A MULTI-GROUP CONFIRMATORY FACTOR ANALYSIS OF THE REACTIVE-PROACTIVE AGGRESSION QUESTIONNAIRE IN A SAMPLE OF YOUNG ADULTS

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ABSTRACT

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Aggression has long-term negative effects on humanity as a whole. Because of this, aggression has become an important topic of study across many disciplines. Originally conceptualized as being either non-impulsive or impulsive, aggression has become dichotomized as being reactive or proactive. Each form of aggression has been linked to a variety of genetic, psychological, physiological, and neurobiological correlates. Because research continues to grow in these fields surrounding proactive and reactive aggression, it is important to make sure that measurement tools are assessing aggression appropriately across a variety of groups. One such tool that has emerged recently is the Reactive-Proactive Aggression Questionnaire (RPQ; Raine et al., 2006). Though a number of studies exist testing the validity of the RPQ in adolescent and child populations. No such studies exist examining the factor structure and measurement invariance of proactive and reactive aggression in young adult male and females from different ethnicities. The current study assesses factor structure and measurement invariance in multiple groups of young North American adults. Results show that a two-factor, proactive-reactive, structure fits the current data overall. However, measurement invariance is not achieved across the majority of these groups. Meaning that interpretation of reactive and proactive scores is not the same across gender and ethnicity. Limitations and future directions are discussed.

KEY WORDS: Aggression, Reactive, Proactive, Neurobiology, Genes, Maoa, 5-ht, Dopamine, Tryptophan, Amygdala, HPA axis, Frontal lobes, Confirmatory factor analysis

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TABLE OF CONTENTS

Page

LIST OF TABLES

CHAPTER I

Introduction

Violent and aggressive acts have devastating and lasting impacts on the socioeconomic order of humanity. In 2014, it was estimated that 5.4 million non-fatal violent crimes occurred in the United States (Langton & Truman, 2014). Direct and indirect costs of violent crimes have been projected to exceed $180 billion/year (McCollister, French, & Fang, 2010). Due to these overwhelmingly negative outcomes, aggressive behaviors are of great interest to scholars across a number of disciplines.

Historically, aggression related traits were dichotomized as either impulsive or non-impulsive (Barratt, Stanford, Dowdy, Liebman, & Kent, 1999).[1] Impulsive aggression was characterized by a variety of neuropsychological correlates such as a lack of strategic processing (Stanford, Greve, & Gerstle, 1997), attentional difficulties, motor and cognitive impulsiveness, increased anger, and feelings of guilt after engaging in violent acts (Barratt et al., 1999). It was suggested that impulsive aggression stems from a dysfunctional stimuli-reward system, such that rewards for aggressive behavior become associated with stimuli that evoke aggressive responses, therefore creating a cycle where specific stimuli intensify aggressive acts (Berkowitz, 1974). Non-impulsive aggression, on the other hand, was associated with lower anxiety in general, dominance over others, and an increased desire for high social status (Barratt et al., 1999). Impulsive and non-impulsive aggression served as a good starting point to understanding the underlying traits of aggression. Recently, however, researchers have moved away from the

[1] Non-impulsive traits have also been referred to as premeditated (Barratt, Stanford, Dowdy, Liebman, & Kent, 1999) but the underlying characteristics are conceptually the same as those related to non-impulsive aggression.

characterization of aggression as being either impulsive or non-impulsive as the result of developing inconsistencies with the operationalization of both the impulsive and non-impulsive facets (Meloy, 1992). These concepts were abandoned for more inclusive constructs, specifically, reactive and proactive aggression. As originally defined by Dodge (1991) and Meloy (1992), reactively aggressive individuals display emotional and uncontrolled violence in response to perceived physical or verbal aggression from others. Proactively aggressive individuals, on the other hand, lack emotion and use purposefully controlled aggressive acts to reach a desired goal (Dodge, 1991; Meloy, 1992). As previously mentioned, reactive aggression is a more encompassing concept than impulsive aggression. Though a core characteristic of reactive aggression is impulsivity (Chase, O'Leary, & Heyman, 2001; Kempes, Matthys, Vries, & Engeland, 2005), empirical research has found a wide range of psychological and physiological correlates to reactive aggression, including attentional difficulties and depression (Vitaro, Gendreau, Tremblay, & Oligny, 1998), high levels of peer victimization (Schwartz et al., 1998), social rejection (Price & Dodge, 1989), higher skin conductance and autonomic arousal (Hubbard et al., 2002), as well as higher levels of self-report anger and low levels of dominance (Chase et al., 2001). Like reactive aggression, proactive aggression is characterized by a series of psychological and physiological correlates such as high levels of dominance and instrumentality (Chase et al., 2001), low autonomic nervous system activity (Hubbard et al., 2002; Kempes et al., 2005), high levels of leadership and peer social status (Price & Dodge, 1989), the expectation of positive outcomes when using aggressive behavior (Schwartz et al., 1998), externalizing problems later in life (Vitaro, Brendgen, & Tremblay, 2002), and lower levels of anxiety (Vitaro et al., 1998).

Evidence shows that reactive and proactive aggression are distinct constructs. Various factor-analytic and meta-analytic studies have shown that both factors (reactive and proactive) are different but can also covary, to a certain degree, within individuals (Polman, Castro, Koops, Boxtel, & Merk, 2007; Poulin & Boivin, 2000; Raine et al., 2006; Salmivalli & Nieminen, 2002). If one were to imagine an aggression spectrum, reactive aggression would exist on one end and proactive aggression would exist on another, with specific characteristics from each overlapping in the middle. Because of the conceptual differences between proactive and reactive aggression, each has been grounded in its own theoretical framework.

Reactive aggression, which comes from the Frustration-Aggression Model (Dollard, Miller, Doob, Mowrer, & Sears, 1939), is speculated to occur when individuals view relatively ambiguous stimuli as hostile, and thus respond in a reactively aggressive manner (Dodge, Lochman, Harnish, Bates, & Pettit, 1997). Support for this comes from a variety of samples and experimental conditions (Atkins, Stoff, Osborne, & Brown, 1993; Crick & Dodge, 1996; Dodge et al., 1997; Hubbard et al., 2002; Quiggle, Garber, Panak, & Dodge, 1992; Scarpa & Raine, 1997). Thus suggesting, to some degree,[2] that reactive aggression is caused by individual perceptions of hostile stimuli.

Proactive aggression, on the other hand, has been framed within Social Learning Theory (Bandura, 1973). The basic idea here is that social information processing patterns lead to a step-wise decision making process that provides a possible means of obtaining a desired goal. Despite empirically driven attempts to test this idea (Atkins et

[2] Hostile attribution bias accounts for reactive aggression when psychological factors are included, as will be discussed in later sections, the specific etiology of reactive aggression is more complex than hostile attribution bias, alone, is able to explain.

al., 1993; Hubbard et al., 2002; Quiggle et al., 1992; Scarpa & Raine, 1997) little evidence has conclusively connected social information processing patterns to the expression of proactive aggression. This is most likely due to the instrumental nature of proactive aggression and the inability for standard social information processing measures to fully capture this nuanced behavior.[3]

Human behavior is dynamic and therefore, at times, can be hard to fully capture and separate. Proactive and reactive aggression are no different in this respect. Though they clearly reflect different behaviors (Polman et al., 2007; Poulin & Boivin, 2000; Raine et al., 2006; Salmivalli & Nieminen, 2002) there are certain characteristics of each aggression factor that correlate highly with the other. When examining the differences between proactive and reactive aggression, Raine and colleagues (2006) found that both forms of aggression were associated with excessive fighting at an early age, as well as stimulant seeking and paranoid ideation throughout adolescence. Similarly, Polman and colleagues (2007) meta-analytic study conclusively determined that proactive and reactive aggression can co-occur within individuals.

Further evidence for the comorbidity of these behaviors can be found in the overlap that characteristic traits of aggression share with various facets of psychopathy (Blair, 1999; Cornell et al., 1996; Frick, Cornell, Barry, Bodin, & Dane, 2003; Frick & Morris, 2004; Loney, Frick, Clements, Ellis, & Kerlin, 2003; Porter & Woodworth, 2006; Yildirim & Derksen, 2013). Specifically, the callous-unemotional (CU) traits that characterize Factor 1 psychopathy have been more strongly related to proactive/instrumental aggression, while reactive/impulsive aggressive behaviors

[3] Similar to reactive aggression, the etiological pathways, to be discussed later, that lead to proactive aggression may explain its development in ways that theory alone cannot.

characterize Factor 2 psychopathic traits (Blair, 1999; Frick et al., 2003; Frick & Morris, 2004; Loney et al., 2003). There is strong evidence to suggest that a core feature of instrumental aggression is callous and unemotional behavioral traits. As previously mentioned, there is little evidence linking proactive aggression to social learning theory, in fact, there is more evidence linking CU traits to proactive aggression than to social learning theory. Because of this, moving forward in the next section, research will focus on the core characteristics of proactive aggression which will often be linked to the CU traits that describe Factor 1 psychopathy.

Because proactive and reactive aggression exist on a spectrum, it is extremely important to have reliable and valid aggression measurement tools. Measurement validity is arguably the focal point of scientific research. A variety of teacher, parental, and self-report rating scales have been created to measure proactive and reactive aggression. One particular rating scale, which has emerged recently, is the Reactive-Proactive Aggression Questionnaire (RPQ; Raine et al., 2006). When developing the RPQ, Raine and colleagues (2006) first tested its measurement validity in a sample of 334 adolescent Caucasian and African-American 16 year old boys. After its development and subsequent release for scientific use, the RPQ was further validated in a variety of populations that included male and female Chinese schoolchildren (Fung, Raine, & Gao, 2009), male and female Italian adolescents (Fossati et al., 2009), and Dutch male and female adolescents (Cima, Raine, Meesters, & Popma, 2013). The studies mentioned show that the RPQ is a reliable and valid measure of aggression in child and adolescent populations across gender and culture. Recently, however, the RPQ has been used more widely across a variety of populations and experimental conditions that extend beyond the samples that

the above studies validated. Specifically, the RPQ has been used to assess proactive and reactive aggression in ethnically diverse, young adult, male and female samples (for examples see: Archer & Thanzami, 2009; Donnellan & Burt, 2015; Gao & Tang, 2013; Goodwin, Sellbom, & Salekin, 2015; Latzman, Vaidya, Clark, & Watson, 2011; Miller, Lynam, & Jones, 2008; Schenk, Fremouw, & Keelan, 2013). This poses a potential limitation because the factor structure and measurement invariance of the RPQ has only been tested in child and adolescent male and female samples with finite cultural differences. The current study seeks to address this limitation by testing the factor structure and measurement invariance of the Reactive-Proactive Aggression Questionnaire in an ethnically diverse sample of young adult males and females from North America.

To provide a basic overview, this paper will continue in three chapters. Chapter II discusses the etiological pathways and development of proactive and reactive aggression. The purpose of this chapter is to provide insight into a variety of genetic and neurobiological substrates that extend beyond theory to explain the onset of both forms of aggression. This chapter also aims to portray just how intricate proactive and reactive aggression are, and why there is a need to ensure that measurement tools, like the RPQ, are interpreted similarly across groups. Chapter III discusses the development of the RPQ and various studies that have validated it. This chapter will discuss limitations and measurement issues that the current study seeks to address. Chapter IV presents the methodology of the study. Participants, measurement, and analytic procedures are discussed within the framework of the provided data. Chapter V discusses the results,

with presentation of figures and tables. Chapter VI provides an overview of the findings with specific areas for future research, etiological meanings, and limitations.

CHAPTER II

The Etiology and Causes of Proactive and Reactive Aggression

Chapter II focuses on the etiological pathways and causes of proactive and reactive aggression. This extends beyond theory by providing specific genetic and neurobiological bases for the development of both forms of aggression. This section begins by examining the genetic heritability of aggression, which leads into a discussion of genetic polymorphisms that have been identified to explain aggression. Genetic effects on neurobiological factors are examined next, with emphasis placed on genetic-neurobiological pathways and gene-environment interactions. Neurobiological structures that potentially mediate the association between genetic variation and aggression subtypes will be included in this section to ensure a thorough review. It should be noted that this chapter provides a historical examination of the genetic and neurobiological factors that have been subject to the most scientific scrutiny. Other genetic and neurobiological explanations for aggression exist, however the topics discussed here are the most prominently researched and empirically sound.

Heritability and Gene Studies

Heritability. Heritability is a term used to describe how much variation in a specific phenotype, within a specific population, is due to genetic variation among individuals in that population. Meta-analysis of total aggression scores has found that genes, shared environmental factors, and non-shared environmental factors account for 44%, 6%, and 50% of the variance in aggression scores, respectively (Rhee & Waldman, 2002).[4] Studies have also shown that genes account for roughly 38%-50% of variation in

[4] This meta-analysis examined the one-factor aggression model rather than the two-factor (proactive-reactive) aggression paradigm.

reactive aggression (Baker, Raine, Liu, & Jacobson, 2008; Laubscher, Odendaal, Schneider, & Spies, 2013; Tuvblad & Baker, 2011)[5]. To the same end, non-shared environmental factors accounted for 40%-64%, while shared environmental factors accounted for 6%-15% of variation in reactive aggression (Baker et al., 2008; Laubscher et al., 2013; Tuvblad & Baker, 2011). When considering proactive aggression, genes account for 12%-50% of the variance, while non-shared environmental factors account for 40%-90% and shared environmental factors account for 6%-15% of the variance (Baker et al., 2008; Laubscher et al., 2013; Tuvblad & Baker, 2011). In short, there appears to be both a genetic and environmental influence on proactive and reactive aggression. However, heritability studies cannot point out which genes, specifically, influence the expression of both factors of aggression.

MAOA. Monoamine Oxidase A (MAOA) is a mitochondrial enzyme that catalyzes the oxidation of amines such as dopamine, norepinephrine, and serotonin. First identified by Sabol and Colleagues (1998), MAOA has been consistently linked to reactive/impulsive aggression. Researchers have found, across a wide range of samples and experimental conditions, that the low expression (L) allele of MAOA (MAOA-L) influences the onset and display of reactive aggression (Buckholtz & Meyer-Lindenberg, 2008; Fan, Fossella, Sommer, Wu, & Posner, 2003; Kuepper, Grant, Wielpuetz, & Hennig, 2013; Meyer-Lindenberg et al., 2006; Passamonti et al., 2006; Reif et al., 2007). Building off these findings, and paired with the knowledge that environmental factors influence reactive aggression (Baker et al., 2008; Laubscher et al., 2013; Tuvblad & Baker, 2011), researchers examined interactions between MAOA-L and various

[5] Of all the studies cited in the section on heritability, only Baker et al. (2008) and Laubscher et al. (2013) utilized the RPQ as a measurement tool in their analysis.

environmental factors. For example, findings suggest that early life trauma interacts with the MAOA-L allele to influence the development of reactive aggression (Frazzetto et al., 2007). This interaction was also observed in males who reported a history of abuse (Huang et al., 2004). The high expression (H) allele of MAOA (MAOA-H) has been found to be associated with reactive aggression in females (Holz et al., 2016), while a gene by environment (G X E) interaction between MAOA-H and childhood physical abuse has predicted the onset of proactive aggression later in life in both males and females (Kolla, Attard, Craig, Blackwood, & Hodgins, 2014). Similarly, individuals carrying the 2 and 3 repeat variant alleles of the MAOA variable number tandem repeat (uVNTR) polymorphism are generally higher in impulsivity, while those carrying the 1 and 4 repeat variant alleles are lower in impulsivity (Manuck, Flory, Ferrell, Mann, & Muldoon, 2000).[6]

In all, MAOA-L seems to be related to reactive/impulsive aggression. Inconsistent evidence exists on the relation of MAOA-H to both reactive and proactive aggression. From a theoretical perspective, it has been proposed that individuals carrying the MAOA-L gene are not only reactively aggressive, but are presenting a hostile-attribution bias to ambiguous stimuli that results in a reactively aggressive response (Dodge, 2006). Currently, this model is purely hypothetical as no direct evidence has examined the relationship between hostile attribution bias, reactive aggression, and the MAOA-L gene. Future studies need to be conducted in order to tease out the exact relationship between these constructs.

[6] Null effects between MAOA and impulsivity have been noted by Skondras and colleagues (2004), but in this specific study, MAOA was measured by its presence in the platelet, which is not the best measure of MAOA.

Serotonin. The serotonergic system is one of the oldest amine systems of the brain. This system includes and consists of anything that pertains to or affects the neurotransmitter 5-HT. The serotonergic system includes a wide variety of transporter and receptor subtypes that innervate on most cortical and subcortical regions of the brain. 5-HT is linked to both reactive aggression (Booij et al., 2010; Coccaro, et al., 1997b; Coccaro, Fanning, Phan, & Lee, 2015; Davidson, Putnam, & Larson, 2000; de Boer & Koolhaas, 2005; Dolan & Anderson, 2003; Frankle et al., 2005; Homberg & Homberg, 2012; Jollant et al., 2007; Reif, et al., 2007; Siever et al., 1999; Soderstrom, Blennow, Manhem, & Forsman, 2001; van de Giessen et al., 2014) and proactive aggression (Davidson et al., 2000; de Boer & Koolhaas, 2005; Dolan & Anderson, 2003; Glenn, 2011; Homberg & Homberg, 2012; Soderstrom et al., 2001). In fact, over the decades that 5-HT has been examined, very few studies have found null effects between 5-HT and aggression (see Marseille, Lee, & Coccaro, 2012).

Genes influencing serotonergic function have long been associated with aggressive behaviors. For example, the 12/12 allele of the serotonergic-transporter variable number tandem repeat (5-HTT VNTR) polymorphism has strongly predicted the onset of reactive aggression (Aluja, Garcia, Blanch, De Lorenzo, & Fibla, 2009; Haberstick, Smolen, & Hewitt, 2006). However, due to 5-HTT VNTR being a relatively new discovery, replication of these findings needs to be conducted in other populations and experimental settings. Like the 12/12 allele of the 5-HTT VNTR polymorphism, the S allele of the serotonin-transporter-linked polymorphic region (5-HTTLPR S) has been related to reactive aggression (Aluja et al., 2009; Cadoret et al., 2003; Haberstick et al., 2006; Retz, Retz-Junginger, Supprian, Thome, & Rösler, 2004), while the L allele (5-

HTTLPR L) has been associated with proactive aggression (Aluja et al., 2009; Brummett, Siegler, Ashley-Koch, & Williams, 2011; Cadoret et al., 2003; Haberstick et al., 2006; McCaffery, Bleil, Pogue-Geile, Ferrell, & Manuck, 2003). From a theoretical standpoint, Sadeh and colleagues (2010) found that low socioeconomic status (SES) individuals homozygote for 5-HTTLPR L were more likely to display callous-unemotional and narcissistic traits that characterize proactive aggression. Keeping within the serotonergic transport system, van de Giessen and colleagues (2014) also found a positive correlation between excessive serotonergic transporter (5-HTT) binding and callous personality traits, thus furthering the evidence for a relationship between the serotonergic transport system, specific gene variants of said system, callous-unemotional traits, and proactive aggression.

Moving away from the serotonergic-transport system and its genetic polymorphisms, genes (HTR1B and HTR1A) coding for serotonergic inhibitory receptors (5-HT1B and 5-HT1A) have also been linked to aggression. Evidence shows that individuals carrying the GG genotype of HRT1A, which increases the expression of 5-HT1A (Benko et al., 2010) and the A-161T locus of HTR1B, which effects transcription of 5-HT1B (Zouk et al., 2007) are more reactively and impulsively aggressive. Similarly, increases in the density of serotonergic excitatory receptors (5-HT2A), which are influenced by the presence of the GC and C genotypes of HTR2A, have been linked to impulsive aggression (Giegling, Hartmann, Möller, & Rujescu, 2006). No research, to the authors knowledge, has yet to examine the relationship between these receptors and proactive aggression.

Though little direct evidence exists, it is worth mentioning that low levels of cerebrospinal fluid (CSF) 5-HIAA, the main metabolite of serotonin, has been linked to the onset and expression of reactive aggression (Coccaro & Lee, 2010; Linnoila et al., 1983), while normal levels of CSF 5-HIAA has been related to proactive aggression (Linnoila et al., 1983). This lends further support for the relationship between serotonin and aggression; however, to draw more conclusions from this more tests of CSF 5-HIAA needs to be conducted.

The serotonergic system, in short, plays a large role in the development and continual expression of reactive and proactive aggression. As mentioned, the serotonergic system innervates on a variety of cortical and subcortical regions of the brain. Later sections will discuss this interactive effect in more depth.

Tryptophan. Tryptophan hydroxylase (TPH) is the principal enzyme in the biosynthesis of serotonin (Zhang, Beaulieu, Sotnikova, Gainetdinov, & Caron, 2004). This enzyme converts L-tryptophan, an essential amino acid, into 5-HTP which is the precursor to the neurotransmitter serotonin. It has been found that carriers of the A218 C allele of the isozyme tryptophan hydroxylase (TPH2) were more likely to react impulsively when confronted with an ambiguous stimulus (Mercedes Perez-Rodriguez et al., 2010). This suggests that TPH2 is not only linked to impulsivity but also a hostile attribution style thought process. This idea was tested more thoroughly by Dougherty and colleagues (1999), who found that individuals with depleted levels of tryptophan were more likely to react in a hostile manner to the perceived change in behavior of others. Further support for this relationship comes from Crockett and colleagues (2008) who experimentally depleted the level of TPH in 20 participants. Manipulation of TPH in this

manner temporarily lowered serotonin levels in these individuals who, compared to matched placebo subjects, were more likely to react negatively to perceived unfair treatment by peers. Dougherty and colleagues (1999) and Crockett et al.'s (2008) experiments provide direct evidence of a hostile attribution bias (Crick & Dodge, 1996) in individuals with low levels of tryptophan. Though evidence is limited, it seems likely to suggest that tryptophan individually (Dougherty et al., 1999), or through an interactive effect on serotonin (Crockett et al., 2008), plays a role in reactive aggression and the display of a hostile-attribution bias in response to ambiguous stimuli.

Dopamine. Dopamine (DA) is a neurotransmitter that plays a major role in reward-motivated behavior. A variety of genes influence different dopaminergic transporter and receptor subtypes. The dopamine transporter (DAT1) has a rate limiting capacity on DA and is coded for by the gene SLC6A3. The 9 repeat allele of SLC6A3 has been linked to aggression in general (Young et al., 2002) and, more specifically, proactive aggression in violent subjects (Gerra et al., 2005). In contrast, Reif and colleagues (2007), through an examination of a male forensic population, found no association between the 9 repeat SLC6A3 allele and aggression

The genes DRD1, DRD2, DRD3, and DRD4, which code for dopamine receptors, have been suggested to influence aggressive behavior. In one of the earliest studies examining the effect that all four DRD genes had on aggression, Sweet et al. (1998) determined that only the DRD1 G polymorphism was associated with physical aggression. Conversely, Zai and colleagues (2012) found that the DRD2 polymorphism was associated with general aggression in children. Along the same lines, DA was examined within the context of a G X E interaction, where it was found that parental

maternal stress, paired with those presenting the DRD4 genotype, were higher in general aggression (Buchmann et al., 2014).

The final gene associated with aggression through its regulatory effect on DA is Catechol-O-methyl transferase (COMT). The COMT gene catalyzes the degradation of DA from the synaptic cleft. When considering reactive aggression, both the high activity Val158 (Perroud et al., 2010) and Met158 (Rujescu, Giegling, Gietl, Hartmann, & Möller, 2003) alleles have been associated with this form of aggression in adult suicide attempters. On the other hand, this same functional polymorphism of COMT has been associated with proactive aggression in schizophrenics (Tosato et al., 2011). Similarly, the COMT single nucleotide polymorphisms rs6269 and rs4818 have been linked to aggression in children, specifically in reference to callous-unemotional traits.

To summarize, evidence is somewhat mixed when considering genes that have a regulatory effect of dopamine and subsequent influence on aggression. There is evidence to suggest that dopamine plays a role in both proactive and reactive aggression, however, future research needs to focus on how exactly the same gene (such as COMT) can produce both proactive and reactive aggression. This could be due to the nuanced nature of aggression, but it could also be caused by the influence that MAOA has on DA. As previously mentioned, MAOA catabolizes various monoamines, including dopamine. Because of this, more research needs to examine whether aggression related traits are a direct effect of DA, or rather dysfunctionality within MAOA.

Hormones. There are a wide variety of hormones, but those most consistently studied in relation to proactive and reactive aggression are testosterone and cortisol. Results from the most conclusive studies of these hormones are mixed. For example,

some have found decreased cortisol to be related to reactive aggression (Cima, Smeets, & Jelicic, 2008; Feilhauer, Cima, Korebrits, & Nicolson, 2013; Poustka et al., 2010; Stadler et al., 2011)[7]. Others have found the opposite, suggesting that high levels of cortisol predict the expression of reactive aggression (Bokhoven et al., 2004; Denson, Mehta, & Ho Tan, 2013), especially in females displaying a hostile attribution bias in response to high and low levels of provocation (Böhnke, Bertsch, Kruk, Richter, & Naumann, 2010; Kobak, Zajac, & Levine, 2009). Still others propose that testosterone moderates cortisol (Popma et al., 2007) thus creating an interactive effect with 5-HT (Kuepper et al., 2010) which leads to the expression of reactive aggression. Taken together, the results of hormone studies and reactive aggression are not very consistent.

When considering proactive aggression, results of studies are less mixed which could be due to the fact that fewer studies have examined the effect that hormones have on proactive aggression (Yildirim & Derksen, 2013). Of the two studies that tested the relationship between hormones and traits leading to aggression, it was found that low cortisol levels in children (McBurnett, Lahey, Rathouz, & Loeber, 2000) and adolescents (Loney, Butler, Lima, Counts, & Eckel, 2006) were associated with callous-unemotional traits, which is a precursor to proactive aggression. This relationship was observed for cortisol only, not testosterone.

Neurobiology

HPA Axis. The hypothalamic-pituitary-adrenal (HPA) axis refers to a set of interconnected structures that interact and provide feedback among the three endocrine glands: the hypothalamus, the pituitary gland, and the adrenal glands. The HPA axis

[7] Of all the studies cited in the section on hormones, only Feilhauer et al. (2013) used the RPQ as a measurement tool for their analysis.

constitutes a major part of the neuroendocrine system which controls, among other things, mood, emotions, and stress responsivity (Gotlib, Joormann, Minor, & Hallmayer, 2008). Due to its effect on stress-responsivity, the HPA axis has been linked to both forms of aggression (Meyer-Lindenberg et al., 2006).

Proactive forms of aggression have been linked to low (hypo) HPA activity (Dolan, Anderson, & Deakin, 2001; O'Leary, Taylor, & Eckel, 2010; Poustka et al., 2010; Stadler et al., 2011). This is most likely caused by the influence that genes have on the HPA axis which results in the display of CU traits and, ultimately, proactive aggression. Evidence for this comes from a meta-analytic study conducted by Miller et al. (2013), who found a significant association between the 5-HTTLPR genotype, such that the S allele displays dysfunctional cortisol activity which is directly related to HPA axis reactivity. Based on the relationship between cortisol and CU traits, Hawes and colleagues (2009) suggest that HPA axis hypoactivity heightens levels of CU traits. Ultimately, callous-unemotional traits would lead to the expression of proactive aggression as discussed by Frick et al. (2003).

HPA axis hyperreactivity has been linked to reactive and impulsive forms of aggression through the interaction between high cortisol levels and fear induced behavior (Lopez-Duran, Olson, Hajal, Felt, & Vazquez, 2008). Though this study revealed a strong relationship between cortisol, HPA axis reactivity, and reactive aggression, others have suggested that aggression is dependent on the ratio of cortisol and testosterone (Pavlov, Chistiakov, & Chekhonin, 2012). Like proactive aggression, a variety of genes that influence HPA axis activity have been linked to reactive aggression. These include the Arginine-Vasopression receptor gene 1B (AVPR1B; Luppino, Moul, Hawes, Brennan, &

Dadds, 2014), minor allele variants of the cholesterol transporter coding gene ABCG1 (Gietl et al., 2007), and the short GGC repeat allele of the gene that codes for androgen receptors (Rajender et al., 2008). Despite the influence that these genes have on the display of reactive aggression, they have not, as of yet, been linked to HPA axis hyperactivity.

Amygdala. The amygdala is an almond shaped cluster of nuclei located in the temporal lobe. The amygdala regulates emotions, emotional behavior, and motivation. Research has found that amygdalar hyporeactivity predicts proactive aggression (Blair, 2006; Deeley et al., 2006; Pardini, Raine, Erickson, & Loeber, 2014). Basically, this means that the less sensitive an amygdalar response is to stimuli (i.e. amygdalar underactivity), such as fear or anger in others faces, the more likely one is to act in a proactively aggressive manner. Research examining genetic effects on the amygdala, and the eventual display of proactive aggression, has supported the above interaction. Specifically, Hariri and colleagues (2002) found that the L allele of the 5-HTTLPR polymorphism was associated with amygdalar hypoactivity. This supports the hypothesized interaction between genetic influences on amygdalar hypoactivity and the development of callous-unemotional traits (Blair, 2006) which leads to the onset and display of proactive aggression (Frick et al., 2003). This interaction is further supported by Kiehl and colleagues (2001) and Marsh et al. (2008) who found that adolescents and adults displaying CU traits had reduced amygdala activity when presented with fearful stimuli.

Amygdalar hyperreactivity, on the other hand, has been linked to reactive aggression (Blair, 2006; Bobes et al., 2013; Carré, Fisher, Manuck, & Hariri, 2012;

Coccaro, McCloskey, Fitzgerald, & Phan, 2007; Montoya, Terburg, Bos, & Honk, 2011)[8]. In other words, individuals with an over active amygdala are more likely to respond reactively/impulsively to threat stimuli. The MAOA-L allele has been associated with amygdalar hyperactivity and the presentation of reactively aggressive traits in response to emotionally charged ques in multiple samples (Buckholtz & Meyer-Lindenberg, 2008; Meyer-Lindenberg et al., 2006) and adults specifically (Sebastian et al., 2010).

Frontal Lobe. The frontal lobe (forebrain) is one of the four major lobes of the cerebral cortex. It is made up of a variety of separate structures which dictate executive orders and decision making processes. Certain areas within the frontal lobe have been implicated in the development of aggression. For example, individuals presenting reactive and impulsive aggression have been found with damage, dysfunction, or lesions in the ventral medial prefrontal cortex (vmPFC; Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Bechara, Damasio, & Damasio, 2000; Bechara, Damasio, Damasio, & Lee, 1999; Bechara, Tranel, & Damasio, 2000; Blair, Mitchell, & Blair, 2005; Buchanan et al., 2010; Eslinger, 1998). Similarly, researchers have found decreases in the volume of the left orbitofrontal cortex compared to the right in reactively aggressive individuals (Antonucci et al., 2006; Blair, 2004; Gansler et al., 2009). This suggests that decreased brain matter in the frontal lobe causes reactively aggressive acts. Further support for this comes from Raine and Colleagues (1998) who found decreased volume in both the left and right prefrontal cortex in overtly aggressive murderers. Genes have also been found to influence certain areas of the frontal lobe with regards to reactive aggression.

[8] Of all the studies cited in the section on the amygdala, only Bobes et al (2013) used the RPQ as a measurement tool in their analysis.

Eisenberger and colleagues (2007), for example, found that individuals carrying the MAOA-L allele showed hyperactivity in the dorsal Anterior Cingulate Cortex (dACC) which led to reactively aggressive traits in a social exclusion task.

Research suggests that proactively aggressive individuals display reduced amygdalar activity (Blair, 2004; Raine et al., 1998) and normal prefrontal activity (Raine et al., 1998) when compared to controls. Further assessment for the effect between genes and neurobiological substrates of proactive aggression needs to be examined before making any generalizations beyond implications of the above mentioned brain imaging studies.

The most salient idea is a full consideration of genetics, brain structures, and environmental factors, though few studies examine the interactive effect between all of these. Buckholtz and Meyer-Lindenberg (2008) found that carriers of the MAOA-L genotype exhibited excessive brain serotonin which led to amygdalar hyperreactivity and dysfunction in the medial prefrontal cortex and, ultimately, reactive aggression. Similarly, Coccaro and Colleagues (2007) found that amygdalar dysfunction and decreased orbiotofrontal volume predicted the onset of reactive aggression. When considering proactive aggression, Finger and colleagues (2008) found that children with CU traits displayed dysfunctional activation of the vmPFC during a role reversal task. Based on findings from Marsh and colleagues (2008) study, it seems that individuals with CU traits demonstrate reduced functional connectivity between the amygdala and vmPFC during a fear recognition task. Though the results of these studies are fairly conclusive, being the only studies of their kind, replication and extension needs to be done to fully

examine the interactive effect between various genes, brain structures, and environmental catalysts.

Collectively, these results show overlapping but distinguishable pathways for proactive and reactive aggression with clear evidence for genetic influences on neurobiological substrates. Despite the general consensus on research presented in this review, there is still some disagreement regarding the exact pathways that lead to the expression of both forms of aggression. Some of the ambiguity in this literature may stem from issues with the measurement of reactive and proactive aggression. Factor structure and measurement invariance of aggression measures, like the RPQ, have not been conclusively demonstrated across different groups. Some researchers continue to questions whether these measures generalize to adults, women, and different ethnic/racial groups. This is the current question that the current study seeks to address.

CHAPTER III

Current Study

Chapter III discusses the development and validation of the Reactive-Proactive Aggression Questionnaire (Raine et al., 2006). As previously mentioned, the RPQ was originally developed to capture the underlying traits of proactive and reactive aggression in an adolescent sample. More recently, the RPQ has been extended to a variety of populations, including young adults, males and females, and multiple ethnicities. This chapter will focus on the specific limitations in current validation studies of the RPQ and will end with an overview of the current research goals, questions, and analytic procedure.

Development and Validation of the RPQ

Raine and colleagues (2006) created the Reactive-Proactive Aggression Questionnaire to address the lack of time efficient self-report measures that examined the differential constructs of proactive and reactive aggression. Development of the RPQ began after the collection of psychosocial and behavioral measures of 503 schoolboys (age 7). Once the participants reached age 16, they were given the first version of the RPQ, which included 26 questions. From there, exploratory factor analysis led to the deletion of three items, due to low endorsement frequencies and item-total correlations, and the final version of the RPQ, which is the 23 item scale used today. Confirmatory factor analysis of the RPQ (reported in Raine et al., 2006) yields a two-factor model of aggression that fits well to the data, however, these results are not without their limitations. As Raine and colleagues (2006) note, their sample was constricted to only male adolescents who were categorized as either Caucasian or African-American. These

sample limitations need to be addressed for three reasons. First, research shows that aggression is expressed differently depending on gender. Various studies suggest that males and females express reactive and proactive aggression differently. Marsee and Frick (2007), for example, found that females did not display a hostile attribution bias when engaging in reactive aggression. Also, female participants were more relationally proactively and reactively aggressive as opposed to males, who were more overtly aggressive. These findings are supported by others who consistently report gender differences in the characteristic expression of reactive and proactive aggression (Archer, 2000, 2004; Connor, Steingard, Anderson, & Jr, 2003; Murray-Close, Ostrov, Nelson, Crick, & Coccaro, 2010). Second, the RPQ was developed specifically for adolescents, however, recently it has been extended to college and adult populations. This poses a potential measurement issue because, as Kempes and colleagues (2005) suggest, age differences in the expression of reactive and proactive aggression exist. Specifically, it seems that older individuals are able to better control both forms of aggression in various situations. Third, factor structure and measurement of the RPQ was not examined across ethnicities other than Caucasian and African-American. It is important to take cultural differences into consideration when developing a psychometric measurement tool to ensure that there is no response bias across different cultures.

In order to validate the RPQ more fully, and address the limitations above, researchers have begun to examine the validity of the RPQ across gender, age, and ethnicity. Fossati and colleagues (2009) tested the validity of the RPQ in a large sample of Italian male and female high school students. Findings show that the RPQ was valid and reliable across ethnicity and gender. Along the same line, Fung et al. (2009) assessed

the RPQ in a large sample of male and female East Asian schoolchildren. Similar to Fossati et al.'s (2009) results, the psychometric properties of the RPQ were generalizable across ethnicity and gender. In the most comprehensive study to date, Cima and colleagues (2013) examined the validity of the RPQ in a large sample of Dutch male and females whose age ranged from 6 to 61 years. Results from the analysis demonstrate the continued replicability in the use of the RPQ in a variety of populations and age groups.

Results from these studies support the notion that the Reactive-Proactive Aggression Questionnaire is valid for use in a certain subset of samples. However, there is still some question as to the measurement invariance of the RPQ in multiple ethnic groups. Where Fossati et al. (2009) and Fung et al. (2009) assessed the RPQ in adolescent samples, the RPQ has been widely used to measure aggression in young adult samples (see Archer & Thanzami, 2009; Donnellan & Burt, 2015; Gao & Tang, 2013; Schenk, Fremouw, & Keelan, 2013). Further, differences in the validity of the RPQ across gender has only been assessed in adult Dutch populations (Cima et al., 2013), which could pose a potential problem when generalizing findings to older populations in North America. Finally, Cima et al. (2013), Fossati et al. (2009), and Fung et al. (2009) tested the ethnic differences of the RPQ in samples outside of the United States. Though the results were generalizable and valid in these populations, it speaks nothing towards the ethnic differences in the U.S. where the RPQ has been applied in samples of Caucasian and African Americans.

Purpose and Research Questions

In all, the RPQ is used quite widely but limited evidence exists for its measurement invariance in young adult samples. Similarly, no evidence exists of its

generalizability across race and gender groups in young adults within North America. The current study seeks to test the factor structure and measurement invariance of the RPQ in a diverse sample of young adults, while taking into account limitations and measurement considerations discussed above. The current study poses three research questions. First, will a two-factor (proactive-reactive) structure fit the current sample? Second, will equivalence of the model be achieved across gender? Finally, will equivalence of the model be achieved across ethnicity?

CHAPTER IV

Methods

Participants

The current sample (see Table 1) consisted of 237 male and 248 female (N= 485) students at a Southern University. Students who participated in this study were enrolled in a variety of undergraduate criminal justice courses. Mean age of participants was 21 years and ethnic differences were broken down as: 17.3% African American, 45.3% Caucasian, 27.6% Hispanic, 2.9% Asian, and 1.3% other. Analytic groups consisted of gender, with females as the reference group (mean male= .49, SD= .500) and ethnicity, with Caucasians as the reference group (mean Hispanic= .28, SD= .500; mean Other= .26, SD= .300). Due to sample size issues, African-Americans, Asians, and other ethnicities were combined into one group labeled "Other."

Measures

The Reactive-Proactive Aggression Questionnaire (RPQ; Raine et al., 2006; see Appendix A) is a 23 item, Likert-type scale that measures the two factor model of aggression, with 11 questions examining reactive aggression and 12 questions assessing proactive aggression. Questions ask participants "how often" they have done something, with response categories ranging from 0 (never), 1 (sometimes), or 2 (often). Example questions from the proactive side include "vandalized something for fun," and "yelled at others so they would do things for you." Example questions from the reactive portion include "reacted angrily when provoked by others," and "damaged things because you felt mad."

Analytic Procedure

The current study aims to test the factor structure of the RPQ across gender and ethnic groups. To begin with, total aggression (one-factor) scores were compared against proactive and reactive (two-factor) scores to ensure that a two-factor model fit the data better overall. Next, in order to make comparisons in regards to the factor structure across groups, a multi-group confirmatory factor analysis (MGCFA) was conducted. This type of analysis relies on tests of measurement invariance to examine whether survey items measure the same things across different groups (Cheung & Rensvold, 2002). Full model and multi-group analyses were conducted using the Robust Weighted Least Squares (WLSMV) estimator because it is robust to normality issues and small sample size.

In Mplus version 7.11 (Muthén & Muthén, 2012), MGCFA measurement invariance of ordered categorical data is tested by examining the difference between a sequence of increasingly restrictive models nested together (Brown, 2015). These models differ with regards to the constraints that are placed on factor loadings, thresholds, or both. Factor loadings illustrate the association between latent and observed variables while thresholds indicate the probability of selecting a particular response category. Muthén and Asparouhov (2002) and Brown (2015) recommend testing measurement invariance by, first, fitting a baseline (or free) model where factor loadings and thresholds are able to vary freely across groups. Second, testing this free parameter model to a model where factor loadings are constrained equal across groups and then to a model where factor loadings and thresholds are constrained to be equal across groups. The models were then nested within each other and compared using a variety of Goodness-of-Fit indices (see Brown, 2015; Cheung & Rensvold, 2002; Muthén et al., 2007;

Vandenberg & Lance, 2000) including chi-square difference test ($\Delta\chi^2$), Root Mean Square Error of Approximation (RMSEA≤ .05), Comparative Fit Index (CFI≥ .9), and the Tucker-Lewis Index (TLI≥ .9).

Table 1

Demographics

	n	**%**
Male	237	48.9
Female	248	51.1
Caucasian	224	46.2
Hispanic	135	27.8
African American	84	17.3
Asian	14	2.9
Other	6	1.3

CHAPTER V

Results

Confirmatory Factor Analysis: Comparing the One- and Two-Factor Models

In order to determine overall model fit of the current data, a one-factor model was compared to a two-factor model. Goodness-of-fit indices and factor loadings are presented in tables 2 and 3, respectively. As can be seen, neither model fits particularly well to the data. The two-factor model (χ^2= 1219.078, df= 229, CFI= .898, TLI= .887, RMSEA= .094) fits more adequately than the one-factor model (χ^2= 1559.517, df= 230, CFI= .863, TLI= .849, RMSEA= .109), which is indicated by a chi-square difference ($\Delta\chi^2$= 120, df= 1, p< .001). Though the two-factor model fits significantly better, neither model fits particularly well based on Goodness-of-fit indices discussed above, specifically RMSEA, CFI, and TLI.

Table 2

Confirmatory Factor Analysis comparing overall fit of the one- and two-factor models without any questions removed or covariances created

	χ^2	df	RMSEA	CFI	TLI	$\Delta\chi^2$, df, p
one-factor	1559.517	230	.109	.863	.849	
two-factor	1214.078	229	.094	.898	.887	120, 1, p<.001

Table 3

Factor loadings comparing overall fit of the one- and two-factor models without any questions removed or covariances created

	Proactive		**Reactive**	
	One-factor	Two-factor	One-factor	Two-factor
Yelled when annoyed			.332	.373
Angry when provoked			.451	.492
Temper tantrums			.243	.269
Damaged things when mad			.374	.416
Angry when frustrated			.385	.428
Mad when lost game			.898	.911
Angry when threatened			.375	.413
Felt good after yelling or hitting			.483	.544
Angry when lost game			.871	.893
Hit to defend			.410	.451
Angry when teased			.477	.535
Had fights to be on top	.378	.478		
Taken things	.373	.459		
Vandalized for fun	.531	.628		
Had a gang fight to be cool	.568	.629		
Hurt others to win	.583	.689		
Used force to control others	.695	.794		
Threatened someone	.423	.507		
Used force to get money	.840	.882		
Made obscene phone calls	.339	.413		
Carried a weapon to fight	.390	.462		
Had others gang up on someone	.412	.501		
Yelled so others would do something for you	.350	.422		

Poor overall model fit led to the examination of modification indices (MI's) and factor loadings. This analysis led to the removal of reactive items five/seven as well as proactive items one/twelve as well as the covariation of reactive items nine with six, one with two, and one with three. After the removal and covariances of items, one- and two-

factor overall models were compared again. Goodness-of-fit indices and factor loadings are presented in tables 4 and 5. As can be seen, both the one-factor (χ^2= 361.114, df= 149, CFI= .976, TLI= .972, RMSEA= .054) and two-factor (χ^2= 264.476, df= 148, CFI= ..987, TLI= .985, RMSEA= .040) models fit better to the data overall. Chi-square difference test shows that the two-factor model fits the data significantly better than the one-factor model ($\Delta\chi^2$= 30.155, df= 1, p<.001). Because of this, the two-factor model was used for multi-group comparisons.

Table 4

Confirmatory Factor Analysis comparing overall fit of the one- and two-factor models with questions removed and covariances created

	χ^2	**df**	**RMSEA**	**CFI**	**TLI**	**$\Delta\chi^2$, df, p**
one-factor	361.114	149	.054	.976	.972	
two-factor	264.476	148	.040	.987	.985	30.155, 1, p<.001

Table 5

Factor loadings comparing overall fit of the one- and two-factor models with questions removed and covariances created

	Proactive		**Reactive**	
	One-factor	Two-factor	One-factor	Two-factor
Yelled when annoyed			.154	.181
Angry when provoked			.330	.370
Temper tantrums			.228	.254
Damaged things when mad			.458	.508
Mad when lost game			.316	.354
Felt good after yelling or hitting			.514	.585
Angry when lost game			.302	.341
Hit to defend			.434	.483
Angry when teased			.519	.588
continued				
Taken things	.455	.492		
Vandalized for fun	.615	.661		
Had a gang fight to be cool	.639	.660		
Hurt others to win	.662	.709		
Used force to control others	.762	.800		
Threatened someone	.478	.512		
Used force to get money	.883	.893		
Made obscene phone calls	.420	.454		
Carried a weapon to fight	.465	.500		
Had others gang up on someone	.487	.524		

Multi-group Confirmatory Factor Analysis: Comparing Gender and Ethnicity

When considering gender (see Table 6), the proactive-reactive subscales were more adequately fit to females (χ^2= 202.261, df= 148, CFI= .989, TLI= .988, RMSEA= .039) than males (χ^2= 266.945, df= 148, CFI= .959, TLI= .952, RMSEA= .057). Chi-square difference test of measurement invariance did not achieve invariance within gender when comparing free parameters to factor loading invariance ($\Delta\chi^2$= 31.970, df=

15, p< .01) or to factor loading/threshold invariance ($\Delta\chi^2$= 97.508, df= 34, p< .001). The same held true when examining factor loading invariance to factor loading/threshold invariance ($\Delta\chi^2$= 75.343, df= 19, p<.001).

Table 6

MGCFA across gender

	χ^2	df	RMSEA	CFI	TLI	$\Delta\chi^2$, df, p
Females	202.261	148	.039	.989	.988	
Males	266.945	148	.057	.959	.952	
Free Parameters	381.621	296	.035	.988	.986	
Factor loading invariance	408.334	334	.036	.986	.985	
Factor loading/threshold invariance	467.505	330	.041	.981	.980	
Free parameters compared to factor loading invariance						31.970, 15, p< .01
Free parameters compared to factor loading/threshold invariance						87.508, 34, p< .001
Factor loading invariance compared to factor loading/threshold invariance						75.343, 19, p< .001

Table 7 reports results from the MGCFA when examining Caucasians compared to Hispanics. Adequate model fit was achieved for both Caucasians (χ^2= 186.617, df= 148, CFI= .979, TLI= .976, RMSEA= .044) and Hispanics (χ^2= 192.446, df= 148, CFI= .991, TLI= .990, RMSEA= .037). Chi-square difference test of measurement invariance

did achieve invariance between Caucasians and Hispanics when comparing free parameters to factor loading invariance ($\Delta\chi^2$= 23.193, df= 15, p= .08). Measurement invariance, however, was not achieved between free parameters and factor loading/threshold invariance ($\Delta\chi^2$= 52.928, df= 34, p< .05) as well as factor loading invariance compared to factor loading/threshold invariance ($\Delta\chi^2$= 37.719, df= 19, p< .01).

Table 7

MGCFA examining Caucasians and Hispanics

	χ^2	df	RMSEA	CFI	TLI	$\Delta\chi^2$, df, p
Caucasian	186.617	148	.044	.979	.976	
Hispanic	192.446	148	.037	.991	.990	
Free parameters	388.310	296	.042	.986	.984	
Factor loading invariance	405.541	311	.041	.986	.985	
Factor loading/threshold invariance	431.264	330	.041	.985	.985	
Free parameters compared to factor loading invariance						23.193, 15, p= .08
Free parameters compared to factor loading/threshold invariance						52.928, 34, p< .05
Factor loading invariance compared to factor loading/threshold invariance						37.719, 19, p< .01

Model fit for the combined ethnicity of the Other group (see table 8) also adequately fit the data (χ^2= 195.133, df= 148, CFI= .987, TLI= .985, RMSEA= .050).

Compared to Caucasians, chi-square difference test of measurement invariance was achieved when testing free parameters to factor loading invariance ($\Delta\chi^2$= 21.814, df= 15, p= .113). But measurement invariance was not achieved when examining free parameters to factor loading/threshold invariance ($\Delta\chi^2$= 72.776, df= 34, p< .001) as well as factor loading invariance to factor loading/threshold invariance ($\Delta\chi^2$= 59.274, df= 19, p< .001).

Table 8

MGCFA examining Caucasians and the combined "Other" group

	χ^2	**df**	**RMSEA**	**CFI**	**TLI**	**$\Delta\chi^2$, df, p**
Caucasian	186.617	148	.044	.979	.976	
Other	195.133	148	.050	.987	.985	
Free parameters	418.084	296	.049	.985	.983	
Factor loading invariance	429.701	311	.047	.986	.984	
Factor loading/threshold invariance	477.376	330	.051	.982	.982	
Free parameters compared to factor loading invariance						21.814, 15, p= .113
Free parameters compared to factor loading/threshold invariance						72.776, 34, p< .001
Factor loading invariance compared to factor loading/threshold invariance						59.274, 19, p< .001

CHAPTER VI

Discussion

This study highlights the importance of the continuous need to examine measurement differences of survey tools across multiple groups. Though the RPQ has been validated in a variety of samples, the current findings suggest that groups, specifically gender and ethnicity, can differ in their variance of responses to survey questions. With the finding that measurement invariance can differ across groups, based on the constraints placed on different parameters, this study hopes to successfully show that measurement issues should be continuously examined when using survey tools in diverse populations.

The objective of the current study was three-fold. First, the current study sought to determine whether or not a two-factor, proactive and reactive, structure of the RPQ fit the current sample. Second, to test the equivalence of the reactive-proactive model across gender. And third, to test the equivalence of the reactive-proactive model across ethnicity. Results from the analysis suggest that a two-factor model of proactive and reactive aggression fits the current sample significantly better than a one-factor, or general aggression, model. This two-factor model was then used to examine group differences between gender and ethnicity. Findings suggest that the two-factor model fits well for both males and females, though there were significant difference in survey measurement between the two groups. This lack of invariance means that the scores of males and females, on the RPQ, cannot be interpreted the same. Comparison of Caucasians to Hispanics, however, shows measurement invariance only when factor loading constraints are imposed. When stricter constraints are enforced (i.e. factor

loadings and thresholds are constrained) measurement invariance is lost, meaning that scores between these groups cannot be interpreted the same. These results held true for the combined "Other" group compared to Caucasians. It seems that model fit was achieved for the group itself, but a difference existed in survey measurement results compared to Caucasians when parameters were constrained. Future research should examine why, exactly, imposing stricter parameterizations leads to the difference in invariance as well as what questions are conceptualized differently between the various groups.

Findings that the two-factor structure fit the data better than the one-factor model support recent validation studies of the RPQ conducted by Raine et al. (2006), Cima and colleagues (2013), Fung, Raine, and Gao (2009), and Fossatei et al. (2009). Results from this study add to the continuously growing research suggesting that proactive and reactive aggression is a full and more encompassing way to think about aggression than a general model. Results from this study, however, were surprising in regards to the level of modification that needed to be done to the RPQ before the authors were able to achieve significant fit to the data. It appears that none of the other RPQ validation studies had to remove items from the analysis to achieve overall fit. Not only were four items removed from this study, but covariances were created between three items. It is also surprising that the sample size of the current study, N= 485, was larger than that of Raine et al.'s (2006) original study, N= 335, where the RPQ was developed. Though this could be due to the mixed gender/ethnic non-clinical population of the current study, future factor analytic studies of the RPQ should take this into account and, perhaps, consider

examining the structure of the RPQ under a more rigorous analytic procedure, such as Item Response Theory.

The discovery that there were significant differences in measurement across gender is not surprising. It seems that research showing differences in the expression of aggression across gender (Archer, 2000; Connor, Steingard, Anderson, & Melloni Jr, 2003; Marsee & Frick, 2007; Murray-Close, Ostrov, Nelson, Crick, & Coccaro, 2010) can be related to differences in response scores on the RPQ between males and females. More research needs to be conducted to determine why variance existed between these groups, and what questions they differed on, in order to develop a more encompassing measure of aggression. In regards to ethnicity, results do not seem to support previous analysis of the RPQ across culture (Raine et al., 2006; Cima et al., 2013; Fung, Raine, and Gao, 2009; and Fossatei et al., 2009). There does seem to be a significant difference in the RPQ's measurement of proactive and reactive aggression between Caucasians and Hispanics as well as Caucasians and Other groups in young adults. Future research should test this finding more thoroughly as it is important to know that items are measuring the same concepts across a wide variety of ethnic groups.

Limitations

The current study was limited in several ways. First, due to the small sample size it was impossible to test measurement invariance in homogenous subgroups. Specifically, there was no way to examine detailed gender-ethnic interactions that might influence the measurement of proactive and reactive aggression within groups. Future research should try to examine subsets of groups (for example Hispanic males) within the larger group paradigm.

Second, due to sample size issues with ethnic groups, we are still unsure as to the generalizability and measurement invariance between other ethnic groups. As mentioned previously, the analysis compared Caucasians to a combined "Other" group. This group consisted of African-Americans, Asians, and other categories. Though there was a difference in measurement between this group and the Caucasian group, it is nearly impossible to tell whether this is caused by one specific ethnic group, or even if this finding is generalizable beyond this study.

Third, levels of aggression in college populations is lower than forensic populations overall. Because of this, it is difficult to determine if these results are generalizable to samples that are at high risk of displaying both proactive and reactive aggression. It would be interesting if, in the future, one were able to examine the factor structure of the RPQ in both college and forensic populations to determine if the group differences found in this study persist.

Fourth, because sample size was limited, the current study employed the WLSMV estimator. This could cause differences in results when compared to previous studies examining the RPQ. It is important for future research to re-test these findings in a larger sample in order to avoid issues with the estimator.

Conclusion

Results from this study show that the two-factor model describes aggression much better than the one-factor structure. There do seem to be significant differences in the interpretation of the RPQ across gender and ethnic groups in a young adult (non-forensic) population. This is quite important as it shows that the etiology of aggression is being measured differently across groups. When looking back to the etiology and causes of

proactive and reactive aggression, it is important to have measurement tools that consistently measure similar constructs in different groups. Future analyses should be conducted to determine why the RPQ assesses the intricacies of proactive and reactive aggression differently across gender and ethnic groups.

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APPENDIX A

Reactive-Proactive Aggression Questionnaire

There are times when most of us feel angry, or have done things we should not have done. Don't spend a lot of time thinking about the items – just give your first response.

How often have you…

-Yelled at others when they have annoyed you
-Had fights with others to show who was on top
-Reacted angrily when provoked by others
-Taken things from other students
-Had temper tantrums
-Vandalized something for fun
-Damaged things because you felt mad
-Had a gang fight to be cool
-Gotten angry when frustrated
-Hurt others to win a game
-Become angry or mad when you lost a game
-Used physical force to get others to do what you want
-Threatened and bullied someone
-Gotten angry when others threatened you
-Used force to obtain money or things from others
-Damaged things because you felt angry
-Made obscene phone calls for fun
-Felt better after hitting or yelling at someone
-Threatened or forced someone to have sex
-Gotten angry or mad when you lost a game
-Hit others to defend yourself
-Carried a weapon to use in a fight
-Gotten angry or mad or hit others when teased

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