24.	I'm full of personal strengths.	1.	2.	3.	4.	5.	6.	7.
25.	I tend to "back down" even if I know I'm right.	1.	2.	3.	4.	5.	6.	7.
26.	I don't have any power over other people's feelings.	1.	2.	3.	4.	5.	6.	7.
27.	I believe that things will work out fine in my life.	1.	2.	3.	4.	5.	6.	7.
28.	It is difficult to form close relationships even with those close to me.	1.	2.	3.	4.	5.	6.	7.
29.	I can adapt to new environments.	1.	2.	3.	4.	5.	6.	7.
30.	Others admire me for being relaxed.	1.	2.	3.	4.	5.	6.	7.

Appendix C2: Afrikaans translation of Appendix C1

INSTRUKSIES:

Beantwoord asb. elk van die onderstaande stellings deur die reaksie te merk wat die meeste ooreenstem met die mate waartoe u saamstem of verskil met daardie stelling. Moenie te lank dink oor die presiese betekenis van elke stelling nie. Daar is geen regte of verkeerde antwoorde nie. Daar is 7 moontlike reaksies op elke stelling wat strek van 'Verskil beslis' (nommer 1) tot 'Stem beslis saam' (nommer 7).

		1……..2……3……4……5……6……7						
		Verskil Beslis						**Stem Beslis Saam**
1.	Dit is vir my maklik om my gevoelens in woorde uit te druk.	1.	2.	3.	4.	5.	6.	7.
2.	Dit is vir my moeilik om dinge uit 'n ander mens se oogpunt te sien.	1.	2.	3.	4.	5.	6.	7.
3.	Ek is 'n hoogs gemotiveerde mens.	1.	2.	3.	4.	5.	6.	7.
4.	Dit is vir my moeilik om my gevoelens te reguleer.	1.	2.	3.	4.	5.	6.	7.
5.	Ek geniet nie die lewe nie.	1.	2.	3.	4.	5.	6.	7.
6.	Ek kan mense maklik hanteer.	1.	2.	3.	4.	5.	6.	7.
7.	Ek verander dikwels van plan.	1.	2.	3.	4.	5.	6.	7.
8.	Ek kan dikwels nie uit pluis wat ek voel nie.	1.	2.	3.	4.	5.	6.	7.
9.	Ek voel dat ek'n hele paar goeie eienskappe het.	1.	2.	3.	4.	5.	6.	7.
10.	Dit is vir my moeilik om vir my regte op te staan.	1.	2.	3.	4.	5.	6.	7.
11.	Ek kan beïnvloed hoe ander mense voel.	1.	2.	3.	4.	5.	6.	7.

Maak gebruik van die volgende respons kategorieë: **1……..2……3……4……5……6……7** **Verskil Beslis** **Stem Beslis Saam**								
12.	Ek het ‘n negatiewe siening oor die meeste dinge.	1.	2.	3.	4.	5.	6.	7.
13.	Mense na aan my kla dikwels dat ek hulle nie reg behandel nie.	1.	2.	3.	4.	5.	6.	7.
14.	Dit is vir my moeilik om my lewe volgens die omstandighede aan te pas.	1.	2.	3.	4.	5.	6.	7.
15.	Ek kan stres hanteer.	1.	2.	3.	4.	5.	6.	7.
16.	Dit is vir my moeilik om aan mense na aan my te wys dat ek omgee.	1.	2.	3.	4.	5.	6.	7.
17.	Ek kan ander mense se gevoelens ervaar.	1.	2.	3.	4.	5.	6.	7.
18.	Dit is vir my moeilik om myself gemotiveerd te hou.	1.	2.	3.	4.	5.	6.	7.
19.	Ek kan my gevoelens beheer wanneer ek wil.	1.	2.	3.	4.	5.	6.	7.
20.	Ek is oor die algemeen tevrede met my lewe.	1.	2.	3.	4.	5.	6.	7.
21.	Ek is ‘n goeie onderhandelaar (ek kan mense oortuig om dinge op my manier te doen).	1.	2.	3.	4.	5.	6.	7.
22.	Ek raak soms betrokke by dinge waaruit ek later wens ek eerder kon kom.	1.	2.	3.	4.	5.	6.	7.
23.	Ek los dikwels dit waarmee ek besig is om eers aan my gevoelens te dink.	1.	2.	3.	4.	5.	6.	7.

Maak gebruik van die volgende respons kategorieë: **1……..2……3……4……5……6……7** **Verskil Beslis** **Stem Beslis Saam**								
24.	Ek het baie persoonlike sterkpunte.	1.	2.	3.	4.	5.	6.	7.
25.	Ek is geneig om terug te tree selfs al weet ek ek is reg.	1.	2.	3.	4.	5.	6.	7.
26.	Ek het geen beheer oor ander mense se gevoelens nie.	1.	2.	3.	4.	5.	6.	7.
27.	Ek glo dat dinge in my lewe reg sal uitwerk.	1.	2.	3.	4.	5.	6.	7.
28.	Dit is moeilik om naby verhoudings te bou selfs met die mense naaste aan my.	1.	2.	3.	4.	5.	6.	7.
29.	Ek kan aanpas by nuwe omgewings en situasies.	1.	2.	3.	4.	5.	6.	7.
30.	Ander mense bewonder my oor ek ontspanne is.	1.	2.	3.	4.	5.	6.	7.

Appendix D1: The Stressful Life Events Screening Questionnaire (SLESQ)

INSTRUCTIONS:

The items listed below refer to events that may have taken place at *any point in your entire life,* including early childhood. **Please mark the applicable response.**

1.	**Have you ever been very ill?**	No	Yes
2.	**Were you ever in a serious accident?**	No	Yes
3.	**Was physical force or a weapon ever used against you in a robbery or mugging?**	No	Yes
4.	**Has a close family member, romantic partner or *very close* friend died as a result of accident, murder, or suicide?**	No	Yes
5.	**When you were a child or more recently, did anyone (parent, other family member, romantic partner, stranger or someone else) ever succeed in *physically forcing* you to do something that you did not want to do?**	No	Yes
6.	**Other than experiences described in item 5, has anyone ever used *physical force or threat* to *TRY* to get you to do something that you did not want to do?**	No	Yes
7.	**When you were a child, did a parent, caregiver or other person ever repeatedly abuse you?**	No	Yes
8.	**Other than the experiences mentioned in item 7, have you ever been kicked, beaten, slapped around or otherwise physically harmed by a romantic partner, date, sibling, family member, stranger or someone else?**	No	Yes
9.	**Other than the experiences mentioned in item 7, have you ever been emotionally abused by a romantic partner, date, sibling, family member, stranger or someone else?**	No	Yes
10.	**Other than the experiences already covered, has anyone ever *threatened* you with a weapon like a knife or gun?**	No	Yes

11.	**Have you ever been present when another person was killed, seriously injured, or sexually or physically attacked?**	No	Yes
12.	**Have you ever been in any other situation where you were seriously injured or your life was in danger (e.g., involved in military combat or living in a war zone)?**	No	Yes
13.	**Have you ever been in any other situation that was very frightening or horrifying that has not been covered above?**	No	Yes
14.	**AS YOU FILLED OUT THIS QUESTIONNAIRE, DID YOU EVER REFER TO THE SAME EVENT MORE THAN ONCE?**	NO	YES
	If yes, please indicate which items refer to the same event. _____		

Appendix D2: Afrikaans translation of Appendix D1

INSTRUKSIES:

Die onderstaande items verwys na gebeurtenisse wat op enige tydstip deur u *hele lewe* mag gebeur het, insluitend u vroeë kinderjare.

1.	**Was u al ooit baie ernstig siek?**	Nee	Ja
2.	**Was u ooit in 'n baie ernstige ongeluk?**	Nee	Ja
3.	**Is fisiese geweld of 'n wapen ooit teen u gebruik in 'n rooftog of 'n straatroof?**	Nee	Ja
4.	**Is daar al ooit 'n naby familielid, romantiese maat of *baie naby* vriend van u dood as gevolg van 'n ongeluk, moord of selfmoord?**	Nee	Ja
5.	**Gedurende u kinderjare of meer onlangs, het enige iemand (ouer, ander familielid, romantiese maat, vreemdeling of iemand anders) dit ooit reggekry om u fisies te forseer om iets te doen wat u nie wou doen nie?**	Nee	Ja
6.	**Behalwe vir ervaringe wat in item 5 beskryf word, het enige iemand al ooit fisiese geweld of dreigemente gebruik om te probeer om u iets te laat doen wat u nie wou doen nie?**	Nee	Ja
7.	**Gedurende u kinderjare, het 'n ouer, versorger, of ander person u ooit herhaaldelik mishandel?**	Nee	Ja
8.	**Behalwe vir die ervarings wat in item 7 genoem is, is u ooit geskop, geslaan, rond geklap of andersins fisies seer gemaak deur 'n romantiese maat, iemand van 'n romantiese afspraak, broer of suster, familielid, vreemdeling of iemand anders?**	Nee	Ja
9.	**Behalwe vir die ervarings wat in item 7 genoem is, is u ooit emosioneel mishandel deur 'n romantiese maat, iemand van 'n romantiese afspraak, broer of suster, familielid, vreemdeling of iemand anders?**	Nee	Ja

10.	**Behalwe vir die ervarings wat reeds na verwys is, het enige iemand u al ooit *gedreig* met 'n wapen soos 'n mes of 'n geweer?**	Nee	Ja
11.	**Was u al ooit teenwoordig terwyl 'n ander person doodgegaan het, ernstig beseer is, of seksueel of fisies aangerand is?**	Nee	Ja
12.	**Was u al ooit in enige ander situasie waar u ernstig beseer is of u lewe in gevaar was (bv. betrokke in millitêre gevegte of in 'n oorlogsone geleef)?**	Nee	Ja
13.	**Was u al ooit in enige ander situasie wat baie skrikwekkend was wat nie in bogenoemde vrae gedek is nie?**	Nee	Ja
14.	**TERWYL U DIE VRAELYS INGEVUL HET, HET U OOIT MEER AS EEN KEER NA DIESELFDE GEBEURTENIS VERWYS?**	NEE	JA
	Indien ja, dui asseblief aan watter items verwys na diesefde gebeurtenis _____		

Appendix E: Data from sequencing reactions

The sequences obtained for the different samples following sequencing is presented below. Full repeats are highlighted in red, whilst half repeats are highlighted in blue. The sample numbers do not all follow one another exactly numerically, because certain samples are heterozygotes not included here. Also, sample 13 and sample 19 had to be excluded from the study due to incomplete questionnaires, and are thus also not included here.

SAMPLE 1: 4.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCA
GTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCG

SAMPLE 2: 4.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCA
GTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCG

SAMPLE 3: 3.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCGAGCGCAAGGCGG
AGGGCCCG

SAMPLE 4: 3.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCGAGCGCAAGGCGG
AGGGCCCG

SAMPLE 6: 4.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCA
GTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCG

SAMPLE 7: 3.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCGAGCGCAAGGCGG
AGGGCCCG

SAMPLE 8: 3.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCGAGCGCAAGGCGG
AGGGCCCG

SAMPLE 9: 5.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACC
AGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCAGT
ACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCG

SAMPLE 10: 4.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCA

GTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCG

SAMPLE 11: 4.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCA
GTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCG

SAMPLE 12: 3.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCGAGCGCAAGGCGG
AGGGCCCG

SAMPLE 14: 4.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCA
GTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCC

SAMPLE 16: 4.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCA
GTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCG

SAMPLE 17: 4.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCA
GTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCG

SAMPLE 21: 4.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCAGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCA
GTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCG

SAMPLE 22: 4.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCA
GTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCG

SAMPLE 23: 4.5 REPEATS

GGCGGCACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCAC
CAGTACCGGCACCGGCACCAGTACCCGCACCAGTACCGGCACCGGCACCAGTACCCGCACCA
GTACCGGCACCGGCACCGAGCGCAAGGCGGAGGGCCCG