A Multi-Method Assessment of Effortful Self-Regulation in Personality Research: Temperamental, Neuropsychological, & Psychophysiological Concomitants.

Dissertation

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Abstract

The self-regulatory construct known as *effortful control (EC)* has garnered considerable support from childhood psychopathology research (e.g. Muris, de Jong, & Engelen, 2004), which has relied upon multiple methods of data acquisition, including questionnaires and performance-based measures. Corroborative findings have emerged from adult research indicating that deficits in effortful control may serve as a risk factor for the development of anxiety and depression (e.g. Dinovo & Vasey, 2003, 2005), yet adult research exploring this construct has relied almost exclusively on self-reports. An important step in remedying this deficit would be additional validation of existing self-reports of EC. Fortunately, means for assessing a self-regulatory construct like effortful control are plentiful within the extant scientific literature, particularly from research on executive functions: neurological processes that permit self-regulation. Moreover, converging findings from physiological investigations of executive function and the cardiovascular system suggest that the processes underlying self-regulation can be indirectly assessed via measures of heart-rate variability (HRV), since many of the neural structures implicated in executive function also modulate heart rate (Ruiz-Padial et al., 2003).

Using correlation- and regression analyses within an undergraduate student sample, this investigation found that self-reported EC held significant relations with

physiological measurements of heart-rate variability. By contrast, neither self-reported EC nor indices of HRV were related to performance-based measures of executive functioning. Thus, while providing some important support for the validity of self-report instruments used in the extant adult literature exploring EC, not all findings were consistent with expectations. Implications for the construct of EC and future directions for research are discussed.

Dedication

This document is dedicated to the three great loves of my life: my wife, Rebecca, for her infinite love, respect, and support for me throughout this endeavor; my daughter, Charlotte, for her irrepressible spirit and imagination; and my mother for her unwavering belief in me throughout this long journey. Without you, none of this would have been possible.

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Chapter 1: Introduction

Recent decades have born witness to an emergent interest in self-regulatory processes in human personality development (e.g. Carver & Scheier, 1981, 1998; Gollwitzer & Bargh, 1996; Mischel, 1973; Posner & Rothbart, 1998). Although discussion of self-regulation dates back to the psychoanalytic framework of Freud (1940/1949) and even to ancient formulations of personality (Galen, 150 A. D.; see Carver & Scheier, 2004), recent work in the areas of biological, developmental, and personality psychology has greatly elucidated the mechanisms underlying self-regulation and its consequences for both normal and abnormal personality development. Although a comprehensive review of all the pertinent findings and issues (e.g. promotion-focused vs. prevention-focused self-regulation; see Higgins, 1997) within the self-regulatory literature is beyond the scope of the present inquiry (for a review, see Bronson, 2000), an attempt will be made in the current study to integrate some of the key findings from research in the areas of cognition, development, physiology, and personality. Moreover, it is my hope that through the proposed investigation additional insights might be gleaned as to the utility of adopting a self-regulatory construct of some importance in the area of child development, known as *effortful control (EC)*, for use with adults.

Though various definitions of *self-regulation* have been promulgated in the psychological literature, they share much in common. Therefore, for simplicity the present study will adopt the viewpoint espoused by Posner and Rothbart (1998). Namely, that *self-regulation* refers to the control of lower order processes responsible for the planning and execution of behavior by higher order (i.e. executive) processes within the brain. Such executive control includes the modulation of a person's reactivity to environmental stimuli, including emotional responses, and the initiation and inhibition of approach behavior (Rothbart & Rueda, 2005). The present paper will also make a distinction between this more voluntary form of self-regulation and the reactive regulation of behavior, whereby mechanisms governing a person's initial reactions to environmental events regulate behavior automatically, without personal volition (Derryberry & Rothbart, 1997).

In this vein, self-regulation will be treated as a major component of the organization of *temperament*: the seat of biologically based individual differences in persons' reactions to internal and external events that are influenced over time by both genes and the environment (Rothbart & Bates, 1998; for an alternate though not incompatible information-processing perspective on self-regulation, see Hoyle, 2006). Such temperamental dispositions purportedly stem from evolutionarily conserved systems (Strelau, 1983) that are shared by all humans, but differ in terms of both strength and sensitivity between individuals. In other words, dispositions differ in both degree and valence across persons such that one's particular constellation of inborn responses serves as a foundation from which individual differences emerge.

These individual differences in behavior become more and more distinct through transactional developmental processes in which people assimilate information from their environments and adapt their behaviors accordingly and in which environments are modified by or in response to the individual. By extension, no two individuals are identical in terms of *personality*: the complex organization of psychophysical systems within the person that give rise to characteristic patterns of behavior, thoughts, and feelings (Carver & Scheier, 2004). Thus, in addition to reflecting temperamental dispositions, personality has many other facets, including an individual's perceptions of others, self-concepts, attitudes, beliefs, morals, and coping strategies that develop over the lifespan. Accordingly, temperament *is* personality in its most rudimentary form; the individual differences in childhood behavior that one sees before the development of the more cognitive and socialized aspects of personality (Rothbart, Ellis, & Posner, 2004).

In the area of personality research, theorists have long posited that temperament consists of a strong self-regulatory component that is driven by underlying differences in individuals' level of arousal or constitutional differences in the reactivity of affective-motivational systems (Bronson, 2000; for review, see Rothbart et al., 2004). In two of the more influential accounts of temperament (Eysenck, 1967; Gray, 1970), individual differences in both approach and avoidance behavior are believed to be mediated through reactive self-regulatory processes. In Eysenck's (1967) biological trait theory, variations in personality characteristics are purportedly traceable to inherited differences in the human nervous system, giving rise

to three broad dimensions of personality. These biological differences are said to create variations in people's typical levels of physiological arousal and in their sensitivity to stress and other environmental stimulation. The first of these personality dimensions, Introversion-Extraversion (or E), reflects an implicit self-regulatory connection between arousal, on the one hand, and distress and pleasure, on the other. Eysenck postulated that the quintessential introvert has inherited a nervous system that is more sensitive and arousable to environmental stimulation than that inherited by a prototypical extravert. As stimulation increases in quantity, intensity, or duration, an optimum level of stimulation is reached more quickly by the introvert than the extravert, and once this stimulation threshold is surpassed, distress will ensue. Consequently, introverts are believed to enjoy low-intensity pleasures to a greater degree than do extraverts, who are likely to be bored with low levels of stimulation. By implication, then, the introvert will automatically regulate their behavior so as to avoid overstimulation, whereas the extravert will be a sensation seeker.

Not only did Eysenck (1967) posit that there were individual differences in arousal sensitivity, but he also believed that people inherited biological sensitivities to stress that are captured in a second major personality dimension, Emotional Stability-Neuroticism (or N). Seen as orthogonal to the first major dimension (for review, see Rothbart et al., 2004), this second trait dimension contrasted individuals who have inherited nervous systems that are relatively insensitive to stressors, with those who have nervous systems that are highly reactive to stressors. Consequently, persons who score toward the neuroticism side of the continuum will generally be more emotional

than those who are emotionally stable, and adjust their behavior to minimize contact with stressors. Eysenck later added a third dimension, Psychoticism, which reflects a predisposition toward psychological detachment from other people. This third dimension contrasts such habitual responses as being hostile and impulsive, found amongst those high in psychoticism, with the behaviors of those low in psychoticism, who tend to be open and careful (Carver & Scheier, 2004). Therefore, Eysenck's third dimension includes aspects of psychopathy or disinhibition (i.e. Conscientiousness factor of the Big Five Model of Personality; see McCrae & Costa, 1987), and as such is related to the ability to regulate action (Watson, 2000).

Jeffrey Gray (1970) followed Eysenck's general framework, but offered a different explanation of the biological factors underlying behavioral approach and avoidance (for review, see Rothbart et al., 2004). According to Gray (1991), differences among people in introversion-extraversion (E) and emotional stability (N) stem from two related brain systems: the behavioral approach system (BAS) and behavioral inhibition system (BIS). The BAS purportedly consists of brain regions that affect people's sensitivity to rewards and their motivation to seek those rewards (Pickering & Gray, 1999) and, as such, has been called the "go" system because it is responsible for how impulsive or uninhibited a person is. By contrast, the BIS is the "stop" system, in that is responsible for how fearful or inhibited a person is. The BIS purportedly involves brain regions that underlie sensitivity to potential punishment (and nonreward) and the motivation to avoid punishment. Gray also postulated (1981) that when there is a mismatch between expectation and outcome, the so-called 'control

mode' of the BIS comes into play by mentally identifying stimuli to resolve the mismatch and interrupting the current execution of behavioral programs until the mismatch can be resolved. Through its connections with physiological systems underlying arousal, the BIS regulates ongoing behavior by increasing arousal past an optimum level when in the presence of punishment, thereby inhibiting approach to the source of potential punishment. It is important at this point to distinguish between the more anxiety-driven forms of self-regulation carried out by the BIS and more voluntary forms of self-regulation (Derryberry & Rothbart, 1997; Eisenberg, Smith, Sadovsky, & Spinrad, 2004) that will be addressed shortly. Thus, while Gray followed Eysenck's general approach when it came to explaining approach and avoidance, Gray rotated the axes of Eysenck's Extraversion-Neuroticism structure in proposing separate approach and avoidance systems: the former of which ranges from low Extraversion-low Neuroticism to high Extraversion-high Neuroticism (i.e. the BAS), the latter of which ranges from high Extraversion-low Neuroticism to low Extraversion-high Neuroticism (i.e. the BIS; Rothbart et al., 2004). Although Gray's theory has its critics (e.g. Corr, 2002), it is now more widely accepted than Eysenck's framework, primarily because Gray's formulation is consistent with some – but not all (e.g. Demaree, Everhart, Youngstrom, & Harrison, 2005) – contemporary findings from neuroscience research (e.g. Canli, Zhao, Desmond, Kang, Gross, & Gabrieli, 2001).

Notwithstanding the debate over whether Eysenck or Gray's temperamental formulation is correct, any credible foray into the issue of self-regulation and

temperament would look for corroborative evidence in the childhood research literature, since one would expect to see individual differences in temperamental substrates at even an early age. Much of our current understanding of temperament in infancy and childhood is owed to the seminal work of Mary K. Rothbart and colleagues. Through a comprehensive program of research involving both questionnaire (e.g. Rothbart, Ahadi, Hershey, & Fisher, 2001) and performance-based (e.g. Rothbart, 1988) measures, three broad factors of children's temperament have consistently emerged (Rothbart et al., 2004). The first broad factor (Surgency/Extraversion), combines positive emotionality and approach, including activity level, impulsivity, positive anticipation, sensation seeking, and a negative loading for shyness, whereas the second broad factor (Negative Affectivity) is defined by anger/frustration, discomfort, fear, sadness, shyness, and a negative loading for soothability. It is worth noting the congruence between these findings and those derived from studies on adult personality (see Watson, Clark, & Harkness, 1994) showing considerable overlap between positive affectivity (PA) and negative affectivity (NA), on the one hand, and the Surgency/Extraversion and Emotional Stability/Neuroticism factors of the Big Five Model of Personality (McCrae & Costa, 1987), respectively.

By contrast, the third broad factor (Orienting/Regulation) reflects only modest affective content in the form of low-intensity pleasure, but is also defined by attentional focusing, inhibitory control, and perceptual sensitivity (Ahadi, Rothbart, & Ye, 1993). This third dimension has been consistently found (e.g. Rothbart, et al.,

2001) to be orthogonal to, or negatively related to, fearfulness, the form of temperamental self-regulation purportedly carried out by Gray's (1970) BIS. These three factors have been replicated in a longitudinal study of infants combining both parent-report and laboratory measures (Rothbart, Derryberry, & Hershey, 2000) and by factor analytic studies (e.g. Gartstein & Rothbart, 2003) on parent-reported infant temperament. Thus, it would appear that as early as infancy, there are at least three broad temperamental dimensions defined by positive reactivity and approach, negative affectivity, and self-regulation, respectively. However, it is not until 6 or 7 years of age that another form of self-regulation clearly begins to emerge and stabilize: a dimension that Rothbart and colleagues have labeled *effortful control* (Derryberry & Rothbart, 1997; Eisenberg et al., 2004).

Subsumed under the broad self-regulatory construct of effortful control (EC) are processes implicated in the executive control of attention and behavioral responding (Evans & Rothbart, 2007). These processes include the flexible distribution of attention between threatening and rewarding sensory inputs: a capacity known as *attentional control* (Derryberry & Reed, 2002). As elaborated on in the ensuing discussion, this capacity to both focus and shift attention when desired is viewed (Mathews, 2004; Rothbart & Rueda, 2005) as important in the voluntary execution of behavior, since attention to environmental stimuli is essential to the execution of responses to those stimuli (Corbetta & Shulman, 2002; Peake, Hebl, & Mischel, 2002; Posner & Rothbart, 2000; Rothbart & Bates, 2006; Rueda, Posner, & Rothbart, 2004, 2005). The regulation of these behavioral responses can assume two

broad forms (Evans & Rothbart, 2007): (1) performing an action despite reactive tendencies to avoid said action (i.e. *activation control*); and (2) inhibiting reactive behavioral tendencies when they are maladaptive or do not concord with the immediate context (i.e. *inhibitory control*). All three self-regulatory processes have been shown to be distinct (Evans & Rothbart, 2007), yet are interrelated and share common neural circuitry (Banfield, Wyland, Macrae, Münte, & Heatherton, 2004; Bronson, 2000), primarily within the prefrontal cortex (PFC).

Approach and Reactive Self-regulation

Consistent individual differences in approach behavior appear by 6 months of age, including differences in smiling and laughter that are related to short latencies in approaching objects (Rothbart, 1988). Moreover, in the aforementioned longitudinal study by Rothbart, Derryberry, and Hershey (2000), individual differences in approach and positive emotionality in infancy were predictive of approach behavior and emotionality at 7-year follow up. In this study, infants' reactions to nonsocial and social-eliciting stimuli were videotaped, and the smiling and laughter present in these infant responses were coded for duration, intensity, and duration. These measures were then aggregated into measures of positive affect. Infants' latency to grasp low-intensity toys, such as small squeeze toys, were used as an index of approach, whereas children's movement amongst toys distributed throughout a room were treated as an index of activity level. In this study, smiling and laughter in infancy predicted approach tendencies at seven years of age. Moreover, when parents of a subset of the study infants completed the Children's Behavior Questionnaire (CBQ; Rothbart, et al.,

2001) at 7-year follow-up, infant approach at 6, 10, and 13 months of age predicted paternal reports of their child's anger, aggression, approach, impulsivity, and sadness (Rothbart et al., 2004). These findings are consistent with the positive relations between anger and activity level found in infancy (Rothbart, et al., 2001). Fear did not predict later frustration/anger, and was negatively related to approach, impulsivity, and aggression at age 7, which is consistent with the idea (see Rothbart et al., 2004) that fear may be involved in the regulation of these tendencies (Gray & McNaughton, 1996), a point that will be addressed below. Thus, approach tendencies present as early as infancy may be related to both the short-term and long-term expression of negative and positive emotions (Derryberry & Reed, 1994a).

By the last quarter of the first year of life, individual differences in inhibited approach to both unfamiliar and intense stimuli begin to emerge (for review, see Derryberry & Rothbart, 1997). Anxiety and inhibition at 21 months can be predicted by a measure of combined crying and motor reactivity to stimulation taken at 4 months (Calkins, Fox, & Marshall, 1996; Kagan, Snidman, & Arcus, 1992). Longitudinal research suggests considerable stability of anxiety-related inhibition from age 2 to age 8 (Kagan, Reznick, & Snidman, 1988), from 3 years of age to 18 years of age (Caspi & Silva, 1995), and from early adolescence to early adulthood (Gest, 1997). Such findings are consistent with the proposition that approach tendencies are relatively consistent across the lifespan, and concords with evidence gleaned from neuroscience research indicating that anxiety plays an important role in the regulation of approach behavior (Fowles, 1994).

Some theorists (Derryberry & Rothbart, 1997; Fowles, 1994; Gray, 1987) have posited that the neural circuitry related to anxiety may influence appetitive approach behavior via inhibitory connections to structures implicated in approach. Structures related to anxiety are believed by some to be situated primarily within the septohippocampal system and Papez Circuit (i.e. Gray's BIS; Gray 1987, 1991), whereas the neurological substrates implicated in approach include dopaminergic neurons within the brainstem's ventral tegmental area and the nucleus accumbens (i.e. Gray's BAS; Gray 1987, 1991). It has been argued that once the circuitry underlying anxiety has received information from sensory registers and the thalamus indicating the presence of a potential threat, such inhibitory connections allow the anxiety system to suppress approach responses that may place the organism in contact with harmful stimuli (Derryberry & Rothbart, 1997). Anxiety-driven inhibition, thus, has potential implications for behavior both within and beyond the social realm, such as preventing a person from walking into oncoming traffic, or suppressing reward-seeking behaviors considered inappropriate in many social contexts (e.g. interpersonal violence).

By implication, a relative lack of anxiety-driven inhibition may lead a person to respond with too much impulsivity. For instance, a relatively fearless child with strong approach tendencies may initially respond with hope and enthusiasm at the prospect of forcefully procuring candy from its sibling, and act accordingly. However, such impulsive behavior would likely place the child in contact with potential sources of punishment, including reprimands from its parents and reprisals by its sibling. Therefore, while it would be easy for such a person to anticipate the

positive consequences of approach behavior, it would be considerably more difficult for them to anticipate any negative outcomes that might result. By contrast, the child with both strong approach tendencies and anxiety-driven inhibition would presumably be better at anticipating any potential problems that may result from their appetitive behavior, and be able regulate their behavior accordingly (Derryberry & Rothbart, 1997).

While the foregoing discussion highlights the adaptive value of anxiety-driven inhibition, it is also worth noting that anxiety could result in an overregulation of approach motivation. This could be particularly problematic in persons with weak approach tendencies, who either do not anticipate or derive as much pleasure from positive and novel experiences as those with strong approach tendencies. For instance, an adolescent who anticipates the social threats they may encounter from attending a party (i.e. strong fear motivation) may tend to avoid the party and other social encounters, regardless of any potential rewards that may be obtained through behavioral approach. Such avoidance would presumably maintain social anxiety by denying the adolescent opportunities for positive, varied experiences that could help him or her develop the skills necessary for coping with their social fears (Derryberry & Rothbart, 1997). The finding that anxious individuals show enhanced attention to threat cues (e.g. Derryberry & Reed, 1996; Vasey, Daleiden, Williams, & Brown, 1995) is congruent with the idea that high levels of anxiety could promote overregulation of approach and maintain anxiety through the negative reinforcing consequences of operant avoidance (Brown, Chorpita, & Barlow, 1998).

Approach and Effortful Self-Regulation

Although anxiety-driven regulation of behavior can be highly reflexive in that it enhances attention to both anticipated and immediate sources of threat, and inhibits approach accordingly, extreme anxiety may lead to the rigid overcontrol of behavior and its attendant developmental sequelae (Derryberry & Rothbart, 1997; Kremen & Block, 1998). By contrast, an altogether different form of self-regulation that implicates voluntary, executive control processes within the brain has been proposed as a more flexible means for adapting behavior to meet contextual demands. Captured in the broad capacity for "effortful control (EC)", this self-regulatory dimension of temperament is posited to reflect a person's capacity for inhibiting a dominant response so that a subdominant response can be performed (Rothbart & Bates, 1998; Rothbart, Ellis, Rueda, & Posner, 2003, Rothbart & Rueda, 2005, p. 169). In other words, EC is conceptualized as an individual's ability to both temporarily stop performing an action that he/she ordinarily performs or is reactively inclined to execute, and replace that action with a response that is presumably more adaptive. As such, EC allows a person to resist the immediate influence of his/her emotions and either approach feared situations (i.e. activation control) or flexibly resist what he/she is inclined to do (i.e. inhibitory control). Hence, effortful control has been proposed as allowing for the voluntary control of behavior and emotion, including the flexible distribution of attention between threatening and rewarding sensory inputs (i.e. stimuli), and by implication is viewed as offering a more flexible means of self-regulation than that afforded by reactive, anxiety-driven inhibition.

Effortful control has been characterized by some as being "situated at the intersection of the temperament and behavioral regulation literatures (Kochanska, Murray, & Harlan, 2000, p. 220; see Rothbart et al., 2004, p. 362)." Much of the original impetus for research on EC stemmed from the emergence of a factor in parental measures of childhood temperament that captured seemingly voluntary forms of attentional and behavioral regulation (Rothbart & Bates, 1998). Similarly, significant correlations were found among self-report measures of attentional focusing, attentional shifting, and inhibitory control in adults (Derryberry & Rothbart, 1988). Such findings led to the development of experimental tasks testing effortful control under conflict conditions (e.g. Gerardi-Caulton, 2000; Posner & Rothbart, 2000). More specifically, Rothbart and colleagues (2003) devised a task wherein children 2-to-3 years of age were required to resolve a conflict between the identity of an object and its spatial location (for a more detailed account of this study, see Rothbart et al., 2003). In short, using a computerized touch screen, children were asked to press one of two keys that matched the identity of a stimulus presented on the left or right side of the screen. Under such task conditions, there is a reported tendency for persons across the lifespan to press the key located on the same side of the screen as the stimulus itself, even when such a key does not match the identity of said stimulus (Gerardi-Caulton, 2000). Subjects who perform well on this task are apparently able to suppress the conflicting spatial information and thus inhibit the dominant response tendency to respond with the hand corresponding to the location of the stimulus. While anticipatory eye movements toward object locations were present

by 24 months, children could successfully perform the spatial conflict task only by the 30th-month of life. Nonetheless, individual differences on the task emerged between children by 30 months, such that children who performed well on the task were described by their caregivers as more skilled at attentional shifting and focusing, as well as less impulsive, less prone to frustration, and as having lower levels of negative affectivity. Other studies (Gerardi-Caulton, 2000) have demonstrated that performance on this same task improves considerably between 27 and 36 months of age, which is in line with the hypothesis that effortful control is a function of the relatively late-developing "anterior attentional system" responsible for both the regulation of attention (Derryberry & Rothbart, 1997), the conscious control of behavior, and the ability to regulate more reactive motivational functions (Posner & Rothbart, 1992).

Many of the primary brain structures responsible for the executive control of action, emotion, and thought are believed to reside within the prefrontal cortex (PFC), the anterior portion of the frontal lobe, which undergoes considerable maturational processes during early childhood (Banfield, Wyland, Macrae, Münte, & Heatherton, 2004; Bunge, Dudokovic, Thomason, Vaidya, & Gabrieli, 2002). Of particular importance is the ventromedial prefrontal cortex (VPFC), which is believed to be important in emotional processing and the control of behavioral output during social interactions (Dolan, 1999); the anterior cingulate cortex (ACC), a structure connected to the PFC that is purportedly involved in error detection (Miller, 2000), the resolution of conflicts between executive attention and prepotent attentional responding to threat-

relevant stimuli (Mathews, 2004), the translation of intentions into actions (Banfield et al., 2004), and the executive control of attention (Bush, Luu, & Posner, 2000); and the dorsolateral prefrontal cortex (DPC), which is implicated in the ability to compare an achieved outcome with an intended goal (Dimitrov, Granetz, Peterson, Hollnagel, Alexander, & Grafman, 1999). All of these structures develop in both functional and structural complexity across the lifespan, but undergo the most profound changes during early childhood (Bunge, et al., 2002), especially during the second and third years of life, when individual differences in effortful control begin to emerge (Derryberry & Rothbart, 1997; Eisenberg et al., 2004).

Recent analyses (e.g. Kopp, 1992; Bronson, 2000) of the development of self-regulation in childhood provide converging evidence that increased impulse control becomes available during the second year of life, as children attempt to influence both objects and other people (Rothbart et al., 2004). However, self-regulatory skills and patience remain underdeveloped and it is not uncommon for children of this age to respond with anger when expectations are not met. Nonetheless, by 3-to-4 years of age, more advanced self-control beings to emerge as children are able to comply with caregiver requests and maintain self-control when caregivers are absent (Kopp, 1992). Some researchers have argued that these and other developmental changes during this period are directly related to the development of executive attention and effortful control (Rueda, Posner, & Rothbart, 2005). Although the temporal stability of effortful control has received surprisingly little research attention, some theorists (e.g. Derryberry & Rothbart, 1997) cite studies that have found children who are better at

delaying gratification during conflict tasks in preschool to be more attentive and resistant to stress as teenagers (Shoda, Mischel, & Peake, 1990). Additional follow-up studies have found that preschoolers' delay of gratification behavior predicted their goal-setting and self-regulatory abilities when they reached their early-30s (Ayduk, Mendoza-Denton, Mischel, Downey, Peake, & Rodriquez, 2000). What is more, in a longitudinal study by Caspi and Silva (1995), children who were inhibited (as measured by behavioral ratings) at 3 years of age, when differences in EC begin to emerge, scored low on self-report measures of impulsivity, danger seeking, aggression, and interpersonal alienation at age 18.

While the foregoing discussion highlights the regulation of approach behavior, it has been argued that effortful control holds an essential function in the regulation of anxiety itself (Derryberry & Rothbart, 1997; Eisenberg et al., 2004). This is attributable, in large part, to EC's role in the executive control of finite information processing resources essential to the regulation of behavior (MacCoon, Wallace, & Newman, 2004). In cases where a person has strong anxiety-driven inhibition and a low capacity for the effortful control of action, attention will operate under predominantly reactive control and favor negative informational input, especially in situations where threat is anticipated. For instance, if an anxious person narrowly focuses finite attentional resources on threatening stimuli within the environment, feelings of anxiety are likely to occur and increase in intensity, thereby limiting his/her capacity to devote attention to information relevant to safety and relief. By implication, such individuals will be less able to flexibly shift attention between

sources of threat, relieving inputs, and information relevant to coping within their cognitive representations, hindering their capacity to confront the external stressor effectively. By contrast, anxious individuals with high effortful control would be better able to disengage attention from both environmental threats and internal feelings of anxiety (i.e. attentional control), allowing them to deal with the stressor effectively (Derryberry & Reed, 2002; Derryberry & Rothbart, 1997; Lonigan & Vasey, 2009). This proposition is supported by evidence obtained from research with adults (e.g. Derryberry & Rothbart, 1988), infants (e.g. Rothbart, Ziaie, & O'Boyle, 1992), and children (Wells & Matthews, 1994) indicating that the disengagement of attention from environmental threats attenuates feelings of anxiety and general negative affect.

The Measurement of Attention Biases and Effortful Control in Cognitive Psychology

Experimental paradigms used to test attention biases have their roots in cognitive psychology, where much of the empirical study of mental processes has originated. One method that has been used extensively in the attention bias literature is the *probe detection task* developed by MacLeod and colleagues (1986), where two stimuli are presented simultaneously on a computer display for brief intervals (e.g. 500 msec): stimuli that are either threat-relevant (e.g. the word "death") or emotionally-neutral (e.g. the word "trees") in content. These presentation trials consist of either (a) two emotionally-neutral words, or (b) one threatening word and one neutral word. This design is adopted so that one can test whether a person

preferentially allocates his or her attention to certain stimuli when *multiple* stimuli compete for processing priority, a situation analogous to those encountered by individuals in their daily lives. On many of these trials, the presentation of these two words is followed by dot probes that appear in the location previously occupied by one of the two words. Response times for probe detection are used as a measure of attention to a given word, where quicker (i.e. temporally shorter) response times across trials are taken as evidence of greater attentiveness to specific word content over the other (e.g. greater attention to threatening words than to emotionally-neutral words). Clinically anxious (Taghavi, Neshat-Doorst, Moradi, Yule, & Dalgliesh, 1999; Vasey, Daleiden, Williams, & Brown, 1995) and high test-anxious children (Vasey, El-Hag, & Daleiden, 1996), as well as high-trait anxious children and adolescents (e.g. Schippell, Vasey, Cravens-Brown, & Bretveld, 2003) have been found to be significantly faster at detecting probes in locations previously occupied by threatening words versus neutral words (see Lonigan, Vasey, Phillips, & Hazen, 2004). Similar findings have emerged in the adult literature, where dental phobics (Johnsen, Thayer, Laberg, Wormnes, Radaal, Skaret, Kvale, & Berg, 2003), clinically-anxious (MacLeod & Mathews, 1991; Mogg, Mathews, & Eysenck, 1992), and high trait-anxious adults (e.g. Mogg, Bradley, & Hallowell, 1994) have exhibited attentional biases favoring threat cues. By contrast, low trait-anxious children (e.g. Vasey & Schippell, 2002) and low-anxious adults (e.g. Mogg et al, 1994) seemingly have an attentional bias favoring neutral stimuli over threat-relevant stimuli (see Lonigan et al., 2004).

Notwithstanding these similar findings across the lifespan, conflicting accounts have emerged that suggest elevations in state anxiety are characterized by attentional shifts away from threat cues (e.g. Mogg, Bradley, Williams, & Mathews, 1993; see Lonigan et al., 2004). To account for this incongruity, some researchers have posited (Mathews & MacLeod, 1994; Mogg et al., 1993) that the time interval of stimulus presentation determines whether or not the cue has entered conscious awareness for a sufficient duration, and as such the stimulus presentation interval may engage different stages of attentional processing: stages that fall under the broad rubrics of *reactive* and *effortful* attention. In other words, if cues are presented for a time interval (e.g. 250 msec) insufficient to permit the effortful control of attention, persons who are highly reactive to threat cues (i.e. high NA) will demonstrate the aforementioned attentional bias toward said stimuli. By contrast, when a threat cue is presented for a comparatively longer period that is sufficient to permit the effortful control of attention (e.g. 500 msec), individual differences in attention allocation emerge among those with reactive biases toward threat (i.e. high NA/N), such that persons with a greater capacity for *effortful control (EC)* are better able to override the reactive control of attention and redirect their attention away from threat cues than persons with less EC capacity.

A review of the experimental literature supports this proposition. Whereas studies that present stimulus information for very brief intervals consistently demonstrate the aforementioned bias toward negative stimuli in persons with high NA/N, studies utilizing presentation intervals sufficiently long to engage effortful

processing yield smaller estimates of bias on average, suggesting the operation of differences in the effortful control of attention as a moderator of the association between NA/N and threat bias (Bar-Haim, Lamy, Pergamin, Bakersman-Kranenburg, & van Ijzendoorn, 2007; Lonigan et al., 2004). This conclusion is further bolstered by a study (Derryberry and Reed, 2002), where they measured the attention allocation of high trait anxious (i.e. high NA/N) individuals toward and away punishment cues at different stimulus presentation intervals. When signals of punishment were presented for intervals too brief to permit the effortful control of attention, participants demonstrated delayed disengagement from these punishment cues regardless of their attentional control (i.e. effortful control) capacity. By contrast, high trait anxious (i.e. high NA/N) individuals with concurrently *high* levels of attentional control (AC) were able to disengage their attentional bias from threat and, thereby, resemble low trait anxious subjects in their attention allocation when punishment cues were presented at intervals permitting the effortful control of attention. High trait anxious (i.e. high NA/N) individuals with concurrently *low* levels of AC, on the other hand, still demonstrated delayed disengagement from threat at these longer presentation intervals.

Similarly, in a recent study by Lonigan and Vasey (2009), participants between the ages of 9 and 18 completed a dot probe detection task (PDT) consisting of word-pair trials presented supraliminally (1250 msec). Excluding filler trials where no dot probe appeared subsequent to word-pair presentation, half of the testing trials consisted of two words of neutral emotional valence (e.g. “invent”), whereas the

remaining word-pair trials consisted of both threatening (e.g. "murder") and neutral words (i.e. threat-neutral critical trials). On the average, youths with concurrently high levels of NA/N and low EC, in contrast to those with high NA/N and high EC, evidenced significant reaction time biases (i.e. short response latencies) for probes following threatening words in comparison to probes following neutral words. In effect, these results extended previous findings (e.g. Derrryberry & Reed, 2002) showing that individual differences in biases toward threat at stimulus presentation intervals sufficient for effortful attention are a function not only of NA /N or trait anxiety but also of EC. Thus, it would appear that (i.e. high NA/N) persons with reactive biases favoring threatening cues differ amongst themselves in terms of a second, self-regulatory dimension (i.e., EC) that helps override the reactive control of attention.

Temperament and the Development of its Attendant Neural Circuitry

The foregoing discussion highlighted how effortful self-regulation contributes to a number of important developmental processes, including the ability to flexibly adjust approach behaviors and regulate anxiety, both of which are implicated in major theories of personality (Carver & Scheier, 2004). In particular, aspects of the Five-Factor Model of Personality (McCrae and Costa, 1987) implicate these developmental processes. For instance, individual differences in Factor 1 (Extraversion) partly reflects variability in approach behavior by contrasting such personality characteristics as being assertive, self-confident, spontaneous, and socially adaptable with such traits as timidity, diffidence, cautiousness, and retire. Anxiety would also seem to be

implicated in Factor 1, but would also ostensibly underlie individual differences in Factor 4 (Neuroticism) which, itself contrasts being calm, secure, and composed with being anxious, insecure, and excitable (Rothbart, et al., 2000). The regulation of both approach and fear would also seemingly have implications for individual variability in Factor 5 (Openness to Experience) which reflects curiosity, independence, and a preference for variety on the one hand, with lack of interest, conformity, and a preference for routine (Rothbart & Bates, 1998).

Though not the focus of the present study, it is worth devoting some attention to the purported neurobiological substrates of effortful control. A primary goal of temperamental approaches is to understand how the development of specific neural systems is tied to the emergence of major dimensions of personality (Rothbart & Bates, 1998). Although our understanding of how underlying differences in temperament translate into long-term personality differentiations is still in its infancy, a compelling account of how this process might unfold is provided by Derryberry and Rothbart (1997). Central to their account is the notion that underlying differences in affective-motivational systems within the brain progressively shape an individual's personality in ways that are consistent with underlying temperament. Their basic proposition is that cortical synapses progressively stabilize over time as the child's brain develops in response to environmental events (see Derryberry & Rothbart, 1997). As cognitive representations within the cortex progressively develop, neural plasticity allows for extreme diversity in neuronal interconnectivity, such that more active synapses tend to be strengthened and stabilized upon exposure to the

environment, whereas those synapses that are underutilized will regress. Attention to certain types of environmental information will depend upon internal, selective information processes that arise from a child's motivational systems and constrain the impact of environmental events. Thus, the resulting representational networks within the brain are shaped by active processes rather than passive instruction by the environment. Such selective processes help determine which synapses are likely to be stimulated by the environment and subsequently stabilized upon repeated activation.

One form of motivational selection proposed within Derryberry and Rothbart's (1997) account arises from a person's inborn response tendencies, such that individual differences in temperament will expose them to specific types of information. For instance, a fearful child may avoid contact with stimulating experiences and favor more familiar and calm environments. By contrast, a child with strong approach tendencies will seek out novel and stimulating experiences. Moreover, children with different response tendencies will also differ with respect to the types of information they evoke from their environments. Fearful children may be treated in a gentle and protective manner by others, whereas children with strong approach tendencies may elicit intense social stimulation from others. What is more, fearful versus approach-oriented children will differ in their exposure to interoceptive emotional information, such that fearful children will be prone to negative affect and approach-oriented children will tend to experience positive affect. Since underlying differences in temperament will expose persons to different types of information, such selective

processes will give rise to differential content across person's cognitive representations.

A more specific form of selective information processing proposed by Derryberry and Rothbart (1997) involves the regulation of attention by motivational systems. This is rooted in the idea that people with different temperaments will be motivated to attend to different types of information. Citing evidence from both developmental psychology (e.g. Ruff & Rothbart, 1996) and neuroscience (e.g. Singer, 1990), Derryberry and Rothbart (1997) argue that attention plays a significant role in enabling cortical plasticity. By extension, since individuals with different temperaments will selectively attend to different types of information, as a consequence they will selectively store different information within their representational networks. Consistent with this hypothesis, individuals with strong approach tendencies (e.g. neurotic extraverts) are slow to disengage attention from cues signaling the opportunity to gain points during a simple detection response, whereas anxious persons with weak approach tendencies (e.g. neurotic introverts) are slow to disengage attention from cues signaling the possibility of losing points (Derryberry & Reed, 1994b). Moreover, anxious subjects are slow to disengage attention from locations containing negative trait adjectives and shift attention to targets in another location (Derryberry & Reed, 1996), which in concert with findings of negative attentional biases favoring threat in trait anxious and clinical anxious subjects (e.g. Vasey et al., 1995; Wells & Matthews, 1994), bolsters the proposition

that people with different temperaments will preferentially attend to different types of information.

Additional evidence (see Derryberry & Rothbart, 1997) indicates that anxious persons form stronger short-term memory representations for attended negative words (Reed & Derryberry, 1995) and show enhanced recall of negative information (e.g. Eysenck & Byrne, 1994; Wells & Matthews, 1994). Derryberry and Reed (1997) maintain that findings of such attentional effects make adaptive sense, in that motivational systems would be expected to promote processes that facilitate attention to- and storage of information that promotes survival advantage and may prove useful for functioning in the future. By implication, then, they argue that selective motivational biases in attention will progressively shape a person's cognitive representations (i.e. schemas) in ways that reflect underlying temperament and give rise to increased personality differentiation (Derryberry & Rothbart, 1997).

Limitations of Previous Research

Discussions of self-regulation as a key facet of personality are hardly unique to the literature on effortful control, as theorists from various perspectives have wrestled with self-regulation's place within the overall "architecture of personality" (Cervone, 2004). Much of the contemporary discussion on this topic has emphasized the adaptive value of self-regulation in governing human actions, yet debate continues as to whether self-regulation should be viewed as an enduring capacity of personality or as a ever-changing cognitive operation stemming from the dynamic interplay between knowledge structures, cognitive appraisals, and other mental processes (Pervin,

Cervone, & John, 2004). Much of the conceptual and empirical work adhering to the view that self-regulation is a dispositional facet of personality has been carried out by Roy Baumeister, whose extensive program of research has greatly elucidated the importance of self-regulation in governing numerous domains of behavior, including aggression (e.g. DeWall, Baumeister, Stillman, & Gailliot, 2007), impression management (e.g. Vohs, Baumeister, & Ciarocco, 2005), thought suppression (e.g. Gailliot, Schmeichel, & Baumeister, 2006), and sexual (in)fidelity (Baumeister, Catanese, & Vohs, 2001). According to his view, self-regulation relies on a limited resource that can become depleted through intense or prolonged periods of use (Muraven & Baumeister, 2000). As a capacity with strength-like properties, efforts at self-regulation are believed to be prone to failure under conditions of "ego depletion", leading to potentially less-than-optimal outcomes (Baumeister, Heatherton, & Tice, 1994). While an impressive body of evidence has been brought to be bear supporting this self-regulatory strength model (for review, see Baumeister, Gailliot, DeWall, & Oaten, 2006), much of this work has focused on the state-dependent properties of self-regulation and the effects of temporarily depleting self-regulatory resources. Though posited as a personality trait within Baumeister's framework (Schmeichel & Baumeister, 2004), forays into potential self-regulatory differences among individuals are notably lacking, save for examinations (e.g. Tangney, Baumeister, & Boone, 2004) of individual differences in factors believed to interact with ego depletion (e.g. psychological adjustment). An examination of the dimensional features of a self-

regulatory construct like effortful control may lend insight into the purported trait-like properties of self-regulation.

It is worth underscoring, however, that the only studies to date that have been expressly intended to measure individual differences in effortful control with adults (e.g. Skowron & Dendy, 2004; Dinovo & Vasey, 2003, 2005) have relied to a large extent on self-reports. Though potentially fruitful, such approaches do not accord with those promoted by such scholars as Fiske and Campbell (1992), who advocate the use of multiple methods when investigating psychological constructs. Arguably, such monomethodism does not control for method variance wholly attributable to self-reports as an assessment technology, nor does it permit one to disentangle this extraneous variance from that attributable to the constructs of interest. Moreover, the reliance on self-reports is rooted in the presumption that participants are capable of introspectively apprehending the processes that underlie their behaviors and emotions when it is not clear that self-reports accurately describe these processes. To the contrary, some experimental evidence suggests that people are often very inaccurate when asked to introspectively report their patterns of cognition (Nisbett & Wilson, 1977), which raises some poignant concerns about the utility of such measures. Thus, there remains a compelling need to determine whether or not findings from the extant adult effortful control literature are mere artifacts of self-report methodology. A crucial step in this process is validation of the self-report measures of effortful control used to date.

Fortunately, means for assessing self-regulation are plentiful within the extant scientific literature, particularly in the area of research on *executive functions (EFs)*: "…the principle classes of behavior that we use toward ourselves for purposes of self-regulation...(Barkley, 2004; p. 304)." To date, attempts at measuring self-regulation in adults have run the gamut from studies investigating regulatory control of interpersonal functioning (Vohs, Baumeister, & Ciarocco, 2005) to investigations of visual attention control during goal pursuit (Diehl, Semegon, & Schwarzer, 2006). Studies of this nature have utilized not only normative community samples (e.g. Fan, McCandliss, Sommer, Raz, & Posner, 2002), but also persons with Attention-Deficit/Hyperactivity Disorder (e.g. Happe, Booth, Charlton, & Hughes, 2006) and those with neuropsychiatric disorders, including Schizophrenia (e.g. Perry, Potterat, & Braff, 2001; Twamley, Palmer, Jeste, Taylor, & Heaton, 2006). While forays into the self-regulation literature provide countless options for measuring the construct using performance-based tasks, the prospect of choosing any one of these tasks is, nonetheless, quite daunting. Moreover, the utilization of performance based measures that have been adopted in the extant self-regulation literature may ironically prove quite limiting, as they generally tap certain types of self-regulation (e.g. perseveration, emotion regulation) rather than *self-regulation* writ large. Therefore, for purposes of the present study, it would seem that validation of existing measures of a self-regulatory construct like effortful control should, at this stage, entail measuring those cognitive processes (or "classes of behavior") necessary for all manner of self-regulatory action. Provided that the results of this study lend support for the use of

existing self-report inventories of effortful control with adults, subsequent studies could implement any number of the methods described in the self-regulation literature, particularly those utilized in studies investigating effortful control in childhood.

At the heart of effortful control is the ability to override reflexive responses to environmental stimuli or delay a behavior for which reinforcement – either positive or negative – is immediately available. Collectively, these conditioned responses and operants are referred to as *prepotent responses.* These behaviors are highly reactive in nature, are often ingrained after years of use, and stem from the dynamic interplay of biological propensities and environmental shaping. These behaviors comprise a large part of a person's psychological make-up, as inborn temperament interacts with learning in the emergence of personality. With regard to voluntary prepotent responses, such operants can be either positively or negatively reinforced, such that certain responses are geared toward obtaining immediate rewards (i.e. positive reinforcement), or directed at avoiding and/or escaping aversive or undesirable stimuli (i.e. negative reinforcement). Inhibition of a prepotent response – or the interruption of an ongoing prepotent response – involves the evaluation of information from one's immediate context, often with reference to long-term plans and more distal opportunities for reinforcement (i.e. goals). This often requires maintenance of information in consciousness so that both the execution and sequencing of mental operations can be carried out (Baddeley, 1986); hence the self-regulatory importance of both working memory and the shifting of attentional resources (i.e. cognitive flexibility) between mental operations. By extension, some have argued (e.g. Engle,

Conway, Tuholski, & Shisler, 1995) that increases in working memory load (not capacity) bring about concomitant decreases in the ability to engage in response suppression. The proposition that working memory and inhibitory processes work in tandem to regulate prepotent responses was supported in a recent study by Mitchell, Macrae, and Gilchrist (2001), who found increased oculomotor antisaccadic errors during the inhibitory component of an *n*-back task (see Banfield et al., 2004). Nonetheless, it is worth noting that debate remains as to whether working memory and behavioral inhibition engage the same or different portions of the PFC (see Miller & Cohen, 2001).

The above discussion also underscores the centrality of response inhibition within the effortful control construct. Subsumed under this broad rubric is the capacity to both select and inhibit appropriate subsets of information obtained from the environment. Studies investigating this key component of self-regulation have often focused on the process of thought suppression in both normal and disordered populations. Although problems with thought suppression are hardly unique to clinical syndromes, particular deficits in this arena have been found in persons with lateral PFC lesions (e.g. Shimamura, Jurica, Mangels, Gershberg, & Knight, 1995), who demonstrate considerable problems suppressing previously learned material. Other studies that have utilized measures of inhibitory processes – including the Stroop and antisaccadic tasks – in these populations have generated comparable deficits (see Alvarez & Emory, 2006), bolstering the proposition of some that such inhibitory problems are reflective of a "general dysexecutive deficit" that manifests in

multiple cognitive tasks (Stuss, Floden, Alexander, Levine, & Katz, 2001). Whether such effects would be found in low EC adults without frontal lobe lesions remains to be seen, but the concordance of low EC with childhood syndromes (e.g. attention-deficit/hyperactivity disorder) involving deficits in impulse control lends preliminary support to this hypothesis (Auerbach, Berger, Atzaba-Poria, ArbelÍe, Cypin, Friedman, & Landau, 2008; Martel, Nigg, & Lucas, 2008; Martel, Nigg, & Von Eye, 2009; Muris, Meesters, & Rompelberg, 2007; Wiersema & Roeyers, 2009).

Another potentially worthwhile means of assessing effortful control may be found in psychophysiological explorations of executive function and the cardiovascular system. Particular attention has been directed at fluctuations in the interbeat-intervals (IBI) between heartbeats in human subjects, otherwise known as heart rate variability (HRV). Measurements of HRV have been taken in several studies involving cognitive tasks that require utilization of executive processes. Among such executive processes are those that engage working memory, as well as selective and sustained attention. In particular, tasks requiring participants to maintain attention on environmental stimuli over extended time periods (i.e. vigilance), or those involving increasing mental workloads, have been used by researchers to assess executive function, since such paradigms are believed to tap limited capacity cognitive processes involved in the execution of behaviors during non-routine tasks (see Hansen, Johnsen, & Thayer, 2003).

An investigation on the relationship between HRV and cognitive processes in healthy normal participants was recently carried out by Hansen and colleagues (2003).

Utilizing both a continuous performance test (CPT), as well as a working memory test (WMT), as measures of executive functioning, the study required that participants correctly identify the presentation of previously shown stimuli and complete a simple addition test. Participants identified as being high in resting HRV via a median split evidenced more correct responses, faster mean reaction times (mRT), and fewer errors on these executive function tests than were subjects identified as having low HRV. Moreover, a study with dental phobics found that those with high resting HRV had shorter response latencies (i.e. RT) during a Stroop task than their low HRV counterparts to color-incongruent words and words related to dental threat (Johnsen et al., 2003). These findings are consistent with those obtained in an earlier study with GAD patients and nonanxious controls, wherein both acceleration in heart rate and failures in cardiac orienting habituation to nonthreatening words were characteristic of those with low resting HRV (Thayer, Friedman, Borkovec, Johnsen, & Molina, 2000). By contrast, participants high in HRV habituated more quickly and had larger cardiac orienting responses to nonthreatening words than their low HRV counterparts. Heart rate variability (HRV) also appears to be associated with a variety of indices related to psychological and physical functioning, such that low HRV is involved in a variety of disease states, including cardiovascular disease (Stein & Kleiger, 1999), depression (Thayer, Smith, Rossy, Sollers, & Friedman, 1998), generalized anxiety disorder (Thayer, Friedman, & Borkovec, 1996), and posttraumatic stress disorder (Cohen, Matar, Kaplan, & Kotler, 1999). Low HRV has also been found to be associated with poor attentional control and emotion regulation (especially anxiety), as well as

behavioral inflexibility (Friedman & Thayer, 1998a, 1998b; for review see Thayer & Friedman, 2004).

As mentioned previously, many of the neural structures believed to be implicated in executive function are thought to reside within the prefrontal cortex (PFC), which itself consists of structures that constitute a portion of what has been coined the *central autonomic network* (CAN; Benarroch, 1997; Bob, Susta, Gregusova, & Jasova, 2009; Thayer & Broschott, 2005; Thayer & Friedman, 2004). Consisting of various subdivisions of the hindbrain (e.g. ventrolateral medulla, pons) and forebrain (cerebral cortex, amygdala, hypothalamus), the CAN is involved in goal-directed behavior and the modulation of autonomic processes (e.g. heart beat, blood pressure) through sympathetic and parasympathetic neuronal innervation of the stellate ganglia and vagus nerve. These latter structures modulate heart rate through cardiac vagal activity and parasympathetic inputs and, in so doing, underlie the variability that one sees in heart rate over time (Ruiz-Padial, Sollers, Vila, & Thayer, 2003). Problems in self-regulation are viewed by some as a failure in the negative feedback functions of inhibitory vagal processes, resulting in positive feedback loops that promote hypervigilance and other deleterious consequences that deplete the processing resources needed to carry out other behavioral and cognitive functions (Thayer & Friedman, 2004). Since many of the neural structures associated with the regulation of attention and emotion are also implicated in the functions of the CAN, it is possible that the processes underlying self-regulation – including effortful control – can be indirectly assessed vis-à-vis measures of HRV.

In light of the above possibilities, the purpose of the present study was twofold. First, I endeavored to determine the extent to which self-report measures of effortful control concord with performance-based measures of executive function in adults. In pursuit of this first aim, efforts were made to determine whether effortful control, as measured by self-report, concords with the executive functions presumed to underlie self-regulation. It was hypothesized that effortful control and its various facets (i.e. activation control, attentional control, and inhibitory control), as measured by the effortful control subscale of the Adult Temperament Questionnaire (ATQ-EC; Rothbart, 2001; Evans & Rothbart, 2007) and the Attentional Control Scale (ACS; Derryberry and Reed, 2002), would have significant relations to measures of executive function, where persons with high levels of EC would perform better on executive function measures than those reporting low levels of EC. Specifically, it was predicted that persons who score low on the ATQ-EC and ACS, relative to those who score high on these self-reports, would demonstrate deficits in various areas of executive function, as evinced by the following: longer response latencies (i.e. lower Interference T-Scores) to color-incongruent words on the Stroop Color Word Interference Test (Stroop, 1935); higher numbers of perseverative errors (i.e. repeating incorrect responses) during sorting rule shifts on the Wisconsin Card Sorting Task (WCST; Heaton, Chelune, Talley, Kay, & Curtiss, 1993); and lower working memory index scores on the Wechsler Memory Scale – 3rd Edition (WMS-III; Wechsler, 1997a). With respect to the Stop-Signal Task (Logan et al., 1997), it was expected that, relative to those self-reporting high EC, persons with low EC would evidence

longer reaction times on stop-trials owing to deficits in response inhibition, as well as longer and more variable reaction times on go-trials owing to deficits in attentional focusing.

A second aim of the proposed study was to determine the degree of concordance between self-report measures of effortful control and a non-invasive physiological measure of executive function, heart rate variability (HRV). It was hypothesized that effortful control and its facets (i.e. activation control, attentional control, and inhibitory control), as measured by self-report (e.g. ATQ-EC, ACS), would have positive relations with HRV, such that persons who score high on EC self-reports would have concurrently high HRV at resting state, as demonstrated by high variability in their interbeat-intervals (IBI). IBI variability was indexed by the time-domain measure of root mean square successive differences (RMSSD) between R-waves, as well as natural-logarithmic transformed heart period variability in high-frequency (0.15 to 0.30 Hz) spectral power. Consistent with the findings of Johnsen and colleagues (2003), those with high resting HRV were expected to demonstrate shorter response latencies (i.e. higher Interference T-Scores) during the Stroop Color Word Interference Test than their low HRV counterparts to color-incongruent words. It was also expected that persons with high HRV, compared to those with low HRV, would have fewer numbers of perseverative errors on the WCST; higher working memory index scores on the WMS-III; and shorter RTs on stop-trials, as well as shorter and less variable RTs on go-trials, on the Stop-Signal Task.

To control for the possibility that EC is merely an indicator of intelligence (a construct that involves executive functions and prior knowledge), the WAIS Sattler-2 Composite (Sattler, 2001) was included as a proxy measure of intelligence in the test-administration battery. My hypothesis was that the WAIS Sattler-2 Composite would not correlate significantly with heart-rate variability, effortful control, or any facet of effortful control (i.e. activation control, attentional control, inhibitory control), since self-regulation is viewed as a capacity independent from intelligence that involves executive functions but not other constructs (e.g. vocabulary, mathematical knowledge) subsumed under the higher-order construct of intelligence (Wechsler, 1997b). It was my expectation that such findings would not only provide evidence-based support for using a self-report measure of effortful control, like the ATQ-EC, in adults, but also that the findings of this study would lend tentative support to the construct validity of effortful control.

Chapter 2: Method

Participants

Data were obtained from introductory psychology students enrolled at The Ohio State University, excluding pilot data from participants that were gathered prior to formal data acquisition. A priori power analyses indicated that we needed to procure data from at least 115 partcipants for a moderate effect size ($d = 0.50$; $r = 0.243$; $r^2 = 0.059$) and adequate power ($1 - \beta = 0.80$) at the 0.05 significance (α) level (Cohen, Cohen, West, & Aiken, 2003). For inclusion in this study, participants had to be 18 years of age or older. No additional exclusion criteria were used.

Procedure

Introductory psychology students were recruited via the Research Experience Program (REP) website, which manages participation in departmental studies for persons receiving credit in partial fulfillment of a course research requirement. Prior to enrollment, a brief description of the study was provided which informed subjects that participation required them to attend one assessment session of approximately two hours duration, during which questionnaires, tests of cognitive ability, and heart rate measurements were to be administered. To facilitate better attendance, an email reminder was sent to participants one day prior to their scheduled appointment.

Upon arriving to their scheduled session, each individual participant provided his or her name to the researcher to ensure that research credit was given. Shortly

thereafter, subjects read and signed a consent form that reiterated participation requirements and confidentiality procedures, and informed them that they had the option to withdraw from the study at any time without penalty. After providing their written consent, participants were instructed in how to put on the HRV recording device (i.e. a POLAR heart-rate monitor) and given privacy so that they could attach it to their person.

The HRV device consisted of a WearLink coded transmitter and chest strap that sends short wave signals to a wristwatch receiver. The chest transmitter strap was to be placed in direct contact with a participant's upper body, such that the WearLink transmitter was situated at the thoracic diaphragm. Sensors on the chest strap were moistened with room-temperature tap water to improve skin conductance. To ensure that the HRV device was properly attached, an individual member of the research staff asked the participant to explain how the HRV device was attached and subsequently activated the POLAR watch receiver to ensure that it was receiving heart-rate measurements from the WearLink transmitter. The POLAR wristwatch was held by the assigned staff member so that he or she could both monitor the functioning of the transmitter on a continuous basis, and press buttons on the watch so that HRV data could be time-stamped at the beginning and end of each assessment phase. The watch receiver was situated within 5 feet of the participant for the entirety of the protocol to facilitate reception of heart-rate signals sent by the WearLink transmitter.

Assessment involved four phases, three of which were completed in a randomized order to control for fatigue and order effects. In all cases, the first phase

was a 10 minute resting period during which participants sat alone in a darkened, sound-attenuated room so that measurements of resting heart-rate could be procured. Participants were instructed to minimize physical movement (e.g. no foot tapping, no rocking in chair) and avoid standing upright during the resting period. Participants were made aware that physical movements could elevate their heart-rate and, thereby, render their heart-rate data invalid. To help further ensure that accurate measurements of resting heart-rate were being taken, this initial assessment phase began no earlier than 10 minutes after the participant arrived. This procedure was implemented so that heart-rate could return to baseline from any elevations that covaried with pre-assessment physical activity (e.g. walking to assessment location).

Following this initial resting period were three assessment phases: (1) questionnaire completion; (2) completion of manually administered executive function measures; and (3) completion of computerized executive function measures. Assessment administration was hierarchically randomized, such that both the order of phase completion, and measure administration within these phases, was randomly determined. Heart-rate measurements were taken continuously throughout completion of all phases, but only HRV data from the initial resting period were utilized in our analyses (note: remaining HRV data were collected for analytic purposes not germane to the present study). Details regarding each of the assessment measures utilized follow.

Self-Report Instruments

Self-report measures were included in a questionnaire packet and arranged in a randomized order. All of these measures have been used extensively in undergraduate samples. A demographic information sheet will also be included as the first document of the questionnaire battery. In light of our use of self-report measures, the Balanced Inventory of Desirable Responding (BIDR) was used as an index of social desirability response bias.

The Adult Temperament Questionnaire – Short Form (ATQ; Evans & Rothbart, 2007; Rothbart, 2001; Rothbart, Ahadi, & Evans, 2000): The ATQ is a 77 item self-report originally adapted from the Physiological Reactions Questionnaire (Derryberry & Rothbart, 1988), a putative measure of temperamental constructs related to affect, arousal, and attention. Items on this measure are rated on a 7-point Likert response scale from 1 (extremely untrue of you) to 7 (extremely true of you), with higher scores indicating greater levels of a corresponding temperamental construct. Each of four temperamental constructs – Negative Affect, Extraversion/Surgency, Orienting Sensitivity, and Effortful Control – is represented by a corresponding subscale on the ATQ. Nineteen of the 77-items make up the Effortful Control subscale (ATQ-EC), which is internally consistent ($\alpha = 0.77$) and itself consists of subscales corresponding to Attentional Control, Inhibitory Control, and Activation Control. Attentional control refers to the ability to voluntarily focus and shift attention, whereas Inhibitory Control reflects the capacity to suppress inhibitory approach behavior. By contrast, the capacity to perform an action, despite prepotent tendencies to avoid it, is captured by Activation Control. The ATQ-EC

holds both inverse (r = -0.38) and positive (r = 0.60) relations with the Neuroticism and Conscientiousness dimensions of the Big Five, respectively (Evans & Rothbart, 2007).

The Attentional Control Scale (ACS; Derryberry & Reed, 2002): The ACS is a 20-item measure that assesses the general capacity for attentional control, a key function of the more broadly defined *effortful control*. Items on the ACS require participants to rate their degree of voluntary attentional control on a 4-point Likert response scale (1=almost never, 2=sometimes, 3=often, 4=always). The scale is internally consistent (alpha = 0.88), and is positively associated with indices of Positive Emotionality (r = 0.40), for instance Extraversion, and is inversely associated with facets of Negative Emotionality (r = - 0.55), such as Trait Anxiety (Derryberry & Reed, 2002). Scores on the ACS have also been shown to be positively correlated with activity in the anterior cingulate cortex (ACC) during the control of attention to threat-relevant stimuli (Mathews, 2004).

The Balanced Inventory of Desirable Responding (BIDR; Paulhus, 1984): The BIDR is a 40-item measure of two constructs: self-deception positivity (the tendency to provide honest yet positively biased self-reports) and impression management (deliberate self-presentation). Each item is stated as a proposition, requiring respondents to indicate on a 7-point Likert response scale their level of agreement with each of the statements. The BIDR is internally consistent (alpha = 0.83) and has adequate test-retest reliability over a 5-week period for the self-deception (r = 0.69) and impression management (r = 0.65) scales, respectively.

Task Completion Questionnaire (TCQ; Dinovo, 2007; not published): The TCQ is a 15-item self-report designed for this study to measure the extent to which participants believed that task performance was affected by factors extraneous to the test measures. From these items one can compute not only a total score, but the items were designed to relate to five types of factors that could potentially impact task performance (i.e. fatigue, distraction, stress, effort, and task attention), and three subscales corresponding to task completion phases (i.e. questionnaires, computerized measures of executive function, and researcher-administered measures of executive function).

Executive Function Measures

Executive function measures were completed in two separate phases: manual test-administration and computerized administration. Manual administration was conducted by the author, who had extensive testing experience with each of these measures. The decision to assign manual test administration to the primary investigator alone was made to both minimize the costs associated with training research personnel in the proper use of these instruments, and to maximize uniformity in both test administration and scoring. The battery of manually-administered tests of executive function included the (1) Stroop Color-Word Interference Test, (2) Letter-Number Sequencing and Spatial Span subtests of the WMS-III (i.e. working memory subscales), and (3) Vocabulary and Matrix Reasoning subtests of the WAIS-III (i.e. Saddler-2 Composite). The administration and scoring of these (and our

computerized) tests were conducted without knowledge of either participant responding to questionnaire items or individuals' resting HRV.

The computerized testing battery consisted of the Wisconsin Card Sorting Task (WCST) and a Stop-Signal Task. While the WCST is often manually administered, a computerized version was chosen due to the primary investigator's limited experience administering this measure and attendant concerns about measurement reliability. Thus, a commercially-available automated, computer-based platform was chosen to help ensure uniformity in test administration and scoring, and minimize the costs associated with training research personnel in the complicated administration of the WCST. By contrast, the Stop-Signal Task can only be administered in a computerized format.

These computerized tests of executive function were completed in a randomized order in a darkened, sound-attenuated room. Participants sat directly facing the computer monitor and viewed stimuli from approximately 3 feet (i.e. 0.9 meters) away. Responses on both tasks were collected via the computer's keyboard while participants viewed stimuli presented on a Dell monitor controlled by a Dell Optiplex GX240 computer.

Computerized Stop-Signal Paradigm (Logan et al., 1997): A measure of inhibitory control, the Stop-Signal Paradigm involves two tasks that are completed concurrently; a *Go Task* where subjects are to discriminate one stimulus (i.e. "X") from another stimulus (i.e. "O"), and a *Stop Task* that is interspersed with go-task trials and accounts for 25% of all trials. Each trial involves the presentation of one

stimulus (an "X" or "O"). On *Go Trials,* participants are required to identify which stimulus is presented by pressing one of two keyboard buttons corresponding to the stimuli themselves. However, on *Stop Trials* a *1000-Hz tone of 100-ms duration* (i.e. the *Stop Signal)* is sounded that indicates that a participant should refrain from pressing the keyboard button that corresponds to the stimulus presented on the computer screen. Thus, the *Stop Task* requires participants to inhibit a *Go Trial* response. Whether or not the participant is able to inhibit a *Go Trial* response depends on a race between the stop task and the go task. Stimuli are presented in eight blocks, each consisting of 32 trials. The delay in sounding the *Stop Signal* following the presentation of the go stimulus varies between trials, and was continuously adjusted throughout the entire test based on the participant's success or failure in inhibiting a response on the previous *Stop Trial.* If a response was successfully inhibited during the Stop Trial, the delay between onset of the go stimulus and the *Stop Signal* was lengthened by 50 ms (i.e., the participant was given less time to inhibit the go response). By contrast, if a response was not successfully inhibited during the previous Stop Trial, the stop-signal delay was shortened by 50 ms (i.e., the participant was given a longer interval in which to inhibit the go response). Thus, the reaction time algorithm adopted for this study (see Logan, Schachar, & Tannock, 1997) adjusted the stop-signal delay based on performance on the preceding Stop Trial (with the exception of the first Stop Trial, on which the Stop-Signal Delay was fixed at 250 ms for every participant). *Stop-Signal Reaction Time* is computed as the difference

between a participant's reaction time (i.e. latency of button pressing) on *Go Trials* and his/her mean *Stop Signal Delay*, and serves as an index of inhibitory control.

Computerized Wisconsin Card Sorting Task (WCST; Heaton, Chelune, Talley, Kay, & Curtiss, 1993): The WCST is widely cited (e.g. Baddeley, 1996; Stuss & Levine, 2002) as the most frequently used measure of executive function and is regarded by many as a key measure in the diagnosis of frontal lobe dysfunction (e.g. Bornstein, 1986; Stuss, Levine, Alexander, Hong, Palumbo, Hamer, Murphy, & Izukawa, 2000). Participants are presented with a series of geometric figures that vary not only by shape, but also by color and number. This task requires participants to sort cards according to a rule that must be deduced from feedback ("right" or "wrong") provided by the test administrator. Cards are to be sorted according to the shape, color, or number of figures presented on each trial, but the sorting rule shifts after correct placements on ten consecutive trials. The sorting rule change can only be gleaned by the change in feedback provided by the test administrator, such that ten consecutive "right" placements followed by a "wrong" placement implicitly indicates a change in the sorting rule. This procedure is repeated until six sorting rule shifts have been achieved or all the trials have been completed. In this study, a 64-trial (i.e. card) computerized version of the WCST was utilized (Psychological Assessment Resources, Inc., 2006). Numerous studies indicate that adults with frontal lobe lesions perform worse on the WCST than healthy controls (e.g. Van Den Broek, Bradshaw, & Szabadi, 1993), although this performance differential has been notably absent in many studies comparing patients with frontal and non-frontal brain lesions (e.g.

Axelrod, Goldman, Heaton, Curtiss, Thompson, Chelune, & Kay, 1996). These latter findings, in concert with neuroimaging studies indicating that WCST performance activates both frontal and non-frontal brain regions (e.g. Bermen, Ostrem, Randolph, Gold, Goldberg, Coppola, Carson, Herscovitch, & Weinberger, 1995; Nagahama, Fukuyama, Yamauchi, Matsuzaki, Konishi, Shibasaki, & Kimura, 1996), would seem to suggest that performance on the WCST is a sensitive, though not specific, indicator of frontal lobe functioning (for review, see Alvarez & Emory, 2006). The number of perseverative errors (i.e. repeating incorrect responses despite feedback) on this task provides an index of inhibitory functioning and cognitive flexibility (Kongs, Thompson, Iverson, & Heaton, R. K., 2000, p. 44), key components of effortful control.

Stroop Color Word Interference Test (Stroop, 1935): One of the most extensively studied measures of selective attention for over 70 years, the Stroop is a classic test of cognition that has generated over 1000 published articles (for review, see MacLeod, 1991; Stuss et al., 2001). Although multiple versions of the Stroop exist, the test often consists of three sets of stimuli: (a) color words (e.g. "RED") printed in black ink; (b) color patches or colored X's; and (c) color words printed in ink colors that are incongruent with the meaning of the word (e.g. the word "GREEN" printed in red ink). The Stroop requires participants to read the actual color words themselves as they are presented in the first set, and subsequently name the colors presented (not the words) in the second and third sets. It is during the latter set, in which color words are printed in incongruous colored ink, that one generally sees a

slowing in reaction time (RT) known as the "Stroop effect". This significant slowing of performance owes itself to the tendency of participants to name the color words themselves rather than the ink color in which the words are printed. Consequently, the Stroop may be a useful measure of response inhibition, a key component of effortful control. The colors are often highly differentiable and the different color words (typically 3 to 5, depending on the test utilized) are usually presented in sets of 100. Persons with frontal lobe lesions typically perform worse on the Stroop than normal, healthy controls (Stuss et al., 2001; Vendrell, Junque, Pujol, Jurado, Molet, & Grafman, 1995), although these effects appear to be limited to the lateral and superior medial areas of the frontal lobes (Alvarez & Emory, 2006). In particular, persons with left frontal lobe lesions usually perform worse on the incongruent color naming portion of the Stroop than persons with lesions in the right frontal, right non-frontal, and left non-frontal regions (Stuss et al., 2001). Though many methods for analyzing reaction time on the Stroop exist (MacLeod, 1991), the index for color-naming interference utilized in this study was a participant's color-word score minus his or her predicted color-word score: a value which is estimated on the basis of a participant's obtained scores on the color and word sections of the test. The difference between these two values is then converted into an Interference T-score, where higher T-scores correspond to less color-word interference (i.e. better response inhibition) than lower T-scores (Golden & Freshwater, 2002).

Matrix Reasoning & Vocabulary subtests of the Wechsler Adult Intelligence Scale – 3rd Edition (WAIS-III; Wechsler, 1997b): Numerous attempts have been made

in the past two decades to develop a valid measure of IQ that can be administered in less time than currently required by standard intelligence tests like the WAIS-III (Axelrod, 2002). Many of these efforts have involved seeing which portions of the WAIS-III correlate most highly with Full Scale IQ and, at present, the Sattler-2 Composite (Sattler, 2001), which combines the WAIS-III Matrix Reasoning and Vocabulary subtests, offers the most time-efficient and valid measure of IQ available. Combined, these two subtests of the WAIS-III hold correlations of 0.89, 0.90, and 0.93 with Verbal IQ, Performance IQ, and Full IQ, respectively (Brooks & Weaver, 2005). The Vocabulary subtest asks that respondents define words, whereas the Matrix Reasoning subtest requires participants to look at incomplete pictures involving abstract patterns and identify which of 5 response options completes the pattern. The Wechsler Adult Intelligence Scale itself is a well validated, standardized assessment device used in medical, psychiatric, and educational settings for the purposes of measuring individual differences in intelligence. The WAIS-III is one of the most widely used instruments for measuring IQ employed by professionals (Brooks & Weaver, 2005; Lange et al., 2006).

Letter-Number Sequencing & Spatial Span subtests of the Wechsler Memory Scale – 3rd Edition (WMS-III; Wechsler, 1997a): These two subtests of the WMS-III form a combined measure of working memory, a key executive function. Letter-Number Sequencing involves the presentation of letters and numbers and requires that respondents repeat the letters and numbers in a different order: the numbers in increasing order, then the letters in alphabetical order. Spatial Span consists of a

series of blocks situated on a flattened board. The test is divided into two parts. First, on each trial the test administrator touches a pattern of blocks that the respondent must replicate. By contrast, during the second part of this test the respondent must touch the blocks in reverse order from the way they were touched by the test administrator. Higher numbers of correctly-responded trials on both parts are believed to be indicative of greater working memory capacity. The Wechsler Memory Scale itself is a well validated, standardized assessment device used in medical, psychiatric, and educational settings for the purposes of measuring individual differences in memory (Lange et al., 2006).

Assessment of Heart Rate Variability (HRV)

Electrocardiographic (ECG) signals were obtained during a 10 minute resting period from a POLAR RS800 Running Computer Wristwatch and WearLink Transmitter (Heart Rate Monitors USA, Inc., 2007). Data obtained from the POLAR watch were downloaded to a computer using an IrDA Interface Port. Using the ECG signal, heart period was defined as the time interval between sequential R-waves, as measured in milliseconds. Per the joint recommendations of the Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology (1996), we adopted the spectral power in high-frequency (0.15 to 0.30 Hz) heart period variability (HF-HPV) as a frequency domain measure, since the vagus is the only autonomic influence known to exist at this high frequency and, thus, modulation of heart rate reflected cardiac vagal activity and parasympathetic input (Ruiz-Padal et al., 2003). Vagally mediated HRV was indexed, in part, using this

frequency-domain measure. Spectral power estimates were natural-log transformed prior to statistical analysis to ameliorate distributional violations. Frequency-domain analyses used an autoregressive statistical method. Resting HRV measurements were analyzed using the KUBIOS HRV Analysis Software (Biosignal Analysis and Medical Imaging Group, 2008).

Resting HRV measurements were taken during a 10 minute resting period where each participant was sequestered individually in a sound-attenuated room. Resting periods began 10 minutes subsequent to each participant's arrival to the study. During this resting period, all participants were instructed to sit silently in a chair and minimize motor movements while resting their heads on a table situated in front of them. Upon completion of the study, heart-rate variability measurements were submitted to KUBIOS, whereby a Hamming window was used to linearly detrend, mean center, and taper the time series. Vagally-mediated (parasympathetic) cardiac control was then measured using both the time-domain measure, square root of mean successive differences (RMMSD) between R-waves, and the frequency-domain measure, natural logarithm of high frequency power (Ln (Resting HF Power). Higher scores on both measures are associated with higher heart rate variability (HRV) in contrast to lower scores.

Data Analytic Strategy

Internal consistencies for the self-report measures of effortful control were computed as Cronbach's alpha (α) coefficients, which effectively function as indices of inter-item response consistency to all items in a test measure (Anastasi & Urbina,

1997, p. 97). Split-half reliabilities were calculated for our performance-based measures of executive function by dividing odd and even numbered test items into separate halves (rather than correlating the first and second halves of the tests) to help minimize differences not only in the nature and difficulty of test items, but also to minimize cumulative effects related to practice, fatigue and other factors the vary from the beginning to the end of test administration. The reliability of HRV was assessed via intraclass correlation coefficients (ICC) computed between the first and last five-minute periods of measurement during our initial resting phase.

To assess the degree of concordance between the three self-regulation measurement modalities, several data analytic procedures were adopted. First, the overall agreement between these measures (i.e. self-report, EF tests, HRV) was computed via Pearson correlation coefficients. Correlations between these measures served as proxy validity coefficients, where statistically significant, high correlations between measures were indicative of a high degree of correspondence. Second, any significant correlations identified between the effortful control self reports (ATQ-EC and ACS) and non-questionnaire measures (i.e. executive function measures, HRV measures) were explored further via multiple linear regression to control for demographic characteristics, intelligence, social desirability response bias (i.e. *self-deception positivity* and *impression management*, as indexed by the BIDR), and factors extraneous to the test measures that may have affected performance (i.e. TCQ Total). Similarly, non-significant correlations relevant to predictions were re-examined via multiple regression to determine whether extraneous variance associated

with task performance (i.e. TCQ Total), socially desirable responding (i.e. BIDR Impression Management, BIDR Self-Deception), or sex-based variable differences (i.e. sex differences in HRV) suppressed zero-order relations.

These multiple linear regressions were conducted in a hierarchical fashion, where a main effect model was first computed, followed by the subsequent addition of covariates in a "Full Model" that included the main effect variable and six covariates. Semi-partial correlation (sr) coefficients were computed for each of the full models, which provided estimates of the linear relationships between two variables after partialling out (or removing) the variance that those two variables share with other covariates included in the regression models (Cohen et al., 2003, pp. 72-73).

In light of the fact that all of the TCQ subscales were intended to measure extraneous factors (e.g. fatigue, inattention) that could have affected task performance, a decision had to be made regarding their inclusion in our regression analyses. This decision was particularly important because one can utilize up to nine separate indices from the TCQ. It was ultimately my view that it was important to limit the number of TCQ variables in our regression equations to minimize the possibility of committing Type I error (see Cohen et al., 2003, pp. 185-187). Additionally, as discussed in the Results section, these subscales lacked adequate distributional properties relative to the TCQ Total. Moreover, the TCQ Total alone arguably provided an adequate basis for assessing the impact of extraneous performance factors, since the eight remaining subscales generally correlated substantially with one another and the TCQ Total evidenced good internal consistency (0.86).

Multiple linear regression analyses were first limited to those correlations which achieved statistical significance (see Tables A.3 – A.6, Appendix A). However, in the likely event that significant sex differences emerged for key study variables (see Table A.1), a second set of regression analyses were conducted for all variable relationships to test for statistical suppression effects for both significant and nonsignficant zero-order correlations. The only relations for which there was no explicit retesting via multiple regressions were those between key study variables and subscales of the BIDR and TCQ, since the latter variables (BIDR Impression Management, BIDR Self-Deception, TCQ Total) were included as covariates in each of the regression analyses.

Continuous measures were standardized and dichotomous predictors were effect coded to reduce multicollinearity (Aiken & West, 1991).

Chapter 3: Results

Preliminary Analyses

Sample Characteristics

Data were initially obtained from 131 introductory psychology students enrolled at The Ohio State University. From this initial sample, data from 7 participants were excluded from analyses owing either to technical errors (e.g. computer failure, test/task administration errors) that occurred during data collection, or insufficient or missing data. The resulting sample consisted of 124 participants, the majority of whom were Caucasian (77.2%). The mean age for the sample was 19.05 years (SD = 1.02) and the composition of our sample consisted of approximately the same number of males (n = 63; 50.8%) and females (n = 61; 49.2%). The final sample (n = 124) met the sample size requirements determined by *a priori* power analyses.

Means and standard deviations for all variables are shown for the full sample and separately for males and females in Table A.1 (see Appendix A [Note: all tables and figures appearing in Appendix A are designated with an 'A' preceding their number]). With the exception of HRV and ACS scores, no sex differences emerged. With regard to ACS scores, females on the average reported lower levels of attentional control than males. In contrast, mean HRV, as indexed by the root mean square of the successive differences between heart beats (rMSSD; time-domain measure), and the natural log of high frequency power (LogHF; frequency-domain measure) was

significantly higher in females than males. This finding is consistent with sex-differences observed in other studies using frequency-domain (Britton, Shipley, Malik, Hnatkova, Hemingway, & Marmot, 2007; Huikuri, Pikkujamsa, Airaksinen, Ikaheimo, Rantala, Kauma, Lilja, & Kesaniemi, 1996; Koskinen, Kähänon, Jula, Laitinin, Keltikangas-Järvinen, Viikari, Välimäki, & Raitakari, 2009; Kuo, Lin, Yang, Li, Chen, & Chou, 1999; Liao, Barnes, Chambless, Simpson, Sorlie, & Heiss, 1995; Zhang, 2007), and both time- and frequency-domain measures (Sinnreich, Kark, Friedlander, 1998; Sztajzel, Jung, & de Luna, 2008) of HRV in adults.

Variable Distribution Diagnostics: Pre-Transformations

Diagnostics were performed on all measures to determine whether distributional problems (e.g. skew, kurtosis) would impede the interpretation of correlation and regression coefficients (see Appendix B for a detailed discussion). The issues of normality and distributional similarity are particularly important within the context of correlation and linear regression (Cohen et al., 2003; Hays, 1994), since the magnitude of the Pearson product moment correlation coefficient (r) can only range from –1.00 to 1.00 when the frequency distributions of two variables (say, X and Y) are identical ($z_x = z_y$). Indeed, while it is widely recognized that true normality is exceedingly rare in behavioral science research (Bradley, 1982; Levine, 2005; Micceri, 1989), extreme deviations from distributional *similarity* between two variables can limit the maximum possible value of r_{xy} to 0.3 or less (Carroll, 1961; Hays, 1994). In light of the multimethodism of this study and the possibility that correlation coefficients between different measures might be attenuated owing to their

differing methods of data acquisition (e.g. self-reports vs. physiological measurements), considerations of statistical normality and distributional similarity are particularly patent.

Methods for evaluating the distributional properties of sample data are numerous and opinions vary widely concerning which method (or combination of methods) is best suited for this purpose (Thode, 2002; interested readers are referred to Appendix B for a detailed discussion of these issues). Whereas some researchers prefer "eyeballing" or visual inspection of order data (e.g. Cleveland, 1984; Orr et al., 1991), others prefer more objective assessments of normality (e.g. Gan & Koehler, 1990; Henderson, 2006) that can range from the examination of either probability-probability (P-P) plots or quantile-quantile (Q-Q) plots, to quantitative indices of kurtosis and skewness and inferential tests of normality, such as the Kolmogorov-Smirnov test (Cabaña, 1996; Drew, Glen, & Leemis, 2000; Massey, 1951; Wilcox, 1997) and its modifications (e.g. Lilliefors modification; Lilliefors, 1967). Still others prefer utilizing multiple criteria to determine the extent to which observed data deviate from a Gaussian distribution (Cohen et al., 2003; D'Agostino et al., 1990; Judd et al., 1995; Wang, Yam, & Zuo, 2004), and it is this multi-criterion approach that was adopted in the present case.

The methods used to assess for problems associated with distributional skewness, kurtosis, and general non-normality included (a) visual inspection of univariate frequency histograms (see Figures B.1 – B.5 in Appendix B [Note: all tables and figures appearing in Appendix B are designated with an 'B' preceding their

number]; (b) examination of probability-probability (P-P) and quantile-quantile (Q-Q) plots (see Figures B.6 – B.15); and (c) the computation of Kolmogorov-Smirnov (Lilliefors modification), as well as skewness and kurtosis, test statistics both before and after variable transformations (refer to Tables B.1 and B.2, respectively).

With a few noteworthy exceptions (e.g. ATQ-EC subscales, Stroop Interference, WMS-Working Memory) nearly all variables in this study evidenced significant signs of statistical non-normality. Of the 18 variables whose distributions deviated from the normal curve, 14 of them displayed signs of significant skew. By contrast, only five variables evidenced significant signs of kurtosis: a distributional feature of particular importance to covariance and correlational analyses since it can attenuate coefficient values (Browne, 1982, 1984; DeCarlo, 1997; Jobson, 1991, p. 55; Mardia, Kent, & Bibby, 1979, p. 149).

Variable Transformations & Post-Transformation Distributional Diagnostics

Although the foregoing distributional analyses provided evidence of non-normality, distributional dissimilarity between study variables was the primary concern, since correlation and regression coefficients are particularly susceptible to attenuation under such conditions (Carroll, 1961; Cohen et al., 2003; Hays, 1994). In the absence of statistical tests for comparing empirical data distributions to *one another*, the transformation strategy utilized aimed to maximize distributional similarity by transforming all non-normal variables to Gaussian-distributions. For a detailed discussion of the transformation methods utilized in this study, the reader is directed to Appendix C.

Kolmogorov-Smirnov, skewness, and kurtosis indices were then re-computed after variable transformations to assess for non-normality. These statistics are presented in Table B.2. Through the usage of outlier deletion and logarithmic transformations, seven variables no longer displayed evidence of significant distributional non-normality. Only two of these variables (BIDR Self-Deception and WCST – Perseverative Errors) continued to evidence some quantitative signs of distributional non-normality, but neither of these variables displayed evidence of kurtosis: a feature that could have substantially impacted our correlational analyses (Browne, 1982, 1984; DeCarlo, 1997; Jobson, 1991, p. 55; Mardia, Kent, & Bibby, 1979, p. 149). Similarly, two key study variables (BIDR Impression Management and WAIS – Saddler 2 Composite) that did not display marked improvement from following transformation attempts and whose original values were consequently retained (see Table B.2), displayed no significant signs of skew or kurtosis despite evincing significant K-S statistics.

In summary, whereas attempts at transforming variable distributions had mixed results, the measures of key constructs (e.g. HRV, executive function test performance) on the whole either benefitted significantly from these transformations and/or their distributions were sufficiently normal as to minimize concerns about attenuated correlation coefficient values.

Data Acquisition-by-Date Analyses

To determine whether sample characteristics varied by date of data acquisition, the sample was temporarily divided for purposes of data analysis into four groups

organized nominally by time-period of participant involvement (see Table A.2). These analyses were conducted since problems with planning and goal implementation are associated with self-regulatory deficits (for review, see Gollwitzer, Fujita, & Oettingen, 2004). This general observation, combined with the statistical observation that the responses of participants are more likely to be similar when their data are collected at similar, as opposed to different, times (see Cohen et al., 2003, p. 120; Judd et al., 1995, p. 459), gave rise to concerns that individual differences in effortful control could have varied systematically by participation date (e.g. low effortful control, late participation date). These individual differences, in turn, could have interacted with other factors extraneous (e.g. weather, participant stress varying with period academic term) to study protocols and, consequently, could have compromised measurement accuracy. Thus, it was viewed as important to examine whether variable scores varied significantly by data acquisition date.

Data acquisition groups were organized on the basis of 15 day intervals over the course of the Winter Quarter 2007 academic term. As determined by the Department of Psychology's Research Experience Program, students were eligible to participate between 7 January 2008 and 6 March 2008 (i.e. 60 days). Thus, one group consisted of subjects who participated during the first 15 days of eligibility, another group consisted of participants whose data were collected during the second 15 day eligibility interval, and so forth. As shown in Table A.2, none of the comparisons between group means for any study measure achieved statistical significance. Since

participant data did not vary systematically on the basis of acquisition time-period, no post-hoc analyses were necessary to explore specific group comparisons.

Correlation Results

Intercorrelations: Primary Variables

For ease of interpretation, intercorrelations between all the primary variables in this study are presented in Table A.3.

Correlations among self-report measures of EC. As expected, all correlations between the ATQ-EC subscales achieved statistical significance. Similarly, the Attentional Control Scale (ACS) correlated significantly with the ATQ-EC total score and with all ATQ-EC subscales, most especially (r = 0.78) the attentional control subscale of the ATQ (i.e. ATQ-EC Attentional Control).

Correlations among performance measures of executive control. Unexpectedly, the performance measures of different aspects of executive control did not correlate significantly with one another. For example, although measures derived from the Stop-Signal Task correlated significantly with one another, none of those measures correlated significantly with Stroop performance, WCST – Perseverative Errors, or WMS - Working Memory. Among these measures, only Stroop performance was significantly correlated with WMS-Working Memory; r = 0.26).

HRV and self-reports of EC. Consistent with expectations, resting HRV, as measured in the time-domain (rMSSD), evinced significant correlations with aggregate effortful control (ATQ-EC Total; r = 0.27), inhibitory control (ATQ-EC Inhibitory Control; r = 0.23) and activation control (ATQ-EC Activation Control; r =

0.27). These correlations were consistent with those obtained with the HRV frequency-domain measure (LogHF), which also correlated significantly with aggregate effortful control (r = 0.25), inhibitory control (r = 0.18), and activation control (r = 0.26). In striking contrast to these correlations, self-reports of attentional control (i.e., ATQ-EC Attentional Control and ACS) were not significantly related to HRV. Indeed, these correlations approached zero.

HRV and performance measures. Contrary to predictions, no significant correlations were found between measures of resting HRV and the performance measures of executive control.

Performance measures and self-reported EC. Similar to the findings involving measures of HRV, performance-based measure of executive functioning were generally not found to be significantly correlated with self-reports of EC or its facets. However, two significant correlations were observed. First, the working memory subscale of the Wechsler Memory Scale had a modest significant relation with the ATQ–EC Attentional Control (r = 0.19). By comparison, the correlation between WMS-Working Memory and the ACS (r = 0.15) was smaller and consequently did not achieve significance. Second, a modest-though-significant correlation was found between Stop-Signal – Go-Trial RT Sd. and ATQ-EC Inhibitory Control (r = −0.19) indicating that higher self-reported levels of inhibitory control were associated with less variability in responses to Go Trials in the Stop Signal Task. The correlation was similar between Stop-Signal – Go Trial RT and ATQ-EC Inhibitory Control (r = −0.18), although that correlation did not achieve significance.

Correlations between measures of EC and intelligence. In general, results were consistent with the prediction that EC is not merely a proxy for intelligence. With regard to self-reports of EC and its facets, only ATQ-EC Activation Control had a significant zero-order correlation with the WAIS Saddler 2 Composite ($r = –0.34$). However, it should be noted that this correlation shows that lower levels of activation control in this sample were associated with higher levels of itelligence. No other self-report measure of effortful or attentional control bore a significant relation with intelligence.

With respect to the relations between intelligence and performance measures of executive functioning, the WAIS Saddler 2 Composite evidenced significant correlations with working memory (i.e. WMS-Working Memory; $r = 0.49$) and several measures of response inhibition: Stroop Interference ($r = 0.23$) and go-trial performance on the Stop-Signal Task (i.e. Stop-Signal – Go-Trial RT, $r = −0.19$; Stop-Signal – Go-Trial RT Sd., $r = −0.31$).

Finally, measures of resting HRV were not significantly correlated with intelligence.

Self-deception and impression management. Self-deception (i.e. the tendency to provide honest yet positively biased self-reports), as indexed by the self-deception subscale of the Balanced Inventory of Desirable Responding (i.e. BIDR – Self-Deception), was moderately related to attentional control ($r = 0.37$) and all facets of effortful control (ATQ-EC Total and subscales) with the exception of inhibitory control (ATQ-EC Inhibitory Control). Moreover, no significant relationship emerged

between inhibitory control and impression management (i.e. deliberate self-presentation), as indexed by the impression management subscale (i.e. BIDR Impression Management) of the BIDR. Neither self-deception, nor impression management, evidenced statistically significant relationships with any physiological or performance measure.

Unlike BIDR Self-Deception, which exhibited significant concurrent relations with attentional control and most facets of effortful control, BIDR Impression Management was only moderately related to aggregate effortful control (r = 0.24) and the activation control subscale of the ATQ (r = 0.23). However, like BIDR Self-Deception, BIDR Impression Management did not covary with any performance measure, yet both self-deception (r = 0.37) and impression management (r = 0.24) correlated significantly with effortful control (ATQ-EC Total).

Intercorrelations: Primary Self-Reports & Task-Completion Questionnaire

As discussed in Appendix C, the individual TCQ subscales had problematic distributional properties whereas the TCQ Total had adequate properties. However, as shown in Table A.4, the TCQ subscales were generally moderately to strongly correlated with one amother. Thus, as shown in Table A.4, the TCQ total score had good internal consistency (Cronbach's alpha = .86). Therefore, analyses were limited to the TCQ total score.

As shown in Table A.4, with but a few exceptions, all primary self-report measures correlated significantly with TCQ total. The reader is reminded that high scores on the TCQ correspond to the endorsement of fewer problems with extraneous

performance factors (i.e. fatigue, attention, distraction, stress, effort). Thus, positive correlations between these measures would be consistent with the idea that participants who endorsed fewer problems with factors that could affect performance (i.e. fatigue, attention, distraction, stress, effort) endorsed higher levels of effortful control and attentional control than those who endorsed more problems with performance factors. However, as also shown in Table A.4, responses on the TCQ were also positively correlated with measures of socially desirable responding, as indexed by the impression management and self-deception subscales of the BIDR. This finding, combined with the aforementioned significant relations between socially desirable responding and facets of self-reported attentional- and effortful control, suggests that both the BIDR and TCQ should be included as covariates in regression models where self-reports of effortful and attentional control are treated as predictor variables.

Intercorrelations: Heart-Rate Variability Measures & Task-Completion Questionnaire

Whereas self-report measures of effortful control and socially desirable responding correlated almost uniformly with the TCQ Total, Table A.5 shows that measures of HRV held no significant relations with the TCQ. These null findings are not unexpected since measurements of resting HRV were procured prior to the completion of all self-reports and tests of executive functioning, before the assessment phases about which items on the TCQ inquired.

Intercorrelations: Executive Function Measures & Task-Completion Questionnaire

For ease of interpretation, relationships between measures of executive functioning and the TCQ are shown in Table A.6. As in the case of HRV, scores on the performance measures of executive functioning were not significantly correlated with TCQ total.

Multiple Linear Regression Results

Although the foregoing correlational analyses helped shed some light on the relations between multiple combinations of two variables, it was necessary to explore significant zero-order correlations further via multiple linear regression to control for demographic characteristics, intelligence, social desirability response bias (i.e. *self-deception positivity* and *impression management*, as indexed by the BIDR), and factors extraneous to the test measures that may have affected performance (i.e. TCQ).

Results: Regression Diagnostics

When conducting these regression analyses, it was necessary to calculate diagnostics relevant to the assumptions of multiple linear regression (MLR). What follows is a summary of the major findings from the regression diagnostics conducted as part of this study's data-analytic procedure. For a detailed discussion of the procedures utilized in the regression diagnostics performed, please refer to Appendix D (Note: all tables and figures appearing in Appendix D are designated with a 'D' preceding their number).

No evidence of multicollinearity emerged for any of the study variables included in the regression analyses. Similarly, none of the residual scatterplots displayed in Figures D.1-D.29 indicated significant deviations from *linearity* for of

any the multiple regression analyses (see Tables A.7-A.20). However, visual inspection of these residual scatterplots unconvered potential problems with *heteroscedasticity* for 5 regressions (see Figures D.5, D.6, D.7, D.13, and D.14). Nevertheless, post-hoc analyses, first in the form of *modified Levene tests*, and later through *slicing* (see Cohen et al., 2003, pp. 145-147), determined that the magnitude of the nonconstant variance (i.e. heteroscedasticity) was not high enough to warrant corrective action (e.g. logarithmic transformation of Y, weighted least squares regression).

To assess residual *independence*, Durbin-Watson coefficients (d) were computed for each linear regression (see Appendix D). All Durban-Watson coefficients (see Tables A.7-A.20) were within acceptable limits (ranged from 1.757-to-2.276) and suggestive of residual independence. Moreover, as shown in Figures D.1 – D.29, no evidence of residual *non-normality* manifested, as the residual plots adequately followed the linear patterns indicative of a normal curve.

In summary, regression diagnostics indicated that the multiple regression analyses met all necessary data-analytic assumptions. Consequently, these results lent support to the veracity of our multiple linear regressions as bases for verifying the correlation coefficient values obtained.

Results: Multiple Linear Regression Analyses

As depicted in Tables A.7 – A.20, a series of hierarchical multiple linear regressions were conducted to further ascertain whether the magnitude of the linear relation between sets of two variables would be affected through the inclusion of

covariates. These results are further summarized in Table A.21, which presents the primary zero-order correlations (r) and semi-partial correlations (sr) from the regression analyses. For all regression analyses, a zero-order correlation between the primary predictor variable and criterion variable was first computed (see Tables A.7-A.20, "Zero Order Model). After this initial calculation, six covariates were added, resulting in the "Full Model" (see Tables A.7-A.20) that included the primary predictor variable and six covariates.

With only two exceptions, these correlations remained statistically significant even after the inclusion of six covariates. Furthermore, even when statistical significance changed, the magnitude of the observed effect remained similar to the zero-order relation.

The first of the two exceptions was the correlation between ATQ-EC Attentional Control and WMS-Working Memory (r = 0.19; see Table A.3), which was no longer significant (sr = 0.116; see Table A.9) after the inclusion of covariates – especially BIDR Self-Deception ($sr_{\text{ATQ-EC Attentional Control}} = 0.229$) and Task Completion TOTAL ($sr_{\text{ATQ-EC Attentional Control}} = 0.201$).

The second exception was the relation between ATQ-EC Inhibitory Control and Stop-Signal – Go-Trial RT Sd., which was rendered statistically nonsignificant with the inclusion of covariates (see Table A.12). However, the actual magnitude of this relation changed very little (sr = -.192 to sr = -.180), suggesting the change in significance largely reflected reduced degrees of freedom.

One other change is potentially of interest. The relation between ATQ-EC Inhibitory Control and HRV – Resting RMSSD (see Table A.10) became stronger with the addition of the covariates. This may reflect the inclusion of participant sex as a covariate, because the semi-partial correlation increased markedly with the inclusion of participant sex: a variable previously shown to have significant relations with HRV-Resting RMSSD (see the discussion of suppression effects below).

Suppression Analyses

Recall that sex differences were found in HRV and ACS scores, with HRV scores being higher in females and ACS scores being higher in males. This pattern of results suggested that participant sex could operate as a suppressor variable (Cramer, 2003; Cohen et al., 2003, p. 78; MacKinnon, Krull, & Lockwood, 2000; Shrout & Bolger, 2002; Tzelgov & Henik, 1991) in regression models involving either the ACS or measures of HRV and provided justification for examining possible sex-based, statistical suppression effects for correlations involving our two measures of HRV, and scores on the ACS.

To test for the possibility of *statistical suppression* on the basis of participant sex, a series of analyses was conducted in which sex was included as a covariate. Specifically, this was done for all relations between ACS and HRV as well as between either ACS or HRV and performance measures. For each analysis I first computed the zero-order "regression" (i.e. correlation) and then followed-up by adding sex as a covariate (i.e. mixed model). Then I compared the partial correlations, standardized betas, and significant levels between the two models.

Evidence of a sex-based suppression effect emerged in several cases. However, in only one case, the aforementioned findings with respect to HRV-Resting RMSSD and ATQ-EC Inhibitory Control (see Table A.10) was the suppression effect of sufficient magnitude to increase coefficient values to such a degree that it resulted in a change in statistical significance. In other words, in all but this one case (Table A.10), when the original correlations were significant they remained significant at the same level (i.e. alpha: 0.05 or 0.01) when sex was partialled out. Similarly, in cases when the original correlations were nonsignificant, partialling out sex resulted in higher semi-partial correlations that, nonetheless, remained statistically nonsignificant.

Thus, it is likely that participant sex contributed irrelevant variance to ATQ-EC Inhibitory Control that was subsequently partialled-out in the regression equation when participant sex was included as a covariate: the result of which was an increased semi-partial correlation between ATQ-EC Inhibitory Control and HRV – Resting RMSSD. The finding of a potential suppressor effect in HRV (Resting RMSSD only) data should perhaps not be too surprising, given that suppressor variables are very common biological models where feedback mechanisms are present (Cohen et al., 2003, p. 457; Tzelgov & Henik, 1991).

Given that a number of significant correlations were found between self-reports of EC and the two BIDR scores (impression management and self-deception), whereas the BIDR scores were unrelated to HRV or performance measures, it is possible that the BIDR variables might serve to suppress correlations between EC questionnaire scores and both HRV and performance-measures. However,

examination of the regression analyses reported above (see Tables A.7 – A.20) suggests little evidence for this possibility. Nonetheless, it remains possible that some non-significant correlations between self-reports of EC and either HRV or performance measures might become significant after controlling for impression management and self-deception. This possibility was tested following the strategy described fort possible suppression due to sex differences. In no case was any non-significant correlation substantially changed nor did any achieve significance.

Chapter 4: Discussion

The overarching purpose of the present study was to investigate the convergent validity of self-report measures of effortful control (EC): a self-regulatory construct that has garnered considerable support from research on development and psychopathology in childhood and adolescence that has relied upon multiple modes of data acquisition. Although some of the findings gleaned from this child and adolescent research have been replicated in adults, these replications have been limited primarly to studies which have relied solely on self-reports (e.g. ATQ-EC): a mode of data acquisition that is reliant upon the insight and veracity of participant responding. Consequently, questions have remained concerning the accuracy and attendant validity of findings gleaned from adult EC research; questions that were ultimately addressed in this study by assessing the degree of concordance of these self-reports with both a well-established measure of self-regulation (i.e. heart-rate variability) and cognitive operations (i.e. executive functions) that subserve self-regulation. It is my view that the results of this investigation provide *qualified* support for the construct validity of effortful control and for the use of two self-report measures (i.e. ATQ-EC, ACS) of this self-regulatory construct in young adults.

I. Relations among Self-Reports of EC

Before testing hypotheses concerning covergent validity of self-reports of EC with other measurement modalities, it was necessary to examine the convergent

validity among the self-report measures under investigation and their correlations with other factors that might influence the correlations among variables. These questionnaires included the (1) EC scale of the Adult Temperament Questionnaire (ATQ-EC) and (2) Attentional Control Scale (ACS), as well as two measures of: (3) social desirability response bias (i.e. Balanced Inventory of Desirable Responding; BIDR) and (4) extraneous performance factors (i.e. Task Completion Questionnaire; TCQ).

After conducting variable transformations to achieve distributional *similarity*, correlational analyses on the ATQ-EC revealed statistically significant relationships at the 0.01 significance level among all ATQ-EC subscales and ATQ-EC Total. These correlations ranged in magnitude from 0.25-to-0.50, which was consistent with the idea that attentional control, activation control, and inhibitory control are related, though distinct facets of the broader construct of effortful control. The distinctness of these facets is particularly illustrated by the observed relation between ATQ-EC Activation Control and ATQ-EC Inhibitory Control, which achieved the lowest zero-order correlation ($r = 0.25$): a coefficient which suggests that the capacity to perform an action for which there is a strong tendency to avoid (i.e. activation control; overriding behavior inhibition or prepotent tendencies to engage in less behavioral approach) is distinct from the capacity to suppress non-optimal approach behavior (i.e. inhibitory control; overriding behavioral activation). The ACS also achieved statistically significant relations with all subscales of the ATQ-EC, especially ATQ-EC Attentional Control ($r = 0.78$), which measures the same facet of effortful control

as the ACS. This was consistent with expectations and provides support for the convergent validity of both scales.

This pattern of intercorrelations was consistent with previous research both with the ATQ-EC subscales (Evans, 2004; Evans & Rothbart, 2007; Rothbart, 2001; Rothbart, Ahadi, & Evans, 2000) and concurrent usage of the ACS and self-reported effortful control (Dinovo & Vasey, 2003, 2005). Nevertheless, questions of convergent validity remained as to whether or not self-reported effortful control would manifest concurrent relations with an objective measure of self-regulation and standardized measures of executive functioning.

Giving rise to potential doubts about these ATQ-EC and ACS intercorrelations, however, were the significant correlations that emerged between these measures and the BIDR and TCQ subscales. As shown in Table A.3, the *self-deception* subscale of the BIDR correlated significantly with ATQ-EC Total, ATQ-EC Attentional Control, ATQ-EC Activation Control, and ACS. Similarly, the *impression management* subscale of the BIDR held significant correlations with ATQ-EC Total and ATQ-EC Activation Control. Only the inhibitory control subscale of the ATQ displayed no significant relations with the BIDR, suggesting that participant responses on ATQ-EC Inhibitory Control may not be related to either deliberate (i.e. BIDR Impression Management) or indeliberate (i.e. BIDR Self-Deception) positive self-presentations.

By contrast, as shown in Table A.4, ATQ-EC Inhibitory Control was significantly related to responses on the TCQ. These results were similar to those of

the ACS and the remaining ATQ-EC subscales, which without exception held significant correlations with the TCQ. Since high scores on the TCQ correspond with few self-reported problems with task performance, these combined results suggested that participant responses on the ATQ-EC and ACS were systematically related to the endorsement of fewer problems with extraneous performance factors (i.e. fatigue, attention, distraction, stress, effort). Whether these significant correlations were due to the fact that people high in EC actually experience fewer problems with distraction, task effort, et cetera could not be gleaned from these correlational findings.

In light of these significant relations between the ATQ-EC, ACS, the two BIDR scales, and the TCQ, the latter three scales were included as covariates in regression models to control for method variance attributable to self-report responding. This allowed for the computation of semi-partial correlations, which involved the partialling-out of such shared variance in models where correlations between the ATQ-EC, ACS, and other key study variables (e.g. heart-rate variability) were reexamined. These semi-partial correlations (sr) among self-report measures are displayed in Tables A.7 – A.15 and are discussed below.

II. Effortful Control & Executive Functioning

As summarized in Tables A.3 and A.21, with only two exceptions, none of the self-report measures of EC or its facets evidenced significant relations with any facet of executive or cognitive functioning tapped by the performance measures used in this study. These measures included indices of response inhibition (i.e. Stop-Signal Task, Stroop Color-Word Interference Test, Wisconsin Card Sorting Test: Perseverative

Errors), working memory (i.e. Wechsler Memory Scale – Working Memory Index), cognitive flexibility (i.e. Wisconsin Card Sorting Test: Perseverative Errors), and general intelligence (WAIS – Saddler 2 Composite), the latter of which was included to rule out the possibility that effortful control is simply the joint application of a person's knowledge and executive functioning (i.e. intelligence).

The only zero-order correlations which attained statistical significance were (a) ATQ-EC Attentional Control and WMS-Working Memory ($r = 0.19$), and (b) ATQ-EC Inhibitory Control and Stop-Signal – Go-Trial RT Sd ($r = -0.19$). However, as shown in Table A.21, these zero-order relations were qualified by non-significant semi-partial (sr) correlations after including self-report and demographic covariates. Thus, little support was found in this study for hypotheses with respect to the relations between self-reported effortful control and executive functioning.

The general lack of significant relations between EC self-reports and performance measures ran counter to the expectation that such self-reports of EC would be related to (a) the inhibition of prepotent responses (i.e. response inhibition); (b) the maintenance of information in consciousness so that both the execution and sequencing of mental operations can be carried out (i.e. working memory); and (c) the shifting of attentional resources between mental operations (i.e. cognitive flexibility). This latter finding was particularly surprising, since attentional shifting – a facet of attentional control – is conceptually similar to cognitive flexibility (Barcelo, Perianez, & Knight, 2002; Greve, Ingram, & Bianchini, 1998; Loose, Kaufmann, Tucha, Auer, & Lange, 2006; Mueller, Dreisbach, Goschke, Hensch, Lensch, & Brocke, 2007;

Ravizza & Carter, 2008; Stemme, Deco, Busch, & Schneider, 2005). A consideration of why these performance measures did not relate to self-reported effortful control, including the possibility that scores were insensitive to individual differences in executive functioning in our college student sample, is addressed later.

By contrast, the absence of a correlation between the WAIS – Saddler 2 Composite and most measures of self-reported self-regulatory capacity – ATQ-EC Total, ATQ-EC Attentional Control, ATQ-EC Inhibitory Control, and ACS – was consistent with expectations, since general intelligence was not expected to be empirically related to effortful control or any of its subfacets. However, conclusions about the relations between effortful control and general intelligence remained qualified by the significant zero order ($r = -0.34$) and semi-partial correlations ($sr = -0.326$) between ATQ-EC Activation Control and the WAIS Saddler-2 Composite (see Table A.15). On its face, this unexpected result suggests that high intelligence may be associated with a lesser capacity to override behavioral disengagement (i.e. low activation control). Given that ATQ-EC Activation Control did not correlate significantly with any individual measure of executive functioning but correlated significantly with general intelligence, this could mean that activation control is subserved by several executive functions that work in tandem (i.e. general intelligence), not in isolation.

In light of the possibility that the self-report measures of effortful control and attentional control may hold significant relations with executive functions that work in tandem (rather than in isolation), a composite measure of executive functioning was

computed. This composite measure was an aggregate of the standardized scores of the four following measures: (1) WMS-Working Memory Index; (2) Stroop Interference T-Score; (3) WCST Perseverative Errors (Log10); and (4) Stop-Signal RT (Log 10). To ensure that all four measures were coded in the same direction, the latter measure (i.e. Stop-Signal RT) was recomputed (i.e. Stop-Signal RT x −1). The same data-analytic procedures were adopted when computing these correlation and semi-partial correlation statistics as had been implemented in the previous regression analyses, including residual diagnostics and inclusion of six covariates. No MLR violations came to light through these diagnostic procedures. As shown in Table A.22, only one variable (i.e. Attentional Control Scale) held a significant correlation ($r = 0.201$) with our composite measure of executive functioning. However, with the inclusion of covariates, the semi-partial correlation attained for this measure ($sr = 0.075$) did not achieve statistical significance, effectively invalidating the aforementioned significant correlation.

III. Effortful Control & Heart-Rate Variability

Although the validity of the ATQ-EC, ACS, and perhaps even the construct of effortful control (EC), could be challenged on the basis that they appear to lack empirical relations to executive functions (processes that underlie self-regulation), such challenges could be countered by the manifest correlations that emerged in this study between self-reported effortful control and heart-rate variability (HRV). As shown in Tables A.3 and summarized in Table A.21, resting heart-rate variability indexed in the time (RMSSD)- and frequency (Log HF Power)-domains was

significantly related to effortful control (r_{RMSSD} = 0.27; sr_{RMSSD} = 0.257; r_{LogHF} = 0.25; sr_{LogHF} = 0.230), inhibitory control (r_{RMSSD} = 0.23; sr_{RMSSD} = 0.244; r_{LogHF} = 0.18; sr_{LogHF} = 0.185), and activation control (r_{RMSSD} = 0.27; sr_{RMSSD} = 0.227; r_{LogHF} = 0.26; sr_{LogHF} = 0.207). These findings are particularly compelling because HRV is a robust measure of self-regulation (Segerstrom, & Nes, 2007; Thayer, Hansen, Saus-Rose, & Johnsen, 2009), lending some compelling support to the validity of self-reported effortful control. The convergence of these self-report and physiological measures also support the trait-like properties of effortful control (Derryberry & Rothbart, 1997; Eisenberg et al., 2004; Rothbart et al., 2000, 2003, 2004) and resting heart-rate variability (Thayer et al., 2009), since EC self-reports inquire about longstanding patterns of behavior and resting HRV has been shown to be temporally stable (Li, Snieder, Su, Ding, Thayer, Trieber, & Wang, 2009) and to have significant genetic influences (Neumann, Lawrence, Jennings, Ferrell, & Manuck, 2005; Snieder, van Doornen, Boomsma, & Thayer, 2007; Wang, Thayer, Treiber, & Snieder, 2005).

Qualifying the relationship between HRV and EC, however, was the absence (see Table A.3) of any significant relations between HRV and attentional control (ATQ-EC Attentional Control, Attentional Control Scale): a facet of effortful control which, like the other facets of EC, did not correlate (sr) significantly with executive functioning with the inclusion of covariates. This null finding between self-reported attentional control and HRV, and between self-reported attentional control and standardized measures of executive functioning, potentially raise doubts about the

validity of attentional control as a self-regulatory construct or, alternatively, challenge the validity of both the *attentional control* subscale of the ATQ-EC and the ACS.

In light of these findings, it might be of interest to the reader that neither index of heart-rate variability correlated significantly with any individual (see Table A.3) or composite (see Table A.22) measure of executive functioning. Although these latter results do not reconcile the absence of a relationship between attentional control and HRV, they do raise questions about the validity or usage of the executive function measures in this study, since one would expect an objective measure of self-regulation (i.e. HRV) to be related to executive processes (e.g. response inhibition, working memory) fundamental to self-regulation (Goldman-Rakic, 1998; Thayer et al., 2009). Indeed, measures of cardiac vagal tone (e.g. HRV) have been shown in previous research (Hansen et al., 2003; Richards, 1987; Suess, Porges, & Plude, 1994) to be significantly related to executive functioning, and heightened activity in the prefrontal cortex (a key structure of the central autonomic network, or CAN) has been demonstrated repeatedly during the completion of executive function tasks (Aron, Robbins, & Poldrack, 2004; Diwadkar, Carpenter, & Just, 2000; Garavan, Ross, & Stein, 1999; Konoshi, Nakajima, Uchida, Kikyo, Kameyama, & Miyashita, 1999; Nystrom et al., 2000; Stern, Owen, Tracy, Look, Rosen, & Petrides, 2000).

Thus, while the absence of a significant relationship between attentional control and HRV in this study may raise doubts about the convergent validity of this one facet of effortful control, this null finding may instead be attributable to other factors (e.g. measurement error, range restriction) specific to our investigation, since it

does not accord with an extensive body of research. This body of research includes evidence supporting the self-regulatory relevance of attentional control in quasi-experimental tasks (e.g. Derrberry & Reed, 2002; Diehl, Evans, 2004; Semegon, & Schwarzer, 2006; Posner & Rothbart, 1998; Rueda, Posner, & Rothbart, 2005); the high degree of convergence between attentional control and other facets of effortful control, which themselves have been shown to be related to HRV in this study; and the aforementioned results of other studies demonstrating the convergence of executive functioning with HRV and prefrontal cortex activity. Consequently, it is important to consider two additional factors that may have attenuated the correlation coefficients observed in our study: measurement reliability and variance.

IV. Measurement Reliability

One assumption in ordinary least squares regression is that each independent variable is measured without error (Cohen et al., 2003, p. 119). Although findings of imperfect reliability are quite typical in behavioral sciences research, where many of the concepts that form the theoretical foundation of a study are only indirectly (and thereby imperfectly) measured (McDonald, 1999; Nunnally & Bernstein, 1993), a consequence is that all indices of a partial relationship (e.g. standardized β, sr) between two or more variables will often be attenuated. Hence, the empirical relation between variables can be underestimated as a consequence of measurement error. Although problems of measurement error in *dependent variables* do not impact the magnitude of *unstandardized* beta (β) coefficients (Cohen et al., 2003, p. 124),

significance testing, statistical power, and the estimation of confidence intervals can still be aversely impacted.

Owing to our study procedures, where measurements were procured from single administrations, the temporal stability (i.e. test-retest reliability) could not be ascertained. However, our mode of data acquisition still permitted the computation of internal consistencies. As shown in Table A.3, no variable in our study attained perfect (1.00) reliability, which could have resulted in attenuated correlation and regression coefficients. However, it is worth noting that all study measures achieved indices indicative of moderate-to-good reliability (Anastasi & Urbina, 1997; McDonald, 1999; Nunnally & Bernstein, 1993). With respect to the reliability of our HRV measurements, the intraclass correlation coefficients (ICCs) obtained indicated substantial – though not excellent (ICC $\geq$ 0.80) – agreement (Pinna, Maestri, Torunski, Danilowicz-Szymanowiczm, Szwochm, La Rovere, & Raczak, 2007). Although some of our measures of executive function (e.g. Stroop Interference, WMS-Working Memory) attained among the lowest reliability coefficients in our study, it is also worth noting that these reliability statistics were still comparably higher than those observed in comprehensive psychometric reviews of cognitive tests (e.g. Miyake et al., 2000; see Alvarez & Emory, 2006).

Thus, while it is certainly possible that the correlation and regression coefficients obtained in this study were attenuated and thereby underestimated the degree of association between variables, there is little evidence that the extent of unreliability in our study measures surpassed conventional standards. Nevertheless,

the noted lack of high reliability ($r \geq 0.90$; $ICC \geq 0.80$) in our study measures, and the attendant likelihood of some attenuation in our measures of inter-variable association (Cohen et al., 2003, p. 119), may provide a partial explanation for the null findings for some key study hypotheses. These findings might also serve as a reminder that, especially in the absence of perfect measurement reliability, the interpretation of correlations and regression coefficients between variables must be carefully distinguished from the interpretation of the theoretical constructs those variables represent.

V. Measurement Variance & Dispersion

Closely related to the problem of measurement unreliability is the issue of whether or not the range and variance of variables was restricted by one's sampling procedures (Cohen et al., 2003, p. 57). This issue is particularly salient given the nature of our sample, which exclusively consisted of college students: a population that is arguably more homogenous on potentially relevant variables (e.g. level of cognitive functioning) than the general population. Like the absence of perfect reliability, low variance or range restriction can attenuate correlation and regression coefficients (Aiken & West, 1991; Atkinson, 1985; Belsley et al., 1980; Berk, 2003; Berry, 1993; Fox, 1991; Kahane, 2001). Thus, a key question that remained was how the dispersion of scores on various measures in this study compared to studies investigating individual differences in effortful control, heart-rate variability, and executive functioning.

As shown in Table A.23, none of our variables consisted of values that spanned the range of possible values, providing some evidence of range restriction. In general this was most pronounced on the lower ends of variable continuums, especially with our performance measures of executive functioning, where no participant obtained extremely low scores consistent with acute cognitive deficits. These findings were consistent with our regression diagnostics in which visual inspection of residual scatterplots suggested that there were few observations for lower variable values (see *Regression Diagnostics: Relationship Linearity & Homogeneity of Variance*). With respect to our measures of executive functioning, the presence of few observations on the lower ends of their distributions is perhaps not too surprising given the nature of our sample, which consisted exclusively of college undergraduates, who tend to score higher on measures of cognition than members of the general population (Wechsler 1997a, 1997b). Thus, it is possible that because our sample consisted of persons who, in general, possess higher cognitive functioning than the general population, there were too few observations on the lower ends of the executive functioning continuum for an adequate comparison with concurrently poor self-regulation, thus attenuating our correlation and regression coefficients.

Indeed, this interpretation is consistent with findings obtained from recent work with children and adolescents showing poor correlations between measures of executive functioning and child- and parent-reported EC. For example, Verstraeten and associates (2010) found that poor correlations between measures of EC and Tests of Everyday Attention (TEA-Ch) were attributable, at least in part, to ceiling effects

and low variability in the TEA-Ch performance of older children and adolescents. Such findings were consistent with earlier child- and adolescent based community research, where child-reported EC evidenced low correlations to performance on executive functioning tasks (Muris, Mayer, van Lint, & Hofman, 2008; Muris, van der Pennen, Sigmond, & Mayer, 2008). Thus, the results of this study are both consistent and contribute to a growing body of evidence that tests of executive function may fail to correlate with measures of EC owing to their insensitivities to individual differences in effortful self-regulation in specific samples.

VI. Summary & Implication of Findings:

In summary, the results of this investigation provide some compelling support for one measure of the self-regulatory trait effortful control (EC), the Adult Temperament Questionnaire – Effortful Control Scale (ATQ-EC). This support came in two forms: (1) high correlations between the ATQ-EC and all of its subscales with another measure of self-regulation, the Attentional Control Scale (ACS); and (2) the high degree of association between ATQ-EC Total, ATQ-EC Activation Control, and ATQ-EC Inhibitory Control and both the time- and frequency-domain measure of heart-rate variability (HRV). Not only is the former finding impressive because the ATQ-EC and ACS were developed independently, but the latter findings collectively, for the first time, provide evidence that self-reported EC is related to an objective physiological measure of self-regulatory capacity.

By contrast, although some support was provided for the ACS, a self-report measure of attentional control (a facet of effortful control), this support came by way

of its associations with the ATQ-EC. Like the ATQ-EC, the ACS held no significant relations to any measure of executive functioning, but unlike the ATQ-EC held no relation to either measure of heart-rate variability. It is worth noting that this null finding held constant even when the ACS was divided into its *attentional shifting* ($r_{RMSDD} = -0.140$; $r_{Log(HF\ Power)} = -0.046$) and *attentional focusing* ($r_{RMSSD} = 0.43$; $r_{Log(HF\ Power)} = 0.077$) subfacets. This finding with respect to HRV could engender some doubts about the self-regulatory relevance of the ACS, given the extensive body of evidence attesting to the validity of HRV as an index of self-regulatory capacity (Thayer et al., 2009).

Not only did the absence of a relationship between the ACS and HRV challenge the validity of the ACS, but the construct validity of *attentional control* can arguably be challenged because the attentional control subscale of the ATQ-EC (i.e. ATQ-EC Attentional Control) also did not correlate significantly with either measure of HRV. Nevertheless, one could attribute the absence of a relation between attentional control and HRV to their measuring differing aspects of effortful control. Indeed, countervailing evidence abounds that *attentional* control may attenuate temperamental vulnerability to diffuse negative affect and low positive affect (e.g. Ayduk, Zayas, Downey, Cole, Shoda, & Mischel, 2008; Derryberry & Reed, 2002; Dinovo, 2003, 2005; Meesters, Muris, & van Rooijen, 2007; Muris, de Jong, & Engelen, 2004; Muris, Mayer, van Lint, & Hofman, 2009). Similarly, the lack of a relationship between attentional control and HRV is not consistent explanatory accounts of this (and closely related) constructs, since discussions of attentional

control (e.g. Derryberry & Reed, 2002; Posner & Rothbart, 1998) implicate many of the same neural systems (e.g. anterior cingulate cortex, rostral limbic system) central to the neurovisceral integration perspective (Thayer & Friedman, 2004; Thayer et al., 2009; Thayer & Lane, 2000): a comprehensive account of the relationship between cardiac vagal tone and self-regulation that implicates the executive control of attentional processes. Thus, definitive statements about the construct validity of attentional control within the realms of psychopathology and health cannot be made at the present time.

The relations between measures of HRV and the ATQ-EC are noteworthy, since one could not attribute their correlations to shared methods variance. That is, whereas self-reports require respondents to answer questions about self-regulation and its attendant consequences, measures of heart-rate variability have the advantage of being objective measures of parasympathetic functioning that do attempt to glean the same information as self-reports based on differences in question construction, because cardiac vagal tone was not indexed on the basis of conscious, verbal (written or spoken) responding by participants. Nevertheless, even if one were to still argue that some of the shared variance between these measures is attributable to shared methods variance, these correlations at worst still provide evidence of the convergent validity of subjective self-reporting, since objective physiological measurements have already shown to be reliable measures of self-regulation (Thayer et al., 2009). Thus, it remains plausible that self-reported EC reflects the outcome or byproduct of latent self-regulatory capacity: a capacity captured by measurements of HRV.

Evidence supporting a delineation between two subfacets of EC, inhibitory control and activation control, also emerged in this study. Both inhibitory control and activation control evidenced relations of comparable magnitude and direction to HRV (time- and frequency-domain), attentional control (ACS and ATQ-EC Attentional Control), and effortful control (ATQ-EC Total), and neither inhibitory control nor activation control were related to executive functioning. One could argue that this parallel pattern of inter-relations to other measures supports the convergent validity of both inhibitory control and activation control, whereas the significant – though modest – correlation between them attests to their discriminant validity within the nomological network (Cronbach & Meehl, 1955) of effortful control. This distinction between inhibitory control and activation control is congruent with the idea that suppression of prepotent responses by the prefrontal cortex can assume a variety of forms (Evans & Rothbart, 2007), including the suppression of inappropriate approach behavior (i.e. inhibitory control) or the overriding of prepotent tendencies to avoid performing an action (i.e. activation control). One might speculate that both inhibitory and activation control could either work together to attenuate maladaptive tendencies in some conditions (e.g. obsessive-compulsive disorder: inhibitory control over compulsive behaviors, and activation control overriding behavioral avoidance of feared stimuli), or work independently in conditions, for instance, that are characterized by pronounced impulsivity (e.g. inhibitory control: ADHD, susbstance use disorders) or social withdrawal (e.g. activation control: unipolar depression, social anxiety disorder). Such questions may prove to be fertile ground for future research.

Additional directions for future research that could provide further support for the convergent validity of effortful control, inhibitory control, and activation control have their roots in several lines of research. For example, despite compelling evidence that HRV may have strong genetic bases (Neumann et al., 2005; Snieder et al., 2007; Thayer, Merritt, Sollers, Zonderman, Evans, Yie, & Abernethy, 2003; Wang et al., 2005), resting HRV can be increased through a variety of changes in diet, physical activity, and stress reduction techniques (Thayer et al., 2009; Thayer & Lane, 2009). In this light, future research may want to focus on whether changes in resting HRV over time relate to predictable changes in self-reported EC, inhibitory control, and activation control.

VII. Closing Remarks

The results of our study provide the first evidence that effortful control, a self-regulatory construct that has thus far been measured only through questionnaire (e.g. ATQ-EC) in adults, is not an artifact of self-report responding: a mode of data acquisition that relies upon the honesty, insight, and comprehension of participants. This evidentiary support resides in the convergence of self-reported effortful control with heart-rate variability (HRV): a construct that has been shown to be a reliable and valid measure of self-regulation across multiple domains (Thayer et al., 2009). Notwithstanding the informative and promising nature of our findings, it is worth noting that these conclusions are constrained by the absence of relevant comparative information concerning variables known to influence HRV. These clinical variables include body mass index (Antelmi et al., 2004; Gutin, Howe, Johnson, Humphries,

Snieder, & Barbeau, 2005; Reed et al., 2006), alcohol consumption (Ingjaldsson, Laberg, & Thayer, 2003; Reed, Porges, & Newlin, 1999), hypothalamic-pituitary-adrenal (HPA) axis dysregulation (Thayer, Hall, Sollers, & Fischer, 2006), tobacco use (Nabors-Oberg, Sollers, Niaura, & Thayer, 2002), and physical activity (Carter, Banister, & Blaber, 2003; Gutin et al., 2005; Reed et al., 2006; Rossy & Thayer, 1998). Not only would such information be useful from the standpoint of elucidating the relations between these conditions and effortful control, but such information would be important in helping us identify potential moderators that could have impacted participants' heart-rate variability during data acquisition.

Moreover, given the influence of physical activity (Carter, Banister, & Blaber, 2003; Gutin et al., 2005; Reed et al., 2006; Rossy & Thayer, 1998) and stress (Bernardi, Wdowczyk-Szulc, Valenti, Castoldi, Passino, Spadacini, & Sleight, 2000; Berntson, Cacioppo, Binkley, Uchino, Quigley, & Fieldstone, 1994; Lackschewitz, Hüther, & Kröner-Herwig, 2008; Shapiro, Sloan, Bagiella, Kuhl, Anjilvel, & Mann, 2000) on short-term measurements of cardiac-vagal functioning, it may have been more prudent to take measurements of resting HRV near the end of our study protocol rather than at the beginning. That is, by measuring resting HRV near the beginning of our study procedure we may have introduced nuisance variance associated with participants' activities prior to participation, including physical and psychological stresses associated with arriving to the study promptly, as required of them according to Research Experience Program (REP) protocols. Thus, by procuring resting HRV measurements near the termination of the study procedure, we may have been able to

minimize such nuisance variance by assuring some degree of continuity in participant activity prior to measurement. On the other hand, it could be argued that taking resting HRV measurements near the end of the study protocol could have introduced nuisance variance in the form of mental stress associated with extensive testing and/or motivations to leave the study. Thus, an alternate strategy might have been to procure resting HRV by counterbalancing resting periods among participants.

Given the absence of any significant correlation between our measures of executive functioning and both EC and HRV, it could similarly be argued that our choice of cognitive measures were not adequate for investigating our study hypotheses. While such a critique may not extend to some of the more well-validated measures (e.g. WMS-III, WCST) of executive functioning utilized in our study, a few of our other measures (e.g. Stop-Signal Task) may not have been appropriate choices for indexing facets of executive functioning. For instance, it has been argued (e.g. Cowan, 1988, 1994) that simple reaction time and choice reaction time tasks, like the Stop-Signal Task, do not require executive cognitive processes like working memory and are, instead, driven reflexively by stimulus properties (see Thayer et al., 2009, p. 146). Indeed, the Stop-Signal Task did not correlate significantly with our other measures of executive functioning, suggesting that individual differences on this measure may have been driven by reactive, inhibitory processes. Consequently, it remains possible that certain facets of executive functioning are related to effortful control. Future research should be directed at addressing this important question.

What is more, some caution should be exercised when attempting to generalize our findings given the nature of our sample. Although the primary motivation of this study was to investigate the validity of self-reported effortful control in young adults, since previous adult research (Dinovo, 2003, 2005; Skowron & Dendy, 2004) had relied almost exclusively on this age group, it remains an open question regarding the extent to which EC would correlate with resting HRV in other (perhaps more heterogeneous) samples. For example, previous research has shown age, sex, and ethnic-related differences in short-term HRV measurements (e.g. Britton et al., 2007; Choi, Hong, Nelesen, Bardwell, Natarajan, Schubert, & Dismale, 2006; Zhang, 2007). Consequently, while our study may lend support to the validity of the ATQ-EC in young adults, additional research utilizing different types of samples (e.g. community-based samples, clinical samples) is likely warranted to establish the external validity of our findings.

Notwithstanding the limitations of this study, it is my belief that the results of this investigation lend support to the construct validity of effortful control: a self-regulatory construct with broad applications across the lifespan. Given the noted applicability of effortful control in realms as ostensibly disparate as normative childhood development and psychopathology, it seems likely that the self-regulatory processes subsumed under the umbrella of effortful control implicate a common inhibitory network (Aron et al., 2004; Chikazoe, Konishi, Asari, Jimura, & Miyashita, 2007) discussed in other theoretical frameworks. Most notably, the neurovisceral

intergration perspective (Thayer et al., 2009) upon which the link between HRV and self-regulation has been explicated.

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Appendix A
Tables: Correlation & Regression Analyses

Measure	Male (n = 63)	Female (n = 61)	Sample (n = 124)
ATQ – EC Total			
M	82.04	81.16	81.59
SD	15.20	14.25	14.66
ATQ – EC Attentional Control			
M	20.91	19.17	20.01
SD	5.83	4.91	5.42
ATQ – EC Inhibitory Control			
M	28.95	27.85	28.38
SD	6.56	6.59	6.57
ATQ – EC Activation Control			
M	32.18	34.15	33.20
SD	7.16	6.64	6.94
Attentional Control Scale			
M	54.63	51.04	52.77*
SD	9.42	8.82	9.25
BIDR Self-Deception			
M	6.21	5.35	5.77
SD	3.49	2.98	3.25
BIDR Impression Management			
M	5.46	6.19	5.84
SD	3.76	3.10	3.44
HRV – Resting RMSSD			
M	47.28	61.04	54.36**
SD	37.17	33.02	35.60

Continued

Table A.1. Descriptive Statistics on Primary Measures. *Note:* * = Sex difference significant at $p < 0.05$, two-tailed. ** = Sex difference significant at $p < 0.01$, two-tailed.

Table A.1. Continued

Measure	Male (n = 63)	Female (n = 61)	Sample (n = 124)
HRV – Ln (Resting HF Power)			
M	6.33	7.08	6.71**
SD	1.52	1.23	1.42
Stop-Signal RT			
M	185.38	180.62	182.93
SD	65.76	59.02	62.13
Stop-Signal Go-Trial RT			
M	515.31	509.27	512.20
SD	120.35	116.45	118.35
Stop-Signal Go-Trial RT Sd.			
M	120.35	116.45	118.35
SD	42.38	33.32	37.86
Stroop Interference			
M	58.16	56.80	57.46
SD	7.93	6.25	7.11
WCST Perseverative Errors			
M	58.55	56.33	57.41
SD	9.85	9.87	9.88
WMS Working Memory Index			
M	112.94	108.09	110.45
SD	12.38	11.70	12.22
WAIS Saddler-2 Composite			
M	24.02	23.00	23.50
SD	3.88	3.00	3.48
TCQ – Total Score			
M	50.73	50.37	50.54
SD	6.40	6.81	6.59
TCQ – Computer Tests			
M	16.43	16.27	16.34
SD	2.26	2.57	2.41
TCQ – Hand Tests			
M	17.13	16.58	16.84
SD	2.55	3.00	2.79

Continued

Table A.1. Continued

Measure	Male (n = 63)	Female (n = 61)	Sample (n = 124)
TCQ – Self-Reports			
M	17.18	17.52	17.35
SD	2.53	2.57	2.55
TCQ – Tired/Fatigued			
M	8.45	8.12	8.28
SD	2.37	2.31	2.33
TCQ – Task Attention			
M	10.72	10.88	10.81
SD	1.50	1.43	1.46
TCQ – Distracted			
M	10.46	10.27	10.36
SD	1.62	1.97	1.81
TCQ – Stressed			
M	9.98	9.97	9.98
SD	1.89	1.78	1.82
TCQ – Task Effort			
M	11.11	11.13	11.12
SD	1.50	1.35	1.42

Measure	Group 1 (n = 14)	Group 2 (n = 24)	Group 3 (n = 31)	Group 4 (n = 55)	Sample (n = 124)
ATQ – EC Total					
M	85.10	81.58	79.02	82.46	81.59
SD	11.14	12.98	16.60	14.30	14.66
ATQ – EC Attentional Control					
M	21.17	19.13	19.42	20.42	20.01
SD	4.76	5.97	6.14	4.78	5.42
ATQ – EC Inhibitory Control					
M	28.19	27.75	27.41	29.28	28.38
SD	5.85	6.44	6.68	6.53	6.57
ATQ – EC Activation Control					
M	35.75	34.71	32.20	32.76	33.20
SD	7.47	6.84	7.18	6.43	6.94
Attentional Control Scale					
M	53.42	51.12	50.46	54.40	52.77
SD	9.10	10.21	7.70	9.03	9.25
BIDR Self-Deception					
M	5.42	6.16	5.51	5.94	5.77
SD	2.68	3.44	2.65	3.62	3.25

Continued

Table A.2. Descriptive Statistics on Primary Measures: Data Acquisition Date Comparisons. *Note:* Group 1 = January 7 – January 21, Group 2 = January 22 – February 5, Group 3 = February 6 – February 20, Group 4 = February 21 – March 6; No between-group differences achieved statistical significance ($p < 0.05$, two-tailed).

Table A.2. Continued

Measure	Group 1 (n = 14)	Group 2 (n = 24)	Group 3 (n = 31)	Group 4 (n = 55)	Sample (n = 124)
BIDR Impression Management					
M	5.33	5.44	6.66	5.39	5.84
SD	3.39	3.08	3.54	3.50	3.44
HRV – Resting RMSSD					
M	48.05	59.13	49.38	54.10	54.36
SD	22.00	35.07	35.70	36.53	35.60
HRV – Ln (Resting HF Power)					
M	6.74	6.96	6.59	6.64	6.71
SD	1.34	1.55	1.26	1.48	1.42
Stop-Signal RT					
M	172.46	201.78	173.18	178.20	182.93
SD	73.03	74.71	41.69	58.00	62.13
Stop-Signal Go-Trial RT					
M	508.22	528.11	500.82	519.08	512.20
SD	143.84	96.79	113.09	103.00	118.35
Stop-Signal Go-Trial RT Sd.					
M	110.70	125.73	114.92	120.26	118.35
SD	35.29	38.21	37.97	36.47	37.86

Continued

Table A.2. Continued

Measure	Group 1 (n = 14)	Group 2 (n = 24)	Group 3 (n = 31)	Group 4 (n = 55)	Sample (n = 124)
Stroop Interference					
M	59.17	58.20	56.39	57.58	57.46
SD	5.02	8.05	7.98	6.55	7.11
WCST Perseverative Errors					
M	58.50	57.24	55.93	58.33	57.41
SD	7.78	9.67	9.89	9.87	9.88
WMS Working Memory Index					
M	109.67	109.36	108.16	111.51	110.45
SD	10.76	11.18	11.48	13.36	12.22
WAIS Saddler-2 Composite					
M	24.18	22.56	23.48	23.36	23.50
SD	3.19	3.85	3.21	3.73	3.48
TCQ – Total Score					
M	50.33	51.04	51.74	49.39	50.54
SD	3.28	5.57	5.47	8.07	6.59

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. ATQ – EC Total	(.81**)															
2. ATQ – EC Attentional Control	.81**	(.76**)														
3. ATQ – EC Inhibitory Control	.73**	.44**	(.60**)													
4. ATQ – EC Activation Control	.77**	.50**	.25**	(.72**)												
5. Attentional Control Scale	.68**	.78**	.44**	.40**	(.86**)											
6. BIDR Self-Deception	.37**	.38**	.15	.33**	.38**	(.67**)										
7. BIDR Impression Management	.24**	.15	.17	.23*	.13	.30**	(.69**)									
8. HRV – Resting RMSSD	.27**	.09	.23*	.27**	–.01	.06	.13	(.75**)								
9. HRV – Ln (Resting HF Power)	.25**	.13	.18*	.26**	.04	.08	.14	.95**	(.74**)							
10. Stop-Signal RT	–.07	–.01	–.02	–.10	–.10	–.12	–.05	.08	.08	(na)						
11. Stop-Signal – Go-Trial RT	–.01	.05	–.18	.11	.08	.12	–.14	–.02	–.05	–.59**	(na)					
12. Stop-Signal – Go-Trial RT Sd.	–.02	.05	–.19*	.10	.08	.10	–.13	–.09	–.11	–.57**	.87**	(na)				
13. Stroop Interference	.09	.04	.10	.08	.07	.07	.01	.01	.01	–.03	–.05	–.08	(.65**)			
14. WCST – Perseverative Errors	.08	.06	.12	.01	.18	–.08	.02	.05	.01	–.15	.09	.04	.05	(na)		
15. WMS – Working Memory	.05	.19*	.11	–.16	.15	.11	–.09	–.11	–.05	–.06	–.01	–.09	.26**	.16	(.71**)	
16. WAIS – Saddler 2 Composite	–.13	–.09	.14	–.34**	–.03	–.14	.06	–.07	–.07	–.11	–.19*	–.31**	.23*	.07	.49**	(.82**)

Table A.3. Correlations among Primary Measures after Transformations. *Note:* * = Correlation significant at $p < 0.05$, two-tailed. ** = Correlation significant at $p < 0.01$, two-tailed. Internal consistencies are shown on the diagonal, unless otherwise indicated (na; individual measurements/items not available for computing internal consistency). --- = previously reported correlation; na = correlation not calculated.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. ATQ – EC Total	(---)															
2. ATQ – EC Attentional Control	---	(---)														
3. ATQ – EC Inhibitory Control	---	---	(---)													
4. ATQ – EC Activation Control	---	---	---	(---)												
5. Attentional Control Scale	---	---	---	---	(---)											
6. BIDR Self-Deception	---	---	---	---	---	(---)										
7. BIDR Impression Management	---	---	---	---	---	---	(---)									
8. TCQ Total	.40**	.36**	.27**	.30**	.33**	.41**	.35**	(.86**)								
9. TCQ – Computer Tests	na	na	na	na	na	na	na	.85**	(.62**)							
10. TCQ – Hand Tests	na	na	na	na	na	na	na	.85**	.60**	(.74**)						
11. TCQ – Self-Reports	na	na	na	na	na	na	na	.85**	.60**	.55**	(.75**)					
12. TCQ – Tired/Fatigued	na	na	na	na	na	na	na	.77**	.67**	.63**	.65**	(.74**)				
13. TCQ – Task Attention	na	na	na	na	na	na	na	.84**	.75**	.65**	.75**	.53**	(.71**)			
14. TCQ – Distracted	na	na	na	na	na	na	na	.80**	.66**	.72**	.66**	.44**	.66**	(.66**)		
15. TCQ – Stressed	na	na	na	na	na	na	na	.60**	.45**	.61**	.46**	.30**	.27**	.42**	(.70**)	
16. TCQ – Task Effort	na	na	na	na	na	na	na	.73**	.65**	.56**	.66**	.42**	.81**	.49**	.19*	(.76**)

Table A.4. Correlations among Task Completion Questionnaire (TCQ) Subscales after Transformations. *Note:* * = Correlation significant at $p < 0.05$, two-tailed. ** = Correlation significant at $p < 0.01$, two-tailed. Internal consistencies are shown on the diagonal, unless otherwise indicated (na; individual measurements/items not available for computing internal consistency). --- = previously reported correlation; na = correlation not calculated.

	1	2	3
1. HRV – Resting RMSSD	(---)		
2. HRV – Ln (Resting HF Power)	---	(---)	
3. TCQ Total	.06	.07	(---)

Table A.5. Correlations among Heart-Rate Variability Measures and Task Completion Questionnaire (TCQ) after Transformations. *Note:* * = Correlation significant at $p < 0.05$, two-tailed. ** = Correlation significant at $p < 0.01$, two-tailed. --- = previously reported correlation

	TCQ Total
1. Stop-Signal RT	−.09
2. Stop-Signal – Go-Trial RT	−.01
3. Stop-Signal – Go-Trial RT Sd.	−.04
4. Stroop Interference	.16
5. WCST – Perseverative Errors	.01
6. WMS – Working Memory	.09
7. WAIS – Saddler 2 Composite	.00

Table A.6. Correlations among Executive Function Measures and Task Completion Questionnaire (TCQ) after Transformations. *Note:* * = Correlation significant at $p < 0.05$, two-tailed.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.072*	(1, 109)	0.005**		
i. HRV – Resting RMSSD				.268**	.268**
Full Model	0.285**	(7, 103)	0.537		
i. HRV – Resting RMSSD				.272**	.257**
ii. Self-Deception				.220*	.193*
iii. Impression Management				.056	.050
iv. Task Completion TOTAL				.269**	.232*
v. Age				.068	.066
vi. Sex				−.100	−.094
vii. Race				−.054	−.052
Durban-Watson Statistic = 2.276					

Table A.7. Hierarchical regression predicting ATQ-EC Total from HRV – Resting RMSSD. *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.064*	(1, 109)	0.008**		
i. HRV – Ln (Resting HF Power)				.253**	.253**
Full Model	0.272**	(7, 103)	0.583		
i. HRV – Ln (Resting HF Power)				.242**	.230*
ii. Self-Deception				.216*	.189*
iii. Impression Management				.054	.049
iv. Task Completion TOTAL				.270**	.233*
v. Age				.068	.066
vi. Sex				−.087	−.082
vii. Race				−.049	−.047
Durban-Watson Statistic = 2.256					

Table A.8. Hierarchical regression predicting ATQ-EC Total from HRV – Ln (Resting HF Power). *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.037*	(1, 109)	0.044*		
i. WMS – Working Memory				.192*	.192*
Full Model	0.234**	(7, 103)	0.975		
i. WMS – Working Memory				.120	.116
ii. Self-Deception				.263**	.229*
iii. Impression Management				.017	.015
iv. Task Completion TOTAL				.234*	.201*
v. Age				−.051	−.050
vi. Sex				−.135	−.131
vii. Race				.003	.003
Durban-Watson Statistic = 2.117					

Table A.9. Hierarchical regression predicting ATQ-EC Attentional Control from WMS – Working Memory. *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.054*	(1, 109)	0.016*		
i. HRV – Resting RMSSD				.232*	.232*
Full Model	0.149*	(7, 103)	0.854		
i. HRV – Resting RMSSD				.257**	.244**
ii. Self-Deception				.007	.006
iii. Impression Management				.079	.071
iv. Task Completion TOTAL				.223*	.192*
v. Age				.006	.006
vi. Sex				−.172	−.162
vii. Race				.018	.017
Durban-Watson Statistic = 2.092					

Table A.10. Hierarchical regression predicting ATQ-EC Inhibitory Control from HRV – Resting RMSSD. *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.034	(1, 109)	0.059		
i. HRV – Ln (Resting HF Power)				.183*	.183*
Full Model	0.124	(7, 103)	0.814		
i. HRV – Ln (Resting HF Power)				.195*	.185*
ii. Self-Deception				.006	.005
iii. Impression Management				.081	.073
iv. Task Completion TOTAL				.225*	.194*
v. Age				.002	.002
vi. Sex				−.152	−.143
vii. Race				.023	.022
Durban-Watson Statistic = 2.088					

Table A.11. Hierarchical regression predicting ATQ-EC Inhibitory Control from HRV – Ln (Resting HF Power). *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.037*	(1, 109)	0.047*		
i. Stop-Signal – Go-Trial RT Sd.				−.192*	−.192*
Full Model	0.122	(7, 103)	0.930		
i. Stop-Signal – Go-Trial RT Sd.				−.184*	−.180
ii. Self-Deception				.054	.047
iii. Impression Management				.071	.064
iv. Task Completion TOTAL				.214*	.184*
v. Age				−.016	−.016
vi. Sex				−.104	−.102
vii. Race				.009	.008
Durban-Watson Statistic = 2.090					

Table A.12. Hierarchical regression predicting ATQ-EC Inhibitory Control from Stop-Signal – Go-Trial RT Sd.. *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.072**	(1, 109)	0.005**		
i. HRV – Resting RMSSD				.269**	.269**
Full Model	0.249**	(7, 103)	0.193		
i. HRV – Resting RMSSD				.239**	.227*
ii. Self-Deception				.225*	.197*
iii. Impression Management				.049	.045
iv. Task Completion TOTAL				.173	.149
v. Age				.175	.170
vi. Sex				.093	.088
vii. Race				−.117	−.114
Durban-Watson Statistic = 2.108					

Table A.13. Hierarchical regression predicting ATQ-EC Activation Control from HRV – Resting RMSSD. *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.066**	(1, 109)	0.008**		
i. HRV – Ln (Resting HF Power)				.257**	.257**
Full Model	0.240**	(7, 103)	0.216		
i. HRV – Ln (Resting HF Power)				.218*	.207*
ii. Self-Deception				.221*	.193*
iii. Impression Management				.048	.043
iv. Task Completion TOTAL				.175	.150
v. Age				.175	.170
vi. Sex				.103	.098
vii. Race				−.113	−.109
Durban-Watson Statistic = 2.106					

Table A.14. Hierarchical regression predicting ATQ-EC Activation Control from HRV – Ln (Resting HF Power). *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.115**	(1, 109)	0.000**		
i. WAIS – Saddler 2 Composite				−.339**	.339**
Full Model	0.304**	(7, 103)	0.073		
i. WAIS – Saddler 2 Composite				−.336**	−.326**
ii. Self-Deception				.182	.157
iii. Impression Management				.109	.098
iv. Task Completion TOTAL				.180	.155
v. Age				.152	.148
vi. Sex				.132	.128
vii. Race				−.158	−.152

Durban-Watson Statistic = 1.890

Table A.15. Hierarchical regression predicting ATQ-EC Activation Control from WAIS – Saddler 2 Composite. *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.037*	(1, 109)	0.046*		
i. WAIS – Saddler 2 Composite				−.193*	−.193*
Full Model	0.101	(7, 103)	0.100		
i. WAIS – Saddler 2 Composite				−.183*	−.177
ii. Self-Deception				.175	.152
iii. Impression Management				−.177	−.159
iv. Task Completion TOTAL				−.028	−.024
v. Age				.038	.037
vi. Sex				.041	.040
vii. Race				−.165	−.158
Durban-Watson Statistic = 1.818					

Table A.16. Hierarchical regression predicting Stop-Signal Go-Trial RT from WAIS – Saddler 2 Composite. *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.096**	(1, 109)	0.001**		
i. WAIS – Saddler 2 Composite				−.310**	−.310**
Full Model	0.136*	(7, 103)	0.188		
i. WAIS – Saddler 2 Composite				−.308**	−.299**
ii. Self-Deception				.127	.110
iii. Impression Management				−.120	−.107
iv. Task Completion TOTAL				−.057	−.049
v. Age				.036	.035
vi. Sex				−.025	−.025
vii. Race				−.129	−.124
Durban-Watson Statistic = 1.880					

Table A.17. Hierarchical regression predicting Stop-Signal Go-Trial RT Sd. from WAIS – Saddler 2 Composite. *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.065**	(1, 109)	0.007**		
i. WMS – Working Memory				.256**	.256**
Full Model	0.115	(7, 103)	0.067		
i. WMS – Working Memory				.219*	.212*
ii. Self-Deception				.033	.028
iii. Impression Management				−.004	−.003
iv. Task Completion TOTAL				.106	.091
v. Age				−.041	−.040
vi. Sex				.014	.014
vii. Race				−.178	−.172
Durban-Watson Statistic = 2.087					

Table A.18. Hierarchical regression predicting Stroop Interference from WMS – Working Memory. *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.051*	(1, 109)	0.020*		
i. WAIS – Saddler 2 Composite				.225*	.225*
Full Model	0.113	(7, 103)	0.083		
i. WAIS – Saddler 2 Composite				.213*	.207*
ii. Self-Deception				.096	.083
iii. Impression Management				−.056	−.050
iv. Task Completion TOTAL				.117	.101
v. Age				−.054	−.053
vi. Sex				.003	.003
vii. Race				−.173	−.166
Durban-Watson Statistic = 2.121					

Table A.19. Hierarchical regression predicting Stroop Interference from WAIS – Saddler 2 Composite. *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Model and Predictor	R^2	*df*	*Sig. F Change*	*β*	sr
Zero Order Model	0.235**	(1, 109)	0.000**		
i. WAIS – Saddler 2 Composite				.485**	.485**
Full Model	0.314**	(7, 103)	0.647		
i. WAIS – Saddler 2 Composite				.512**	.497**
ii. Self-Deception				.214*	.185
iii. Impression Management				−.182	−.163
iv. Task Completion TOTAL				.059	.051
v. Age				−.057	−.056
vi. Sex				−.085	−.083
vii. Race				−.040	−.038
Durban-Watson Statistic = 2.090					

Table A.20. Hierarchical regression predicting WMS – Working Memory from WAIS – Saddler 2 Composite. *Note:* R^2 = squared multiple correlation. *Sig. F. Change* = p-value of significance of change in R^2 with addition of predictor. *B* = standardized beta. sr = semi-partial correlation. * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed. Self-Deception = BIDR Self-Deception. Impression Management = BIDR Impression Management.

Variable 1	Variable 2	r_{12}	sr_{12}
7. ATQ-EC Total	HRV – Resting RMSSD	0.27**	0.257**
8. ATQ-EC Total	HRV – Ln (Resting HF Power)	0.25**	0.230**
9. ATQ-EC Attentional Control	WMS – Working Memory	0.19*	0.116
10. ATQ-EC Inhibitory Control	HRV – Resting RMSSD	0.23*	0.244**
11. ATQ-EC Inhibitory Control	HRV – Ln (Resting HF Power)	0.18*	0.185*
12. ATQ-EC Inhibitory Control	Stop-Signal – Go-Trial RT Sd.	−0.19*	−0.180
13. ATQ-EC Activation Control	HRV – Resting RMSSD	0.27**	0.227**
14. ATQ-EC Activation Control	HRV – Ln (Resting HF Power)	0.26**	0.207*
15. ATQ-EC Activation Control	WAIS – Saddler 2 Composite	−0.34**	−0.326**
16. Stop-Signal Go-Trial RT	WAIS – Saddler 2 Composite	−0.19*	−0.177
17. Stop-Signal Go-Trial RT Sd.	WAIS – Saddler 2 Composite	−0.31**	−0.299**
18. Stroop Interference	WMS – Working Memory	0.26**	0.212*
19. Stroop Interference	WAIS – Saddler 2 Composite	0.23*	0.207*
20. WMS – Working Memory	WAIS – Saddler 2 Composite	0.49**	0.497**

Table A.21. Summary of Statistical Significant Correlation and Semi-Partial Correlations. *Note:* Integer to the left of variable name corresponds with table # in Appendix A from which the coefficients were obtained. r_{12} = zero-order correlation derived from zero-order model multiple linear regression. sr_{12} = semi-partial correlation derived from full model multiple linear regression (with all covariates included). * = Significant at $p < 0.05$, two-tailed. ** = Significant at $p < 0.01$, two-tailed.

Variable 1	Variable 2	r_{12}	sr_{12}
Composite Executive Functions	ATQ-EC Total	0.112	0.030
Composite Executive Functions	ATQ-EC Attentional Control	0.137	0.000
Composite Executive Functions	ATQ-EC Inhibitory Control	0.149	0.079
Composite Executive Functions	ATQ-EC Activation Control	−0.011	−0.020
Composite Executive Functions	Attentional Control Scale	0.201*	0.075
Composite Executive Functions	HRV – Resting RMSSD	−0.063	−0.028
Composite Executive Functions	HRV – Ln (Resting HF Power)	−0.062	−0.053

Table A.22. Summary of Correlations and Semi-Partial Correlations between Composite Executive Function and Key Measures of Self-Regulation. *Note:* Composite Executive Functions = Sum of Standardized Scores (WMS-Working Memory Index, Stroop Interference T-Score, Log10WCST Perseverative Errors, Log10Stop-Signal RT). r_{12} = zero-order correlation between Variable 1 and Variable 2. sr_{12} = semi-partial correlation derived from full model multiple linear regression (with all covariates included). * = Significant at $p < 0.05$, two-tailed.

Variable	Range Observed (a)	Range Possible/Typical (b)
ATQ-EC Total	43 – 123	19 – 133
ATQ-EC Attentional Control	7 – 35	5 – 35
ATQ-EC Inhibitory Control	13 – 43	7 – 49
ATQ-EC Activation Control	16 – 49	7 – 49
Attentional Control Scale	32 – 80	20 – 80
BIDR Self-Deception	0 – 15	0 – 20
BIDR Impression Management	0 – 15	0 – 20
HRV – Resting RMSSD	6.32 – 182.45	typical
HRV – Ln (Resting HF Power)	2.17 – 9.63	typical
Stop-Signal RT	57.89 – 456.51	na
Stop-Signal – Go-Trial RT	332.27 – 797.58	na
Stop-Signal – Go-Trial RT Sd.	57.95 – 226.58	na
Stroop Interference	40 – 80	21 – 80
WCST – Perseverative Errors	28 – 79	19 – 80
WMS – Working Memory	79 – 146	49 – 155
WAIS – Saddler 2 Composite	12 – 33	2 – 38

Table A.23. Summary of Variable Range Values Obtained and Variable Range Values Possible. *Note:* a = Range of values observed in our sample. b = Range of possible values that could have been assumed by measure. Typical = range is consistent with general expectations for an undergraduate sample. na = no standard range available.

Appendix B
Distributional Analyses

Visual Inspection of Univariate Frequency Histograms

The most frequently used visual tool for *qualitatively* assessing a sample's normality is the histogram (Cohen et al., 2003; Hays, 1994; Henderson, 2006), which allows for a quick visual inspection of data symmetry and spread. Figures B.1 – B.5 show separate data distributions for every major variable included in this study. To facilitate interpretation, overlaying each sample distribution is a normal curve with the same mean and standard deviation as its corresponding variable.

An examination of the plots in Figures B.1 − B.5 indicated possible asymmetry (i.e. skewness) within the distributions of some variables. Variables that demonstrated potential signs of *positive* skew (i.e. where much of the distribution is concentrated on the left of the figure and the right tail is longer) included BIDR Self-Deception (Figure B.2), HRV – Resting RMSSD (Figure B.3), and all three measurements from the Stop-Signal Task (Figure B.4). By contrast, only HRV – Ln (Resting HF Power), shown in Figure B.3, evidenced possible signs of *negative* skew (where much of the distribution is concentrated on the right of the figure and the left tail is longer).

Regarding the extent to which sample data clustered around a central point, or resembled the probability densities of the normal distribution, several variables

evidenced potential signs of kurtosis. Among those variables whose distributions showed possible signs of *positive* kurtosis, or *leptokurtosis* (where order data assume values closer to the mean, and the extremes, of a distribution than normally distributed data, as evidenced by a more pronounced "peak" near the mean and "fatter" tails, than the normal curve), were both measures of heart-rate variability (Figure B.3), HRV – Resting RMSSD and HRV - Ln (Resting HF Power), Stop-Signal RT (Figure B.4), and Stroop Interference and the WAIS – Saddler 2 Composite (Figure B.5). One variable that displayed potential signs of *negative* kurtosis, or *platykurtosis* (where order data assume values not as close to the mean, nor the extremes, of a distribution than normally distributed data, as evidenced by a wider "peak" near the mean and "thinner" tails than a normal curve), was ATQ-EC Inhibitory Control (Figure B.1).

Visual Inspection of P-P Plots & Q-Q Plots

Whereas frequency histograms provide a useful, qualitative way to assess the distributional properties of sample data, probability-probability (P-P) and quantile-quantile (Q-Q) plots can provide *semi-quantitative* evidence of deviations from a specified distribution (in this context, the normal distribution). Both methods do so by plotting order statistics (i.e. observed data) against the *expected values* of those order statistics from the distribution of interest (Henderson, 2006). However, since the particular parameters of any distribution are rarely known, both methods must estimate these unknown parameters before the expected values themselves can be estimated.

While the particulars of how these expected values are estimated are beyond the scope of the present discussion, it is worth noting that both methods derive these values from a cumulative distribution function (CDF) for each random variable. Consequently, both plots are interpreted similarly when drawing inferences about how closely a data set approximates a specified distribution (e.g. normal distribution) and, thus, either method could be used to assess deviations from statistical normality. However, inclusion of both plots was viewed as being potentially useful in the present context, since the P-P plot is believed to be better at assessing goodness-of-fit near the center of a data distribution compared to the Q-Q plot, which by contrast is believed to be a better goodness-of-fit measure at the tails of a data distribution (Gan, Koehler, & Thompson, 1991; Holmgren, 1995).

Probability-probability (P-P) plots for every major variable included in this study can be found in Figures B.6 – B.10. Deviations from normality are indicated by data patterns that approximate an "S-curve" and/or "banana" or "crescent" shaped data plot (Henderson, 2006). Variables that demonstrated possible deviations from normality (especially near the center of their respective data distributions) were both BIDR subscales (Figure B.7), both heart-rate variability indices (Figure B.8), and all three measurements from the Stop-Signal Task (Figure B.9).

Corroborating the visual evidence of non-normality gleaned from the P-P plots are the quantile-quantile (Q-Q) plots shown in Figures B.11 − B.15, which, like the P-P plots, evidenced possible deviations from normality (especially near the tails of the data distributions) for all measurements of socially desirable responding (BIDR;

Figure B.12), heart-rate variability (Figure B.13), and Stop-Signal Task performance (Figure B.14). These Q-Q plots also showed potential deviations in normality in WCST – Perseverative Errors and WAIS – Saddler 2 Composite (Figure B.15) performance, the latter inference of which would be consistent with our observation of kurtosis in the WAIS – Saddler 2 Composite distribution in the previous section.

Left unclear by the visual inspections of the histograms presented in the previous section, and the P-P plots and Q-Q plots in this section, was whether these *potential* deviations from normality were genuine and, if so, were they of a sufficient magnitude to have attenuated our correlation coefficients. This was especially the case with the ATQ subscales, which displayed (see Figures B.1, B.6, and B.11) seemingly minor deviations from statistical normality (e.g. signs of *platykurtosis* in ATQ-EC Inhibitory Control) but were less clearly non-normal than many of the other variables discussed. Such ambiguity both underscores the near-ubiquity of non-normality in behavioral science data (Bradley, 1982; Levine, 2005; Micceri, 1989) and highlights the limited utility of visual inspection in determining the extent to which a data distribution approximates a distribution of interest. Thus, while visual inspection of data plots may be useful when clear signs of kurtosis, skewness, and other factors (e.g. outliers) that can significantly affect statistical tests are present, quantitative indices of normality might be of particular value when visual evidence of non-normality is far from compelling.

Quantitative Assessment of Normality

Since the advent of modern statistics many tests have been devised to surmise the distributional properties of sample data (for review, see Henderson, 2006). These tests have ranged from the classical chi-square (χ^2) goodness-of-fit test (Pearson, 1900) and tests of central (C-moment), linear (L-moment), and absolute moments (Cornish & Fisher, 1937; Cramér, 1946; Hosking, 1990), to more contemporary approaches based on statistical regression or empirical distribution functions, like the Shapiro-Wilk (Shapiro & Wilk, 1965) and modified Kolmogorov-Smirnov (Lilliefors, 1967) tests, respectively. Although one must certainly consider a variety of factors (e.g. hypothesized distribution specifications, sample size, data scale) when choosing among tests of departure from normality, comprehensive studies comparing the effectiveness of these tests (Gan & Koehler, 1990; Filliben, 1975; Pearson, D'Agostino, & Bowman, 1977; Shapiro, Wilk, & Chen, 1968; Stephens, 1974) have generally found the Shapiro-Wilk test to be a superior test of non-normality for samples of up to 50 participants, and the Kolmogorov-Smirnov (Lilliefors modification) test to be best suited for measuring non-normality in samples consisting of more than 50 participants (Henderson, 2006; Thode, 2002). Therefore, our *quantitative* test of choice for measuring statistical non-normality was the Kolmogorov-Smirnov (Lilliefors modification) test (Lilliefors, 1967; Mason & Bell, 1986). For a detailed discussion of the mathematical theory underlying the Kolmogorov-Smirnov (Lilliefors modification) test statistic, please refer to Henderson (2006).

Consistent with standard statistical practices and recommendations (Balanda & MacGillvray, 1988; Cohen et al., 2003; D'Agostino, 1986), we also calculated kurtosis and skewness statistics to accompany results from the modified Kolmogorov-Smirnov (K-S) test to further ascertain the extent to which variable distributions deviated from normality. Whereas the Kolmogorov-Smirnov test provides a robust *general* estimate of non-normality, separate skewness and kurtosis statistics (not provided by the K-S test) provide specific information concerning *how* a data distribution deviates from a hypothetical distribution of interest (e.g. normal distribution). Indices of skewness and kurtosis are widely available for use in most statistical packages, and have been consistently shown to have excellent statistical properties (Balanda & MacGillvray, 1988, p. 114; Cohen et al., 2003; D'Agostino et al., 1990; DeCarlo, 1997), including those normality indices provided by SPSS (SPSS Inc., 2008): the statistical program utilized for these and many of our other analyses.

Presented in Table B.1 one will find the results obtained from our modified Kolmogorov-Smirnov tests, as well as our skewness and kurtosis test statistics. When interpreting these test statistics, the reader should note that a normal distribution would have skewness and kurtosis values of 0, whereas positive and negative test statistic values are indicative of positive skew (or kurtosis) and negative skew (or kurtosis), respectively. As shown in Table B.1, the data distributions of no self-report measure of effortful control or attentional control deviated significantly ($\alpha = 0.05$) from a Gaussian distribution, allaying concerns about skewness and kurtosis within those key study variables. However, consistent with the evidence of non-normality

presented in Figures B.2, B.7, and B.12, it would appear that both BIDR subscales deviated significantly from normality. The self-deception subscale of the BIDR, in particular, evidenced significant signs of positive skew (0.638), whereas the impression management subscale of the BIDR appeared to have been significantly non-normal in the aggregate but, nonetheless, displayed no statistically significant signs of either skew or kurtosis.

With respect to measures of heart-rate variability, only HRV – Resting RMSSD showed evidence of significant positive skew (1.261) and kurtosis (2.087), and had a correspondingly high Kolmogrov-Smirnov (K-S) test statistic value. By contrast, our other measure of heart-rate variability, HRV – Ln (Resting HF Power), displayed significant signs of negative skew (–0.480) but did not yield a significant K-S value. This finding with respect to HRV – Ln (Resting HF Power) could indicate that while the data distribution for this variable was negatively skewed, the skewness was not of magnitude sufficient to yield a significant difference between the empirical distribution function and normal cumulative distribution for this variable (perhaps because kurtosis was so minimal; see Henderson, 2006 and Mason & Bell, 1986).

Notwithstanding these complicated findings with respect to HRV – Ln (Resting HF Power), it was our view that the evidence of skew – a near ubiquitous property of heart-rate variability distributions (Agelink, Boz, Ullrich, & Andrich, 2002; Bhattacharyya, Whitehead, Rakhit, & Steptoe, 2008; Britton et al., 2007; Carney, Blumenthal, Stein, Watkins, Catellier, Berkman, Czajkowski, O'Connor, Stone, & Freedland, 2001; Goldberger, Challapalli, Tung, Parker, & Kadish, 2001;

Koskinen et al., 2009; Kuo et al., 1999; Kupper, Willemsen, van der Berg, de Boer, Posthuma, Boomsma, de Geus, 2004; Maestri, Pinna, Porta, Balocchi, Sassi, Signorini, Dudziak, & Raczak, 2007; McCraty, Atkinson, Tomasino, & Stuppy, 2001; Nunan, Jakovljevic, Donovan, Hodges, Sandercock, & Brodie, 2008; Reed, Warburton, Whitney, & McKay, 2006; Sinnreich et al., 1998; Stein, Barzilay, Chaves, Domitrovich, & Gottdiener, 2009; Stein, Carney, Freedland, Skala, Jaffe, Kleiger, & Rottman, 2000; Stein, Domitrovich, Ambrose, Lyden, Fine, Gracely, & Clauw, 2004; Sztajzel, 2008; Umetani, Singer, McCraty, & Atkinson, 1998; Vuksanovic & Gal, 2007) – provided an adequate justification for later transforming this variable to minimize the potential effects of non-normality on our second set of correlation/regression analyses.

Consistent with the visual evidence of non-normality gleaned from the previous sections, the data distributions for all three measures of Stop-Signal Task performance attained not only significant Kolmogorov-Smirnov test values, but also index values indicative of positive skew (see Table B.1). These findings are consistent with both the ubiquity of positive skew in response times on the Stop-Signal Task (Band, van der Molen, & Logan, 2003; Clark, Blackwell, Aron, Turner, Dowson, Robbins, & Sahaklan, 2007; Li, Chao, & Lee, 2009; Logan, 1994), and extensive research showing that reaction time distributions are often positively skewed (see Luce, 1986). Moreover, Stop-Signal RT attained an extremely high index of positive kurtosis (6.594), which was congruent with our interpretation of its corresponding frequency histogram presented in Figure B.4. Combined, these

findings provided ample justification for transforming the data obtained from the Stop-Signal Task for subsequent analyses.

Additional performance-based measures that showed significant departures from statistical normality were WCST – Perseverative Errors and WAIS – Saddler 2 Composite, both of which attained significant K-S values (see Table B.1). These values were consistent with our observations of non-normality in the Q-Q plots for these variables (see Figure B.15). However, running counter to the aforementioned visual inspection of the WAIS – Saddler 2 Composite (Figure B.5) frequency histogram (where possible signs of leptokurtosis emerged), neither of these variables attained significant skewness or kurtosis values. Combined, these findings may indicate that while the WAIS – Saddler 2 Composite and WCST – Perseverative Errors distributions are significantly non-normal, neither display signs of significant skew or kurtosis. As was the case with BIDR Impression Management, it was our view that the evidence of non-normality provided by the modified Kolmogorov-Smirnov test justified subsequent data transformations for these two variables to ascertain whether their distributions could have attenuated the correlation coefficients in our first set of analyses.

The reader may have taken notice that in none of the preceding sections were the distributional properties of the Task Completion Questionnaire (TCQ) subscales discussed. Our reasons for these omissions were twofold. First, while the TCQ may provide some useful information about factors that could have affected task performance, these scales were designed mostly for exploratory purposes and not

viewed as central to our hypotheses. As a result, it was our position that it was more important to minimize pagination where possible to facilitate reader comprehension. Second, while the TCQ has the potential to be a useful psychometric measure, any inferences derived from TCQ scores would be highly tentative because its statistical properties are far from clear at the present time.

Whether or not one takes issue with the soundness of this decision is ultimately moot, because as one can see in Table B.1, *quantitative* indicators of normality were computed for all TCQ subscales. Without exception, the modified K-S statistics for all of these subscales achieved statistical significance, indicating substantial departures from normality. Eight out of nine TCQ subscales also attained index values indicative of negative skew, demonstrating that the majority of respondents endorsed having experienced few problems (e.g. distraction, heightened stress) during task completion. Evidence of *platykurtosis* emerged for the *hand test*, *self-report*, and *task effort* subscales of the TCQ, which in combination with the negative skew observed in these variables, suggests that few respondents (a) endorsed substantial motivational difficulties while completing the tasks, or (b) endorsed significant problems when completing the self-reports (e.g. ATQ-EC) and manually administered tests of executive function (e.g. Wechsler Memory Scale).

Summary & Significance of Distributional Analyses

Most variables evidenced signs of significant distributional non-normality. Skewness was a particularly common distributional characteristic of our study variables, with nine variables showing negative skew and five variables evidencing

positive skew. By contrast, kurtosis was not a distributional property within our sample data, with only five variables showing signs of positive kurtosis. Although the high frequency of skew within our sample may have been cause for concern, the *relative* infrequency of kurtosis was even more striking given the nature of our correlational analyses. Sampling (Monte Carlo) studies have generally shown that tests of covariances are affected to a much larger degree by kurtosis than skewness (Browne, 1982, 1984; DeCarlo, 1997; Jobson, 1991, p. 55; Mardia, Kent, & Bibby, 1979, p. 149). While both skewness and kurtosis can substantially impact correlation coefficients, the emergence of kurtosis in only five variables arguably allowed for greater latitude during the data transformation process, which can often necessitate trade-offs between decreasing skewness or kurtosis at the expense (or increasing) of the other (Cleveland, 1984; Ruppert & Aldershof, 1989; Yanagihara & Yuan, 2005). Similarly, while skewness can impact tests of both means and covariances, these effects are much more pronounced in directional (or one-tailed) tests of statistical significance (Hopkins & Weeks, 2000) than in non-directional (or two-tailed) tests, like those utilized in the present study. Therefore, it was our view that while data transformation efforts would attempt to minimize both skewness and kurtosis, our primary concern would be on lessening kurtosis.

While the foregoing analyses dealt explicitly with the matter of distributional normality, it is worth noting that these analyses were ultimately aimed at addressing the matter of variable distribution similarity. Indeed, while extreme deviations from distributional *similarity* between two variables can attenuate their shared correlation or

regression coefficients (Carroll, 1961; Cohen et al., 2003; Hays, 1994), there is no expectation or assumption (Cohen et al., 2003; pp. 110-117) that the distributions of independent or dependent *variables* approximate a normal distribution in ordinary least squares regression or correlational analyses (however, as will be addressed later in our regression analyses, the *residuals* are assumed to be normally distributed). Unfortunately, while many statistical tests are available for comparing distributions of variable data to hypothetical distributions (e.g. Gaussian, Chi-Square, Gamma), tests for comparing empirical data distributions to *one another* are lacking.

In light of these issues, we adopted a two-stage strategy to test and subsequently correct for deviations from distributional similarity. First, we compared each variable's data distribution to the normal (i.e. Gaussian) distribution (i.e. the stage completed in this section). In those cases where significant deviations from statistical normality were evident, we then transformed non-normal distributions to a Gaussian distribution (see next section). It was our hope that this approach would ultimately yield data distributions that approximated the normal curve and, in so doing, increased the level of distributional similarity between study variables to help ensure that adequate assessments of their empirical relations could be conducted.

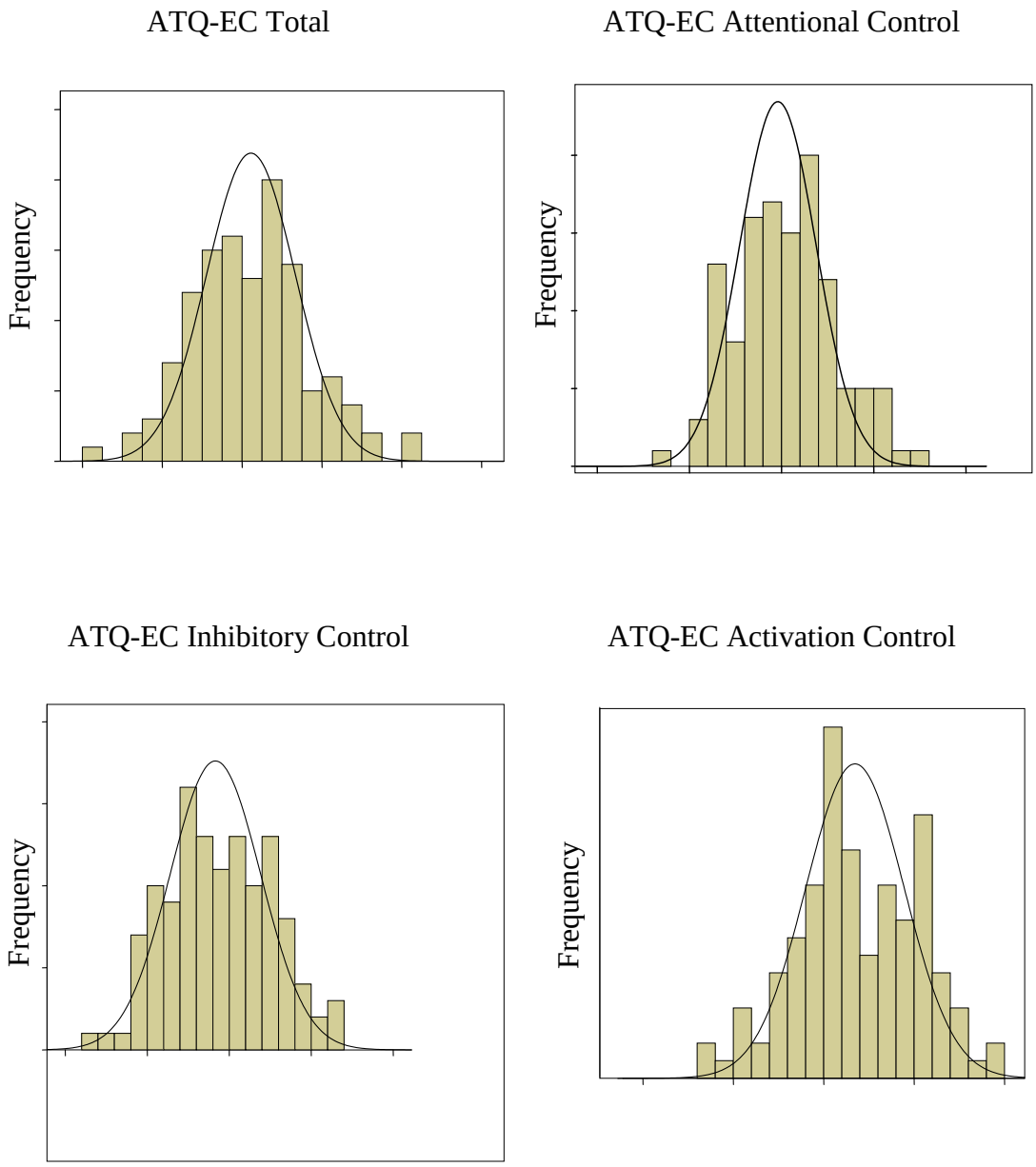


Figure B.1. Frequency Histograms for ATQ-EC and ATQ-EC Subscales.

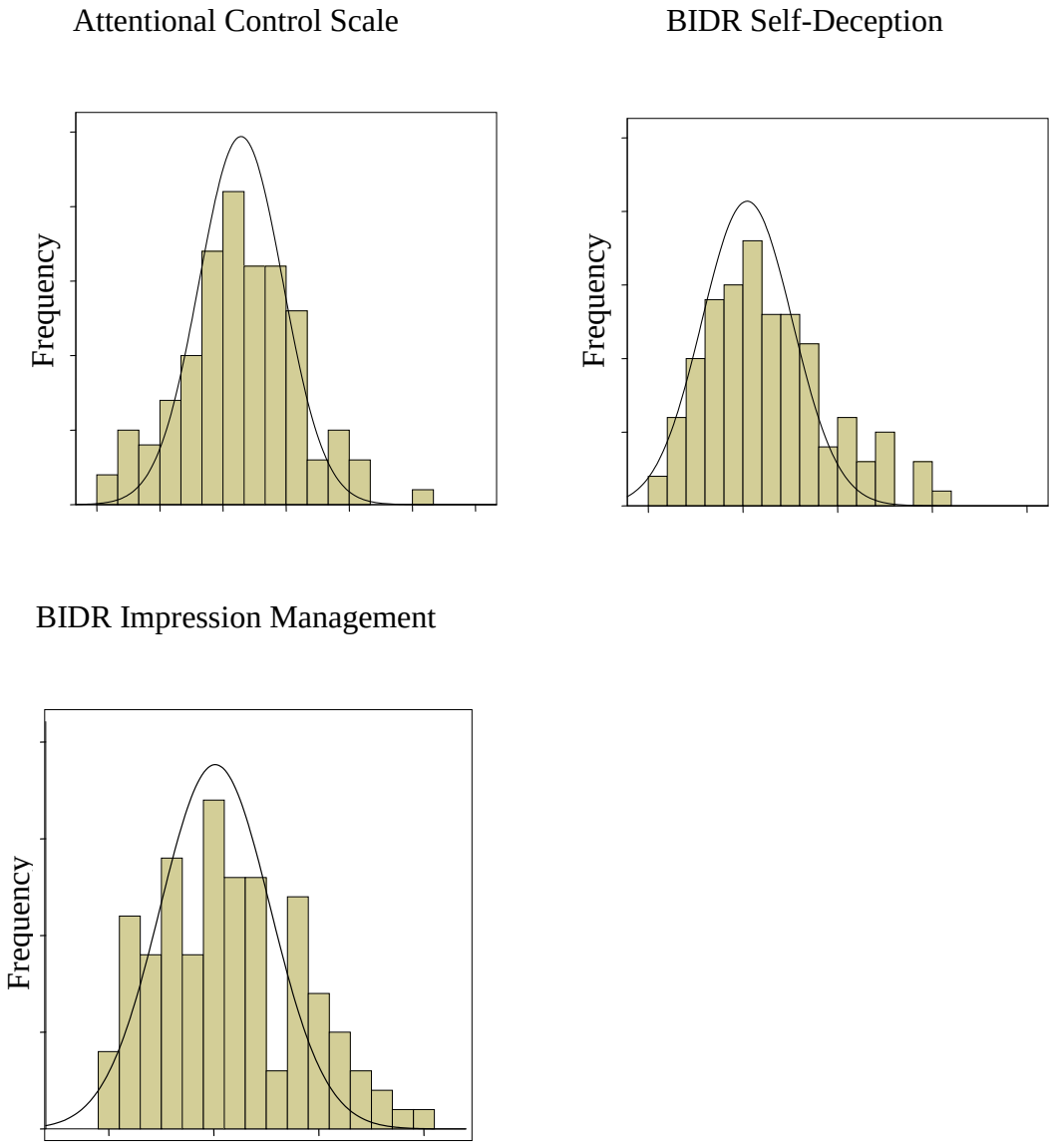


Figure B.2. Frequency Histograms for Attentional Control Scale and Balanced Inventory of Desirable Responding (BIDR).

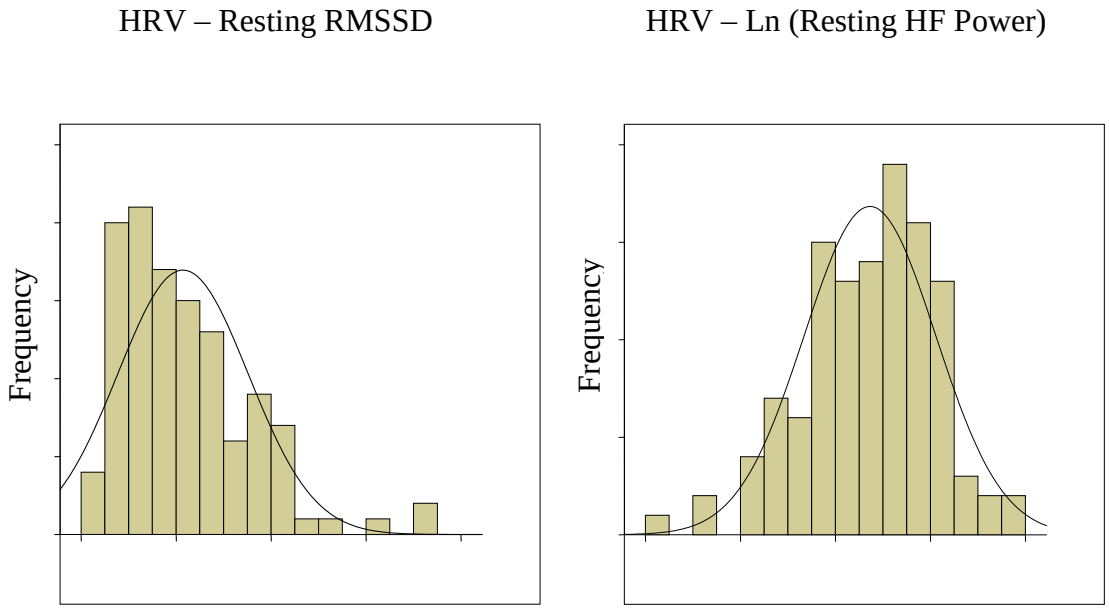


Figure B.3. Frequency Histograms for Heart Rate Variability (HRV) Measurements.

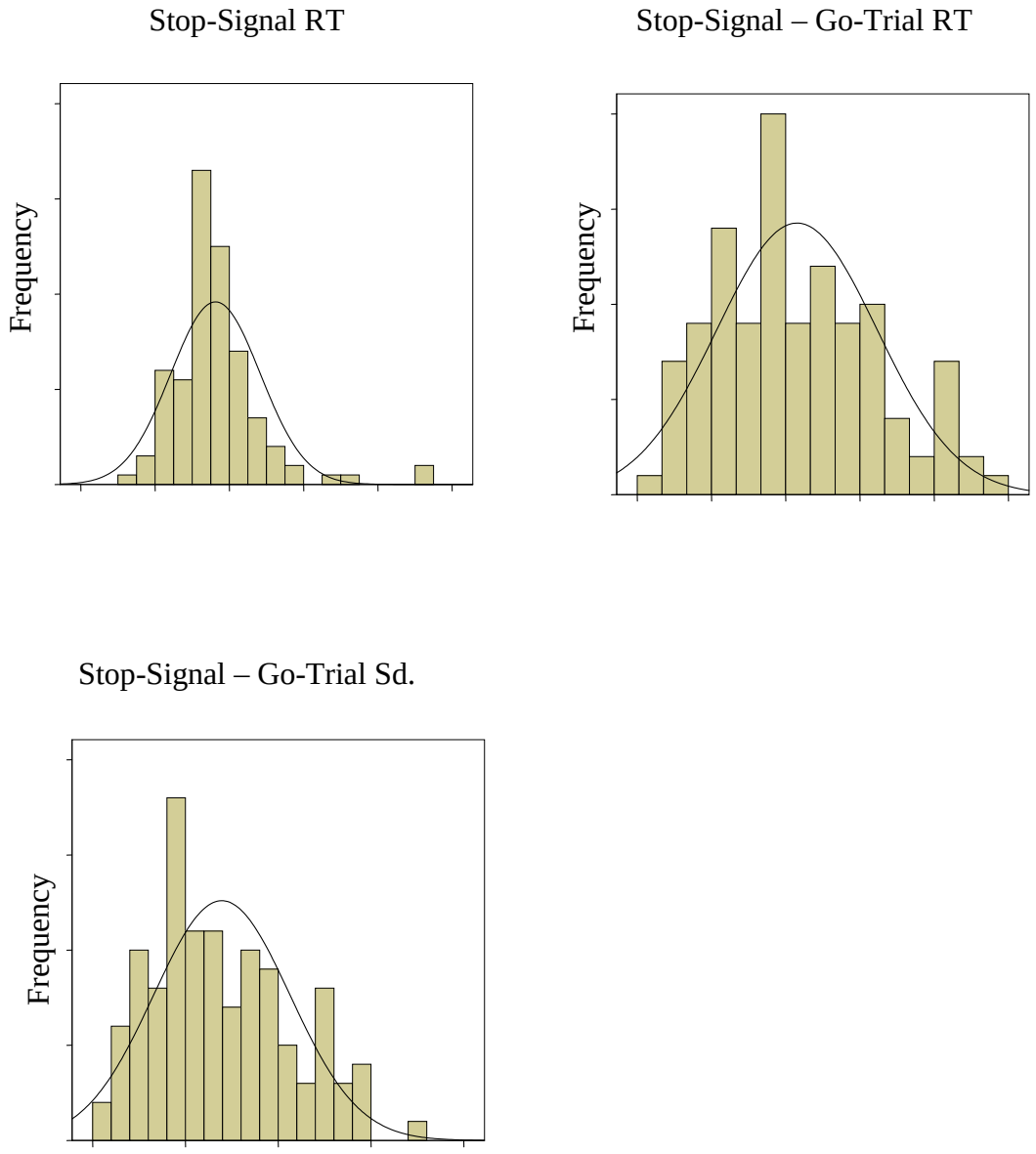


Figure B.4. Frequency Histograms for Stop-Signal Task.

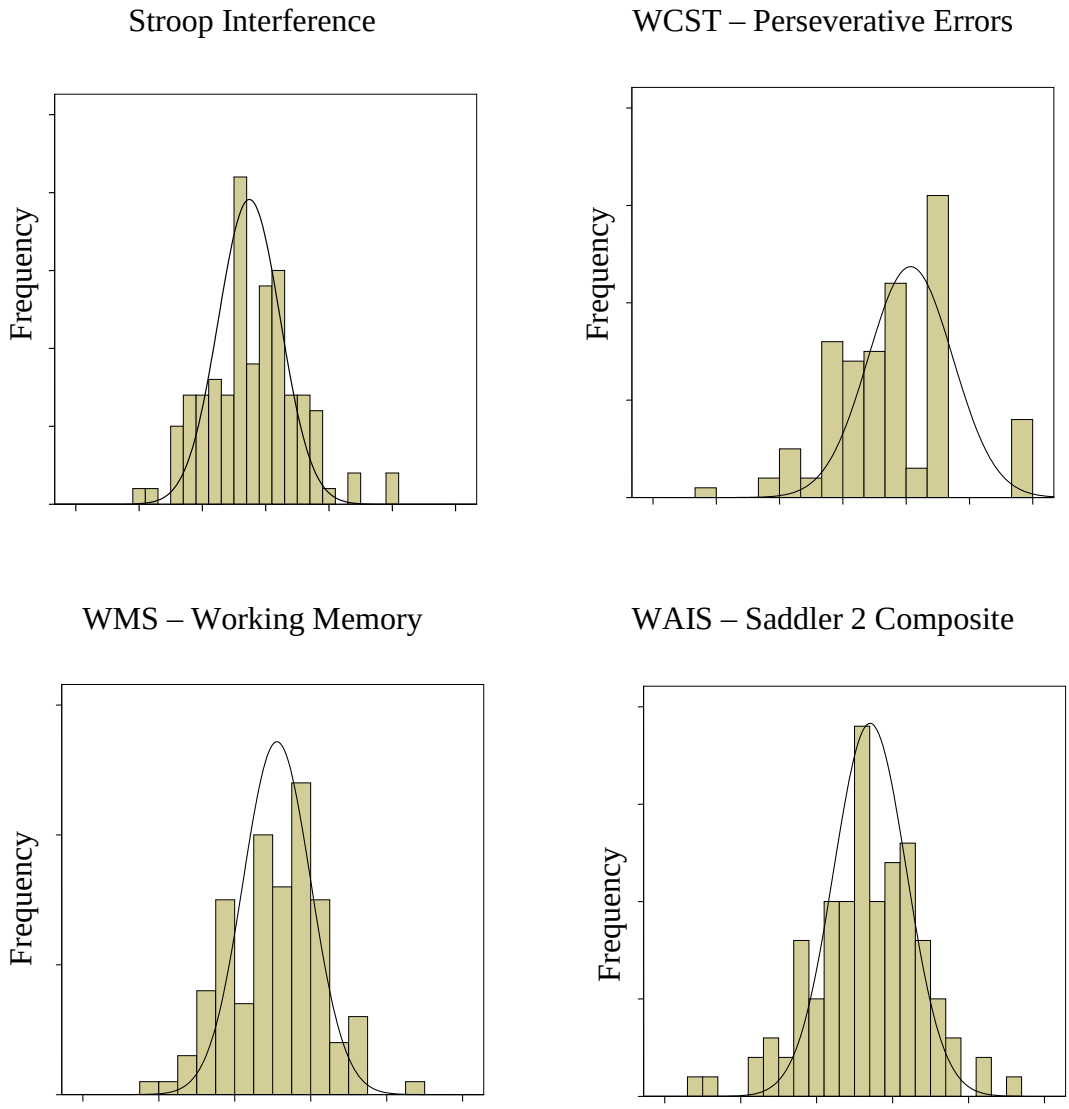


Figure B.5. Frequency Histograms for Stroop Color-Word Interference Test, WCST, WMS, and WAIS Saddler 2 Composite.

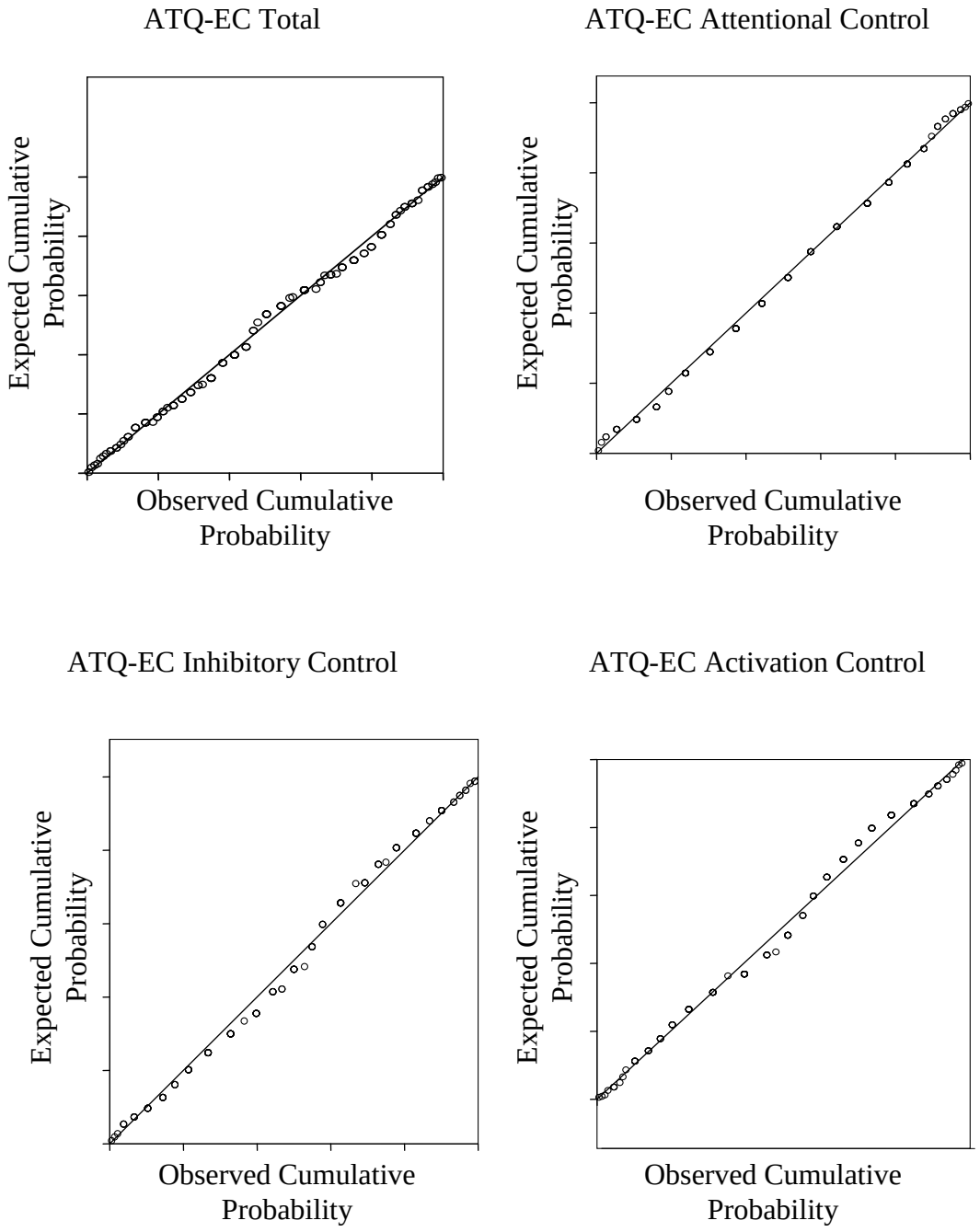


Figure B.6. Probability-Probability (P-P) Plots for ATQ-EC and ATQ-EC Subscales.

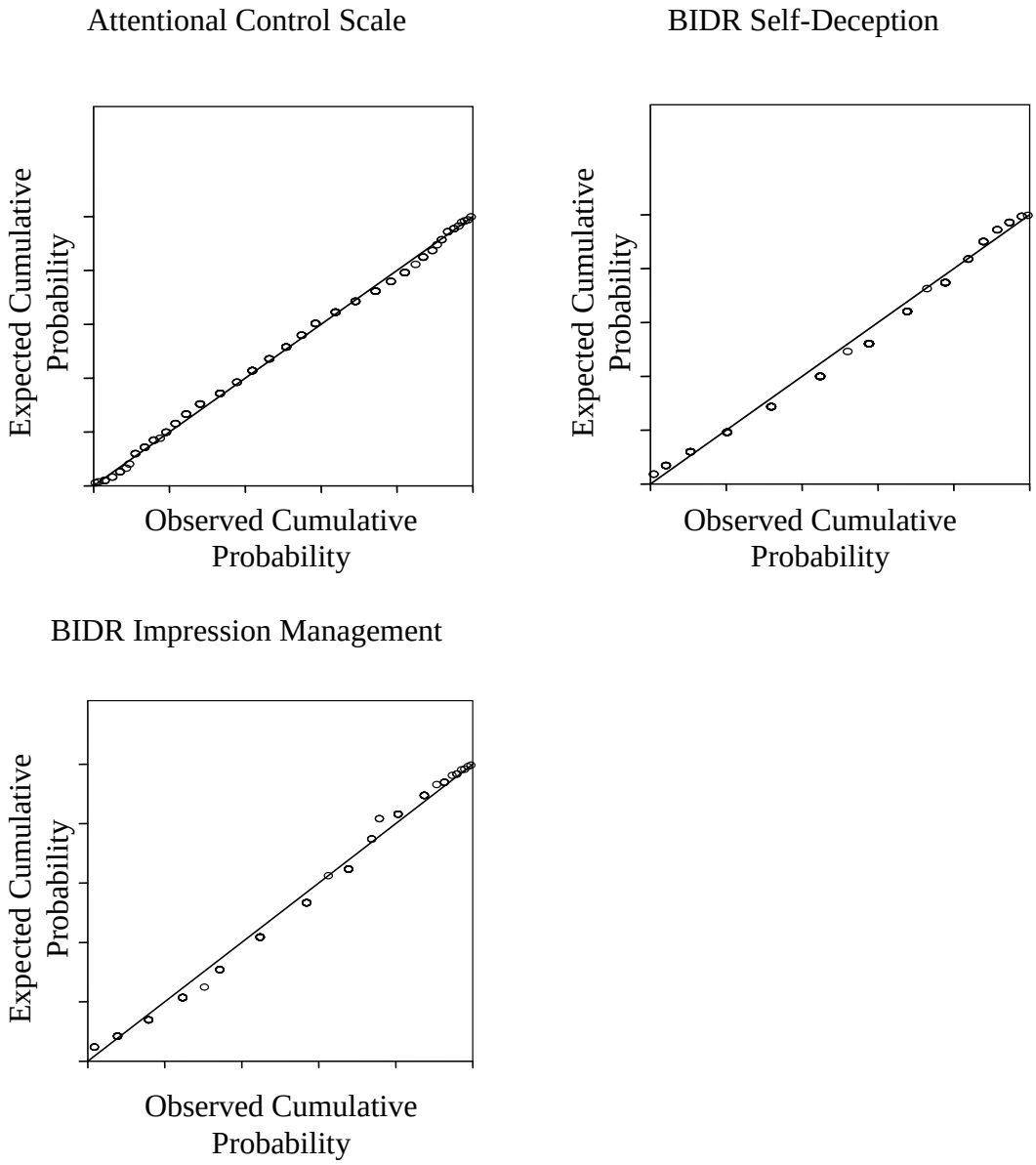


Figure B.7. Probability-Probability (P-P) Plots for Attentional Control Scale and Balanced Inventory of Desirable Responding (BIDR).

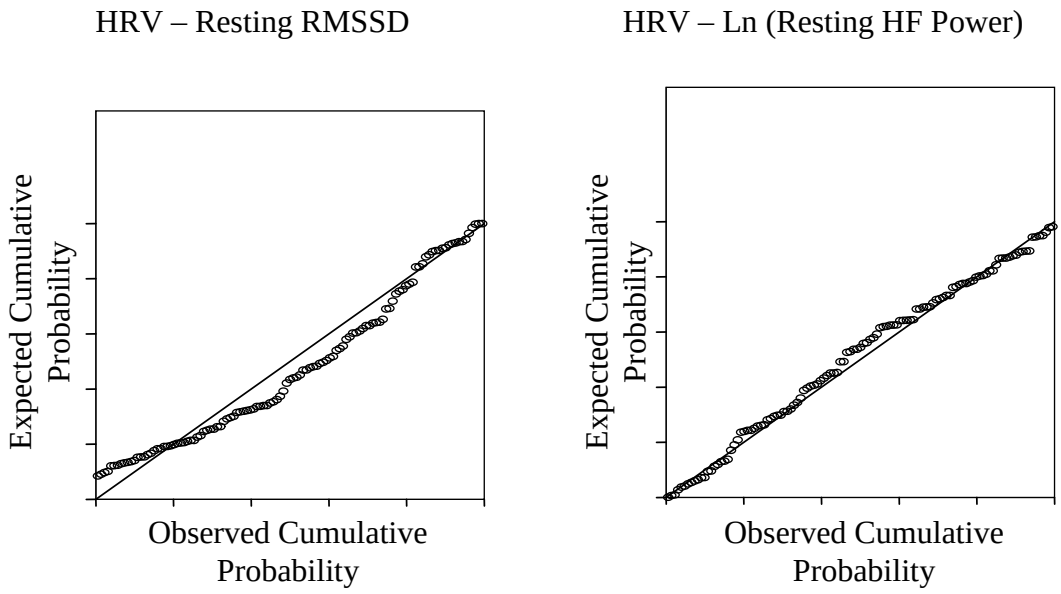


Figure B.8. Probability-Probability (P-P) Plots for Heart Rate Variability (HRV) Measurements.

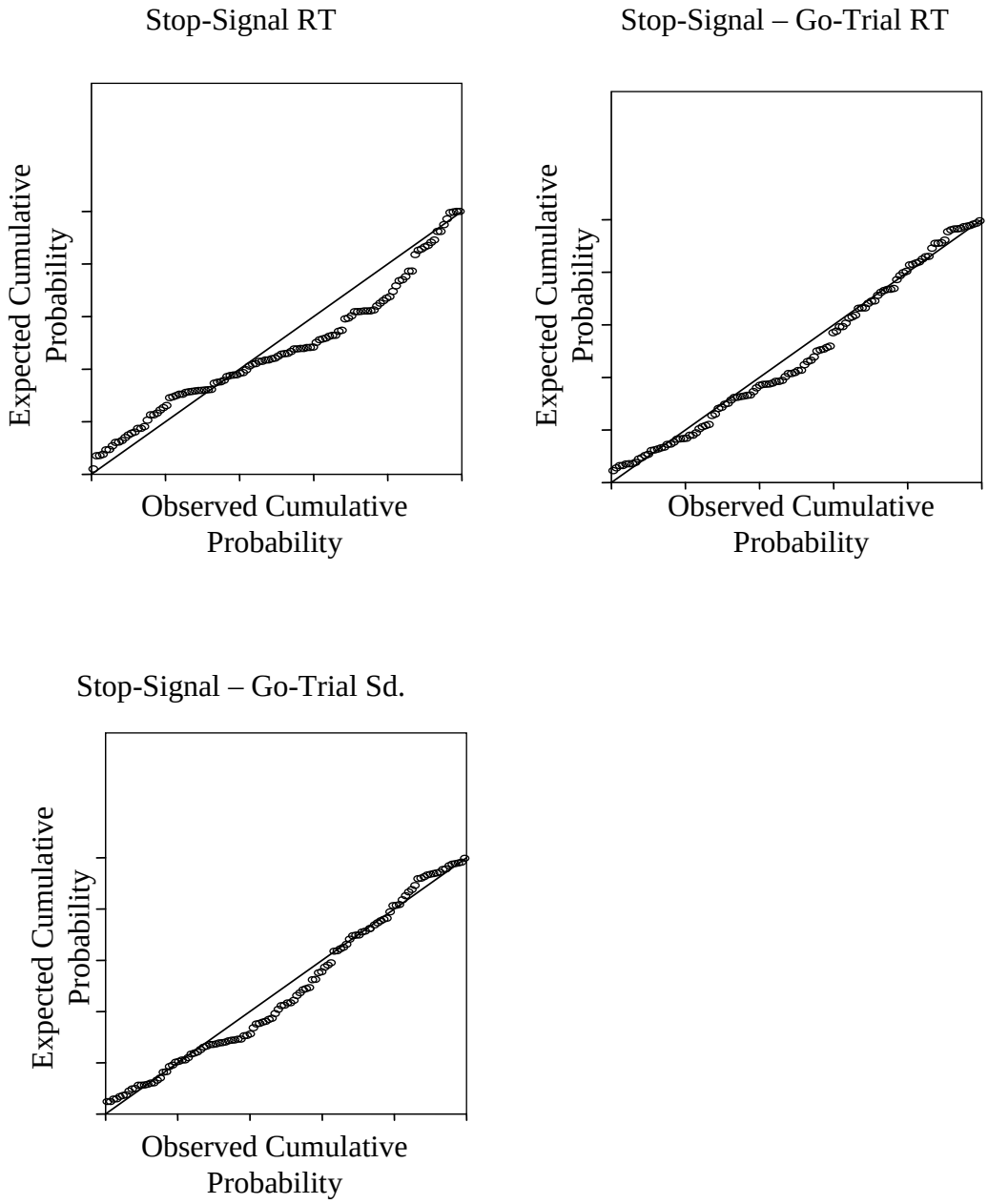


Figure B.9. Probability-Probability (P-P) Plots for Stop-Signal Task.

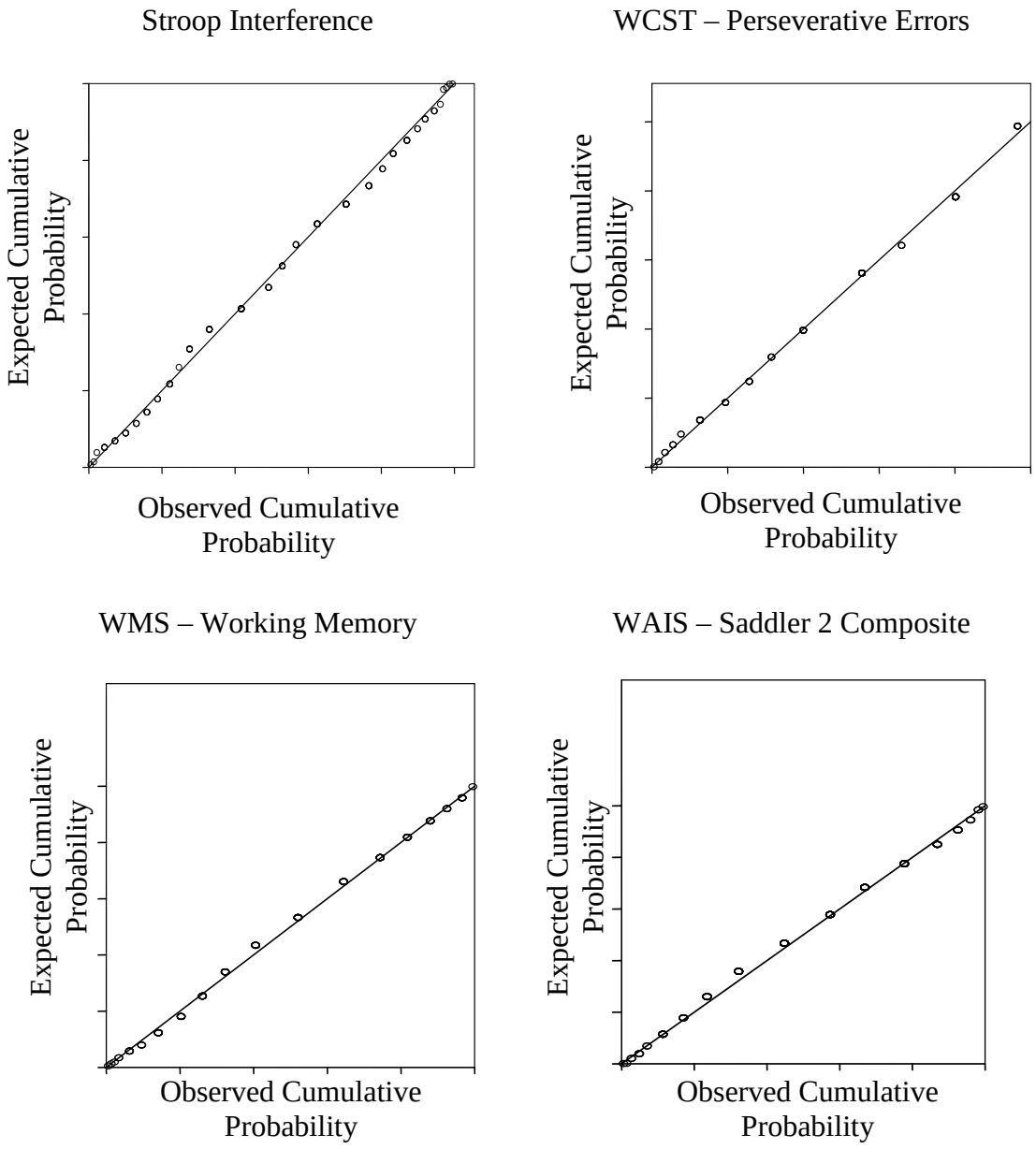


Figure B.10. Probability-Probability (P-P) Plots for Stroop Color-Word Interference Test, WCST, WMS, and WAIS Saddler 2 Composite.

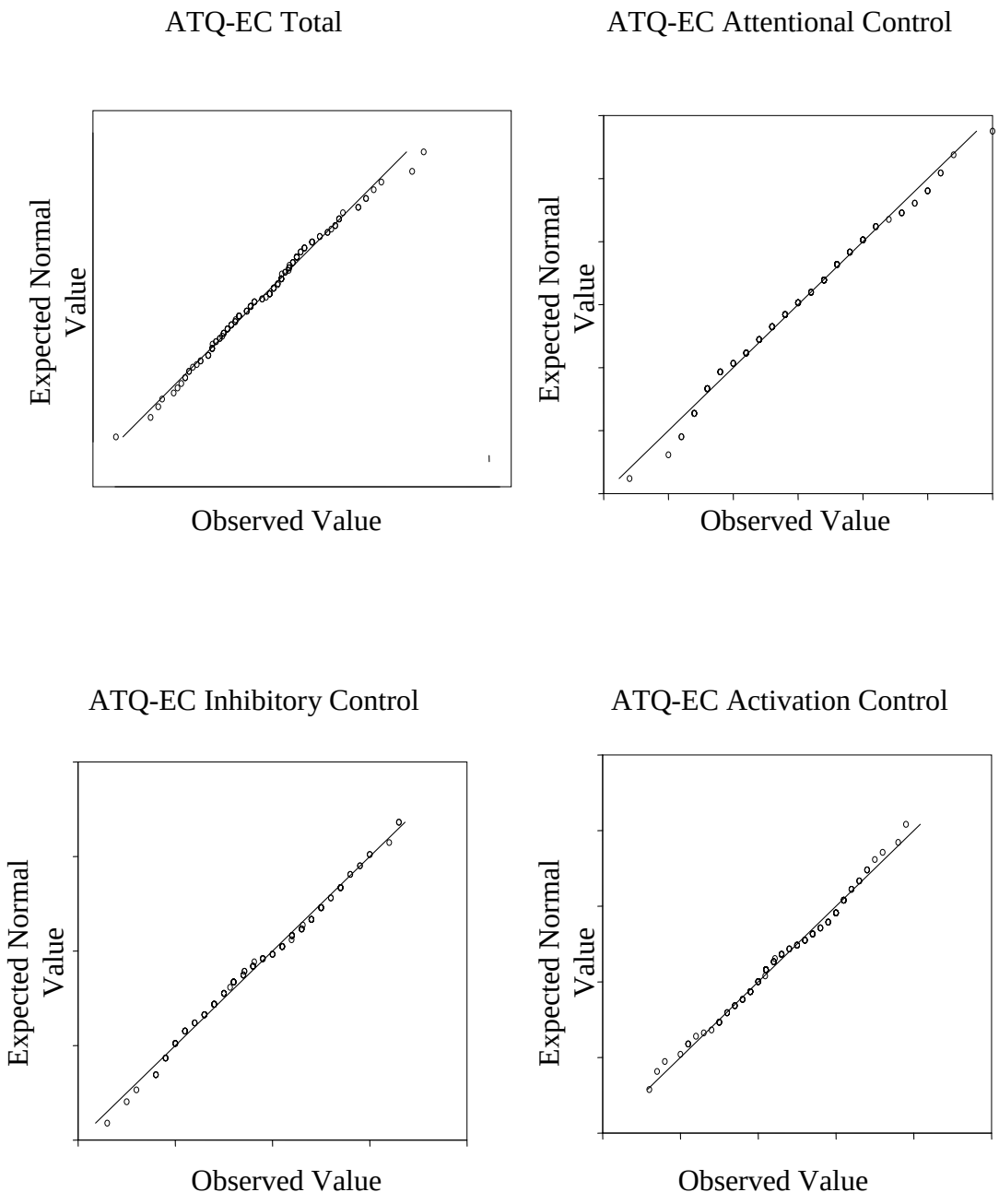


Figure B.11. Quantile-Quantile (Q-Q) Plots for ATQ-EC and ATQ-EC Subscales.

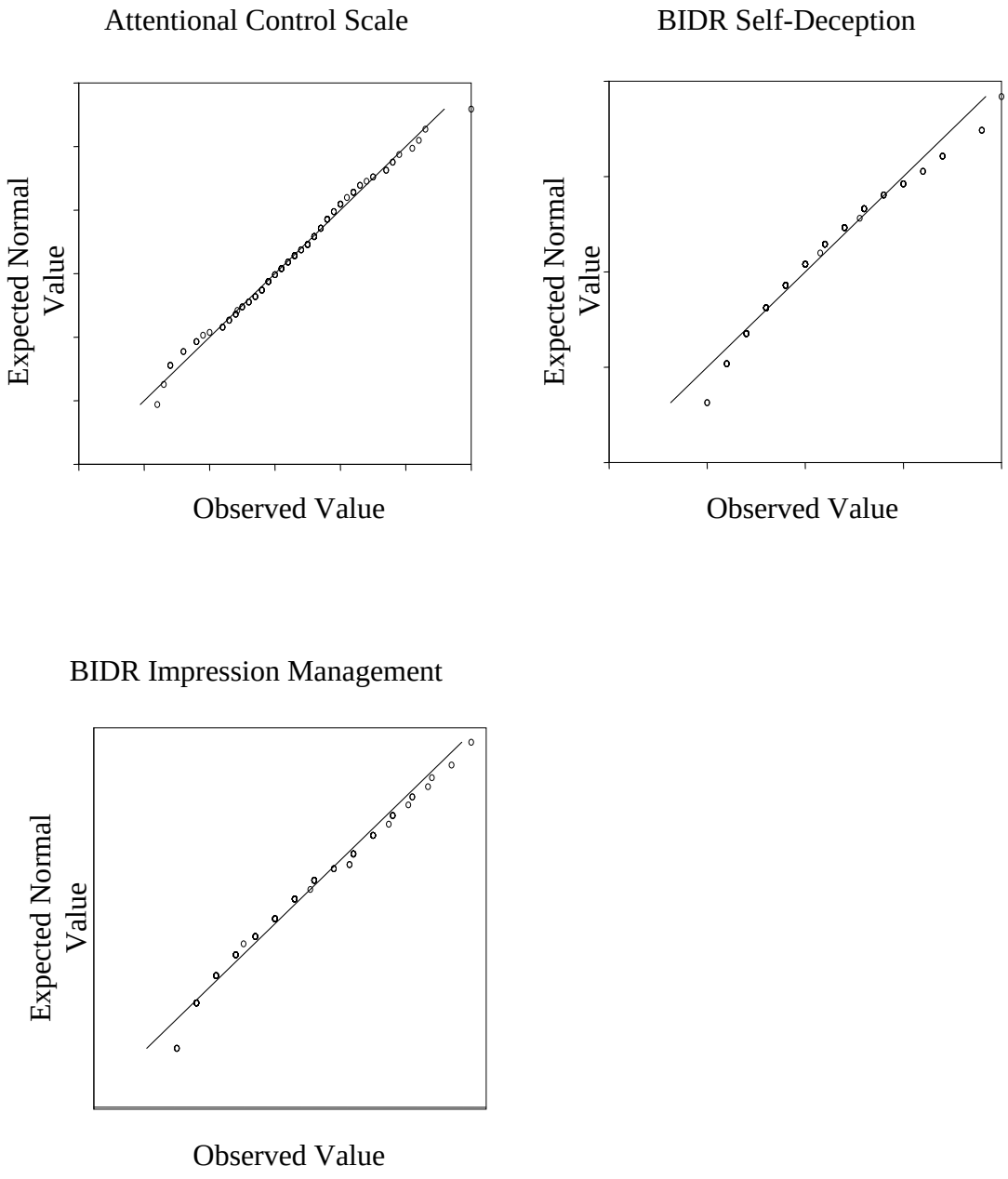


Figure B.12. Quantile-Quantile (Q-Q) Plots for Attentional Control Scale and Balanced Inventory of Desirable Responding (BIDR).

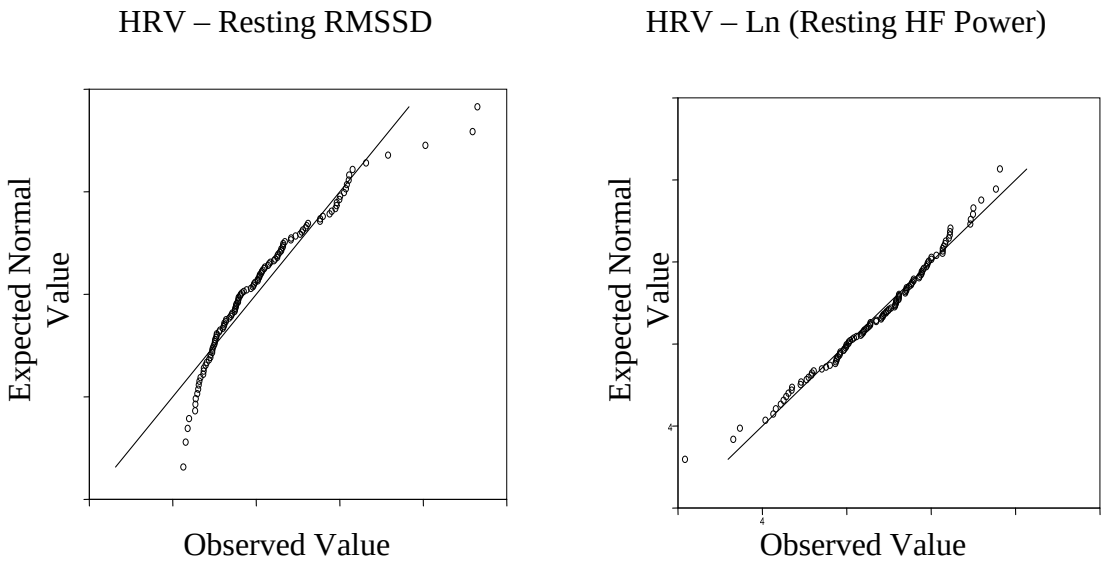


Figure B.13. Quantile-Quantile (Q-Q) Plots for Heart Rate Variability (HRV) Measurements.

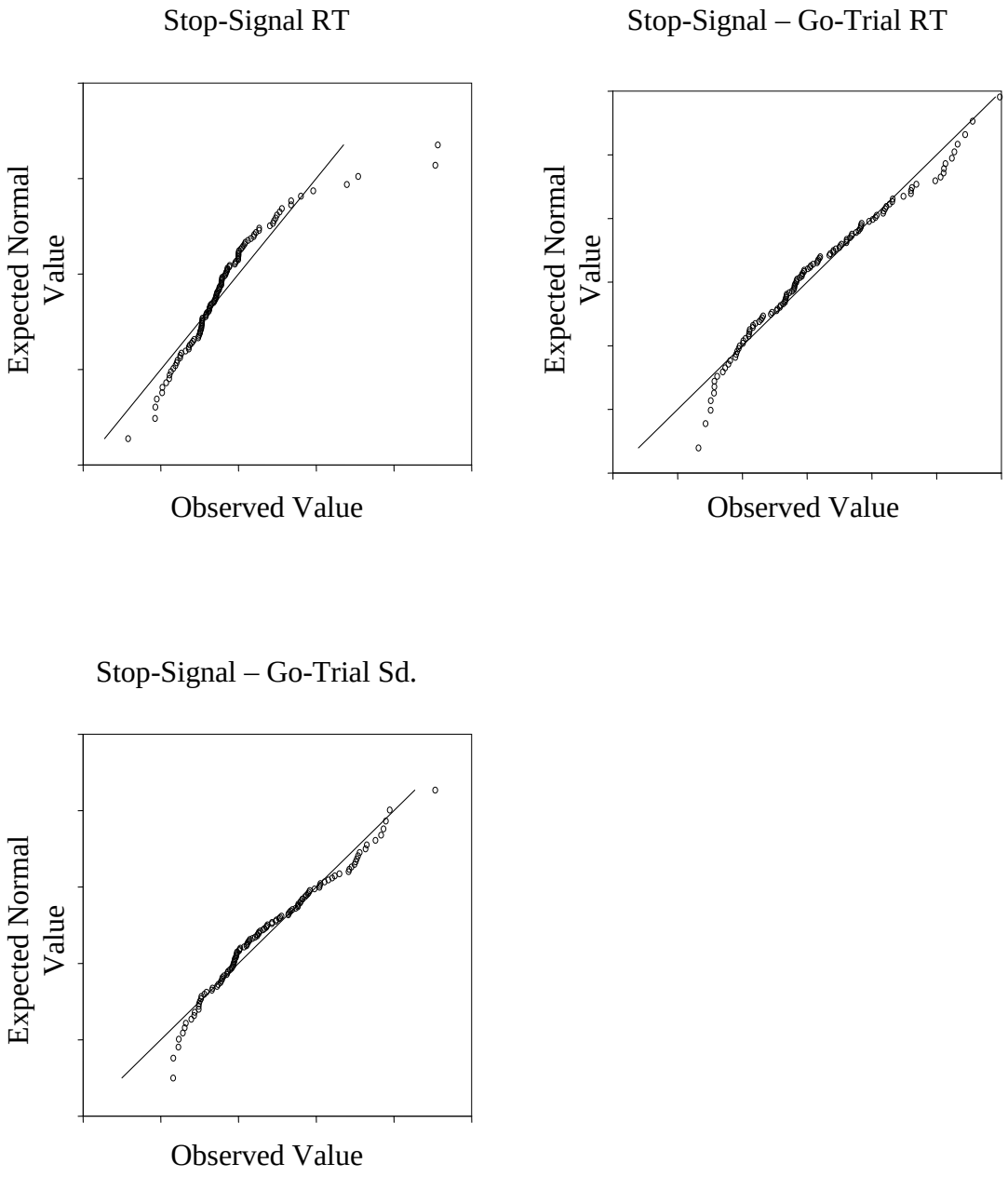


Figure B.14. Quantile-Quantile (Q-Q) Plots for Stop-Signal Task.

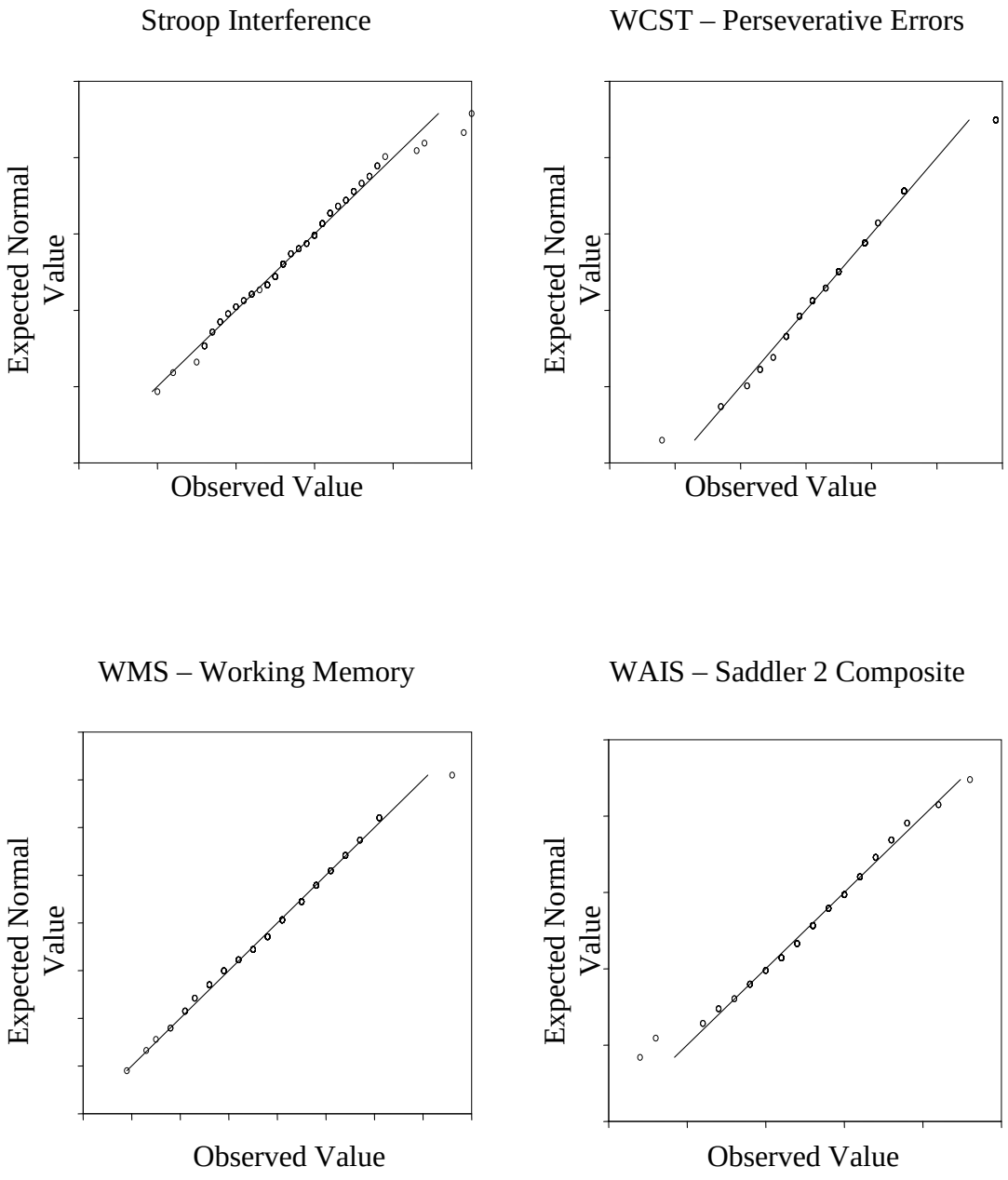


Figure B.15. Quantile-Quantile (Q-Q) Plots for Stroop Color-Word Interference Test, WCST, WMS, and WAIS Saddler 2 Composite.

	Normality	Skewness		Kurtosis	
	Kolmogorov-Smirnov Statistic[a]	Statistic	Std. Error	Statistic	Std. Error
1. ATQ – EC Total	.053	.193	.219	.199	.435
2. ATQ – EC Attentional Control	.054	.242	.219	–.219	.435
3. ATQ – EC Inhibitory Control	.071	.074	.219	–.598	.435
4. ATQ – EC Activation Control	.074	–.129	.219	–.351	.435
5. Attentional Control Scale	.054	.051	.218	.223	.433
6. BIDR Self-Deception	.117*	.638*	.217	.068	.431
7. BIDR Impression Management	.098*	.394	.217	–.471	.431
8. HRV – Resting RMSSD	.109*	1.261*	.224	2.087*	.444
9. HRV – Ln (Resting HF Power)	.069	–.480*	.224	.288	.444
10. Stop-Signal RT	.142*	1.918*	.225	6.594*	.446
11. Stop-Signal – Go-Trial RT	.090*	.490*	.225	–.443	.446
12. Stop-Signal – Go-Trial RT Sd.	.093*	.513*	.225	–.387	.446
13. Stroop Interference	.062	.294	.221	.629	.438
14. WCST – Perseverative Errors	.150*	.088	.222	.537	.440
15. WMS – Working Memory	.079	–.061	.220	–.083	.437
16. WAIS – Saddler 2 Composite	.102*	–.329	.226	.724	.447

Continued

Table B.1. Normality Diagnostics on Measures. *Note:* a = Computed with Lilliefors Significance Correction. * = Value significant at $p < 0.05$, two-tailed.

Table B.1. Continued

	Normality	Skewness		Kurtosis	
	Kolmogorov-Smirnov Statistic[a]	Statistic	Std. Error	Statistic	Std. Error
17. TCQ – TOTAL	.089*	−.614*	.222	−.126	.440
18. TCQ – COMPUTER	.120*	−.544*	.222	.097	.440
19. TCQ – HAND TESTS	.143*	−1.044*	.222	1.693*	.440
20. TCQ – SELF-REPORTS	.149*	–1.127*	.222	1.568*	.440
21. TCQ – TIRED/FATIGUED	.088*	–.102	.222	–.606	.440
22. TCQ – TASK ATTENTION	.267*	–1.042*	.222	.261	.440
23. TCQ – DISTRACTED	.215*	–.876*	.222	–.329	.440
24. TCQ – STRESSED	.187*	–.814*	.222	–.052	.440
25. TCQ – TASK EFFORT	.359*	–1.564*	.222	1.651*	.440

	Normality	Skewness		Kurtosis	
	Kolmogorov-Smirnov Statistic[a]	Statistic	Std. Error	Statistic	Std. Error
Outliers Removed					
HRV – Ln (Resting HF Power)	.066	–.285	.225	.265	.446
Log_{10} Transformed					
BIDR Self-Deception	.098*	–.237	.225	–.581	.446
HRV – Resting RMSSD	.062	–.396	.224	–.108	.444
Stop-Signal RT	.068	–.089	.233	–.051	.461
Stop-Signal – Go-Trial RT	.051	.095	.225	–.717	.446
Stop-Signal – Go-Trial RT Sd.	.055	–.089	.225	–.660	.446
WCST – Perseverative Errors	.141*	–.185	.223	.097	.442
Original Variable Retained					
BIDR Impression Management	.098*	.394	.217	–.471	.431
WAIS – Saddler 2 Composite	.102*	–.329	.226	.724	.447

Continued

Table B.2. Normality Diagnostics on Revised & Retained Measures. *Note:* a = Computed with Lilliefors Significance Correction. * = Value significant at $p < 0.05$, two-tailed.

Table B.2. Continued

	Normality	Skewness		Kurtosis	
	Kolmogorov-Smirnov Statistic[a]	Statistic	Std. Error	Statistic	Std. Error
Original Variable Retained					
TCQ – TOTAL	.089*	–.614*	.222	–.126	.440
TCQ – COMPUTER	.120*	–.544*	.222	.097	.440
TCQ – HAND TESTS	.143*	–1.044*	.222	1.693*	.440
TCQ – SELF-REPORTS	.149*	–1.127*	.222	1.568*	.440
TCQ – TIRED/FATIGUED	.088*	–.102	.222	–.606	.440
TCQ – TASK ATTENTION	.267*	–1.042*	.222	.261	.440
TCQ – DISTRACTED	.215*	–.876*	.222	–.329	.440
TCQ – STRESSED	.187*	–.814*	.222	–.052	.440
TCQ – TASK EFFORT	.359*	–1.564*	.222	1.651*	.440

Appendix C
Variable Transformations & Post-Transformation
Distributional Analyses

What follows is a summary of the variable transformation methods utilized in this study, and a discussion of the method that yielded the best results for each variable transformed (and, in some cases, not transformed).

Provided that the variable distribution diagnostics revealed evidence of distributional non-normality or dissimilarity amongst study variables, it was viewed as necessary to transform these variables to minimize the attenuation of correlation and regression coefficients. Although the finer points of how and whether one should resolve issues of distributional nonnormality remain sources of debate among some psychologists (Games, 1983, 1984; Levine & Dunlap, 1982, 1983; Levine, Liukkonen, & Levine, 1992), the practice of re-expressing variable data through certain variance-stabilizing transformations has become generally accepted within the behavioral sciences (Cohen et al., 2003; Judd et al., 1995). Among the more widely utilized transformations of correlational data are the "Fisher's z' transformation" (Aroian, 1941; Fisher, 1934) and linear “z” transformation (see Cohen et al., 2003, p. 25), but neither transformation was considered appropriate in the present context. Whereas the nonlinear "Fisher's z' transformation" converts the sampling distribution of “Pearson’s

r" to a normal distribution so that one can determine whether one correlation is significantly different from another, linear transformations have no effect on correlation coefficients (or squared proportions of variance in general) because the inclusion of a numerical constant merely alters variable data in a uniform fashion across a numerical scale (Cohen et al., 2003, p. 222).

Fortunately, as discussed at length below, when approaching the issue of how to address distributional nonnormality within our sample, two commonly utilized and viable product-moment remedies remained: (1) the removal and detection of outliers (Orr et al., 1991; Stevens, 1984; Zimmerman, 1994, 1995); and (2) nonlinear data transformations (Cleveland, 1984; (Ruppert & Aldershof, 1989; Yanagihara & Yuan, 2005). In the interest of expediting our discussion of these issues, it is worth noting that it was my intent to both methods (albeit separately) to correct for deviations from distributional normality for all variables necessitating transformations. The distributional properties of each variable would then be re-evaluated after each transformation to ascertain (a) which method best corrected for distributional non-normality and (b) determine whether the variable distributions were adequately transformed so that the appropriate calculation of correlation and regression coefficients could ensue.

Solution #1: Outlier Detection & Removal

While in many cases violations of statistical normality are rooted in general distributional problems within the data, it is sometimes the case that normality violations stem from a few discrepant observations, or *outliers*. However, even when

general distributional problems are minimal or nonexistent, it is worth underscoring that the least-squares minimization procedures that are central to correlation, multiple regression analyses, and other statistical methods are not robust in the presence of even a few outliers (Rousseeuw & Leroy, 1987). Consequently, many statisticians (Atkinson, 1985; Cohen et al., 2003; Hays, 1994; Judd & McClelland, 1989; Stevens, 1984; Zimmerman, 1994, 1995), though certainly not all (e.g. Orr et al., 1991), view the identification and removal of outliers as a crucial step in the data-analytic process (for a compelling justification of this practice, see Judd et al., 1995; pp. 453-454).

Although a variety of useful graphical techniques (e.g. frequency histograms, scatterplot matrices) are available for detecting discrepant observations within sample data, we opted to utilize univariate boxplots to facilitate identification of data outliers, since this method is widely used and particularly effective at identifying cases that fall both within and outside the quartiles of a data distribution (Cohen et al., 2003 p. 108-110). This graphical technique was augmented through the calculation of "Cook's D_i" (Cook, 1977, 1979), which is a widely used index of how the inclusion or deletion of an outlier can affect a data set (Cohen et al., 2003; p. 402).

An examination of these univariate boxplots and "Cook's D_i" coefficients revealed high leverage-value outliers in each of these variables (note: number of discrepant cases in parentheses): BIDR Self-Deception (n = 6); HRV – Ln (Resting HF Power; n = 1); Stop-Signal RT (n = 8); WCST – Perseverative Errors (n = 9); WAIS – Saddler 2 Composite (n = 2); TCQ – TOTAL (n = 1); TCQ – COMPUTER (n = 5); TCQ – HAND TESTS (n = 2); TCQ – SELF-REPORTS (n = 4); TCQ –

TIRED/FATIGUED (n = 6); TCQ – TASK ATTENTION (n = 3); TCQ – STRESSED (n = 6); and TCQ – TASK EFFORT (n = 11). Per the recommendations of those supporting the Gaussian-focused removal of discrepant data observations (Atkinson, 1985; Cohen et al., 2003; Hays, 1994; Judd & McClelland, 1989; Judd et al., 1995; Orr et al., 1991; Stevens, 1984; Zimmerman, 1994, 1995), we adopted a stepwise procedure whereby individual outliers (beginning with the most discrepant observations) were deleted and the distributional properties of the corresponding variable were reassessed via "Cook's D_i" to determine whether outlier removal resulted in a (quasi-) normal distribution. This procedure was repeated for each variable until quantitative indices of normality (i.e. Kolmogorov-Smirnov) indicated that outlier removal yielded a distribution that approximated a normal distribution.

In only one case, HRV – Ln (Resting HF Power), did the removal of outliers alone remedy distributional nonnormality (see Table B.2, Appendix B). By contrast, the deletion of discrepant observations did little to correct for deviations from normality for some variables (e.g. BIDR Self-Deception, Stop-Signal RT, WCST – Perseverative Errors) and, in many cases (e.g. all TCQ subscales), actually appeared to engender greater departures from distributional normality. As a result, we implemented nonlinear data transformations to remedy distributional problems for those variables whose distributions were not adequately or appropriately responsive to outlier detection and removal.

Solution #2: Nonlinear Transformations of Variable Data

While outlier detection and deletion are in many cases all that are needed to remedy problems of distributional nonnormality, by far the most common methods for correcting departures from normality in regression analyses fall under what are collectively known as "nonlinear transformations" (see Cohen et al., 2003, Tabachnick & Fidell, 2007). Although their uses are numerous, within the present context these nonlinear transformations can re-express variable data via exponential, square-root, and/or logarithmic conversions so that the relationships between dependent and independent variables can be meaningfully interpreted, owing to the resultantly decreased risk of committing Type I and Type II error. After an extensive review of the literature on the application and conceptual bases for these nonlinear transformations (see Cleveland, 1984; Cohen et al., 2003; Ruppert & Aldershof, 1989; Tabachnick & Fidell, 2007; Yanagihara &Yuan, 2005), and following multiple attempts at using the assorted nonlinear transformations in question, it was our conclusion that logarithmically-based transformations were best suited for remedying distributional problems within our sample data.

Depending on whether data distributions evidenced signs of positive or negative skew, different procedures were adopted when implementing these logarithmic transformations. For positively skewed variable distributions, it was only necessary to logarithmically transform the variable (with the appropriate base power) since this nonlinear transformation compresses the right side of the distribution more than the left side of the distribution. For negatively skewed variable distributions, it was necessary to first reflect (or reverse) the distribution and, add a numerical

constant to bring its minimum value above 1.0 before logarithmically transforming the variable. After completing this logarithmic transformation, it was necessary to again reflect (or reverse) the variable distribution to restore the original order of the once-negatively skewed variable.

In all cases, a logarithmic transformation base 10 ($\log_{10}$) was used initially. It might be of interest to the reader, however, that per the recommendations of Cleveland (1984), a natural logarithm (L_n) was also applied separately as a precaution when transforming some variables (e.g. Stop-Signal – Go-Trial RT) whose departures from normality were less extreme, since high logarithmic bases (e.g. $\log_{10}$) can sometimes worsen distributional violations in such cases. However, this precautionary measure (i.e. implementing natural logarithmic transformation instead of $\log_{10}$) ultimately provided no appreciable benefit over log base 10 transformations, even for those variables (see Table B.2, Appendix B; *Original Variable Retained*) whose distributions ultimately evidenced no improvements in normality through our transformation processes. Nevertheless, as one can see in Table B.2, the normality diagnostics of six variable distributions (BIDR Self-Deception, HRV – Resting RMSSD, Stop-Signal RT, Stop-Signal – Go-Trial RT, Stop-Signal – Go-Trial RT Sd., WCST – Perseverative Errors) evidenced significant improvements in normality through our log base 10 transformations.

Although our nonlinear transformations were largely successful at remedying problems of distributional non-normality, the reader may note that in Table B.2 two variables (BIDR Self-Deception and WCST – Perseverative Errors) still evidenced

significant K-S statistics. Nevertheless, a comparison of these values to those K-S statistics presented in Table B.1 (see Appendix B) still demonstrated overall improvement in the distributional properties of these two variables. Moreover, BIDR Self-Deception evidenced statistically significant reductions in skew, and WCST – Perseverative Errors evidenced marked improvements in kurtosis after our logarithmic transformations.

Finally, no appreciable or beneficial changes in the distributional properties of the TCQ subscales ever manifested despite numerous and varied transformation efforts (see Table B.2, Appendix B). These did little to ameliorate concerns about attenuated correlation coefficients due to distributional dissimilarity with respect to these variables (Carroll, 1961; Hays, 1994). However, the TCQ total score was found to have adequate distributional properties and that score was therefore used in all analyses.

Appendix D
Multiple Linear Regression Diagnostics

Although correlational analyses helped shed some light on the relations between multiple combinations of two variables, no examination of this study's hypotheses would have been complete without addressing two related questions. First, to what extent are the correlation coefficients that manifested actual indicators of empirical relationships between specific variables? Second, what do the *combinations* of intercorrelations tell us about the relationships between the assorted variables under investigation? Although the former question is addressed to some extent by the null hypothesis significance testing (NHST), left unresolved by the product moment correlations is how or why two variables appear to be related. While definitive answers regarding causality between variables are undoubtedly beyond the scope of our data, product moment correlations alone are not sufficient for examinations of whether one variable is *influenced by* the other, because neither variable is explicitly regarded as the criterion (or dependent) variable whose values can be estimated by the predictor (or independent) variable. This limitation is attributable, at least in part, to the fact that although two correlated variables *could* be causally related, they could alternatively be correlated with each other by virtue of (a) the overlap or redundancy of information they provide or (b) their independent relationships to another variable (or combination of variables), perhaps unidentified.

As discussed previously, questions still abound as to whether the relations between key study variables (e.g. effortful control and heart-rate variability) are the result of socially desirable responding on questionnaire items. To address this matter, one can include measures of socially desirable responding (e.g. BIDR Impression Management) as covariates of predictor variables (e.g. ATQ-EC Total) in regression models of criterion measures (e.g. HRV – Resting RMSSD): 'dependent' measures previously shown to hold significant correlations with the predictor variables. From these stepwise regression analyses one can (a) determine whether the inclusion of the covariate attenuates (i.e. moderates or suppresses) or explains (i.e. mediates) the statistically significant relationship between the predictor and criterion variables, as well as (b) calculate *semi-partial correlations* to examine the relationships between three or more variables at one time.

When conducting these regression analyses, it was necessary to calculate diagnostics relevant to the assumptions of multiple linear regression (MLR), particularly with respect to variable *residuals* (or "errors"). What follows is an overview of the regression diagnostics conducted as part of our data-analytic procedure.

i. Regression Diagnostics: Collinearity

An assumption underlying multiple regression is that each independent variable (or X) included in a regression analysis has the potential to contribute information that can improve prediction of the dependent variable (or Y) under

investigation (for review see Cohen et al., 2003, pp. 419-430). As a result, the inclusion of more predictor variables in a multiple regression not only has the potential to improve the prediction of Y, it also decreases the likelihood that each predictor variable can contribute *unique* information (or variance) not provided by other variables in a multiple regression of Y, especially when those predictor variables are highly related to one another. While this aspect of multiple regression has the advantage of allowing one to investigate variable mediation and moderation, problems in the interpretation of individual regression coefficients can arise as predictors become increasingly correlated. In cases where a predictor variable is perfectly correlated (i.e. exact collinearity) or highly correlated (i.e. multicollinearity) with other X variables, individual regression coefficients become unreliable (as evidenced by large standard errors) and difficult to compute accurately (e.g. resultantly increased or decreased coefficient magnitudes, and/or changes in coefficient directionality). Consequently, testing for collinearity is viewed by some statisticians (e.g. Belsley, Kuh, & Welsch, 1980; Cohen et al., 2003; Hays, 1994) as a crucial component of any regression analysis. In light of the aforementioned correspondence among key variables in this study, such admonishments are particularly patent.

To ascertain whether potential problems with collinearity could hinder the interpretation of regression coefficients, a series of analyses were conducted wherein each predictor was regressed on all of the remaining predictors: an approach that yields separate squared-multiple correlations that can be used to compute a variance inflation factor (VIF) for each of the predictor variables. Per the recommendations of

Cohen and colleagues (2003; p. 423), a criterion was adopted in which any VIF of 10 or more would be considered evidence of serious multicollinearity involving the corresponding regressed independent variable.

Our preliminary analyses revealed evidence of severe multicollinearity when both measures of heart-rate variability (i.e. HRV – Resting RMSSD, HRV – Ln (Resting HF Power)) were included simulataneously in linear regression equations predicting scores on the ATQ-EC subscales ($VIF_{Resting\ RMSSD}$ = 11.050 to and $VIF_{Ln\ (Resting\ HF\ Power)}$ = 11.025). Similar results were obtained for the heart-rate variabilty measures, Resting RMSSD and Ln (Resting HF Power), when the following were treated as criterion variables: Attentional Control Scale ($VIF_{Resting\ RMSSD}$ = $VIF_{Ln\ (Resting\ HF\ Power)}$ = 11.012); WAIS – Saddler 2 Composite ($VIF_{Resting\ RMSSD}$ = 11.789 and $VIF_{Ln\ (Resting\ HF\ Power)}$ = 11.703); WMS – Working Memory ($VIF_{Resting\ RMSSD}$ = 11.017 and $VIF_{Ln\ (Resting\ HF\ Power)}$ = 11.018); Stroop Interference ($VIF_{Resting\ RMSSD}$ = 10.963 and $VIF_{Ln\ (Resting\ HF\ Power)}$ = 10.962); Stop-Signal RT ($VIF_{Resting\ RMSSD}$ = 11.149 and $VIF_{Ln\ (Resting\ HF\ Power)}$ = 11.110); Go-Trial RT ($VIF_{Resting\ RMSSD}$ = 11.308 and $VIF_{Ln\ (Resting\ HF\ Power)}$ = 11.266); Go-Trial RT Sd. ($VIF_{Resting\ RMSSD}$ = 11.308 and $VIF_{Ln\ (Resting\ HF\ Power)}$ = 11.266); and WCST – Perseverative Errors ($VIF_{Resting\ RMSSD}$ = 10.651 and $VIF_{Ln\ (Resting\ HF\ Power)}$ = 10.647). These results affirmed our decision not to include either heart-rate variability measure as a covariate in models wherein its counterpart was treated as a predictor variable.

Additional analyses indicated no signs of serious multicollinearity for any other study variable. Hence, we proceeded to our analyses of variable linearity and residual homoscedasticity, independence, and normality.

ii. Regression Diagnostics: Relationship Linearity & Homogeneity of Variance

Another important assumption of ordinary least squares regression (univariate and multivariate) is that the form or mathematical shape of the relationship between Y and each of the predictor variables has been correctly specified (see Cohen et al., 2003, pp. 117-119). In linear regression and correlation, the relationship between the dependent (Y) and independent (X) variable(s) is expected to follow a straight line across the full range of X values. When this assumption is not satisfied and the relationship between X and Y is curvilinear (e.g. quadratic), their co-relation cannot be adequately represented by a linear regression equation, which would produce both biased regression (or correlation) coefficients and standard errors, and resultantly compromised significance tests.

Subsumed under the linearity assumption is the expectation that the variability of residual (or "error") terms will be constant for every value of a predictor (see Cohen et al., 2003, pp. 119-120). Otherwise stated, when predicting the value of Y in a regression model for every value of a predictor (based on the specifications of the regression equation), the variance of the residuals (e.g. standard deviations of the error terms) is expected to be the same regardless of the value of the predictor variable. When this homogeneity of variance assumption is met, the distribution is said to meet the conditions of *homoscedasticity*. By contrast, when the residual variance changes

across values of the predictor variable, this condition is known as *heteroscedasticity*. Although heteroscedasticity does not directly affect regression coefficients, it can aversely impact standard errors and result in biased significance tests.

A common practice for evaluating the linearity and homoscedasticity assumptions is to examine the scatterplots of the standardized residuals (ordinate) against the predicted residual values (abscissa) for each regression model under investigation (for review see Cohen et al., 2003, pp. 125-126, 130-132). This is possible because a residual represents the portion of a specific case's (i.e. participant's) score on the dependent measure not adequately accounted for by the regression equation. When *systematic* variance remains across residuals, it is likely that the regression model has been misspecified, and these systematic residual variations will reveal certain graphical display patterns. Among these patterns are residual plot curvatures indicative of linearity violations and heteroscedasticity (see Cohen et al., 2003, pp. 130-132).

To assist in the assessment of the linearity assumption, *loess* fit lines (see Cleveland, 1979; Cohen et al., 2003, pp. 111, 131), sometimes referred to as *lowess* lines (*lowess* is an acronym for "locally weighted scatterplot smoother"), were superimposed on the aforementioned scatterplots (note: loess trends computed for 99% of cases with Gaussian-kernel fitting). No assumptions are made in the loess method about the form of the relationship between X and Y. Instead, the loess lines follow the trend of the data, and if the variable relationship under scrutiny is linear in the population, the loess line will roughly approximate (not necessarily follow) a

straight line (see Cohen et al., 2003, p. 111). Per loess fit interpretation guidelines (Alvarez & Emory, 2006, p. 31; Borkowf, Albert, & 2003; Cleveland, 1991, p. 54; Cohen et al., 2003, pp. 111, 131; Sohn, Kim, Hwang, & Lee, 2008, pp. 4111-4113; Wilcox, 2007), none of the residual scatterplots displayed in Figures D.1-D.29 indicated significant deviations from linearity for of any our multiple regression analyses (see Tables A.7-A.20, Appendix A).

To help ascertain whether problems with heteroscedasticity could interfere with the significance testing of our regression coefficients (see A.7-A.20), we examined the residual scatterplots to see if the residual variance changed across predictor variables. Per interpretation guidelines (Broto & Ruiz, 2009, pp. 5-22; Cohen et al., 2003, p. 132; Robinson, 2008; Yang & Tse, 2008, pp. 357-368), visual inspection of these residual scatterplots (see Figures D.1-D.29) revealed potential problems with heteroscedasticity in Figures D.5, D.6, D.7, D.13, and D.14. However, definitive statements regarding heteroscedasticity could not be made on the basis of these visual inspections alone, because the scatterplot patterns indicated few observations for lower predictor values, perhaps indicating problems with range restriction (see ensuing discussion). Thus, *modified Levene tests* were conducted for each regression equation to further assess for nonconstant variance (see Cohen et al., 2003, p. 133), which confirmed the presence of heteroscedasticity in three of these graphical displays (Figures D.5, D.6, D.7).

In light of the aforementioned signs of heteroscedasticity, it was necessary to determine whether or not the magnitude of the nonconstant variance was high enough

to warrant corrective action (e.g. logarithmic transformation of Y, weighted least squares regression). To do so, cases were separately organized from lowest-to-highest value for each predictor variable and divided into five segments or *slices* (for review of method, see Cohen et al., 2003, pp. 145-147). Residual variance was then calculated for each data segment and the ratio of the largest-to-smallest conditional residual variance segments was computed. Since these ratios (ranging from 6.26:1-to-5.97:1) were less than 10:1, the magnitude of nonconstant variance was deemed insufficient to warrant remedial procedures to correct for heteroscedasticity.

iii. Regression Diagnostics: Independence of Residuals

Just as residuals are expected to be constant across values of a predictor variable, there is also the expectation that no relationship exists between the residuals for any two cases in a sample (see Cohen et al., 2003, p. 120; Judd et al., 1995, p. 459). Although the use of random sampling virtually ensures that the assumption of residual/observational independence will be met, when data are gathered in groups and/or are procured at similar times (as they were on occasion when multiple participants were assessed simultaneously in our study), the responses of two participants selected from the same group (e.g. introductory psychology students) or time (e.g. Saturday at 3:30PM) are more likely to be similar than those of two participants from different groups our whose data was gathered at different times (i.e. *autocorrelation*). Although cases of non-independence (or *clustering*) do not give rise to errors in estimating regression coefficients, they can engender effects in standard errors and, resultantly, affect significance testing.

To assess residual independence, Durbin-Watson coefficients (d) were computed for each linear regression. For a detailed discussion of the usage and mathematical theory underlying the Durbin-Watson Test statistic, several excellent resources are available (e.g. Belsley et al., 1980; Berk, 2003; Berry, 1993; Cohen et al., 2003; Cook & Weisberg, 1982; Fox, 1991; Kahane, 2001). In brief, values of "d" range from 0 to 4, where 0 is indicative of extreme *positive autoregression* (i.e. standard errors of regression coefficients too small) and 4 is indicative of extreme *negative autoregression* (i.e. standard errors of regression coefficients too large). While the exact critical value of the Durbin-Watson coefficient (d) is extremely difficult to calculate for a regression model (see Cohen et al., 2003; p. 137), as a general rule test statistic values of between 1.5 and 2.5 are regarded as evidence of independence. These Durban-Watston coefficients are presented along with the independent linear regressions in Tables A.7-A.20. All Durban-Watson coefficients were within acceptable limits (ranged from 1.757-to-2.276) and suggestive of residual independence.

iv. Regression Diagnostics: Normality of Residuals

As discussed in our variable distribution analyses, while the order data from independent and dependent variables is neither expected nor assumed to approximate a normal distribution (Cohen et al., 2003; pp. 110-117, 120), the *residuals* of these variables are expected to be normally distributed. Although regression coefficient estimates are not biased as a consequence of violating this assumption, significance testing can be impacted when residuals are not normally distributed and sample size

does not provide statistical power sufficient for the correlations observed (the reader is reminded that we anticipated correlations approximating r = 0.50: a magnitude that did not manifest for the majority of our coefficients).

To assess residual normality, Q-Q plots of standardized residuals were constructed for each regression equation. These residual plots were constructed in much the same way as they had been when assessing our variable distributional analyses, except that they entailed plotting residuals and expected residual values derived from a cumulative distribution function (Cohen, 2003, p. 137). As shown in Figures D.1-D.29, no evidence of residual non-normality manifested, as the residual plots adequately followed the linear patterns indicative of a normal curve (Cohen et al., 2003, pp. 138-139; Henderson, 2006, pp. 115-116).

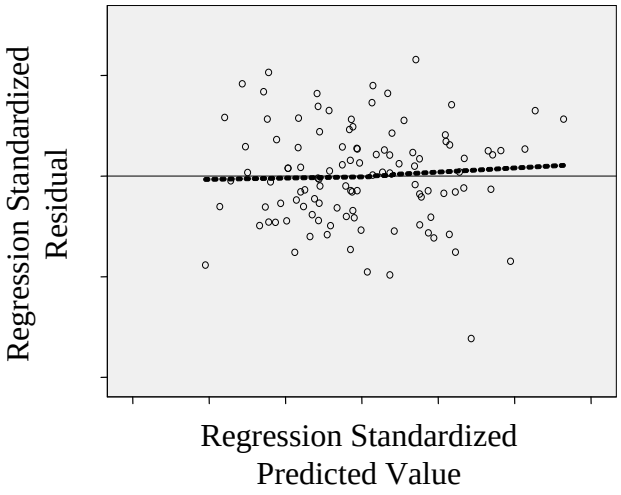
v. Summary of Residual Diagnostics

To further ascertain whether or not the significant correlations presented in Tables A.3-A.6 were indicators of empirical relationships between specific variables, it was necessary to conduct a series of multiple regression analyses: procedures that would help determine whether (a) socially desirable responding or problems with task completion gave risk to spurious correlations, and (b) allow for the calculation of *partial correlations* between three or more variables at one time. When conducting these regression analyses, it was necessary to calculate diagnostics relevant to the assumptions of multiple linear regression (MLR), particularly with respect to variable *residuals* (or "errors").

Our collinearity analyses evidenced signs of significant multicollinearity between our measures of heart-rate variability, indicating that their dual inclusion in regression equations could potentially lead to unreliable regression coefficients, thus affirming our decision not to include either heart-rate variability measure as a covariate in models wherein its counterpart was treated as a predictor variable. Signs of heteroscedasticity also emerged for several regression equations. However, post-hoc analyses indicated that the magnitude of nonconstant variance was deemed insufficient to warrant corrective action (e.g. logarithmic transformation of Y, weighted least squares regression).

Additional residual diagnostics evidenced no signs of assumption violations that could compromise the utility of our regression analyses. As shown in Figures D.1-D.29, residual scatterplots displayed no significant deviations from variable relationship linearity, suggesting that the relationship between Y and each of the predictor variables had been correctly specified. Similarly, none of the residual scatterplots displayed in Figures D.1-D.29 indicated significant evidence of residual non-normality, as the residual plots adequately followed the linear patterns indicative of a normal curve. What is more, the Durban-Watson coefficients presented in Tables A.7-A.20 were all indicative of residual independence, which in concert with the other residual diagnostics, suggested that the multiple regression analyses would provide an adequate basis for verifying the bi-variate correlations obtained in our earlier analyses.

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

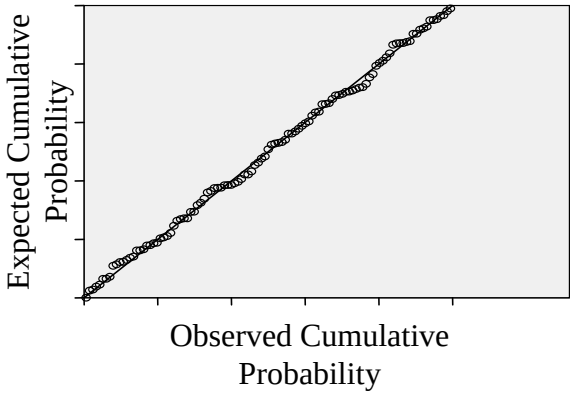
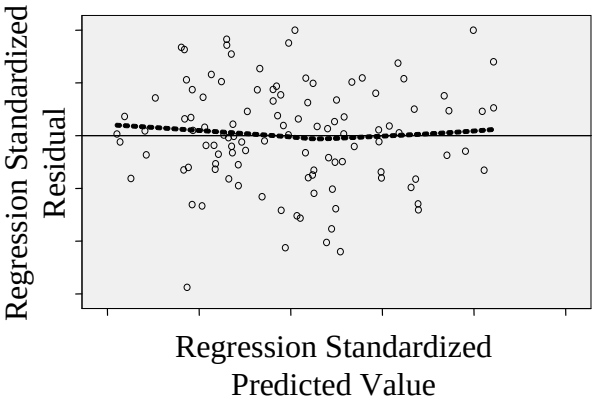


Figure D.1. Graphical Displays Testing Residual Assumptions for ATQ-EC Total and ATQ-EC Attentional Control Multiple Regression (corresponds with Table A.7, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

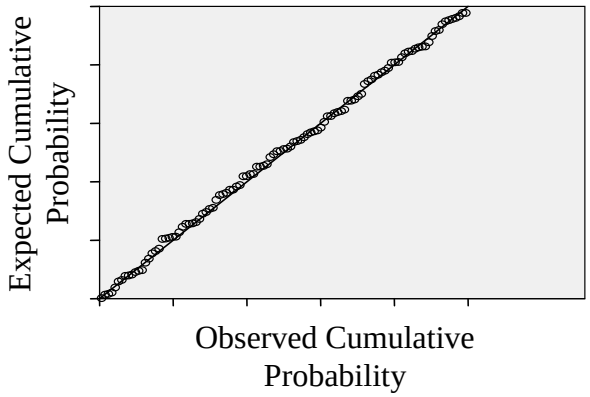
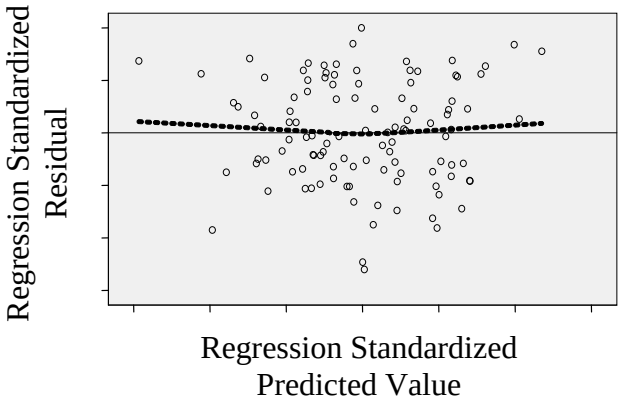


Figure D.2. Graphical Displays Testing Residual Assumptions for ATQ-EC Total and ATQ-EC Inhibitory Control Multiple Regression (corresponds with Table A.8, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

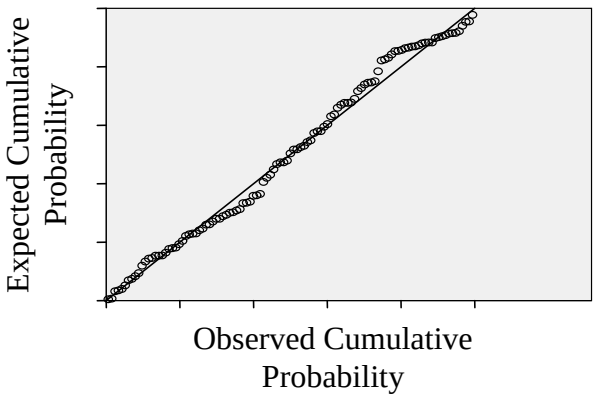
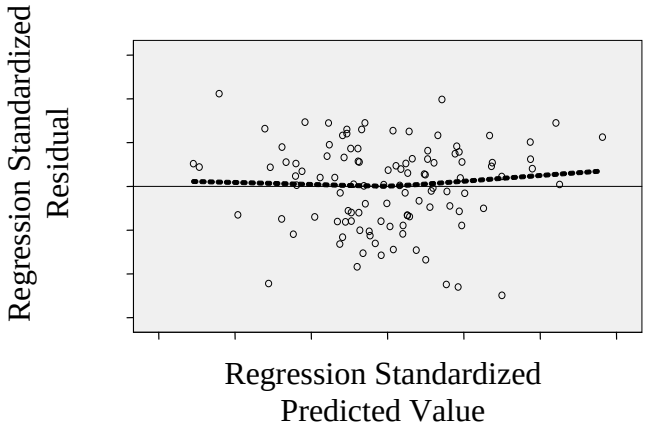


Figure D.3. Graphical Displays Testing Residual Assumptions for ATQ-EC Total and ATQ-EC Activation Control Multiple Regression (corresponds with Table A.9, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

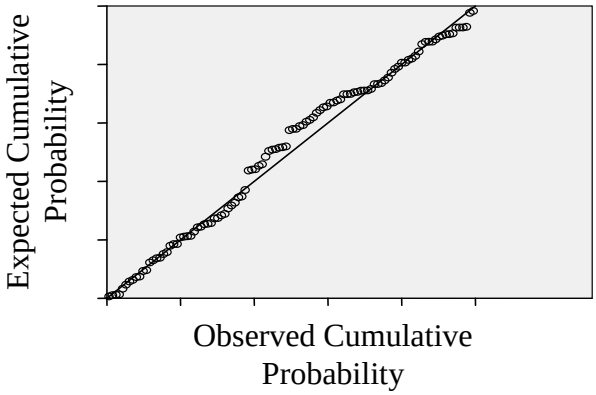
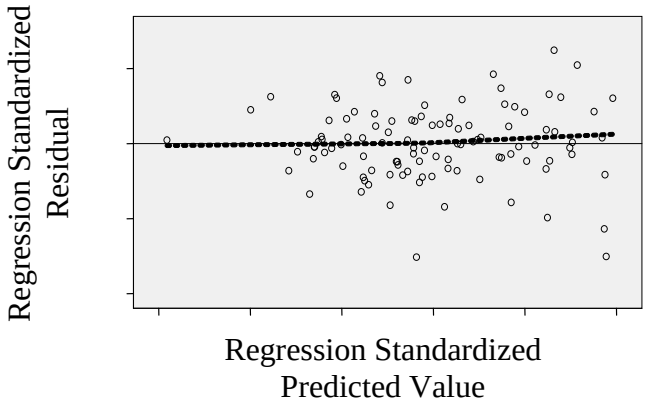


Figure D.4. Graphical Displays Testing Residual Assumptions for ATQ-EC Total and Attentional Control Scale Multiple Regression (corresponds with Table A.10, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

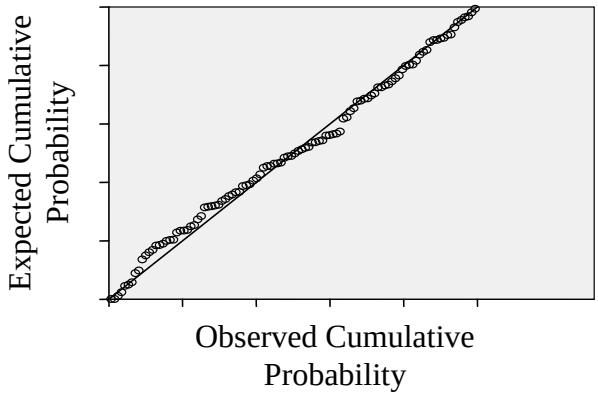
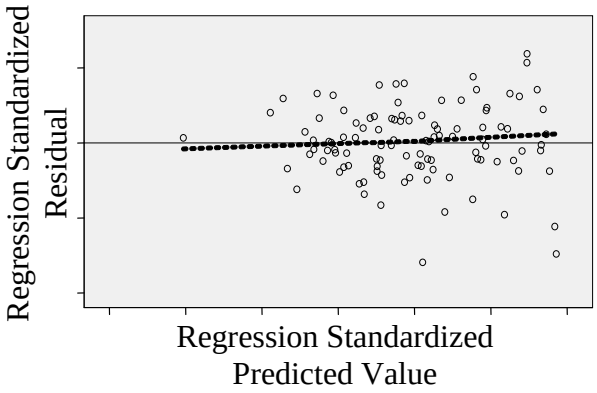


Figure D.5. Graphical Displays Testing Residual Assumptions for ATQ-EC Total and HRV – Resting RMSSD Multiple Regression (corresponds with Table A.11, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

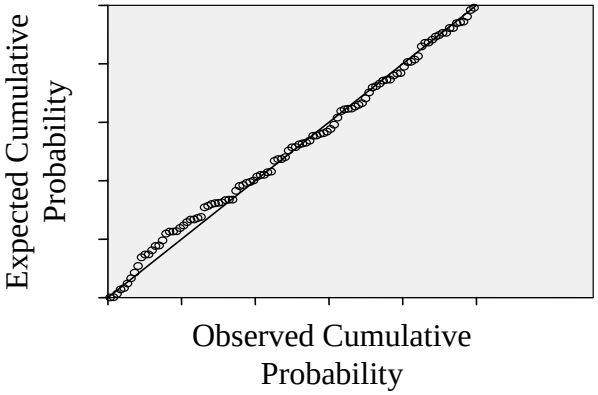
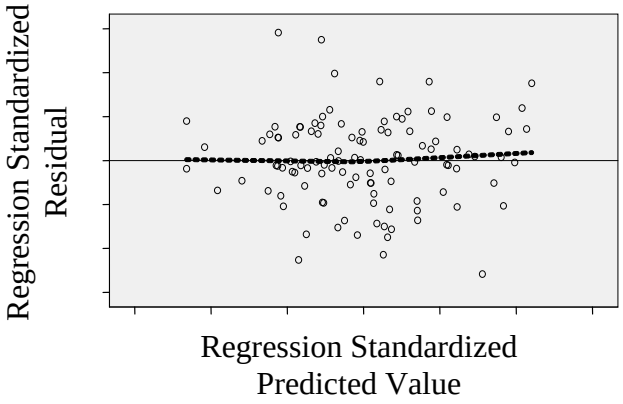


Figure D.6. Graphical Displays Testing Residual Assumptions for ATQ-EC Total and HRV – Ln (Resting HF Power) Multiple Regression (corresponds with Table A.12, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

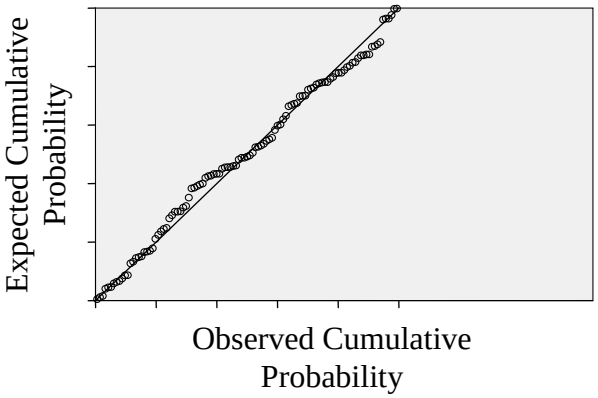
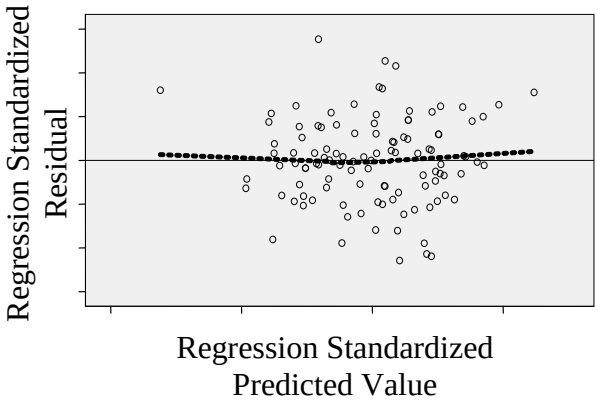


Figure D.7. Graphical Displays Testing Residual Assumptions for ATQ-EC Attentional Control and ATQ-EC Inhibitory Control Multiple Regression (corresponds with Table A.13, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

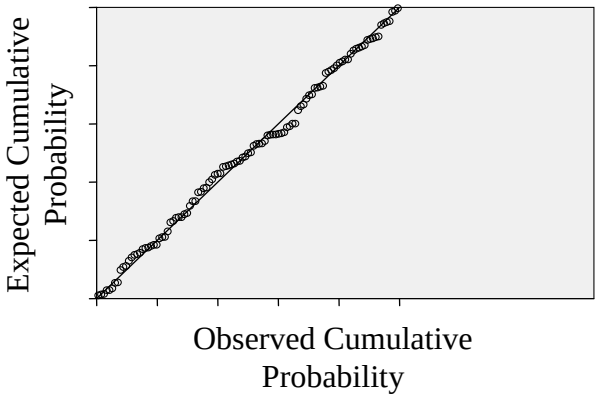
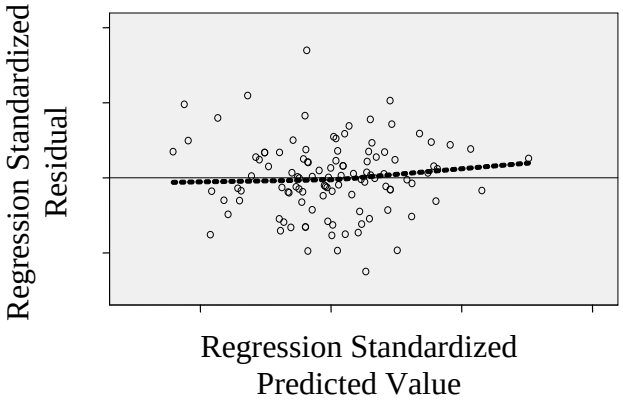


Figure D.8. Graphical Displays Testing Residual Assumptions for ATQ-EC Attentional Control and ATQ-EC Activation Control Multiple Regression (corresponds with Table A.14, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

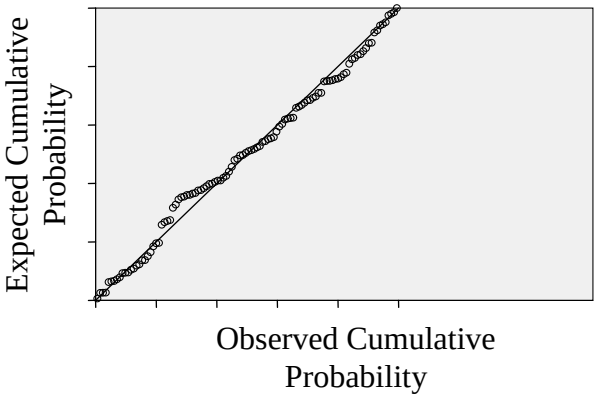
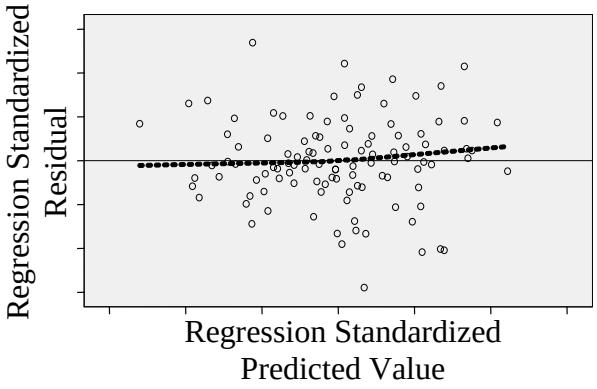


Figure D.9. Graphical Displays Testing Residual Assumptions for ATQ-EC Attentional Control and Attentional Control Scale Multiple Regression (corresponds with Table A.15, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

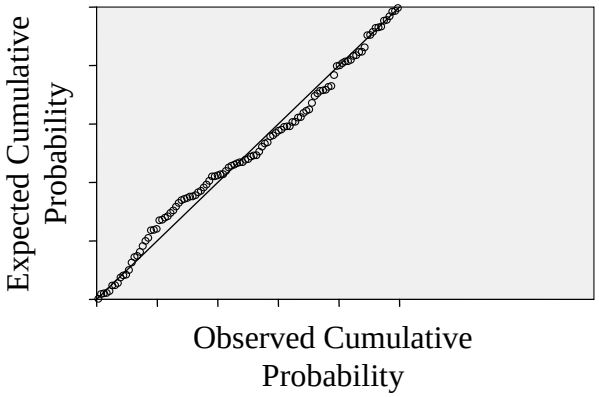
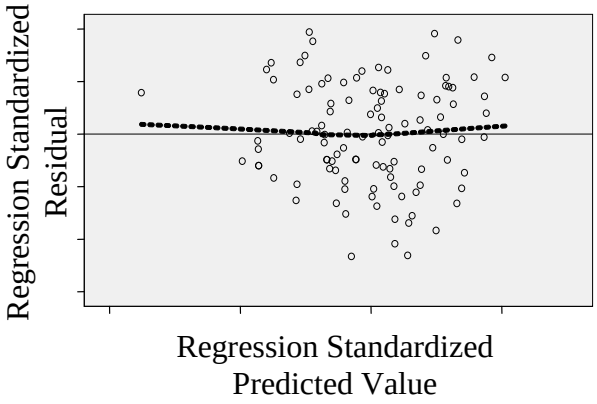


Figure D.10. Graphical Displays Testing Residual Assumptions for ATQ-EC Attentional Control and WMS – Working Memory Multiple Regression (corresponds with Table A.16, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

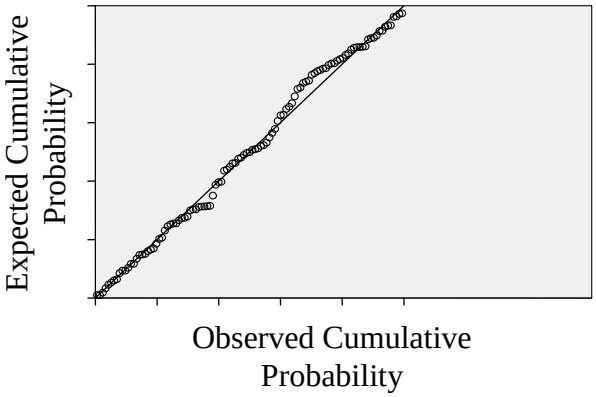
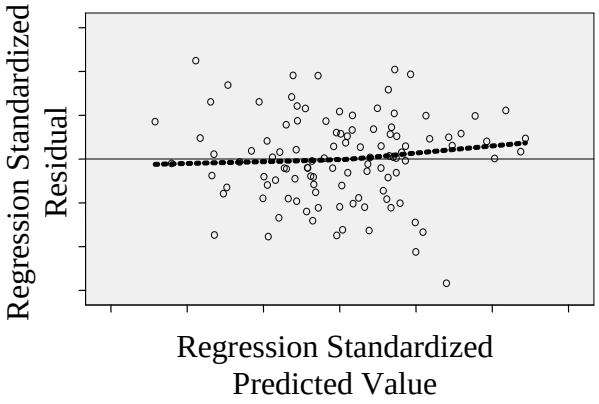


Figure D.11. Graphical Displays Testing Residual Assumptions for ATQ-EC Inhibitory Control and ATQ-EC Activation Control Multiple Regression (corresponds with Table A.17, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

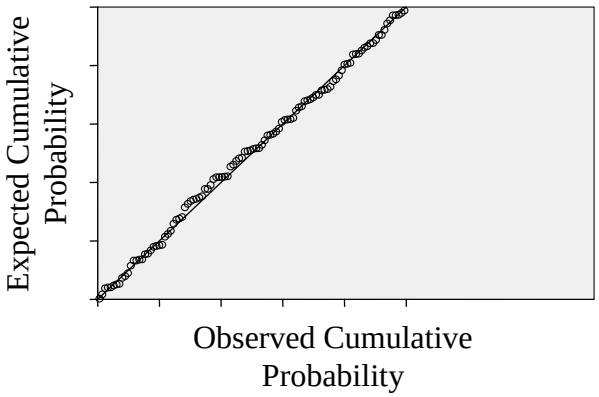
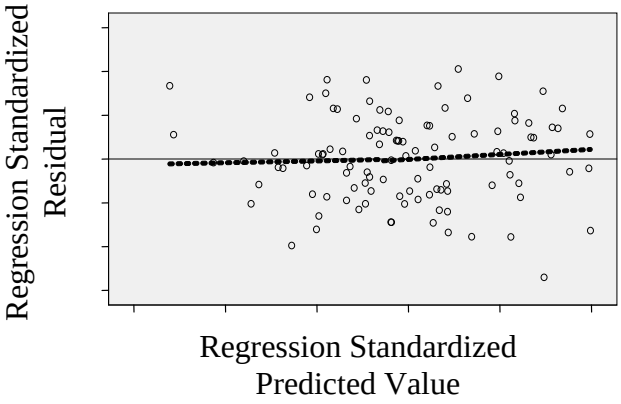


Figure D.12. Graphical Displays Testing Residual Assumptions for ATQ-EC Inhibitory Control and Attentional Control Scale Multiple Regression (corresponds with Table A.18, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

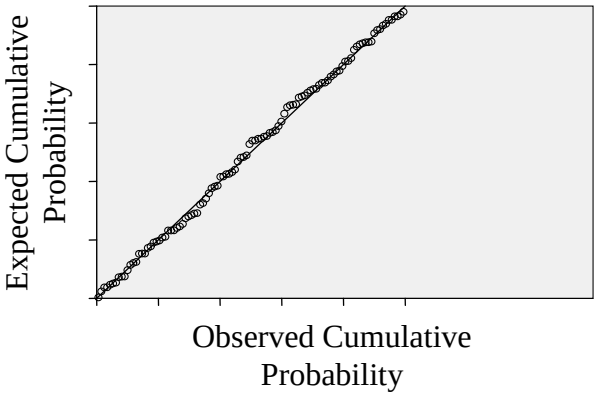
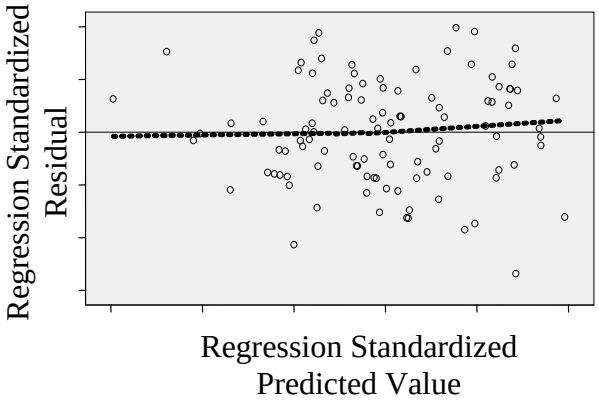


Figure D.13. Graphical Displays Testing Residual Assumptions for ATQ-EC Inhibitory Control and HRV – Resting RMSSD Multiple Regression (corresponds with Table A.19, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

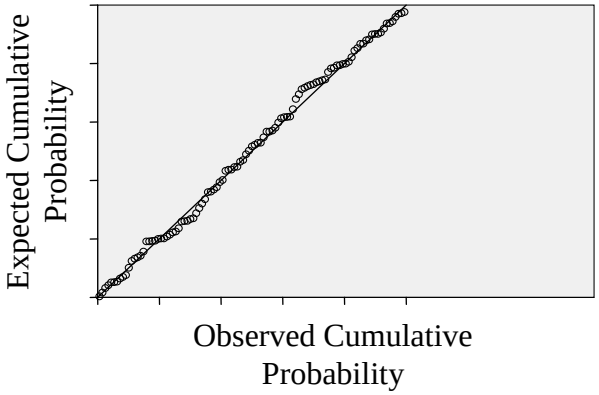
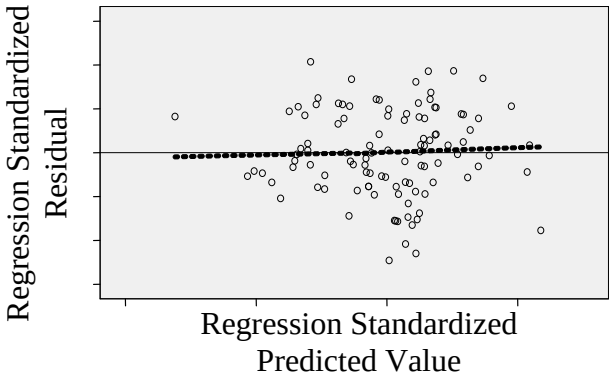


Figure D.14. Graphical Displays Testing Residual Assumptions for ATQ-EC Inhibitory Control and HRV – Ln (Resting HF Power) Multiple Regression (corresponds with Table A.20, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

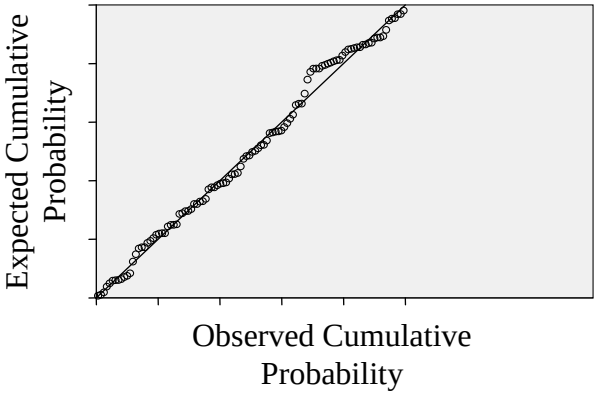
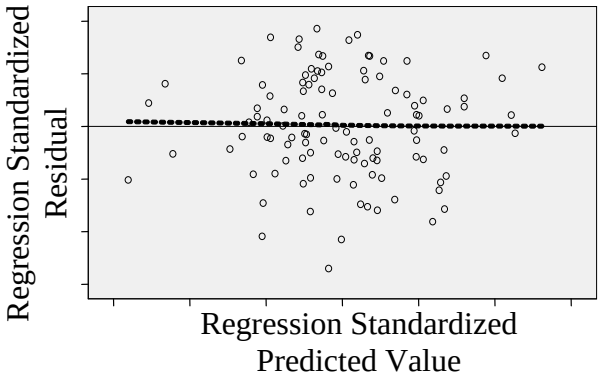


Figure D.15. Graphical Displays Testing Residual Assumptions for ATQ-EC Inhibitory Control and Stop-Signal – Go-Trial RT Sd. Multiple Regression (corresponds with Table A.21, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

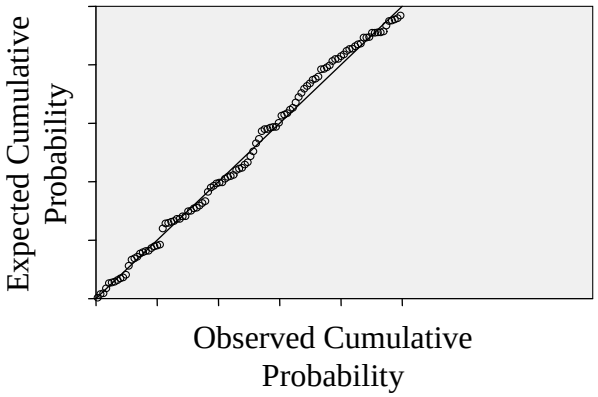
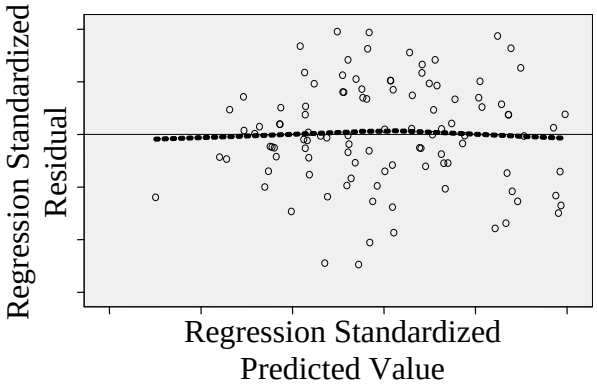


Figure D.16. Graphical Displays Testing Residual Assumptions for ATQ-EC Activation Control and Attentional Control Scale Multiple Regression (corresponds with Table A.22, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

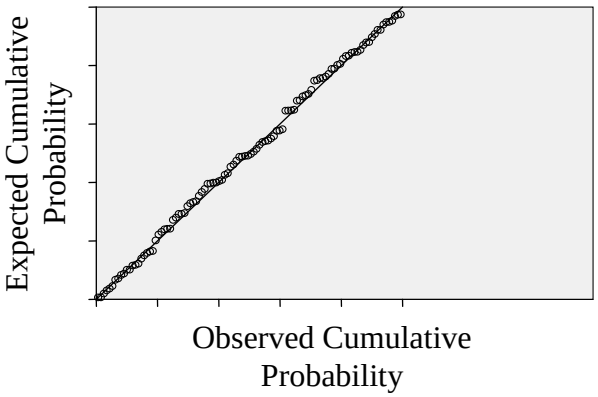
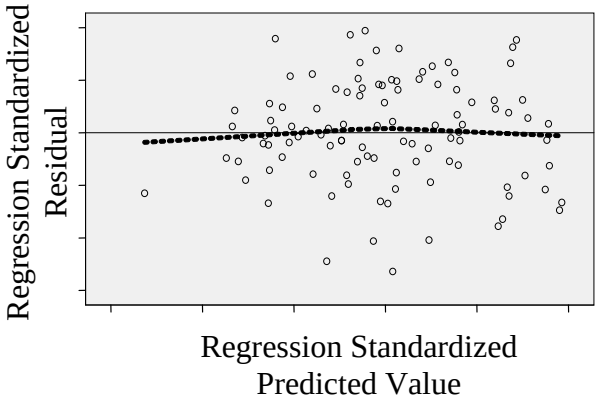


Figure D.17. Graphical Displays Testing Residual Assumptions for ATQ-EC Activation Control and HRV – Resting RMSSD Multiple Regression (corresponds with Table A.23, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

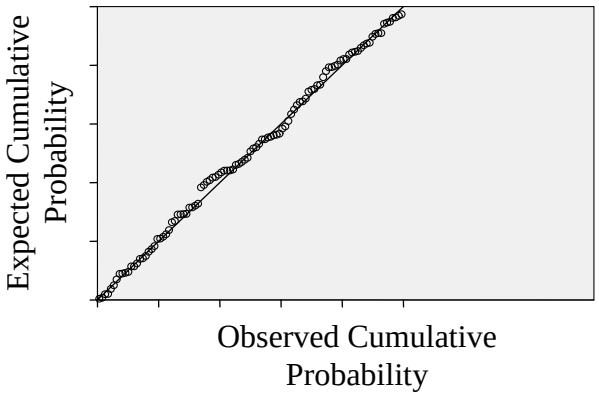
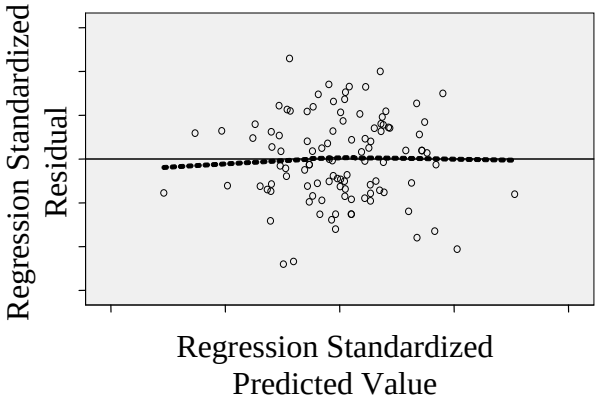


Figure D.18. Graphical Displays Testing Residual Assumptions for ATQ-EC Activation Control and HRV – Ln (Resting HF Power) Multiple Regression (corresponds with Table A.24, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

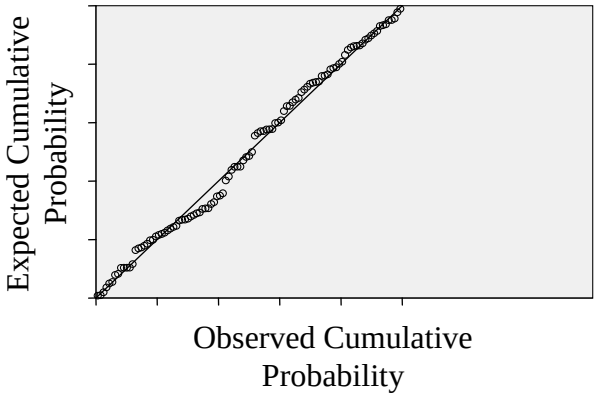
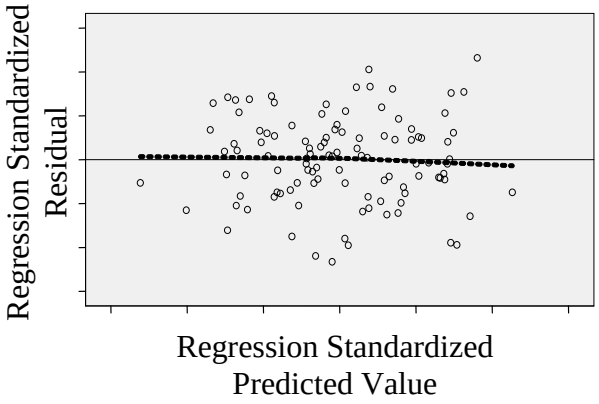


Figure D.19. Graphical Displays Testing Residual Assumptions for ATQ-EC Activation Control and WAIS – Saddler 2 Composite Multiple Regression (corresponds with Table A.25, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

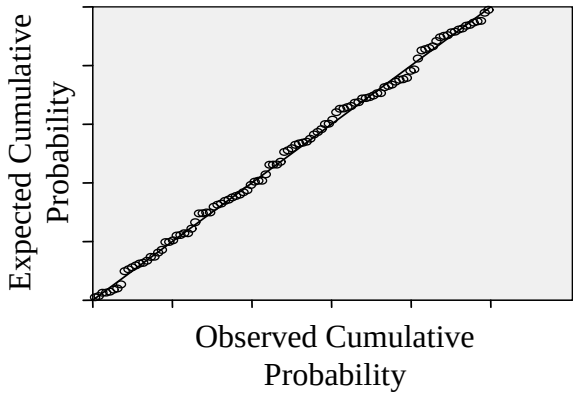
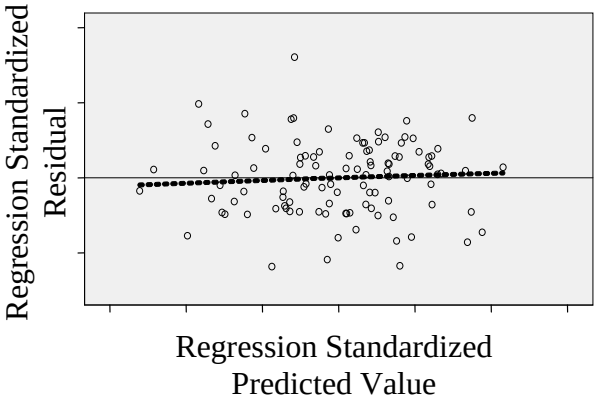


Figure D.20. Graphical Displays Testing Residual Assumptions for Attentional Control Scale and WCST – Perseverative Errors Multiple Regression (corresponds with Table A.26, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

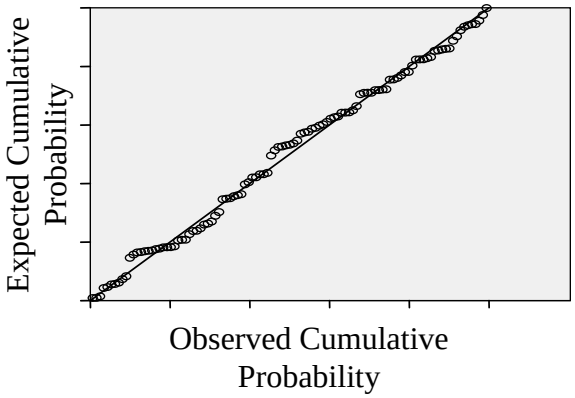
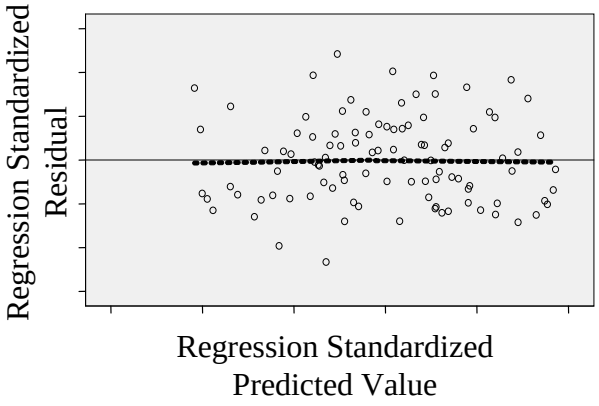


Figure D.21. Graphical Displays Testing Residual Assumptions for HRV – Resting RMSSD and HRV – Ln (Resting HF Power) Multiple Regression (corresponds with Table A.27, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

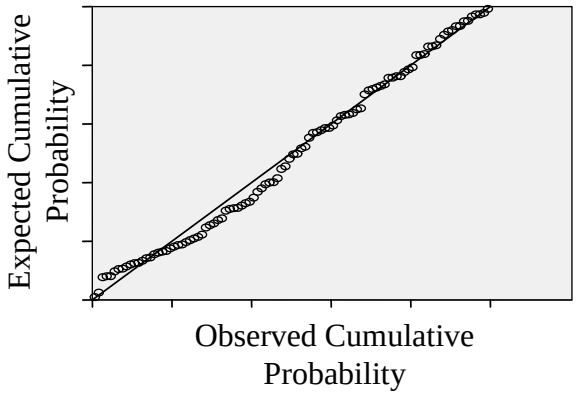
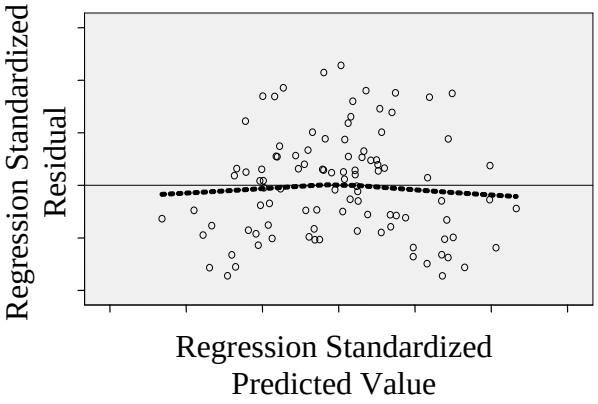


Figure D.22. Graphical Displays Testing Residual Assumptions for Stop-Signal RT and Stop-Signal – Go-Trial RT Multiple Regression (corresponds with Table A.28, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

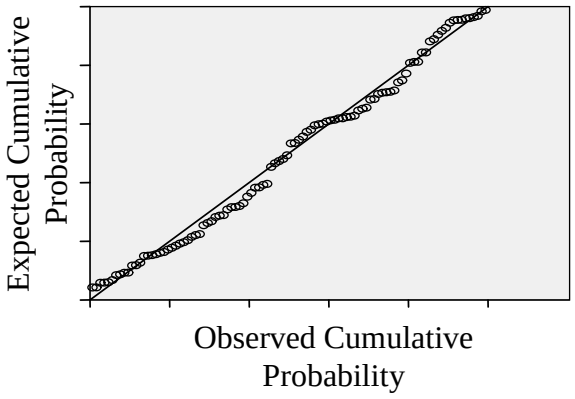
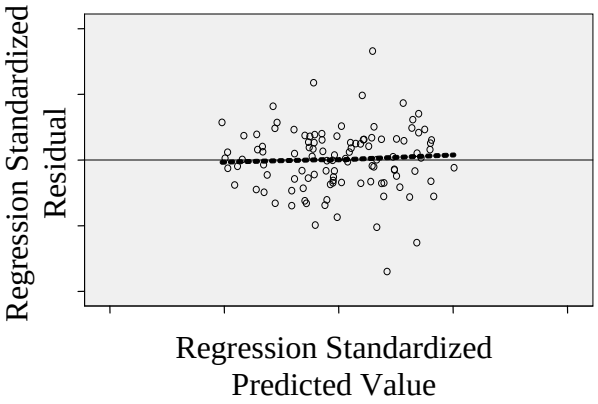


Figure D.23. Graphical Displays Testing Residual Assumptions for Stop-Signal RT and Stop-Signal – Go-Trial RT Sd. Multiple Regression (corresponds with Table A.29, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

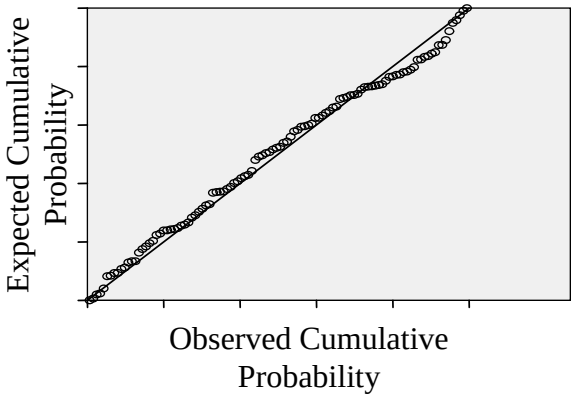
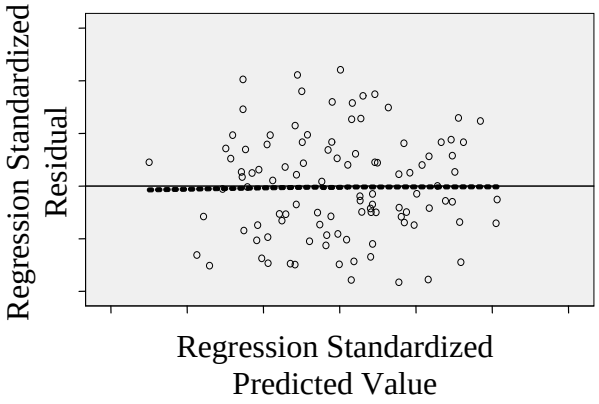


Figure D.24. Graphical Displays Testing Residual Assumptions for Stop-Signal Go-Trial RT and Stop-Signal – Go-Trial RT Sd. Multiple Regression (corresponds with Table A.30, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

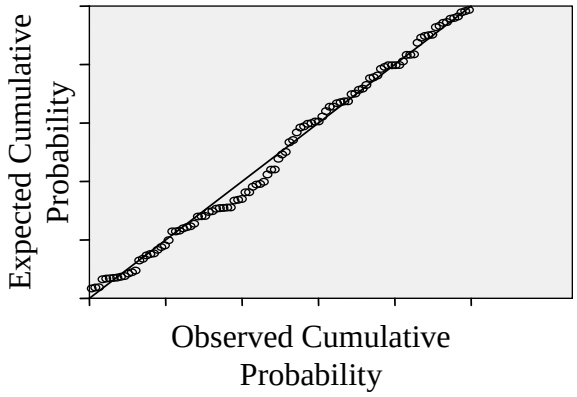
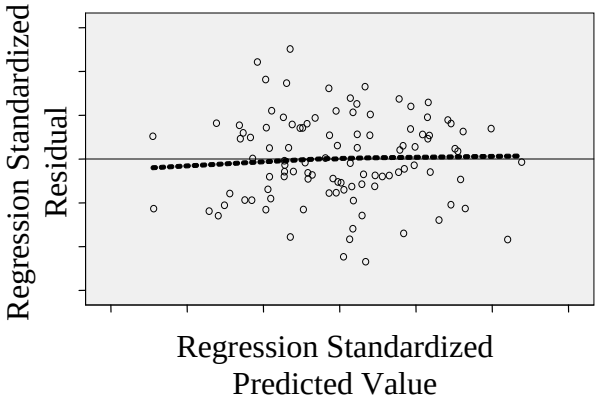


Figure D.25. Graphical Displays Testing Residual Assumptions for Stop-Signal Go-Trial RT and WAIS – Saddler 2 Composite Multiple Regression (corresponds with Table A.31, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

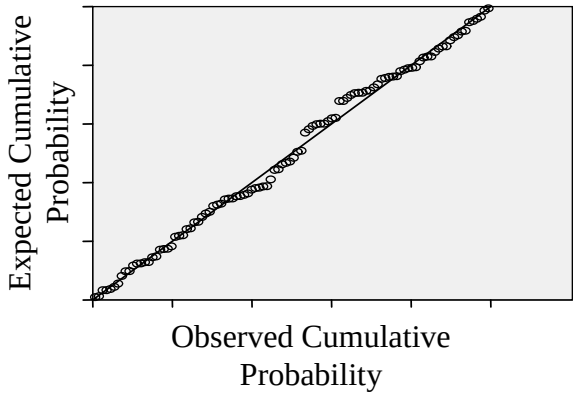
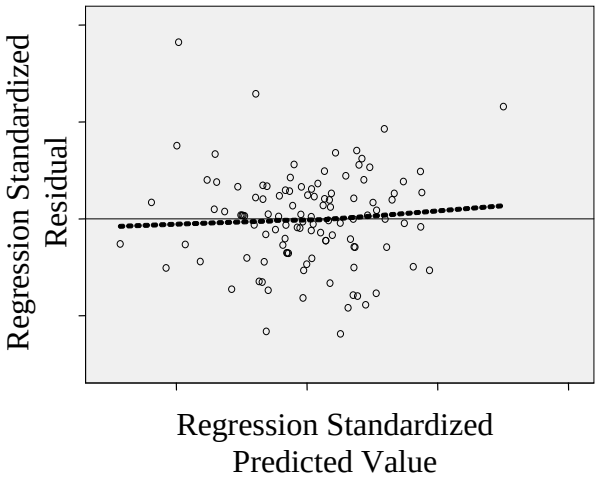


Figure D.26. Graphical Displays Testing Residual Assumptions for Stop-Signal Go-Trial RT Sd. and WAIS – Saddler 2 Composite Multiple Regression (corresponds with Table A.32, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

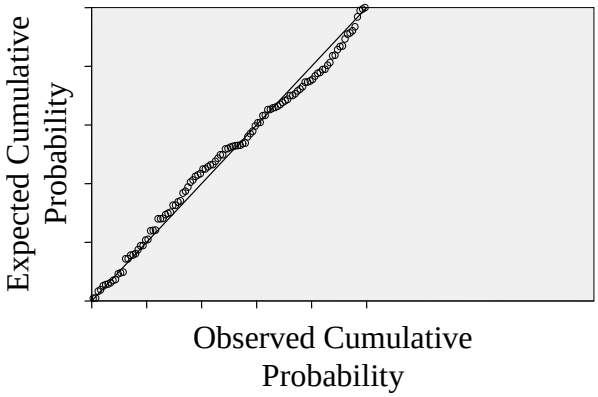
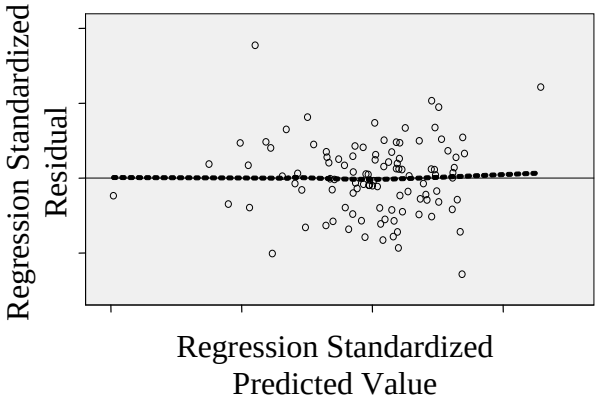


Figure D.27. Graphical Displays Testing Residual Assumptions for Stroop Interference and WMS – Working Memory Multiple Regression (corresponds with Table A.33, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

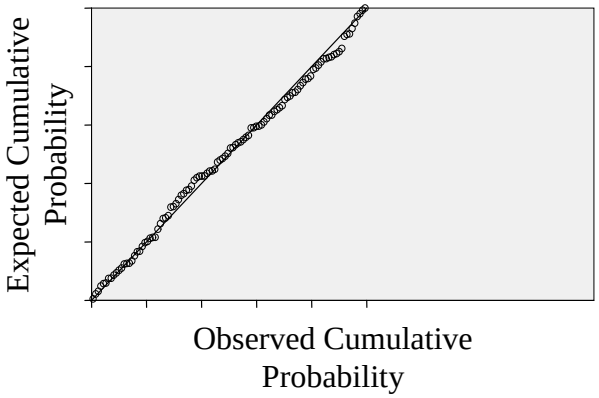
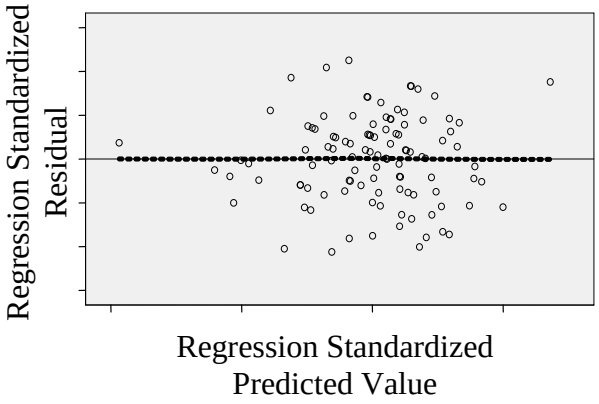


Figure D.28. Graphical Displays Testing Residual Assumptions for Stroop Interference and WAIS – Saddler 2 Composite Multiple Regression (corresponds with Table A.34, Appendix A).

Assumptions: Linearity & Homoscedasticity



Assumption: Normality

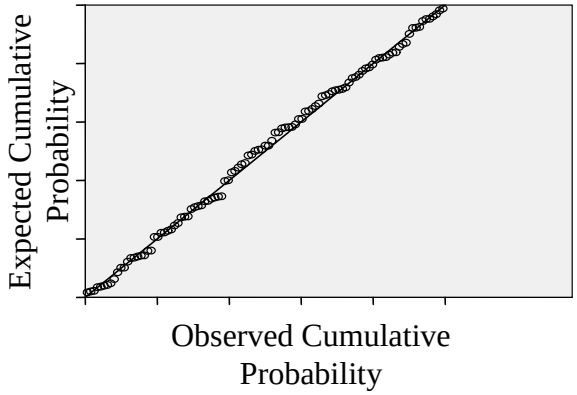


Figure D.29. Graphical Displays Testing Residual Assumptions for WMS – Working Memory and WAIS – Saddler 2 Composite Multiple Regression (corresponds with Table A.35, Appendix A).

Appendix E
Self-Report Measures

Demographic Information

1. Age: _____

2. Gender: Female Male

3. Racial Heritage: African-American or Black

American Indian or Alaska Native

Asian

Hispanic or Latino

Native Hawaiian or other Pacific Islander

White (non-Hispanic)

Other (please describe):_____

TASK COMPLETION QUESTIONNAIRE

We would like to know now about your experience during our study. We are especially interested in how motivated you were to complete each of the measures and whether or not you were distracted while completing these measures. This information can be VERY IMPORTANT and particularly useful in helping us determine if the data we collected is accurate and a true reflection of your abilities.

We also recognize that some participants may be reluctant to provide information related to their study involvement. For example, if a participant was feeling tired and not motivated to put forth his/her best effort, the participant may feel embarrassed if the experimenter knew about this.

Therefore, we also ask that after you have completed this questionnaire, please PLACE THIS QUESTIONNAIRE IN THE ENVELOPE PROVIDED AND SEAL THE ENVELOPE. We will not look at the contents of the envelope until long after you have left the study to help further ensure that we do not know which data belongs to you. ***You should also know that you will still receive 2.0 hours of REP credit, no matter how you respond to the following questionnaire items.***

Using the scale below as a guide, write a number beside each statement to indicate how much you agree with it.

1	2	3	4
Strongly Agree	Moderately Agree	Moderately Disagree	Strongly Disagree

1. I felt **tired (or sleepy)** when completing the computerized tests.

1 2 3 4

2. I did NOT pay **attention** when completing the computerized tests.

1 2 3 4

3. I was **distracted** when completing the computerized tests.

1 2 3 4

1	2	3	4
Strongly Agree	Moderately Agree	Moderately Disagree	Strongly Disagree

4. I was feeling **stressed** when completing the computerized tests.

1 2 3 4

5. I did NOT put for my best **effort** when completing the computerized tests.

1 2 3 4

6. I felt **tired (or sleepy)** when completing the cognitive tests given to me by the experimenter.

1 2 3 4

7. I did NOT pay **attention** when completing the cognitive tests given to me by the experimenter.

1 2 3 4

8. I was **distracted** when completing the cognitive tests given to me by the experimenter.

1 2 3 4

9. I was feeling **stressed** when completing the cognitive tests given to me by the experimenter.

1 2 3 4

10. I did NOT put for my best **effort** when completing the cognitive tests given to me by the experimenter.

1 2 3 4

11. I felt **tired (or sleepy)** when completing the questionnaires.

1 2 3 4

1	2	3	4
Strongly Agree	Moderately Agree	Moderately Disagree	Strongly Disagree

12. I did NOT pay **attention** when completing the questionnaires.

1	2	3	4

13. I was **distracted** when completing the questionnaires.

1	2	3	4

14. I was feeling **stressed** when completing the questionnaires.

1	2	3	4

15. I did NOT put for my best **effort** when completing the questionnaires.

1	2	3	4

Appendix F
Study Materials

RECRUITMENT SCRIPT
(POSTED ON R.E.P. WEBSITE)

Protocol Title: **Effortful Self-Regulation in Personality Research: A Multi-Method Validation of Questionnaire-Based Measures of Effortful Control**

Investigators: Michael W. Vasey, Ph.D. & Salvatore Dinovo, M.A.

Title: Cognition & Self-Regulation

Hours: 2.0

Requirements: Must attend one session lasting approximately 120 minutes, during which questionnaires, tests of cognitive ability, and non-invasive heart rate measurements will administered and completed. Participants must be 18 years of age or older. To participate, you must bring a picture ID confirming your age.

Researcher: Michael Vasey & Salvatore Dinovo

Researcher E-Mail: dinovo.13@osu.edu

INTRODUCTORY SCRIPT

"Hello and welcome to our experiment. The study that you are about to participate in will involve your completing questionnaires and other measures and will last approximately 2 hours. For your participation you will receive 2.0 hours of REP credit."

"First, you will be completing a variety of tests of cognition, some of which measure facets of IQ. We do ask that you try your best on each of these tests so that we can obtain the most accurate results possible."

"After completing these cognitive tests, you will complete a battery of questionnaires. The questionnaire packet consists of questions that require you to disclose information of a personal nature. If you feel at any time uncomfortable answering a particular question, you may leave the item blank."

"While completing both the cognitive tests and questionnaires, you will wear a polar wrist watch that will take measurements of your heart rate."

"To preserve your confidentiality, we ask that NO information related to your personal identity be provided. Instead, your data will be linked to an arbitrary identification number that will NOT be connected to your name in any way. This way, even we would not be able to break confidentiality because we will not know which data is yours."

"You should know that you have the option NOT to participate and may withdraw at any time without penalty. In other words, you'll still be given 2.0 hours of REP credit, whether or not you choose to complete the study."

"Before you leave you will be given a sheet which will explain the study in a little more detail. If you have any questions I will gladly assist you. We greatly appreciate your participation!"

DEBRIEFING

Protocol Title: **Effortful Self-Regulation in Personality Research: A Multi-Method Validation of Questionnaire-Based Measures of Effortful Control**

Principal Investigators: Michael W. Vasey & Salvatore A. Dinovo

We greatly appreciate your participation. The tests you just completed are intended to help us determine the extent to which tests of thinking and cognition are related to questionnaire-based measures of self-regulation: a capacity that helps a person control his or her emotions and behaviors (see your Psychology 100 text, page 483).

Previous research has shown that individuals who have self-regulation deficits think about negative experiences with great frequency are more likely to develop an anxiety or depressive disorder. While most people reflect upon experience from time to time, persons with these disorders experience such rumination as more distressing and disabling than do most people. They also seem to find MORE environmental events distressing. We hypothesize that this disparity reflects individual differences in the ability to control one's emotional reactions to negative stimuli in one's environment. This ability is captured in a self-regulatory construct known as ***effortful control (EC)***.

Unfortunately, most of the research investigating *effortful control* has relied upon questionnaires, which may or may not be accurate measures of self-regulation. An important step remedying this problem is to see if these questionnaires are related to more objective measures of self-regulation, such as the tests of cognition and heart rate measurements you just completed.

As stated at the beginning of today's study, some of the measures you completed measure components of IQ. However, since we are only measuring parts of IQ, we have no adequate basis for providing you reliable scientific feedback on matters related to intelligence or IQ. Moreover, since we are only measuring specific components of IQ, you should not treat your performance on any these measures as a reliable predictor of your intelligence, or probable academic or vocational success.

Should you have ANY questions about this study in the future, you may contact Dr. Michael Vasey at: vasey.1@osu.edu or 614-292-2951.

Moreover, if at any time you feel that anxiety or depression is a significant problem for you, we would gladly assist you in finding help. One source of services you may wish to consider is the OSU Counseling and Consultation

Service, which is located in the Younkin Success Center. Their telephone number is 292-5766. Alternatively, you may wish to contact one of the following agencies in the Columbus area:

Anxiety and Stress Disorder Clinic
Consultation Services
223 Townshend Hall
614-292-2345

OSU Psychological Services Center
Mental Health
105 Psychology Building
614-292-2059

Mental Health Association of Franklin County
614-221-1441
Broad Street

OSU Counseling and

4th Floor, Younkin Success Center
614-292-5766

Columbus Area Community

614-252-0711

For emergencies:
Net Care Access
199 S. Central or 741 E. Broad Street
614-276-2273