IMPULSIGENIC TRAITS AND MIDLIFE CARDIOMETABOLIC RISK: THE MEDIATING ROLE OF MALADAPTIVE HEALTH BEHAVIORS

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Although impulsivity predisposes individuals to engage in maladaptive health behaviors that confer risk for cardiometabolic diseases, limited research has evaluated behavioral pathways through which distinct impulsigenic traits promote cardiometabolic risk. The present study aimed to provide a fuller understanding of the distinct impulsigenic traits most strongly related to cardiometabolic risk and to identify specific behavioral mechanisms driving these relationships. Community adults (N = 1295) between the ages of 30 and 54 years (53% female, 84% Caucasian) completed a battery of impulsivity measures, reported their engagement in health behaviors over the past week (i.e., cigarette smoking, alcohol use, physical activity, energy intake, and dietary composition), and were assessed for measures of cardiometabolic risk (i.e., adiposity, blood pressure, insulin resistance, and dyslipidemia). Structural equation modeling was used to estimate previously established hierarchical models of distinct impulsigenic traits and cardiometabolic risk. Indirect effects through the observed health behaviors were then examined for each association between the seven latent impulsigenic traits identified and the latent cardiometabolic risk factor. Results indicated that Neuroticism/Negative Emotionality was the only latent impulsigenic trait directly related to heightened cardiometabolic risk (β = 0.09, 95% CI [0.01, 0.16], p = 0.02). In addition, Extraversion/Positive Emotionality indirectly related to reduced cardiometabolic risk

through greater physical activity (β = -0.04, 95% CI [-0.06, -0.02], $p < 0.01$), and both Inhibition (β = 0.02, 95% CI [0.001, 0.04], p = 0.05) and Impulsive Decision-Making (β = 0.08, 95% CI [0.001, 0.15], p = 0.05) indirectly related to cardiometabolic risk through saturated fat intake, but in opposing directions. Specifically, individuals low on Inhibition were at reduced cardiometabolic risk as a consequence of less saturated fat intake whereas individuals high on Impulsive Decision-Making were at heightened cardiometabolic risk as a consequence of greater saturated fat intake. These findings indicate that distinct impulsigenic traits differentially relate to cardiometabolic risk through varied behavioral pathways and ultimately serve to clarify both *who* is at cardiometabolic risk and *how* those individuals are at risk.

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1.0 INTRODUCTION

Metabolic syndrome (MetS) refers to a constellation of anthropometric, metabolic, and hemodynamic risk factors that together contribute to heightened risk for cardiometabolic morbidity and mortality (Alberti et al., 2009). MetS is associated with a twofold increase in risk for cardiovascular disease (Mottillo et al., 2010) and a fivefold increase in risk for type 2 diabetes (Ford, Li, & Sattar, 2008). MetS is further associated with dysfunction in the renal (Locatelli, Pozzoni, & Del Vecchio, 2006), hepatic (Hamaguchi, Kojima, Takeda, & et al., 2005), ocular (Chopra, Chander, & Jacob, 2012), respiratory (Leone et al., 2009), and nervous (Akbaraly et al., 2010) systems. Although the prevalence of MetS varies widely across sociodemographic groups (Aguilar, Bhuket, Torres, Liu, & Wong, 2015; Cameron, Shaw, & Zimmet, 2004; Park et al., 2003), epidemiological studies have consistently documented a steady increase in the overall prevalence of MetS in both developed (Aguilar et al., 2015; Ford, Giles, & Mokdad, 2004; Khunti et al., 2010) and developing (Misra & Khurana, 2008) nations. For example, the prevalence of MetS has significantly increased in the United States over the past decade, with nearly 35% of all United States adults currently meeting criteria for the syndrome (Aguilar et al., 2015).

The systemic health effects and rising prevalence of MetS have conjointly led to the consideration of the syndrome as a serious public health concern. The American Heart Association recently issued a call to action for healthcare providers and clinical researchers to improve the cardiovascular health of the nation by developing more effective methods for identification and

treatment of MetS (Mozaffarian et al., 2015). In line with this aim, accumulating evidence has identified biological (Halder, Muldoon, Ferrell, & Manuck, 2007; Marsland, McCaffery, Muldoon, & Manuck, 2010; Scott, Carter, & Grant, 2008), psychological (De Bacquer et al., 2009; Räikkönen, Matthews, & Kuller, 2007; Vitaliano et al., 2002; Weiss et al., 2011), and socioenvironmental (Clark et al., 2013; Manuck, Phillips, Gianaros, Flory, & Muldoon, 2010; Matthews, Räikkönen, Gallo, & Kuller, 2008) mechanisms that interact to differentially predict risk for MetS. Despite the multifactorial nature of MetS, the syndrome continues to be largely considered a disease of unhealthy lifestyle (Alkerwi et al., 2009; Edwardson et al., 2012; Ferreira, Twisk, van Mechelen, Kemper, & Stehouwer, 2005; Fogli-Cawley et al., 2007; Malik et al., 2010; Mozaffarian et al., 2015; Santos, Ebrahim, & Barros, 2007; Sun, Liu, & Ning, 2012; Zhu, St-Onge, Heshka, & Heymsfield, 2004). Accordingly, identifying factors that predispose individuals to engage in maladaptive health behaviors that augment risk for MetS could aid in the development of more targeted prevention and intervention efforts designed to mitigate the health consequences of cardiometabolic diseases.

Personality traits are enduring characteristics that reliably distinguish who is likely to engage in maladaptive health behaviors (Mischel & Shoda, 1995) and have thus received growing attention as relevant indicators for the development and persistence of MetS (Cohen, Panguluri, Na, & Whooley, 2010; Elovainio et al., 2011; Mommersteeg, Kupper, & Denollet, 2010; Räikkönen, Matthews, & Salomon, 2003; Räikkönen, Matthews, Sutton-Tyrrell, & Kuller, 2004; Tziallas et al., 2011). Although the majority of previous research has focused on how personality traits characterized by a proneness towards aggression and negative affectivity relate to MetS (Goldbacher & Matthews, 2007), impulsivity has recently garnered attention as another meaningful personality predictor of MetS (Phillips et al., 2010; Sutin, Costa, et al., 2010).

Impulsivity is defined by a tendency to act on immediate urges either before or despite consideration of negative consequences (DeYoung, 2010) and is associated with a heightened likelihood of habitually engaging in maladaptive health behaviors that confer cardiometabolic disease risk (Brogan, Hevey, O'Callaghan, Yoder, & O'Shea, 2011; Franken & Muris, 2005; Ishizawa, Kumano, Sato, Sakura, & Iwamoto, 2010; Jokela et al., 2014; Lane et al., 2000; Lillis, Levin, & Trafton, 2012; Meule & Platte, 2015; Mobbs, Crépin, Thiéry, Golay, & Van der Linden, 2010; Shipley, Weiss, Der, Taylor, & Deary, 2007). As such, individuals high on impulsivity may be at heightened risk for MetS due to a tendency towards an unhealthy lifestyle. However, limited research has attempted to understand the distinct behavioral pathways through which impulsivity promotes risk for MetS.

The present study aims to examine whether the associations between several related but distinct impulsigenic traits and MetS are differentially driven by varied maladaptive health behaviors. Impulsivity first will be proposed as an important health risk indicator that predisposes individuals to engage in maladaptive health behaviors known to confer risk for MetS. A detailed overview of the measurements commonly used to assess impulsivity also will be provided as will an empirical framework with which to conceptualize measures of impulsivity into distinct impulsigenic traits. The relationship between specific maladaptive health behaviors and risk for MetS subsequently will be reviewed, with a focus on the pathophysiologic mechanisms underlying each risk relation. Finally, the results from a structural equation modeling approach used to test the mediating effects of varied health behaviors on the associations between distinct impulsigenic traits and cardiometabolic risk will be detailed, followed by a discussion of the study implications and future directions.

1.1 PERSONALITY AND METABOLIC SYNDROME

MetS is diagnosed in the presence of several interrelated cardiometabolic risk factors, including elevated blood pressure, insulin resistance, visceral adiposity, and dyslipidemia (Alberti et al., 2009). Though MetS arises from a complex interplay between myriad biopsychosocial factors (Clark et al., 2013; De Bacquer et al., 2009; Halder et al., 2007; Manuck et al., 2010; Marsland et al., 2010; Matthews et al., 2008; Räikkönen et al., 2007; Vitaliano et al., 2002; Weiss et al., 2011), the syndrome largely is promoted through the habitual engagement in unhealthy lifestyle behaviors (Santos et al., 2007; Zhu et al., 2004). Indeed, both prospective and retrospective reports have established cigarette smoking (Sun et al., 2012), heavy alcohol use (Alkerwi et al., 2009), physical inactivity (Ferreira et al., 2005), and unhealthy diet (Fogli-Cawley et al., 2007; Malik et al., 2010) as core maladaptive health behaviors that directly contribute to the pathogenesis of MetS. These findings have subsequently prompted extensive research efforts aimed at identifying factors that predispose individuals to engage in maladaptive health behaviors that increase their vulnerability for cardiometabolic dysfunction and disease (Boersma, Benthem, van Beek, van Dijk, & Scheurink, 2011; Levitsky, 2005).

One promising line of research has focused on the specific role that personality traits play in promoting risk for MetS (Cohen et al., 2010; Elovainio et al., 2011; Mommersteeg et al., 2010; Räikkönen et al., 2003; Räikkönen et al., 2004; Tziallas et al., 2011). Personality traits are considered enduring characteristics (Roberts & DelVecchio, 2000) that reliably predict individual patterns in thinking, feeling, and behaving (Kazdin, 2000; Mischel & Shoda, 1995). Personality traits have therefore received growing attention as valuable determinants of behavioral health outcomes (Harper, 2004) and have been implicated in the etiology and prognosis of myriad physical (Adler & Matthews, 1994; Boersma et al., 2011; Bogg & Roberts, 2004; Jokela et al.,

2014; Jokela et al., 2013) and psychological (Bornovalova, Lejuez, Daughters, Zachary Rosenthal, & Lynch, 2005; de Wit, 2009; Swann, 2009; Waxman, 2009; Winstanley, Eagle, & Robbins, 2006) disorders. Although numerous conceptual models have been proposed to explain how personality promotes or protects against disease states (Adler & Matthews, 1994; Deci & Ryan, 2008; Montano, Kasprzyk, Glanz, Rimer, & Viswanath, 2008; Smith, 2006; Ursin, 1980), there exists widespread agreement that personality traits largely influence health processes through their action on health behaviors (Adler & Matthews, 1994; Smith, 2006). Specifically, individual differences in personality predict general health habits, which, in turn, affect broader health outcomes. Thus, individual differences in personality traits may be especially useful in distinguishing who is likely to develop MetS as a consequence of an unhealthy lifestyle.

Extensive research efforts have assessed the overall relationship between personality traits and MetS, with the majority of this research focusing on personality traits characterized by a proneness towards aggression and negative affectivity (Goldbacher & Matthews, 2007). In particular, trait hostility (Cohen et al., 2010; Elovainio et al., 2011; Räikkönen et al., 2003), trait anger (Cohen et al., 2010; Räikkönen et al., 2004), and Type D personality styles (Mommersteeg et al., 2010; Tziallas et al., 2011) are personality traits associated with the development and persistence of the syndrome. Impulsivity is a related personality trait that has garnered broad attention as an indicator of cardiometabolic risk (Brogan et al., 2011; Franken & Muris, 2005; Ishizawa et al., 2010; Jokela et al., 2014; Lane et al., 2000; Lillis et al., 2012; Meule & Platte, 2015; Mobbs et al., 2010; Shipley et al., 2007), with additional evidence documenting that impulsivity may have specific importance as a predictor of MetS (Phillips et al., 2010; Sutin, Costa, et al., 2010). Despite the compelling evidence linking impulsivity to MetS and related cardiometabolic outcomes, limited work has sought to extend these findings by using a

multidimensional assessment of impulsivity or by examining the specific behavioral mechanisms driving the association between impulsivity and MetS.

1.1.1 Impulsivity and metabolic syndrome

Impulsivity generally is defined by a tendency to act on immediate urges either before or despite consideration of negative consequences and predisposes individuals to engage in maladaptive health behaviors that foster adverse health outcomes (DeYoung, 2010). Impulsivity develops from genetic and environmental factors (Meyer-Lindenberg et al., 2006; Niv, Tuvblad, Raine, Wang, & Baker, 2012) that promote the emergence of dysfunctional neural processes in frontostriatal and limbic systems (Cyders & Smith, 2008b; Davidson, Putnam, & Larson, 2000; Winstanley et al., 2006). In particular, impulsivity is associated with neurobiological mechanisms that potentiate a heightened sensitivity to rewards (Kirby, Zeeb, & Winstanley, 2011; Volkow, Fowler, Wang, & Swanson, 2004) coupled with broad deficits in executive processes associated with attention (Weissman, Roberts, Visscher, & Woldorff, 2006), inhibition (Garavan, Ross, Murphy, Roche, & Stein, 2002), decision-making (Peters & Büchel, 2009; Platt & Huettel, 2008), and emotion regulation (Cyders & Smith, 2007; Cyders & Smith, 2008b). Consequently, individuals high on impulsivity have difficulty maintaining inhibitory control and exhibit a heightened propensity to engage in rewarding and disinhibited behaviors. Impulsivity is indeed related to a greater likelihood of engaging in each of the core maladaptive health behaviors associated with MetS, including cigarette smoking (Dallery & Raiff, 2007; Friedel, DeHart, Madden, & Odum, 2014; VanderVeen, Cohen, Cukrowicz, & Trotter, 2008), heavy alcohol use (Coskunpinar, Dir, & Cyders, 2013; Goudriaan, Grekin, & Sher, 2011; Magid, MacLean, & Colder, 2007; Rubio et al., 2008), physical inactivity (Allen, Walter, & McDermott, 2016; Sutin et al., 2016), and unhealthy

diet (Davis, 2009; Kakoschke, Kemps, & Tiggemann, 2015; Sproesser, Strohbach, Schupp, & Renner, 2011). Accordingly, individuals high on impulsivity may be particularly likely to develop MetS as a consequence of unhealthy lifestyle behaviors.

Despite the substantial evidence linking impulsivity to the core maladaptive health behaviors that confer risk for MetS, limited research has assessed the direct association between impulsivity and the syndrome as a whole. Impulsivity has long been cited as a risk factor for related cardiometabolic diseases, including obesity (Brogan et al., 2011; Emery & Levine, 2017; Franken & Muris, 2005; Lillis et al., 2012; Meule & Platte, 2015; Mobbs et al., 2010), cardiovascular disease (Shipley et al., 2007), and type 2 diabetes (Ishizawa et al., 2010; Jokela et al., 2014; Lane et al., 2000). Impulsivity has been further implicated as a risk factor for each of the component parts of MetS, including elevated blood pressure (Goodwin, Cox, & Clara, 2006), insulin resistance (Ishizawa et al., 2010), visceral adiposity (Armon, Melamed, Shirom, Shapira, & Berliner, 2013; Terracciano et al., 2009), and dyslipidemia (Sutin, Terracciano, Deiana, Uda, et al., 2010). However, findings documenting a relationship between impulsivity and MetS have been equivocal, with some studies showing that impulsive personality traits are associated with a greater likelihood of being diagnosed with MetS (Dermody et al., 2015; Phillips et al., 2010; Sutin, Costa, et al., 2010) and others reporting no relationship between impulsive personality traits and MetS (Ross, Martin, Chen, & Miller, 2011; van Reedt Dortland, Giltay, van Veen, Zitman, & Penninx, 2012).

These mixed findings may be partly explained by limitations associated with the particular measures used to assess impulsive personality traits. For example, each of the aforementioned studies utilized measures of Neuroticism, a broad personality trait characterized by a proneness towards psychological distress, but failed to include more direct assessments of impulsivity.

Although impulsivity is considered a primary component of Neuroticism, Neuroticism itself is a composite measure of several characteristics that encompass, but do not exclusively assess, impulsivity. Given that measures of Neuroticism assess broad personality traits that underlie specific types of impulsive behaviors, they may lack the specificity needed to reliably predict complicated disease states like MetS (Mommersteeg & Pouwer, 2012). In support of this proposition, Sutin and colleagues (2010) found that individuals high on Neuroticism were 15% more likely to meet criteria for MetS than were those low on Neuroticism. However, upon further inspection of the specific personality subfacets comprising the Neuroticism subscale, the impulsivity subfacet was shown to be associated with a 33% greater chance of having the syndrome, making it the strongest personality predictor of MetS in their sample. Thus, impulsivity is a potentially important risk factor for the development and maintenance of MetS. However, understanding whether and how impulsivity relates to MetS likely requires the use of more comprehensive measurements of impulsivity.

1.1.2 Conceptualization and measurement of impulsivity

There historically has been limited consensus regarding the operationalization of impulsivity. Although early personality theorists viewed impulsivity as a unidimensional construct (Guilford, 1939), current conceptualizations recognize impulsivity as a broad personality trait comprising several distinct impulsive phenotypes that manifest in varied behavioral patterns (Sharma, Markon, & Clark, 2014; Whiteside & Lynam, 2001). Despite the widespread agreement that impulsivity is a multifaceted personality trait, there remains ongoing debate surrounding the appropriate definition and identification of impulsivity and its underlying facets (Sharma et al., 2014; Whiteside & Lynam, 2001). Researchers have consequently developed many diverse models and

measures of impulsivity (Carver, 2005; Cyders & Smith, 2008b; Dalley, Everitt, & Robbins, 2011; Gray, 1970; Hamilton et al., 2015; Miyake et al., 2000; Whiteside & Lynam, 2001; Zuckerman, Kolin, Price, & Zoob, 1964), which have ultimately served to further complicate our understanding of both what impulsivity is and how best to quantify it.

Impulsivity is most commonly assessed through two broad measurement modalities, including questionnaire measures of impulsivity and behavioral task measures of impulsivity. Questionnaire measures of impulsivity assess stable personality traits that contribute to impulsive characteristics by asking respondents to self-report the extent to which they engage in impulsive behaviors. Meanwhile, behavioral task measures of impulsivity assess behavioral responses to controlled laboratory paradigms and provide a measure of state variability in impulsive action that is thought to reflect underlying trait characteristics (Cyders & Coskunpinar, 2011). Researchers often use questionnaire and behavioral task measures of impulsivity interchangeably under the supposition that they are measuring the same broad trait. However, it has long been speculated that questionnaire and behavioral task measures of impulsivity are subject to the jingle fallacy, which refers to the erroneous assumption that two different constructs are the same because they share the same name (Whiteside & Lynam, 2001). Indeed, recent psychometric reports have confirmed this suspicion by documenting weak (Cyders & Coskunpinar, 2011; Lane, Cherek, Rhoades, Pietras, & Tcheremissine, 2003; Reynolds, Ortengren, Richards, & de Wit, 2006) to moderate (Duckworth & Kern, 2011) convergence between questionnaire and behavioral task measures of impulsivity, thereby indicating that varied measurement constructs commonly labeled as impulsivity actually comprise a multitude of related but distinct impulsigenic traits.

Several lines of research have separately sought to identify the primary impulsigenic traits assessed by questionnaire and behavioral task measures of impulsivity. The majority of this

research has focused on questionnaire measures of impulsivity, with preliminary findings documenting that questionnaire measures of impulsivity can best be conceptualized as assessing broad tendencies to engage in rash action either through deficits in premeditation and perseverance (Lynam & Miller, 2004; Whiteside & Lynam, 2001), difficulties managing the experience of positive and negative affective states (Carver, 2005; Cyders & Smith, 2007; Cyders & Smith, 2008b; Gray, 1970), or a desire to seek out novel and intense experiences (Zuckerman et al., 1964). Meanwhile, additional evidence from neuropsychological research has suggested that behavioral task measures of impulsivity primarily assess behavioral manifestations of impulsigenic traits that stem either from a general incapacity to inhibit prepotent motor responses or to delay gratification (Dalley et al., 2011; Hamilton et al., 2015) or from a general inability to flexibly shift away from ineffective mental sets or to sustain selective attention (Miyake et al., 2000). Although these independent lines of research have together contributed to a growing understanding of the specific impulsigenic traits that underlie questionnaire and behavioral task measures of impulsivity, limited research has attempted to integrate these findings to develop a more unified theory of impulsivity.

Sharma and colleagues (2014) recently aimed to address this gap in the literature by conducting an extensive meta-analytic principal-components factor analysis of the most commonly used questionnaire and behavioral task measures of impulsivity. Results from their analysis indicated that questionnaire measures of impulsivity aligned with three latent factors. The first of these latent questionnaire factors was labeled Disinhibition versus Constraint/Conscientiousness (DvC/C) and comprised questionnaire measures of impulsivity that assess a general inability to engage in planned and thoughtful action, persevere on difficult or monotonous tasks, and maintain inhibitory control. The second of these latent questionnaire factors was labeled Extraversion/Positive Emotionality (E/PE) and comprised questionnaire measures of

impulsivity that assess sensation and novelty seeking as well as reward sensitivity. The E/PE factor was further theorized to be strongly related to the behavioral activation system, which promotes both a tendency to approach rewarding stimuli and a propensity to engage in impulsive behaviors that are positively reinforcing (Carver, 2005; Gray, 1970). The third and final of these latent questionnaire factors was labeled Neuroticism/Negative Emotionality (N/NE) and comprised questionnaire measures of impulsivity that assess a proneness towards psychological distress and a disposition to act rashly in the face of negative emotions. The N/NE factor was also theorized to be strongly related to the behavioral inhibition system, which results in a predisposition to avoid aversive stimuli and engage in impulsive behaviors that are negatively reinforcing (Carver, 2005; Gray, 1970). Importantly, although the DvC/C, E/PE, and N/NE factors are referred to as distinct impulsigenic traits throughout the present document, they each include general measures of personality traits that contain impulsive features as well as specific measures of impulsigenic traits. Accordingly, the DvC/C, E/PE, and N/NE factors are best conceptualized as being three broad personality traits that underlie unique manifestations of impulsive behavior.

Sharma and colleagues (2014) further found that behavioral task measures of impulsivity aligned with four latent factors. The first of these latent behavioral task factors was labeled Inattention and comprised behavioral task measures of impulsivity that assess a general capacity to engage in selective attention by requiring respondents to selectively attend to a target stimulus while suppressing a distractor stimulus. The second of these latent behavioral task factors was labeled Inhibition and comprised behavioral task measures of impulsivity that assess the overall ability of an individual to inhibit behavioral impulses by requiring respondents to selectively respond to target stimuli while suppressing prepotent motor responses. The third of these latent behavioral task factors was labeled Impulsive Decision-Making and comprised behavioral task

measures of impulsivity that assess both a tendency to make risky decisions and a general inability to tolerate reinforcement delays by requiring respondents either to earn larger rewards at increasing risk of loss or to select between small, immediate rewards and larger, delayed rewards. The fourth and final of these latent behavioral task factors was labeled Set-Shifting and comprised behavioral task measures of impulsivity that assess cognitive flexibility by requiring respondents to change their approach to a given exercise by resisting memory intrusions of no longer relevant information.

Sharma and colleagues went on to evaluate the convergent and divergent validity of each impulsigenic trait they identified. Consistent with previous reports (Cyders & Coskunpinar, 2011; Duckworth & Kern, 2011; Lane et al., 2003; Reynolds et al., 2006), the authors documented weak to moderate convergence among the impulsigenic traits derived from the same measurement modality, with the latent questionnaire factors tending to be more highly correlated than the latent behavioral task factors. The authors further demonstrated that the latent questionnaire factors were largely unrelated to the latent behavioral task factors, indicating that the impulsigenic traits assessed using the two different measurement modalities were largely discriminant. Sharma and colleagues (2014) further documented that the impulsigenic traits they identified uniquely predicted distinct maladaptive health behaviors. However, the authors cautioned that these findings should be interpreted as preliminary due to a lack of sufficient literature relating multiple measures of impulsivity to maladaptive health behaviors. Thus, although these findings provide increasing support for the notion that commonly used questionnaire and behavioral task measures of impulsivity comprise several related but distinct impulsigenic traits, there exists a need for additional research to test the predictive validity of these particular impulsigenic traits in relation to varied health outcomes and behaviors.

Two recent studies have attempted to extend the work of Sharma and colleagues (2014) by relating the impulsigenic traits they identified both to problematic health outcomes (Emery & Levine, 2017) and to maladaptive health behaviors (Creswell, Wright, Flory, Skrzynski, & Manuck, 2018). Results from a comprehensive meta-analysis specifically documented that the DvC/C, Inattention, Impulsive Decision-Making, and Set-Shifting factors were distinctly associated with higher body mass index (Emery & Levine, 2017). A yet unpublished study further demonstrated that the DvC/C, E/PE, N/NE, Impulsive Decision-Making, and Set-Shifting factors each uniquely related to higher engagement in varied externalizing behaviors (Creswell et al., 2018). Together, these findings document the potential utility of using distinct impulsigenic traits to identify subsets of individuals who are likely to develop either problematic health outcomes or to engage in maladaptive health behaviors. However, no studies have attempted to integrate these findings to explore whether distinct impulsigenic traits promote problematic health outcomes through different behavioral pathways. For example, although impulsivity is broadly associated with a greater likelihood of engaging in maladaptive health behaviors that confer risk for MetS, including cigarette smoking (Dallery & Raiff, 2007; Friedel et al., 2014; VanderVeen et al., 2008), heavy alcohol use (Coskunpinar et al., 2013; Goudriaan et al., 2011; Magid et al., 2007; Rubio et al., 2008), physical inactivity (Allen et al., 2016; Sutin et al., 2016), and unhealthy diet (Davis, 2009; Kakoschke et al., 2015; Sproesser et al., 2011), the pattern of these associations may depend on the particular impulsigenic trait assessed. Accordingly, there is a need to better understand the specific behavioral mechanisms through which distinct impulsigenic traits alter risk for MetS.

1.2 MALADAPTIVE HEALTH BEHAVIORS AND METABOLIC SYNDROME

In contrast to the paucity of literature exploring the unique behavioral pathways linking distinct impulsigenic traits to MetS, the pathophysiologic mechanisms driving the associations among maladaptive health behaviors and MetS have been widely investigated. As reviewed below, cigarette smoking (Sun et al., 2012), heavy alcohol use (Alkerwi et al., 2009), physical inactivity (Ferreira et al., 2005), and unhealthy diet (Fogli-Cawley et al., 2007; Malik et al., 2010) have all independently been linked to cardiometabolic abnormalities that contribute to the expression of MetS.

1.2.1 Cigarette smoking and metabolic syndrome

Extensive evidence has documented a positive relationship between cigarette smoking and MetS (Geslain-Biquez et al., 2003; Ishizaka et al., 2005; Kawada et al., 2010; Miyatake et al., 2006; Nakanishi, Takatorige, & Suzuki, 2005; Santos et al., 2007; Sun et al., 2012; Wada, Urashima, & Fukumoto, 2007; Weitzman et al., 2005; Wilsgaard & Jacobsen, 2007; Zhu et al., 2004), with meta-analytic findings showing that individuals who currently smoke cigarettes have a 26% greater chance of meeting criteria for MetS than do nonsmoking individuals (Sun et al., 2012). Additional research has uncovered a positive dose-response relationship between the number of cigarettes smoked daily and risk for MetS in both adult (Nakanishi et al., 2005; Sun et al., 2012; Wada et al., 2007) and adolescent populations (Weitzman et al., 2005). Importantly, several studies have found that former cigarette smoking continues to be associated with a heightened incidence of MetS (Ishizaka et al., 2005; Nakanishi et al., 2005) for up to two decades after smoking cessation (Wada et al., 2007). Thus, although smoking cessation drastically reduces cardiometabolic disease

risk (Bullen, 2008), both current and former cigarette smokers remain vulnerable to developing MetS.

Cigarette smoking contributes to the expression of MetS by promoting cardiometabolic abnormalities related to the pathogenesis of the syndrome, including insulin resistance (Facchini, Hollenbeck, Jeppesen, Chen, & Reaven, 1992; Frati, Iniestra, & Ariza, 1996; Rönnemaa, Rönnemaa, Puukka, Pyörälä, & Laakso, 1996; Targher et al., 1997), elevated blood pressure (Balhara, 2012; Primatesta, Falaschetti, Gupta, Marmot, & Poulter, 2001), dyslipidemia (Criqui et al., 1980; Razay & Heaton, 1995), and visceral adiposity (Mizuno et al., 2005). For example, cigarette smoking directly reduces insulin sensitivity and impairs glucose tolerance by increasing circulating levels of hormones that have antagonistic effects on insulin action (Kapoor & Jones, 2005) and by promoting the release of free fatty acids, which interfere with insulin-mediated glucose uptake (Targher et al., 1997). Cigarette smoking also promotes a narrowing of vascular walls through the release of vasopressin (Ambrose & Barua, 2004; Omvik, 1996), which leads to acute and chronic elevations in blood pressure by increasing cardiac output and total peripheral vascular resistance (Balhara, 2012; Primatesta et al., 2001). Despite additional evidence linking cigarette smoking to elevated plasma triglycerides (Razay & Heaton, 1995) and reduced HDL cholesterol levels (Criqui et al., 1980; Razay & Heaton, 1995), limited research has investigated the specific mechanisms through which cigarette smoking promotes dyslipidemia, though several physiologic pathways related to increased activation of the sympathetic nervous system have been proposed to be involved (Razay & Heaton, 1995).

The overall relationship between cigarette smoking and visceral adiposity has been less consistently demonstrated. Although some research has linked cigarette smoking to a larger waist circumference (Mizuno et al., 2005), cigarette smoking tends to be associated with reduced risk

for visceral adiposity (Albanes, Jones, Micozzi, & Mattson, 1987; Bouros et al., 2006; Flegal, Troiano , Pamuk , Kuczmarski , & Campbell 1995; Huot, Paradis, & Ledoux, 2004). The negative relationship between cigarette smoking and visceral adiposity is often attributed to the stimulating effect of nicotine, a chemical found in cigarettes (Benowitz, 2010) that promotes a negative energy balance. Nicotine limits energy intake by suppressing appetite through increased satiety signaling and reducing motivation to engage in eating behavior (Audrain-McGovern & Benowitz, 2011; Bouros et al., 2006). Nicotine also elevates resting metabolic rate and the thermogenesis of adipose tissue (Andersson & Arner, 2001; Hellerstein et al., 1994), which can increase daily energy expenditure by up to 10% among smokers relative to nonsmokers (Hofstetter, Schutz, Jequier, & Wahren, 1986). These metabolic effects limit weight gain among smokers, and the loss of these effects following smoking cessation can lead to significant weight gain (Audrain-McGovern & Benowitz, 2011), which prospectively increases risk for MetS among former smokers (Nakanishi et al., 2005). Despite the effect of cigarette smoking on body weight regulation, additional evidence has shown that heavy cigarette smoking is associated with greater visceral adiposity (Molarius, Seidell, Kuulasmaa, Dobson, & Sans, 1997; Rosmond & Bjorntorp, 1999) than is light to moderate cigarette smoking. Although the mechanisms linking heavy cigarette smoking to greater visceral adiposity are not fully understood, it is hypothesized to result from heightened sympathetic nervous system activity that encourages visceral fat accumulation through increased cortisol secretion and altered levels of testosterone and estrogen (Balhara, 2012; Cena, Fonte, & Turconi, 2011; Chiolero, Faeh, Paccaud, & Cornuz, 2008). Thus, although light to moderate cigarette smoking can protect against visceral adiposity, this effect reverses with increasing cigarette consumption. Taken together, these findings indicate that cigarette smoking largely

enhances risk for MetS by promoting insulin resistance, elevated blood pressure, and dyslipidemia and can also lead to increased visceral adiposity at high doses.

1.2.2 Alcohol use and metabolic syndrome

Research examining the relationship between alcohol use and MetS is relatively sparse and inconsistent (Alkerwi et al., 2009). Previous research has implicated alcohol use as both a risk factor for (Djousse et al., 2004; Villegas, Creagh, Hinchion, O'Halloran, & Perry, 2004; Yoon, Oh, Baik, Park, & Kim, 2004; Zhu et al., 2004) and a protective factor against (Carnethon et al., 2004; Djousse et al., 2004; Freiberg, Cabral, Heeren, Vasan, & Curtis Ellison, 2004; Gigleux et al., 2006; Yoon et al., 2004; Zhu et al., 2004) MetS, with additional findings documenting no relationship between alcohol use and the syndrome (Buja et al., 2010; Lee, Jung, Park, Rhee, & Kim, 2005; Santos et al., 2007; Villegas et al., 2004; Wannamethee, Shaper, & Whincup, 2006). These discrepant findings are hypothesized to be driven by the complex relationship between alcohol use and the cardiometabolic risk indicators underlying MetS (O'Keefe, Bybee, & Lavie, 2007). Indeed, the amount of and frequency in which alcohol is consumed can have both preventative and exacerbating effects on each component part of MetS (Gigleux et al., 2006; Rimm, Williams, Fosher, Criqui, & Stampfer, 1999), suggesting that the directionality of the risk relationship between alcohol use and MetS likely depends on overall drinking patterns. In support of this proposition, a meta-analysis of observational studies by Alkerwi and colleagues (2009) confirmed that light to moderate alcohol use was associated with reduced risk for MetS but found no relationship between heavy alcohol use and risk for MetS. Meanwhile, a more recent meta-analysis of prospective studies by Sun and colleagues (2014) found that light alcohol use was associated with diminished relative risk for MetS when compared to alcohol abstinence and that heavy

alcohol use was associated with heightened relative risk for MetS when compared to lighter alcohol use. Taken together, these findings indicate that the association between alcohol use and risk for MetS is dependent on the amount of alcohol consumed and tends to follow a J- or U-shaped pattern (Djousse et al., 2004; Yoon et al., 2004; Zhu et al., 2004), whereby light to moderate alcohol use protects against MetS relative to alcohol abstinence and heavy alcohol use promotes MetS relative to less frequent alcohol use.

Given that the cardiometabolic effects of alcohol depend on both the amount of alcohol consumed and the frequency of drinking episodes (O'Keefe et al., 2007), research has begun to uncover how distinct patterns of drinking behavior influence the pathophysiology of MetS (Mukamal et al., 2003; Mukamal, Jensen, et al., 2005; Mukamal, Maclure, Muller, & Mittleman, 2005; O'Keefe et al., 2007; Rehm, Sempos, & Trevisan, 2003). For example, although light to moderate alcohol use reduces blood pressure (Beulens et al., 2007), heavy alcohol use leads to notable elevations in blood pressure and is considered one of the leading preventable causes of hypertension (Beilin & Puddey, 2006). The impact of heavy alcohol consumption on blood pressure specifically results from the stimulating effect of alcohol on the sympathetic nervous system that promotes vasoconstriction, endothelial dysfunction, oxidative stress, and calcification of vascular smooth muscle (Husain, Ansari, & Ferder, 2014; Marchi, Muniz, & Tirapelli, 2014). Light to moderate alcohol consumption is also associated with metabolic effects that reduce insulin resistance by improving insulin signaling and glucose utilization among individuals with (Greenfield, Samaras, Hayward, Chisholm, & Campbell, 2005; Turner, Jenkins, Kerr, Sherwin, & Cavan, 2001) and without type 2 diabetes (Davies et al., 2002), though this effect is reversed when alcohol is consumed in high doses (Flanagan et al., 2000). The divergent effect of alcohol use has similarly been documented in relation to triglyceride concentrations (Greenfield et al., 2005;

Mukamal et al., 2007) but not in relation to HDL cholesterol concentrations (Averina et al., 2004; Nilssen et al., 2005). Rather, alcohol use increases HDL cholesterol in a linear fashion (Davies et al., 2002; Greenfield et al., 2005; Mukamal, Jensen, et al., 2005; Mukamal et al., 2007) by directly altering the activity of enzymes and proteins involved in the synthesis and clearance of HDL cholesterol (De Oliveira et al., 2000; Sillanaukee, Koivula, Jokela, Pitkajarvi, & Seppa, 2000).

Despite evidence directly linking alcohol use to mechanisms that influence blood pressure, insulin resistance, and hypertriglyceridemia, accumulating evidence suggests that these effects may be indirectly driven by the impact of alcohol on visceral adiposity (Bell, Mayer-Davis, Martin, D'agostino, & Haffner, 2000; Freiberg & Samet, 2005; Stampfer et al., 1988). For example, several studies have documented that the positive relationship between alcohol use and insulin resistance does not persist after measures of visceral adiposity are statistically accounted for (Bell et al., 2000; Stampfer et al., 1988). The loss of this effect is not surprising given the substantial and direct impact of visceral adiposity on insulin resistance and related indicators of cardiometabolic risk (Bastard et al., 2006; Carr et al., 2004; Després & Lemieux, 2006; Hall, do Carmo, da Silva, Wang, & Hall, 2015; Thomas et al., 2004). Although the mechanisms linking alcohol use to visceral adiposity are not well-established (Traversy & Chaput, 2015), there is some evidence to suggest that the high thermogenic effect of alcohol may reduce risk for visceral adiposity by increasing daily energy expenditure among individuals who engage in light to moderate alcohol consumption (Klesges, Mealer, & Klesges, 1994; Raben, Agerholm-Larsen, Flint, Holst, & Astrup, 2003). However, alcohol is also high in calories (Shelton & Knott, 2014), stimulates overeating behaviors (Hofmann & Friese, 2008; Yeomans, 2010), and suppresses fat oxidation (Raben et al., 2003; Sonko et al., 1994), which together promote a positive energy balance and may lead to increased risk for visceral adiposity when regularly consumed at high doses. Accordingly, although light to

moderate alcohol use protects against the pathogenesis of MetS, heavy alcohol use promotes the development of MetS by directly influencing factors associated with elevated blood pressure, insulin resistance, and hypertriglyceridemia, and these effects are further enhanced by a heightened likelihood for visceral adiposity.

1.2.3 Physical inactivity and metabolic syndrome

Physical inactivity is generally defined as the failure to achieve recommended levels of weekly physical activity whereas sedentary behavior refers to the engagement in activities, such as sleeping, sitting, and lying down, that do not increase energy expenditure substantially above resting level (Hamilton, Healy, Dunstan, Zderic, & Owen, 2008). Although physical inactivity and sedentary behavior are related constructs, they both uniquely contribute to cardiometabolic disease risk and are therefore important to distinguish as distinct predictors of MetS (Hamilton, Hamilton, & Zderic, 2004; Hamilton et al., 2008; Wittink, Engelbert, & Takken, 2011). Physical inactivity and sedentary behavior are indeed independently associated with the development and progression MetS (Ford, Kohl, Mokdad, & Ajani, 2005; Healy, Dunstan, et al., 2008). For example, relative to cigarette smoking, alcohol use, and dietary intake, physical inactivity is the strongest behavioral predictor of MetS (Santos et al., 2007; Zhu et al., 2004). Meta-analytic findings have further documented that engaging in extended periods of sedentary behavior increases the odds of MetS by up to 73% (Edwardson et al., 2012). Randomized controlled trials designed to increase physical activity and decrease sedentary behavior also are associated with markedly improved cardiometabolic profiles, suggesting that both physical inactivity and sedentary behavior directly relate to factors involved in the pathogenesis of MetS (Lakka & Laaksonen, 2007).

The majority of research examining the mechanisms linking physical inactivity to MetS has focused on the beneficial effects of an active lifestyle on each component part of the syndrome. Treatment trials designed to increase physical activity through aerobic exercise or resistance training have documented concomitant reductions in body weight and visceral adiposity (Donnelly, Jacobsen, Heelan, Seip, & Smith, 2000; Irving et al., 2008; Slentz et al., 2011), improved insulin sensitivity and glucose tolerance (Cuff et al., 2003; Poehlman, Dvorak, DeNino, Brochu, & Ades, 2000; Sigal et al., 2007), lower triglyceride and heightened HDL cholesterol levels (Eriksson et al., 1998; Jones, Doran, Leatt, Maher, & Pirmohamed, 2001; Lokey & Tran, 1989), and decreased blood pressure (Collier et al., 2008; Hagberg, Montain, Martin, & Ehsani, 1989). The protective effect of physical activity on MetS is primarily promoted through favorable alterations in body weight and body composition that lead to associated improvements in cardiometabolic risk factors (Kay & Singh, 2006; Lakka & Laaksonen, 2007; Salonen et al., 2015). Physical activity is indeed associated with a cascade of metabolic effects that enhance fat oxidation (Goodpaster, Katsiaras, & Kelley, 2003; Talanian, Galloway, Heigenhauser, Bonen, & Spriet, 2007), prevent fat deposition (Kay & Singh, 2006; Lamarche, 1993), and build lean muscle tissue (Holloszy & Coyle, 1984), which collectively protect against obesity and visceral adiposity (Donnelly et al., 2003; Irwin et al., 2003; Ross et al., 2000; Slentz et al., 2005). The impact of physical activity on lean muscle tissue also directly enhances insulin sensitivity and glucose tolerance (Holten et al., 2004; Miller et al., 1994; Ryan, Pratley, Goldberg, & Elahi, 1996), decreases glycogen accumulation (Ebeling et al., 1993), and regulates fatty acid uptake and oxidation (Kiens, 2006), thereby improving insulin sensitivity. Physical activity also decreases availability of lipid products (Turcotte & Fisher, 2008), increases lipid oxidative capacity (Pruchnic et al., 2004), and stimulates enzyme production (Campaigne, Fontaine, Park, &

Rymaszewski, 1993; Leaf, 2003) in muscle cells, resulting in modest improvements in lipid profiles (Healy, Wijndaele, et al., 2008; Kodama et al., 2007). Physical activity further promotes meaningful decreases in blood pressure through improved vagal tone (Fagard & Cornelissen, 2007), with meta-analytic findings showing that regular engagement in physical activity can reduce systolic and diastolic blood pressure by up to 7 and 5 mm Hg, respectively (Cornelissen & Fagard, 2005).

In addition to the extensive research examining the physiologic mechanisms driving the association between physical activity and MetS, accumulating research has begun to focus more specifically on inactivity physiology to describe the distinct pathogenic role of sedentary behavior in promoting risk for the syndrome (Hamilton et al., 2008; Wittink et al., 2011). Sedentary behavior is characterized by extended periods of muscular inactivity and reduced thermogenesis, which contribute to obesity risk and associated cardiometabolic abnormalities (Hamilton et al., 2004; Hamilton et al., 2008; Wittink et al., 2011). For example, sedentary behavior is linked to decreased muscle glycogen synthesis (Bergouignan et al., 2016; Petersen et al., 2007), which leads to insulin resistance in lean muscle tissue (Jensen, Rustad, Kolnes, & Lai, 2011; Kida, Esposito-Del Puente, Bogardus, & Mott, 1990). The increased insulin resistance observed in lean muscle tissue subsequently promotes dyslipidemia by diverting energy derived from ingested carbohydrates away from muscle glycogen storage and into hepatic lipogenesis, thereby promoting hypertriglyceridemia and reduced HDL cholesterol levels (Petersen et al., 2007). Although findings linking objectively measured sedentary behavior to elevated blood pressure have been mixed (Gerage et al., 2016; Healy, Wijndaele, et al., 2008; Sohn et al., 2014), animal models suggest that sedentary behavior suppresses lipoprotein lipase action, which leads to increases in both free radical production and inflammatory markers that promote blood pressure elevations

(Hamilton, Hamilton, & Zderic, 2007). Despite a need to further elucidate the specific physiologic effects that sedentary behavior has on cardiometabolic outcomes, these findings indicate that both physical inactivity and sedentary behavior are associated with distinct pathophysiologic mechanisms that promote MetS. Meanwhile, physical activity remains a primary protective factor against MetS through its beneficial effects on each component part of the syndrome.

1.2.4 Energy intake, dietary composition, and metabolic syndrome

Energy intake and dietary composition are important determinants of MetS (Kastorini et al., 2011; Malik et al., 2010; Nicklas, O'Neil, & Fulgoni, 2012; Yamaoka & Tango, 2012; Yosaee et al., 2016; Zhu et al., 2004). Although there is ongoing debate regarding the optimal diet to protect against MetS (Baxter, Coyne, & McClintock, 2006), dietary patterns characterized by high consumption of unsaturated fats, fruits, vegetables, whole grains, fish, and poultry and low consumption of saturated fats, carbohydrates, salt, red meat, fried foods, and sugar-sweetened beverages appear to have the most substantial impact in reducing risk for the syndrome (Kastorini et al., 2011). Such dietary patterns also have been linked to notable improvements in each component part of MetS, including reduced visceral adiposity, improved lipid profiles, decreased blood pressure, and enhanced insulin sensitivity (Kastorini et al., 2011).

The mechanisms through which energy intake and dietary composition affect the primary cardiometabolic components of MetS are highly complex. For example, energy intake is largely believed to contribute to the development of MetS through its influence on body weight and body composition (Astrup, Grunwald, Melanson, Saris, & Hill, 2000; Brehm, Seeley, Daniels, & D'alessio, 2003; Krieger, Sitren, Daniels, & Langkamp-Henken, 2006; Layman et al., 2003; Newby et al., 2003; Yannakoulia et al., 2003). Specifically, individuals who regularly consume

more calories than they expend develop a surplus of energy that is eventually converted to and stored as body fat (Blundell & Cooling, 2000), which can result in substantial increases in adipose tissue and excess weight gain if sustained over time (Epstein, Leddy, Temple, & Faith, 2007). The accumulating fat mass observed among individuals with high calorie diets directly leads to additional impairments in cardiometabolic health, including insulin resistance, dyslipidemia, and elevated blood pressure, that subsequently promote the development of MetS (Bastard et al., 2006; Carr et al., 2004; Després & Lemieux, 2006; Hall et al., 2015; Thomas et al., 2004). However, the impact of a high calorie diet on excess adiposity does not fully account for the relationship between energy intake and MetS. Indeed, dietary composition also directly influences metabolic factors that impact insulin sensitivity (Kennedy, Martinez, Chuang, LaPoint, & McIntosh, 2009; Ludwig, 2002; Riccardi, Giacco, & Rivellese, 2004), lipid profiles (Ford & Liu, 2001; Frost et al., 1999; Pelkman, 2001; Siri & Krauss, 2005), and blood pressure (Appel et al., 2005; He & MacGregor, 2002; Shah, Adams-Huet, & Garg, 2007), independent of adiposity.

Research on dietary composition and MetS has largely focused on the impact of macronutrient consumption on cardiometabolic profiles and has identified fat and carbohydrate intake as being particularly important in predicting risk for the syndrome (Riccardi & Rivellese, 2000; Siri & Krauss, 2005). The type of dietary fat consumed has differential effects on cardiometabolic risk. Whereas diets high in unsaturated fat protect against cardiometabolic risk, diets high in saturated fat increase cardiometabolic risk (Hu, Manson, & Willett, 2001; Mozaffarian, Micha, & Wallace, 2010). Diets high in saturated fat specifically contribute to cardiometabolic risk by altering fatty acid profiles (Riccardi et al., 2004), promoting systemic inflammation (Kennedy et al., 2009), and increasing oxidative stress (Devaraj, Wang-Polagruto, Polagruto, Keen, & Jialal, 2008), which collectively serve to disrupt insulin action and can lead to

insulin resistance. High levels of saturated fat intake also result in dyslipidemia through increased lipid production (Lopez et al., 2011) and impaired lipid metabolism (Rivellese et al., 2003). Although diets characterized by high protein intake derived from red meat consumption also have been linked to insulin resistance and dyslipidemia, this effect is primarily driven by the high levels of saturated fat found in such protein sources (de Oliveira Otto et al., 2012).

Similar to dietary fat intake, varied sources of carbohydrate intake can also have opposing effects on cardiometabolic risk (Barclay et al., 2008; Liu et al., 2000). One means of evaluating carbohydrate sources is the glycemic index, a measurement system that ranks sources of carbohydrates according to their impact on blood glucose levels (Wolever & Mehling, 2003). Relative to carbohydrates with a low glycemic index (e.g., most fruits, non-starchy vegetables, and whole grains), carbohydrates with a high glycemic index (e.g., white bread, starchy vegetables, and white rice) are digested and absorbed quickly into the bloodstream, leading to rapid elevations in blood glucose and subsequent increases in serum insulin levels (Feskens, Bowles, & Kromhout, 1991; Ludwig, 2002). Habitual consumption of carbohydrates with a high glycemic index therefore initiates a cycle of hyperinsulinemia and hyperglycemia that can lead to insulin resistance (Ludwig, 2002). The hyperglycemic state that results from the consumption of carbohydrates with a high glycemic index further prompts the liver to transform excess glucose into triglycerides for more efficient storage, which can lead to hypertriglyceridemia if sustained over time (Parks & Hellerstein, 2000). Frequent consumption of carbohydrates with a high glycemic index also have been linked to low HDL cholesterol concentrations (Ford & Liu, 2001; Frost et al., 1999), which is theorized to partly result from associated reductions in the size and density of HDL cholesterol particles (Brinton, Eisenberg, & Breslow, 1990; Lichtenstein et al., 1994; Schaefer et al., 1995). Although there is some evidence to suggest that both saturated fat and carbohydrate consumption

increase blood pressure (Appel et al., 2005; Shah et al., 2007), this effect is largely driven by the high salt content characteristic of foods that are high in saturated fat and carbohydrates (He, Li, & MacGregor, 2013; Vollmer et al., 2001). Thus, energy intake and dietary composition, particularly fat and carbohydrate intake, largely appear to contribute to the pathogenesis of MetS by altering risk for excess adiposity as well as by directly influencing physiologic mechanisms that impact insulin sensitivity, lipid profiles, and blood pressure.

1.2.5 Summary

Taken together, these findings highlight the complex pathophysiologic mechanisms linking cigarette smoking, heavy alcohol use, physical inactivity, and unhealthy diet to MetS and help to provide a more detailed understanding of why individuals who engage in such maladaptive health behaviors are at risk for the syndrome. Despite the importance of understanding *how* maladaptive health behaviors promote cardiometabolic risk, these findings ultimately do not provide insight into *who* is likely to engage in such maladaptive health behaviors. As such, understanding psychological characteristics that determine who is likely to engage in behaviors that confer risk for MetS has important implications for interventions designed to mitigate the health consequences of cardiometabolic diseases.

1.3 SCOPE OF THE PRESENT STUDY

Given the widespread disease burden of MetS (Alberti et al., 2009), efforts are needed to identify factors that contribute to risk for the syndrome. Impulsivity is a personality trait that has gained

increasing attention as an important health risk indicator that predisposes individuals to engage in the maladaptive health behaviors shown to confer risk for MetS (DeYoung, 2010). Although preliminary evidence found that individuals high on impulsivity were at risk for MetS (Sutin, Terracciano, Deiana, Uda, et al., 2010), more recent findings documenting this relationship have been equivocal (Ross et al., 2011; van Reedt Dortland et al., 2012). These mixed findings may be partly explained by the use of omnibus personality measures that broadly assess impulsive characteristics but lack the specificity needed to reliably predict complicated disease states like MetS (Mommersteeg & Pouwer, 2012). Thus, the use of more comprehensive measurements of impulsivity will bolster our understanding of whether and how impulsivity relates to MetS. Moreover, because varied measures of impulsivity are best characterized as representing modestly associated but distinct impulsigenic traits (Sharma et al., 2014), the relationship between impulsivity and MetS likely depends on the particular impulsigenic trait assessed.

The primary aims of the present study were twofold. First, the present study aimed to assess the overall relationship between distinct impulsigenic traits, conceptualized according to the hierarchical structure found by Sharma and colleagues (2014), and cardiometabolic risk. Second, because the specific behavioral mechanisms linking impulsivity to cardiometabolic risk may differ according to the particular impulsigenic trait assessed, the present study further aimed to explore the extent to which specific health behaviors, including cigarette smoking, alcohol use, physical activity, energy intake, and dietary composition, accounted for the relationships between each of the distinct impulsigenic traits identified by Sharma and colleagues (2014) and cardiometabolic risk. By examining whether the relationships between distinct impulsigenic traits and cardiometabolic risk were mediated by varied health behaviors, the present study ultimately aimed to clarify both *who* is at heightened cardiometabolic risk and *how* those individuals are at risk.

2.0 METHODS

2.1 PARTICIPANTS

Data were derived from the University of Pittsburgh Adult Health and Behavior (AHAB) project, a large registry of behavioral and biological measurements. Participants for the parent study were recruited between 2001 and 2005 via mass-mail solicitation from communities of southwestern Pennsylvania in the United States (principally Allegheny County). Participants included Caucasian and African American individuals of non-Hispanic ethnicity who were between the ages of 30 and 54 years. Exclusion criteria included a reported history of atherosclerotic cardiovascular disease, chronic kidney or liver disease, cancer treatment in the preceding year, neurologic disorders, or psychotic illness. Other exclusions included current pregnancy as well as the use of insulin, nitrate, glucocorticoid, antiarrhythmic, psychotropic, or prescription weight-loss medications. Informed consent was obtained in accordance with approved protocol guidelines of the University of Pittsburgh Institutional Review Board. Data collection occurred over multiple sessions. The total AHAB sample was retained for the present study (N = 1295).

2.2 MEASURES

2.2.1 Demographic characteristics

Participants reported demographic information, including age, sex, race, and number of years of education.

2.2.2 Questionnaire measures of impulsivity

2.2.2.1 Barratt Impulsiveness Scale-11 (BIS-11). The BIS-11 is a 30-item questionnaire that measures a general ability to maintain control over thoughts and behaviors (Patton, Stanford, & Barratt, 1995). The BIS-11 comprises three subscales, including Attentional Impulsivity, Motor Impulsivity, and Non-Planning Impulsivity. The Attentional Impulsivity subscale consists of 8 items measuring a tendency to have difficulty focusing on tasks and to experience intrusive and racing thoughts. The Motor Impulsivity subscale consists of 11 items measuring a tendency to act rashly and to have difficulty maintaining a consistent lifestyle. The Non-Planning Impulsivity subscale consists of 11 items measuring a tendency to have difficulty engaging in careful planning and challenging mental tasks. All items were rated using a 4-point Likert scale, and a sum score was calculated for each subscale, with higher sum scores on each subscale indicating higher levels of impulsivity.

2.2.2.2 Behavioral Inhibition System/Behavioral Activation System (BIS/BAS). The BIS/BAS is a 20-item questionnaire that measures appetitive and aversive motivation (Carver & White, 1994). The BIS/BAS comprises four subscales, including Behavioral Inhibition System, Drive,

Fun-Seeking, and Reward Responsiveness. The Behavioral Inhibition System subscale consists of 7 items measuring a tendency to avoid situations with aversive consequences. The Drive subscale consists of 4 items measuring a tendency to persistently pursue desired goals. The Fun-Seeking subscale consists of 4 items measuring a tendency to desire rewards and to approach potentially rewarding events without forethought. The Reward Responsiveness subscale consists of 5 items measuring a tendency to have a high sensitivity to rewards. All items were rated using a 4-point Likert scale, and a sum score was calculated for each subscale, with higher sum scores on each subscale indicating higher levels of impulsivity.

2.2.2.3 Multidimensional Personality Questionnaire-Brief Form (MPQ-BF). The MPQ-BF is a 155-item questionnaire that measures broad aspects of temperament (Patrick, Curtin, & Tellegen, 2002). The MPQ-BF comprises three broad trait subscales, defined as Constraint, Positive Emotionality, and Negative Emotionality, that each consist of 10 primary trait scales, including Achievement (12 items), Aggression (12 items), Alienation (12 items), Control (12 items), Harm Avoidance (12 items), Social Closeness (12 items), Social Potency (12 items), Stress Reaction (12 items), Traditionalism (12 items), and Wellbeing (12 items). The Constraint subscale consists of 120 items and measures a tendency to exhibit self-control, caution, and avoidance of danger. The Positive Emotionality subscale consists of 120 items and measures a tendency to be positively engaged in interpersonal relationships and to experience positive mood states. The Negative Emotionality subscale consists of 120 items and measures a tendency to be negatively engaged in interpersonal relationships and to experience anxiety, anger, and worry. All items were rated using true or false options, and a weighted sum score of the 10 primary trait scales was calculated for each of the three broad trait subscales (Patrick et al., 2002). Lower weighted sum scores on the Constraint subscale indicate higher levels of impulsivity whereas higher weighted sum scores on

the Positive Emotionality and Negative Emotionality subscales indicate higher levels of impulsivity.

2.2.2.4 NEO Personality Inventory-Revised (NEO-PI-R). The NEO-PI-R is a 240-item questionnaire that measures five broad domains of personality, including Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness (Costa & McCrae, 1992). To remain consistent with the analyses reported by Sharma and colleagues (2014) and in previous research using this study sample (Creswell et al., 2018), only the subfacets of the Conscientiousness, Extraversion, and Neuroticism subscales were included in the present analysis. The Conscientiousness subscale measures a tendency to be thorough, careful, and vigilant and consists of the Achievement Striving (8 items), Competence (8 items), Deliberation (8 items), Dutifulness (8 items), Order (8 items), and Self-Discipline (8 items) subfacets. The Extraversion subscale measures a tendency to be outgoing and energetic and consists of the Activity (8 items), Assertiveness (8 items), Excitement-Seeking (8 items), Gregariousness (8 items), Positive Emotions (8 items), and Warmth (8 items) subfacets. The Neuroticism subscale measures a proneness towards psychological distress and consists of the Angry Hostility (8 items), Anxiety (8 items), Depression (8 items), Impulsiveness (8 items), Self-Consciousness (8 items), and Vulnerability (8 items) subfacets. All items were rated using a 5-point Likert scale, and a sum score was calculated for each subfacet. Lower sum scores on the Conscientiousness subfacet indicate higher levels of impulsivity whereas higher sum scores on the Extraversion and Neuroticism subfacets indicate higher levels of impulsivity.

2.2.2.5 Schedule for Nonadaptive and Adaptive Personality (SNAP). The SNAP is a 375-item questionnaire that measures a range of adaptive and maladaptive personality traits to assess

personality pathology (Clark & Press, 1993). The SNAP consists of three broad personality domains that comprise 15 subscales assessing relatively distinct personality trait dimensions. The three broad personality domains are labeled Disinhibition, Positive Affectivity, and Negative Affectivity. The Disinhibition domain measures disinhibited and impulsive behavior and consists of the Disinhibition (35 items), Impulsivity (19 items), Propriety (20 items), and Workaholism (18 items) subscales. Because the Disinhibition subscale contains several items that overlap with other SNAP subscales, the non-overlapping version of the Disinhibition (16 items) subscale was used in the present analysis. The Positive Affectivity domain measures a tendency to be outgoing and energetic and consists of the Positive Temperament (27 items), Detachment (18 items), Entitlement (16 items), and Exhibitionism (16 items) subscales. The Negative Affectivity domain measures a proneness towards psychological distress and consists of the Negative Temperament (28 items), Aggression (20 items), Dependency (18 items), Eccentric Perceptions (15 items), Manipulativeness (20 items), Mistrust (19 items), and Self-Harm (16 items) subscales. All items were rated using true or false options and a sum score was calculated for each subscale. Higher sum scores on each subscale indicate higher levels of impulsivity, with the exception of the Propriety and Workaholism subscales, in which lower sum scores indicate higher levels of impulsivity. To remain consistent with the analyses reported by Sharma and colleagues (2014) and in previous research using this study sample (Creswell et al., 2018), the Aggression, Dependency, and Entitlement subscales were not included in the present analysis. Moreover, because this measure was introduced late in the parent study, data were only available for 930 participants.

2.2.2.6 Zuckerman Sensation Seeking Scale (SSS). The SSS is a 40-item questionnaire that measures a willingness to take risks and seek out novel and intense experiences (Zuckerman et al., 1964). The SSS comprises four subscales including Boredom Susceptibility, Disinhibition,

Experience Seeking, and Thrill and Adventure Seeking. The Boredom Susceptibility subscale consists of 10 items measuring a general aversion to repetition and routine. The Disinhibition subscale consists of 10 items measuring a desire for uninhibited social and sexual experiences. The Experience Seeking subscale consists of 10 items measuring a desire for unique sensory experiences and a non-conforming lifestyle. The Thrill and Adventure Seeking subscale consists of 10 items measuring a desire to engage in sports or activities involving speed and danger. All items were rated using a forced-choice format, and a sum score was calculated for each subscale, with higher sum scores on each subscale indicating higher levels of impulsivity.

2.2.2.7 Temperament and Character Inventory (TCI). The TCI is a 240-item questionnaire that measures broad aspects of temperament (Cloninger, Svrakic, & Przybeck, 1993). The TCI comprises four broad temperament domains, including Reward Dependence, Harm Avoidance, Persistence, and Novelty Seeking. Although the TCI was not included in the analysis reported by Sharma and colleagues (2014), the Novelty Seeking domain was included in the present analysis to remain consistent with previous research using this study sample (Creswell et al., 2018). The Novelty Seeking domain measures a tendency to experience intense excitement in the presence of novel stimuli and consists of the Exploratory Excitability (11 items), Extravagance (9 items), Disorderliness (10 items), and Impulsiveness (10 items) subscales. All items were rated using true or false response options, and a sum score was calculated for each subscale, with higher sum scores on each subscale indicating higher levels of impulsivity.

2.2.3 Behavioral task measures of impulsivity

2.2.3.1 Delay Discounting Task (DDT). The DDT is a computerized task in which individuals choose between smaller, immediate rewards and larger, delayed rewards. For the parent study, participants were asked to make choices about hypothetical monetary rewards (Mitchell, 1999). For each choice, two options were simultaneously displayed on a computer screen. On one side of the screen, participants were shown variable amounts of money, ranging from 10 cents to 105 dollars, that they could receive immediately. On the other side of the screen, participants were shown a fixed monetary amount of 100 dollars that they could receive following seven different delay intervals, ranging from zero days to five years. For each choice, participants were asked to select which of the two options they would prefer. All combinations of immediate rewards and delay intervals were presented in randomized order. The indifference point (i.e., the point at which the delayed reward was equally as valuable as the immediate reward) was computed for each of the seven delay intervals (Mitchell, 1999). A hyperbolic function was then fit to each indifference point (de Wit, Flory, Acheson, McCloskey, & Manuck, 2007), yielding a k-value. The distribution of k-values was subsequently normalized by logarithmic transformation (Sweitzer, Donny, Dierker, Flory, & Manuck, 2008), with higher k-values indicating higher levels of impulsivity. Because this task was introduced late in the parent study, data were only available for 743 participants.

2.2.3.2 Iowa Gambling Task (IGT). The IGT is a computerized task that assesses decision making under risk and uncertainty by asking individuals to select cards from advantageous and disadvantageous decks to maximize monetary profit over loss (Bechara, 2007). For the parent study, participants were asked to choose a card from one of four decks labeled A through D. The

participants were awarded hypothetical monetary rewards for each correct choice but lost money after incorrect choices. Choices from the A and B decks were disadvantageous as they were associated with big wins and losses while choices from the C and D decks were advantageous as they were associated with small wins and losses. A net score was calculated by taking the difference between the total number of disadvantageous and advantageous cards selected ([C+D] – [A+B]), with lower net scores indicating higher levels of impulsivity. Because this task was introduced late in the parent study, data were only available for 575 participants.

2.2.3.3 Stroop Color Word Test. The Stroop Color Word Test is a task in which individuals are required to name the color of a written color word while inhibiting the impulse to read the word itself (Golden & Freshwater, 1978, 2002). For the parent study, participants were required to read aloud from three pages of color word lists as quickly as possible. Page one required participants to read a list of color names (e.g., "red," "green," "blue"). Page two required participants to name the colors of the inks from a list of congruent color words (e.g., the word "red" printed in red ink). Page three required participants to name the colors of the inks from a list of incongruent color words (e.g., the word "blue" printed in yellow ink). The interference score was then calculated as the difference between the actual number of correct responses on the incongruent trial and the predicted number of correct responses from the control trials (see Marsland et al., 2015), with lower interference scores indicating higher levels of impulsivity.

2.2.3.4 Wisconsin Card Sort Test (WCST). The WCST is a computerized task in which individuals have to match a target card with one of four category cards under changing conditions (Heaton, Chelune, Talley, Kay, & Curtis, 1993). In the parent study, participants sorted 128 cards according to changing matching rules (e.g., matching cards according to their color, shape, or

number). Participants were required to learn the matching rule by trial and error as the computer provided feedback about whether their responses were correct or incorrect. After 10 consecutive correct responses, the matching rule changed unbeknownst to the participants, demanding a flexible shift in set to identify the new matching rule. The task continued until all cards were sorted or a maximum of six correct matching rules were reached. The total number of perseverative errors, defined as the total number of incorrect responses that would have been correct for the preceding matching rule, and the total number of non-perseverative errors, defined as the total number of incorrect responses that did not involve perseveration, were calculated, with a greater number of perseverative and non-perseverative errors indicating higher levels of impulsivity.

2.2.4 Cardiometabolic risk

All components of cardiometabolic risk were assessed in the morning following an 8-hour, overnight fast, as has previously been described (Muldoon, Nazzaro, Sutton-Tyrrell, & Manuck, 2000). All physical and biological assessments were completed by a nurse trained in the parent study protocol.

To adjust for medication effects, specific components of cardiometabolic risk were treated as missing among participants taking antihypertensive, oral hypoglycemic, or cholesterol-lowering medications (n = 142). Specifically, systolic and diastolic blood pressure were treated as missing among participants taking antihypertensive medication (n = 95), fasting glucose and insulin concentrations were treated as missing among participants taking oral hypoglycemic medication (n = 7), and fasting HDL cholesterol and triglyceride concentrations were treated as missing among participants taking cholesterol-lowering medication (n = 62). Given the limitations associated with imputing missing data to adjust for medication effects (Hunt et al., 2002; Tobin, Sheehan, Scurrah,

& Burton, 2005), the present study used the full information maximum likelihood (FIML) approach to account for missing data among the cardiometabolic risk variables. The FIML approach produces an estimate of population parameters by using all available data to infer what the full study sample should look like rather than imputing missing cases (Graham, 2009) and is particularly suited for use in structural equation modeling when missing data are present (Enders & Bandalos, 2001). Because the majority of missing cardiometabolic risk data were deleted to adjust for medication effects, and therefore could not be assumed to be missing completely at random, they were instead assumed to be missing at random (Enders, 2010; Enders & Bandalos, 2001).

To ensure that using the FIML approach to adjust for medication effects did not influence the final results, a sensitivity analysis was conducted using a conservative approach to handling medication effects. The final results obtained using the FIML approach were then compared to the final results obtained using the sensitivity analysis. In the sensitivity analysis, participants who reported taking antihypertensive, oral hypoglycemic, or cholesterol-lowering medications (n = 142) were completely excluded from analysis, and the final results were estimated using only the remaining participants (n = 1153). As expected, the final parameter estimates varied slightly between the two approaches. However, the overall pattern of results was the same, indicating that the FIML approach did not meaningfully alter the final results when compared to the conservative approach used in the sensitivity analysis. Accordingly, the full study sample was retained for the present analysis and the FIML approach was used to account for missing cardiometabolic risk data.

2.2.4.1 Blood pressure. Diastolic and systolic blood pressure were measured in mm Hg by manual sphygmomanometry as the mean of two consecutive readings obtained in a seated position following 20 minutes of rest.

2.2.4.2 Adiposity. Height and weight were measured and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m^2). Waist circumference was measured in inches at the level of the umbilicus.

2.2.4.3 Insulin resistance and blood lipids. A 40 mL sample of blood was obtained from each participant. Fasting serum concentrations of glucose, insulin, HDL cholesterol, and triglycerides were determined by the Heinz Nutrition Laboratory at the University of Pittsburgh Graduate School of Public Health.

2.2.5 Health behaviors

2.2.5.1 Cigarette smoking. Participants reported the average number of cigarettes that they currently smoke daily.

2.2.5.2 Alcohol use. Participants reported the total number of standard alcoholic beverages they consumed in the previous week.

2.2.5.3 Physical activity. Physical activity was assessed using the Paffenbarger Physical Activity Questionnaire (Paffenbarger, Wing, & Hyde, 1978). Participants reported the number of city blocks they walked, flights of stairs they climbed, and the frequency and duration of any sports or

recreational activities they participated in during the previous week. Energy expenditure from physical activity was measured by summing the metabolic equivalents for each activity to estimate the total amount of kilocalories each participant expended during physical activity in the previous week.

2.2.5.4 Energy intake and dietary composition. Energy intake and dietary composition data were gathered among a subset of participants (n = 469) during the second phase of the parent study. Eligibility criteria for the second phase of the parent study were identical to that of the first phase, with several additional exclusionary criteria. Specifically, participants were excluded from the second phase of the parent study if they were taking antihypertensive or cholesterol-lowering medications, had severe hypertension, as indicated by a blood pressure reading greater than 180/110 mm Hg, had secondary hypertension due to chronic renal insufficiency, as indicated by a creatinine level greater than 1.8 mg/dL, had suspected hyperaldosteronism, as indicated by a potassium level less than 3.5 mg/dL, reported consuming more than 21 standard alcoholic beverages per week, had a BMI greater than or equal to 40 kg/m^2, were diagnosed with diabetes, bulimia nervosa, or anorexia nervosa, or reported having previously received bariatric surgery.

Energy intake and dietary composition data were collected for two separate 24-hour periods using the Nutrition Data System for Research interview (NDSR; University of Minnesota, Minneapolis, MN), a computer-assisted interview designed to collect and analyze 24-hour food recalls. The food recall interviews were structured using a multiple-pass approach, which provided participants several opportunities to recall dietary intake for the previous 24-hours. Participants also were given a booklet consisting of standard portion sizes and measurements to allow for more accurate estimation of food and beverage amounts. Energy intake and dietary composition data were directly input into the NDSR program and nutrient values were automatically calculated. To

reduce reporting variability (Beaton, Milner, McGuire, Feather, & Little, 1983; Ma et al., 2009), the nutrient values obtained from the two separate 24-hour periods were averaged to estimate typical energy intake and dietary composition patterns. Energy intake and dietary composition data for participants whose food recall interviews were determined to be invalid (n = 5) or were only available for one 24-hour period (n = 24) were treated as missing. Energy intake and dietary composition data for the remaining participants (n = 440) were extracted from the NDSR program. Energy intake was defined as the average number of kilocalories consumed per day, and dietary composition was defined as the percentage of average energy intake derived from carbohydrates, protein, saturated fat, and unsaturated fat.

3.0 DATA ANALYSIS

Descriptive statistics were computed using version 24 of the SPSS statistical software program (SPSS, Inc., Chicago, IL). The primary aims of the present study were tested with structural equation modeling using version 7.3 of the Mplus statistical software program (Muthén & Muthén, 2010). All statistical tests were evaluated with a two-sided, Type I error rate of 0.05. Prior to analysis, the normality assumptions of the data were examined by inspecting graphical displays of all observed study variables. Several observed study variables, including fasting glucose, insulin, and triglyceride concentrations, perseverative and non-perseverative errors on the WCST, cigarette smoking, and alcohol use, were determined to be highly skewed and were normalized by logarithmic transformation. As previously described, missing data were handled using the FIML approach and were assumed to be missing at random (Enders, 2010; Enders & Bandalos, 2001).

Measurement models for questionnaire and behavioral task measures of impulsivity and for cardiometabolic risk were initially constructed separately. Measurement models were estimated using the maximum likelihood method with robust standard errors to account for nonnormality in the data (Kline, 2015). The measurement models for questionnaire and behavioral task measures of impulsivity were constructed in accordance with the analytic strategy of previous research using this study sample (Creswell et al., 2018) and were closely modeled after the findings reported by Sharma and colleagues (2014). Specifically, an exploratory factor analysis with

oblique geomin rotation was first run on the observed questionnaire measures of impulsivity, and a quasi-confirmatory approach was used to extract a three-factor solution. A confirmatory factor analysis was then run on the observed behavioral task measures of impulsivity, and measurement paths were constrained to coincide with the four-factor structure that Sharma and colleagues (2014) found for behavioral task measures of impulsivity. The measurement model for cardiometabolic risk was also constructed in accordance with the analytic strategy of previous research using this study sample (Dermody et al., 2015; Marsland et al., 2010; McCaffery, Marsland, Strohacker, Muldoon, & Manuck, 2012; McCaffery, Shen, Muldoon, & Manuck, 2007). As such, a confirmatory factor analysis was run on the observed cardiometabolic risk variables, and measurement paths were constrained to load onto four subfactors underlying a single, superordinate factor.

After examining the separate measurement models, a single structural model was estimated to determine the extent to which the observed health behavior variables accounted for the relationships between each latent impulsivity factor and the superordinate cardiometabolic risk factor. The observed health behavior variables included in the structural model were cigarette smoking, alcohol use, physical activity, energy intake, and percentage of energy intake from carbohydrates, protein, saturated fat, and unsaturated fat. The structural model was run using exploratory structural equation modeling, which permits the simultaneous estimation of exploratory and confirmatory factors within the same model (Asparouhov & Muthén, 2009). The structural model was estimated using the maximum likelihood method with robust standard errors to account for nonnormality in the data (Kline, 2015), and all latent and observed study variables were allowed to freely covary. All latent and observed study variables were also conditioned on key demographic variables, including age, sex, race, and number of years of education. Prior to

estimating the structural model, bivariate correlations were calculated among all latent and observed study variables to evaluate their interdependencies. The structural model was then estimated by simultaneously specifying several pathways. Specifically, each of the observed health behavior variables were regressed on each of the latent impulsivity factors, and the superordinate cardiometabolic risk factor was regressed on each of the observed health behavior variables and on each of the latent impulsivity factors. By simultaneously examining each of these pathways in a single structural model, the interdependencies among the latent and observed study variables were accounted for, and the independent effects of each pathway were established. Multiple mediation was then tested using the MODEL INDIRECT command in Mplus to obtain standardized parameter estimates for the direct, indirect, and total effects of the structural model (Muthén & Muthén, 2010). Given that significant direct effects are not necessary to detect mediation (MacKinnon, Fairchild, & Fritz, 2007), indirect effects were examined for the relationships between each latent impulsivity factor and the superordinate cardiometabolic risk factor, regardless of significance.

Model fit for each measurement model and for the structural model was largely determined according to theoretical meaningfulness (Barrett, 2007; Hayduk, Cummings, Boadu, Pazderka-Robinson, & Boulianne, 2007) and further evaluated using conventional model fit indices (Browne & Cudeck, 1993; Hu & Bentler, 1999). The chi-square test was first used to evaluate the congruency between the theorized model and the empirical data from the sample. However, because the chi-square test is highly sensitive to large sample sizes and often results in statistically significant but empirically trivial differences, several additional model fit indices were used to evaluate model fit. These consisted of the comparative fit index (CFI), the standardized root mean residual (SRMR), and the root mean square error of approximation (RMSEA) and its resulting

90% confidence interval (CI). Given the complexity of the measurement model for questionnaire measures of impulsivity and of the structural model, conservative estimates of model fit were not expected to be consistently achieved (Hu & Bentler, 1999). As such, acceptable fit was set at a CFI value greater or equal to 0.95, a SRMR value less than 0.10, and a RMSEA value less than 0.08 (Cangur & Ercan, 2015). The residual matrix for each measurement model and for the structural model was also inspected for any large deviations from zero or negative residual variances that could indicate discrepancies between the theorized model and the empirical data from the sample.

4.0 RESULTS

Descriptive statistics were first calculated for each observed demographic, cardiometabolic risk, and health behavior variable (see Table 1). Descriptive statistics were then calculated for each observed questionnaire measure of impulsivity (see Table 2) and each observed behavioral task measure of impulsivity (see Table 3). Psychometric properties were also calculated for each observed questionnaire measure of impulsivity (see Table 2). As shown in Table 2, the majority (77%, n = 37) of observed questionnaire measures of impulsivity had a Cronbach's alpha value greater than or equal to 0.70, indicating adequate internal consistency (Tavakol & Dennick, 2011).

Table 1. Descriptive statistics for the observed demographic, cardiometabolic risk, and health behavior variables (N = 1295)

Observed Demographic Variables	Mean ± SD or % (n)
Age (years)	44.63 ± 6.74
Education (years)	15.71 ± 2.84
Sex (female)	53% (683)
Race (Caucasian)	84% (1081)
Observed Cardiometabolic Risk Variables	
Systolic Blood Pressure (mm Hg)	115.56 ± 13.12
Diastolic Blood Pressure (mm Hg)	77.85 ± 9.23
Waist Circumference (inches)	36.20 ± 6.27
Body Mass Index (kg/m^2)	27.46 ± 5.77
Insulin (µU/mL)	13.34 ± 7.61
Glucose (mg/dL)	96.00 ± 16.56
Triglycerides (mg/dL)	119.47 ± 81.43
HDL Cholesterol (mg/dL)	53.68 ± 14.68
Observed Health Behavior Variables	
Cigarette Smoking (number of cigarettes per day)	6.14 ± 10.94
Alcohol Use (number of alcoholic beverages per week)	3.80 ± 7.50
Physical Activity (kilocalories per week)	2416.47 ± 1839.74
Energy Intake (kilocalories per day)	2266.83 ± 814.38
Carbohydrate Intake (% energy intake)	49.35 ± 10.24
Protein Intake (% energy intake)	15.58 ± 4.34
Saturated Fat Intake (% energy intake)	11.58 ± 3.38
Unsaturated Fat Intake (% energy intake)	19.90 ± 5.42

Note. HDL = high-density lipoprotein.

Table 2. Descriptive and psychometric statistics for the observed questionnaire measures of impulsivity

Observed Questionnaire Measure	Mean ± SD	α
BIS-11		
Attentional	16.48 ± 3.41	0.68
Motor	19.98 ± 3.74	0.68
Non-Planning	24.33 ± 5.20	0.74
BIS/BAS		
Behavioral Inhibition	19.18 ± 3.62	0.78
Drive	10.81 ± 2.32	0.77
Fun-Seeking	11.59 ± 2.28	0.71
Reward Responsiveness	17.30 ± 1.89	0.66
MPQ-BF		
Constraint	84.09 ± 14.49	0.82
Negative Emotionality	29.39 ± 14.24	0.82
Positive Emotionality	69.15 ± 15.51	0.82
NEO-PI-R Conscientiousness		
Achievement Striving	19.05 ± 4.78	0.76
Competence	23.45 ± 3.75	0.71
Deliberation	18.63 ± 4.31	0.73
Dutifulness	23.74 ± 3.91	0.62
Order	18.54 ± 4.63	0.71
Self-Discipline	21.17 ± 4.85	0.81
NEO-PI-R Extraversion		
Activity	18.31 ± 4.59	0.70
Assertiveness	16.50 ± 5.16	0.79
Excitement-Seeking	17.35 ± 5.03	0.64
Gregariousness	17.13 ± 5.52	0.79
Positive Emotions	21.03 ± 5.13	0.80
Warmth	23.02 ± 4.64	0.82
NEO-PI-R Neuroticism		
Angry Hostility	11.62 ± 5.21	0.81
Anxiety	12.99 ± 5.26	0.80
Depression	11.54 ± 5.77	0.84
Impulsiveness	15.18 ± 4.56	0.72
Self-Consciousness	13.97 ± 4.87	0.73
Vulnerability	9.02 ± 4.17	0.79
SNAP Disinhibition		
Disinhibition[a]	3.20 ± 2.53	0.67
Impulsivity	4.34 ± 3.61	0.80
Propriety	13.11 ± 3.88	0.57
Workaholism	7.17 ± 3.87	0.71
SNAP Positive Affectivity		
Positive Temperament	19.09 ± 5.53	0.87
Detachment	5.78 ± 4.45	0.88
Exhibitionism	5.49 ± 3.63	0.83
SNAP Negative Affectivity		
Negative Temperament	7.75 ± 6.57	0.92
Eccentric Perceptions	2.56 ± 2.72	0.79
Manipulativeness	3.10 ± 2.89	0.75
Mistrust	3.90 ± 4.02	0.87
Self-Harm	1.42 ± 2.17	0.80

Table 2 (continued)

Observed Questionnaire Measure	Mean ± SD	α
SSS		
Boredom Susceptibility	1.98 ± 1.70	0.52
Disinhibition	3.30 ± 2.57	0.76
Experience Seeking	5.14 ± 2.20	0.64
Thrill and Adventure Seeking	5.06 ± 3.06	0.82
TCI Novelty Seeking[b]		
Disorderliness	4.60 ± 1.99	0.51
Exploratory Excitability	6.13 ± 2.42	0.65
Extravagance	4.52 ± 2.38	0.74
Impulsiveness	3.41 ± 2.46	0.72

Note. BIS-11 = Barratt Impulsiveness Scale-11; BIS/BAS = Behavioral Inhibition System/Behavioral Activation System; MPQ-BF = Multidimensional Personality Questionnaire-Brief Form; NEO-PI-R = NEO Personality Inventory-Revised; SNAP = Schedule for Nonadaptive and Adaptive Personality; SSS = Zuckerman Sensation Seeking Scale; TCI = Temperament and Character Inventory.

[a]Because the Disinhibition subscale (35 items) contains several items that overlap with other SNAP subscales, the non-overlapping version of the Disinhibition subscale (16 items) was used in the present analysis.

[b]The TCI was not used in the study by Sharma and colleagues (2014) but was used in previous research using this study sample (Creswell, et al., 2018) and was thus included in the present analysis.

Table 3. Descriptive statistics for the observed behavioral task measures of impulsivity

Observed Behavioral Task Measure	Mean ± SD
DDT Indifference Point	-2.40 ± 0.70
IGT Net Score	19.35 ± 29.35
Stroop Interference Score	-0.49 ± 7.36
WCST Perseverative Errors	9.51 ± 8.54
WCST Non-Perseverative Errors	9.30 ± 9.08

Note. DDT = Delay Discounting Task; IGT = Iowa Gambling Task; WCST = Wisconsin Card Sorting Test.

4.1 MEASUREMENT MODELS

4.1.1 Measurement model for questionnaire measures of impulsivity

A total of 48 observed subscales obtained from seven commonly used questionnaire measures of impulsivity were included as indicators in the measurement model for questionnaire measures of impulsivity. Each of these indicators overlapped with those included in the study by Sharma and colleagues (2014), with the exception of the four indicators from the Novelty Seeking domain of the TCI.

Results from the exploratory factor analysis showed that the three latent factors extracted from the observed questionnaire measures of impulsivity accounted for 46% of the total variance and 71% of the common variance (see Table 4). These three latent questionnaire factors were highly consistent with those found by Sharma and colleagues (2014) as well as with those found in previous research using this study sample (Creswell et al., 2018) and were thus labelled Disinhibition versus Constraint/Conscientiousness (DvC/C), Extraversion/Positive Emotionality (E/PE), and Neuroticism/Negative Emotionality (N/NE). As shown in Table 4, there were 26 indicators that loaded on the DvC/C factor. The DvC/C factor was positively anchored by the

SNAP Impulsivity and SNAP Disinhibition indicators and negatively anchored by the MPQ-BF Constraint indicator. All six indicators from the NEO-PI-R Conscientiousness subfacet also negatively loaded on the DvC/C factor. In addition, there were 18 indicators that loaded on the E/PE factor. The E/PE factor was positively anchored by the MPQ-BF Positive Emotionality indicator and negatively anchored by the SNAP Detachment indicator and also tended to include indicators measuring sensation seeking. All six indicators from the NEO-PI-R Extraversion subfacet also positively loaded on the E/PE factor. Finally, there were 19 indicators that loaded on the N/NE factor. The N/NE factor was positively anchored by the SNAP Negative Temperament indicator. All six indicators from the NEO-PI-R Neuroticism subfacet also positively loaded on the N/NE factor. Similar to the findings reported by Sharma and colleagues (2014), a number of indicators (29%, $n = 14$) cross-loaded on more than one factor.

Given the large number of indicators included in the measurement model for questionnaire measures of impulsivity, it was not expected to achieve conservative estimates of model fit (Hooper, Coughlan, & Mullen, 2008). Indeed, the chi-square test was significant ($\chi^2(987) = 10385.59$, $p < 0.001$), indicating incongruence between the theorized model and the empirical data from the sample. The additional model fit indices also indicated poor model fit (CFI = 0.70; RMSEA = 0.09, 90% CI [0.08, 0.09]), with the exception of the SRMR, which indicated acceptable model fit (SRMR = 0.06). Factor congruence coefficients (Lorenzo-Seva & Ten Berge, 2006) were subsequently calculated to determine the vector similarities between the DvC/C, E/PE, and N/NE factors found in the present study and those found in the study by Sharma and colleagues (2014). The factor congruence coefficients for the DvC/C, E/PE, and N/NE factors were calculated from the factor loadings of shared indicators between the present study and the study by Sharma and colleagues (2014) and were respectively found to be 0.86, 0.78, and 0.92. These findings

Table 4. Factor loadings obtained from the exploratory factor analysis conducted on the observed questionnaire measures of impulsivity

Observed Questionnaire Measure	Scale	DvC/C	E/PE	N/NE
SNAP	Impulsivity	**0.79**	0.04	0.05
MPQ-BF	Constraint	**-0.77**	0.01	0.19
SNAP	Disinhibition[a]	**0.71**	-0.01	0.12
NEO-PI-R	Deliberation	**-0.67**	0.06	-0.18
TCI[b]	Disorderliness	**0.60**	0.09	-0.19
TCI[b]	Impulsiveness	**0.60**	-0.01	0.001
BIS-11	Non-Planning	**0.58**	-0.21	0.11
SSS	Experience Seeking	**0.57**	0.07	-0.21
SSS	Disinhibition	**0.54**	0.14	-0.02
SNAP	Propriety	**-0.53**	0.22	**0.31**
SNAP	Manipulativeness	**0.51**	0.06	0.26
NEO-PI-R	Self-Discipline	**-0.51**	**0.34**	-0.27
NEO-PI-R	Dutifulness	**-0.51**	0.24	-0.17
NEO-PI-R	Order	**-0.50**	0.27	0.01
BIS/BAS	Fun-Seeking	**0.49**	**0.45**	0.04
NEO-PI-R	Competence	**-0.48**	**0.39**	**-0.31**
TCI[b]	Extravagance	**0.44**	0.11	0.02
SSS	Thrill and Adventure Seeking	**0.42**	0.18	-0.21
NEO-PI-R	Excitement Seeking	**0.40**	**0.39**	-0.001
BIS-11	Motor	**0.37**	0.23	**0.36**
SSS	Boredom Susceptibility	**0.34**	0.08	0.15
MPQ-BF	Positive Emotionality	-0.02	**0.80**	-0.15
SNAP	Positive Temperament	-0.07	**0.76**	-0.08
NEO-PI-R	Activity	-0.15	**0.66**	0.02
NEO-PI-R	Assertiveness	-0.01	**0.61**	-0.20
NEO-PI-R	Achievement Striving	**-0.47**	**0.61**	0.03
SNAP	Exhibitionism	0.29	**0.54**	-0.05
BIS/BAS	Drive	0.10	**0.54**	0.09
NEO-PI-R	Positive Emotions	0.13	**0.51**	**-0.30**
TCI[b]	Exploratory Excitability	**0.39**	**0.49**	-0.18
NEO-PI-R	Gregariousness	0.12	**0.48**	-0.17
SNAP	Detachment	-0.04	**-0.48**	**0.32**
NEO-PI-R	Warmth	0.03	**0.47**	-0.29
BIS/BAS	Reward Responsiveness	-0.04	**0.46**	0.13
SNAP	Workaholism	-0.25	**0.45**	**0.36**
SNAP	Negative Temperament	-0.01	0.13	**0.87**
NEO-PI-R	Anxiety	-0.10	-0.07	**0.77**
NEO-PI-R	Depression	0.11	-0.16	**0.76**
MPQ-BF	Negative Emotionality	0.09	0.18	**0.72**
NEO-PI-R	Angry Hostility	0.07	0.04	**0.71**
NEO-PI-R	Self-Consciousness	-0.06	-0.18	**0.66**
NEO-PI-R	Vulnerability	0.15	-0.26	**0.64**
BIS/BAS	Behavioral Inhibition	-0.14	-0.003	**0.61**
SNAP	Mistrust	0.07	0.06	**0.59**
SNAP	Self-Harm	**0.30**	-0.14	**0.47**
NEO-PI-R	Impulsiveness	**0.37**	0.04	**0.44**
SNAP	Eccentric Perceptions	0.23	0.23	**0.39**
BIS-11	Attentional	**0.33**	-0.10	**0.38**

Note. The exploratory factor analysis was estimated using the maximum likelihood method with robust standard errors. An oblique oemin rotation was specified, and a three-factor solution was extracted.

Table 4 (continued)

Standardized factor loadings are displayed. Boldface data indicate factor loadings above |0.30|. DvC/C = Disinhibition versus Constraint/Conscientiousness; E/PE = Extraversion/Positive Emotionality; N/NE = Neuroticism/Negative Emotionality; BIS-11 = Barratt Impulsiveness Scale-11; BIS/BAS = Behavioral Inhibition System/Behavioral Activation System; MPQ-BF = Multidimensional Personality Questionnaire-Brief Form; NEO-PI-R = NEO Personality Inventory-Revised; SNAP = Schedule for Nonadaptive and Adaptive Personality; SSS = Zuckerman Sensation Seeking Scale; TCI = Temperament and Character Inventory.

[a]Because the Disinhibition subscale (35 items) contains several items that overlap with other SNAP subscales, the non-overlapping version of the Disinhibition subscale (16 items) was used in the present analysis.

[b]The TCI was not used in the study by Sharma and colleagues (2014) but was used in previous research using this study sample (Creswell, et al., 2018) and was thus included in the present analysis.

Demonstrate that the latent questionnaire factors found in the present study were largely equivalent to those found in the study by Sharma and colleagues (2014) despite the poor model fit.

4.1.2 Measurement model for behavioral task measures of impulsivity

A total of five observed scores obtained from four commonly used behavioral task measures of impulsivity were included as indicators in the measurement model for behavioral task measures of impulsivity. Each of these indicators overlapped with those included in the study by Sharma and colleagues (2014).

The measurement model for behavioral task measures of impulsivity was constructed using a confirmatory approach and consisted of four latent behavioral task factors analogous to those found by Sharma and colleagues (2014). Given the limited number of observed behavioral task measures of impulsivity included in the present study, three of the four latent behavioral task factors comprised a single indicator. These three latent behavioral task factors are therefore equivalent to the observed variables (Hayduk & Littvay, 2012) and do not fully correspond with the latent behavioral task factors found by Sharma and colleagues (2014). Despite the differences

in available indicators between studies, the four latent behavioral task factors in the present study were labelled Inattention, Inhibition, Impulsive Decision-Making, and Set-Shifting to remain consistent with the terminology used in the study by Sharma and colleagues (2014).

As shown in Figure 1, the Inattention, Inhibition, and Impulsive Decision-Making factors each comprised a single indicator while the Set-Shifting factor comprised two indicators. The Stroop Interference Score, IGT Net Score, and DDT Indifference Point indicators were constrained to respectively load on the Inattention, Inhibition, and Impulsive Decision-Making factors by fixing their factor loadings to one and their variances to zero (Hayduk & Littvay, 2012). Meanwhile, the WCST Perseverative Errors and WCST Non-Perseverative Errors indicators were constrained to freely load on the Set-Shifting factor and both produced strong and significant factor loadings in the expected directions.

The measurement model for behavioral task measures of impulsivity was consistent with that reported in previous research using this study sample (Creswell et al., 2018) and provided a good fit to the data. Because the measurement model for behavioral task measures of impulsivity was almost fully saturated, it was expected to demonstrate near perfect model fit (Kline, 2015). Indeed, the chi-square test was not significant ($\chi^2(2) = 3.04$, $p = 0.22$), indicating congruence between the theorized model and the empirical data from the sample. The additional model fit indices also indicated acceptable model fit (CFI = 0.99; SRMR = 0.01; RMSEA = 0.02, 90% CI [0.00, 0.06]).

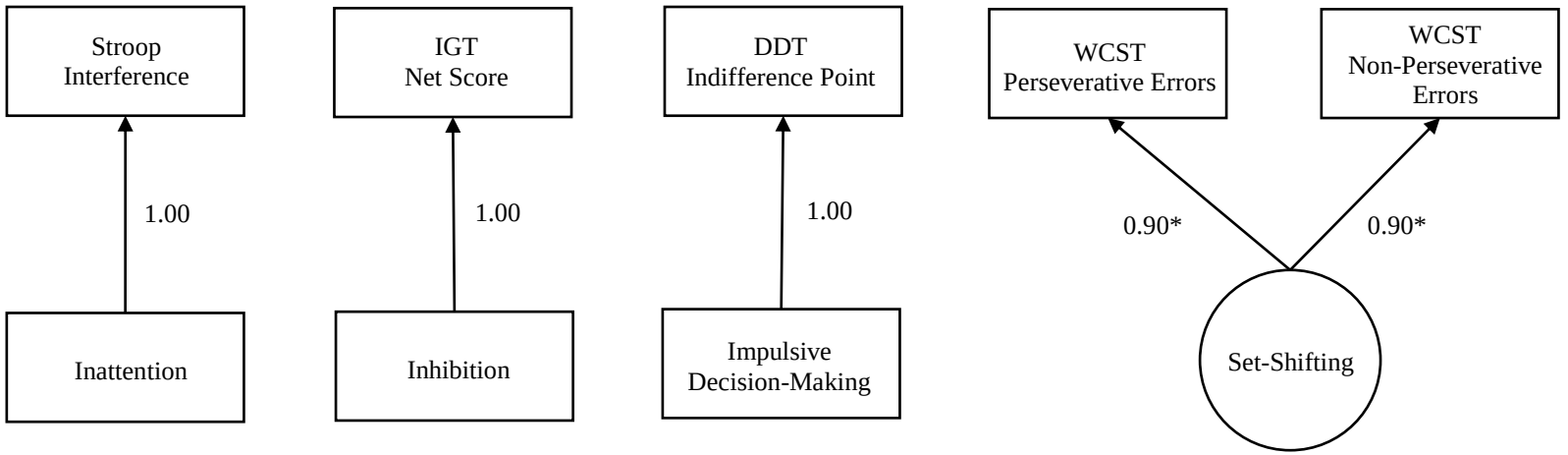

Figure 1. Measurement model for behavioral task measures of impulsivity. *Note.* Standardized factor loadings from the confirmatory factor analysis are displayed. The model was estimated using the maximum likelihood method with robust standard errors. Residual arrows for the observed variables and latent factors are omitted to simplify the figure. DDT = Delay Discounting Task; IGT = Iowa Gambling Task; WCST = Wisconsin Card Sorting Test.
*$p < 0.05$.

4.1.3 Measurement model for cardiometabolic risk

A total of eight observed cardiometabolic risk variables were included as indicators in the final measurement model for cardiometabolic risk. Each of these indicators overlapped with those included in previous research using this study sample (Dermody et al., 2015; Marsland et al., 2010; McCaffery et al., 2012; McCaffery et al., 2007).

The measurement model for cardiometabolic risk was constructed using a confirmatory approach and consisted of four subfactors underlying a single, superordinate factor analogous to the currently accepted definition of MetS (Alberti et al., 2009). As shown in Figure 2, each of the four subfactors comprised two indicators and were labelled Blood Pressure, Insulin Resistance, Adiposity, and Blood Lipids. The systolic and diastolic blood pressure indicators were constrained to freely load on the Blood Pressure subfactor, the insulin and glucose indicators were constrained to freely load on the Insulin Resistance subfactor, and the triglycerides and HDL cholesterol indicators were constrained to freely load on the Blood Lipids subfactor. Although the BMI indicator was constrained to freely load on the Adiposity subfactor, the waist circumference indicator produced a small but non-significant negative residual variance (residual variance = -0.07, $p = 0.60$) and was therefore constrained to load on the Adiposity subfactor by fixing its factor loading to one and its variance to zero (Kline, 2015). The seven indicators that were constrained to freely load on their respective subfactors produced strong and significant factor loadings in the expected directions. The four subfactors were further constrained to freely load on a single, superordinate factor labelled Cardiometabolic Risk. Each of the four subfactors loaded strongly and significantly on the superordinate cardiometabolic risk factor in the expected directions.

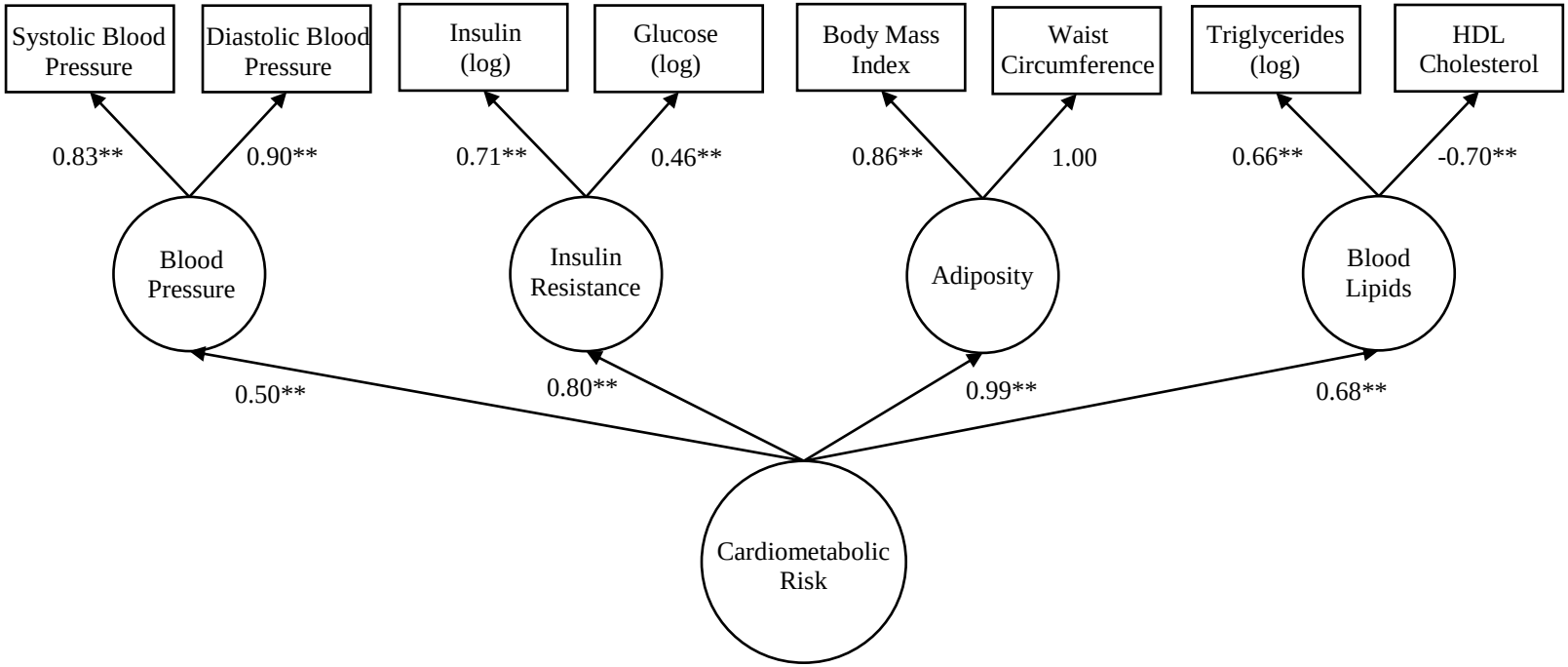

Figure 2. Measurement model for cardiometabolic risk. *Note.* Standardized factor loadings from the confirmatory factor analysis are displayed. The model was estimated using the maximum likelihood method with robust standard errors. Residual arrows for the observed variables and latent factors are omitted to simplify the figure. HDL = high-density lipoprotein.
*$p < 0.05$; **$p < 0.01$

The measurement model for cardiometabolic risk was consistent with the findings reported in previous research using this study sample (Dermody et al., 2015; Marsland et al., 2010; McCaffery et al., 2012; McCaffery et al., 2007) and provided a good fit to the data. Although the chi-square test was significant ($\chi^2(18) = 203.96$, $p < 0.001$), indicating some incongruence between the theorized model and the empirical data from the sample, the additional model fit indices all suggested acceptable model fit (CFI = 0.95; SRMR = 0.05; RMSEA = 0.08, 90% CI [0.07, 0.09]).

4.2 STRUCTURAL MODEL

4.2.1 Bivariate correlations among the latent and observed study variables

Prior to constructing the structural model, bivariate correlations were calculated among all latent and observed study variables (see Tables 5 and 6). As shown in Tables 5 and 6, correlations between the latent and observed study variables ranged in magnitude from weak to strong and were generally in the expected directions. Correlations between the latent impulsivity factors tended to be strongest among factors derived from the same measurement modality. Although DvC/C and E/PE were unrelated, N/NE positively correlated with DvC/C and negatively correlated with E/PE. In addition, N/NE and DvC/C both positively correlated with Impulsive Decision-Making, and N/NE was further shown to positively correlate with Set-Shifting. Impulsive Decision-Making and Set-Shifting were likewise positively correlated and both negatively correlated with Inattention and Inhibition.

The strength and significance of the remaining correlations varied widely. DvC/C, Inattention, and Set-Shifting were the only latent impulsivity factors to significantly correlate with the latent cardiometabolic risk factor. The latent cardiometabolic risk factor also positively correlated with energy intake, protein intake, and saturated fat intake and negatively correlated with physical activity. Although Inattention and Inhibition were unrelated to the observed health behavior variables, the remaining latent impulsivity factors all correlated with distinct health behaviors. Specifically, DvC/C, Impulsive Decision-Making, and Set-Shifting positively correlated with cigarette smoking. DvC/C and Impulsive Decision-Making also negatively correlated with alcohol use, and N/NE negatively correlated with physical activity. Meanwhile, E/PE positively correlated with both alcohol use and physical activity. Contrary to expectations, none of the energy intake or dietary composition variables significantly correlated with the latent impulsivity factors. However, the energy intake and dietary composition variables were strongly correlated with one another and also demonstrated several significant correlations with cigarette smoking, alcohol use, and physical activity. Cigarette smoking, alcohol use, and physical activity were likewise all significantly correlated.

To better understand how the latent impulsivity factors and the observed health behavior variables related to the latent cardiometabolic risk factor, each of the latent impulsivity factors and the observed health behavior variables were correlated with the cardiometabolic risk indicators (see Table 7). As shown in Table 7, correlations ranged in magnitude from weak to moderate. The latent impulsivity factors tended to correlate with fewer cardiometabolic risk indicators than did the observed health behavior variables, and none of the latent impulsivity factors correlated with diastolic blood pressure, glucose concentrations, or HDL cholesterol concentrations. Moreover, Set-Shifting did not correlate with any of the cardiometabolic risk indicators. The remaining latent

impulsivity factors demonstrated varied relationships with the cardiometabolic risk indicators, with some correlations documenting poor cardiometabolic outcomes (e.g., DvC/C positively correlated with waist circumference) and others documenting favorable cardiometabolic outcomes (e.g., DvC/C negatively correlated with insulin concentrations).

Unlike the latent impulsivity factors, the observed health behavior variables were more consistently correlated with the cardiometabolic risk indicators in the expected directions. Alcohol use, physical activity, and energy intake correlated with nearly all of the cardiometabolic risk indicators. Although higher alcohol use was predominantly correlated with poorer cardiometabolic outcomes, it was also correlated with some cardiometabolic benefits, including lower BMI and insulin concentrations and greater HDL cholesterol concentrations. Meanwhile, higher physical activity was uniformly correlated with favorable cardiometabolic outcomes, and higher energy intake was uniformly correlated with problematic cardiometabolic outcomes. In addition, the dietary composition variables were most strongly correlated with waist circumference and BMI. Protein intake and saturated fat intake also positively correlated with insulin concentrations, and protein intake was further shown to negatively correlate with HDL cholesterol concentrations. Finally, cigarette smoking positively correlated with glucose concentrations.

Table 5. Bivariate correlations among the latent impulsivity factors, latent cardiometabolic risk factor, and observed health behavior variables

Study Variable	DvC/C	E/PE	N/NE	Inattention	Inhibition	Impulsive Decision-Making	Set-Shifting
DvC/C	1	--	--	--	--	--	--
E/PE	0.02	1	--	--	--	--	--
N/NE	**0.21****	**-0.20****	1	--	--	--	--
Inattention	0.04	-0.04	-0.03	1	--	--	--
Inhibition	-0.06	-0.05	-0.02	0.07	1	--	--
Impulsive Decision-Making	**0.15****	-0.02	**0.15****	**-0.12****	**-0.29****	1	--
Set-Shifting	-0.03	0.04	**0.08***	**-0.19****	**-0.25****	**0.31****	1
Cardiometabolic Risk	**0.09****	-0.01	0.04	-0.06*	0.01	0.07	**0.11****
Cigarette Smoking (log)	**0.15****	-0.03	0.06	-0.03	-0.07	**0.16****	**0.12****
Alcohol Use (log)	**0.20****	**0.08***	0.03	0.06	0.03	**-0.13****	-0.05
Physical Activity	0.04	**0.18****	**-0.06***	0.04	0.001	0.002	-0.04
Energy Intake	0.02	-0.02	0.09	0.05	0.001	-0.02	-0.07
Carbohydrate Intake	-0.09	0.05	-0.02	0.04	0.06	-0.20	-0.05
Protein Intake	-0.11	0.12	-0.14	0.02	0.09	-0.12	-0.06
Saturated Fat Intake	-0.02	0.01	0.03	0.05	-0.02	-0.06	-0.05
Unsaturated Fat Intake	-0.01	0.05	-0.08	0.03	0.03	-0.05	-0.03

Note. Boldface data indicate significance. DvC/C = Disinhibition versus Constraint/Conscientiousness; E/PE = Extraversion/Positive Emotionality; N/NE = Neuroticism/Negative Emotionality.
*$p < 0.05$; **$p < 0.01$.

Table 6. Bivariate correlations among the latent cardiometabolic risk factor and observed health behavior variables

Study Variable	Cardiometabolic Risk	Cigarette Smoking (log)	Alcohol Use (log)	Physical Activity	Energy Intake	Carbohydrate Intake	Protein Intake	Saturated Fat Intake	Unsaturated Fat Intake
Cardiometabolic Risk	1	--	--	--	--	--	--	--	--
Cigarette Smoking (log)	0.04	1	--	--	--	--	--	--	--
Alcohol Use (log)	0.03	**0.16****	1	--	--	--	--	--	--
Physical Activity	**-0.16****	**-0.07***	**0.12****	1	--	--	--	--	--
Energy Intake	**0.26****	-0.01	**0.18****	0.03	1	--	--	--	--
Carbohydrate Intake	0.10	**-0.12***	**-0.25****	0.04	**-0.14****	1	--	--	--
Protein Intake	**0.18****	0.001	0.02	0.04	**-0.22****	**-0.42****	1	--	--
Saturated Fat Intake	**0.17****	-0.08	-0.06	**-0.13****	**0.12***	**-0.60****	0.001	1	--
Unsaturated Fat Intake	0.02	0.06	-0.04	-0.08	**0.19****	**-0.66****	0.05	**0.46****	1

Note. Boldface data indicate significance.
*$p < 0.05$; **$p < 0.01$.

Table 7. Bivariate correlations among the latent impulsivity factors, observed health behavior variables, and observed cardiometabolic risk variables

Latent Impulsivity Factors	Systolic Blood Pressure	Diastolic Blood Pressure	Waist Circumference	Body Mass Index	Insulin (log)	Glucose (log)	Triglycerides (log)	HDL Cholesterol
DvC/C	-0.03	-0.04	**0.19****	-0.08	**-0.11****	0.02	0.05	-0.06
E/PE	-0.01	-0.03	-0.08	0.07	0.05	-0.004	**-0.07***	-0.01
N/NE	**-0.10***	0.03	-0.01	0.09	-0.02	0.02	0.03	-0.02
Inattention	-0.05	0.01	0.04	**-0.10***	-0.01	-0.03	0.04	-0.003
Inhibition	0.04	-0.05	-0.05	0.06	**-0.10**	-0.004	**0.14***	0.002
Impulsive Decision-Making	0.10	-0.07	-0.09	**0.14***	0.03	0.04	-0.02	-0.03
Set-Shifting	0.07	0.001	0.10	-0.02	0.001	0.03	-0.03	-0.03
Observed Health Behavior Variables								
Cigarette Smoking (log)	0.02	-0.03	0.05	0.02	-0.02	**0.10****	0.05	0.01
Alcohol Use (log)	**0.10****	**0.07***	0.03	**-0.09****	**-0.11****	**0.12****	**0.10****	**0.09****
Physical Activity	-0.05	-0.05	**-0.16****	**-0.15****	**-0.13****	**-0.09****	**-0.12****	**0.13****
Energy Intake	**0.18****	**0.20****	**0.22****	0.04	0.07	**0.13****	**0.16****	**-0.23****
Carbohydrate Intake	-0.09	-0.07	**-0.16****	**-0.15****	-0.07	-0.04	-0.05	0.02
Protein Intake	-0.03	-0.01	0.08	**0.12***	**0.10***	-0.03	-0.03	-0.001
Saturated Fat Intake	0.02	0.03	**0.17****	**0.21****	**0.12***	-0.01	0.06	**-0.12***
Unsaturated Fat Intake	0.01	0.01	**0.11***	**0.11***	0.02	0.04	-0.02	-0.07

Note. Boldface data indicate significance. DvC/C = Disinhibition versus Constraint/Conscientiousness; E/PE = Extraversion/Positive Emotionality; N/NE = Neuroticism/Negative Emotionality; HDL = high-density lipoprotein.
*$p < 0.05$; **$p < 0.01$.

4.2.2 Structural analysis of the latent impulsivity factors, observed health behavior variables, and latent cardiometabolic risk factor

The structural model was estimated using exploratory structural equation modeling and examined the extent to which the observed health behavior variables accounted for the direct effects between each latent impulsivity factor and the latent cardiometabolic risk factor. As previously described, all latent and observed study variables included in the structural model were conditioned on key demographic variables, including age, sex, race, and number of years of education. Each of the observed health behavior variables were then simultaneously regressed on each of the latent impulsivity factors, and the latent cardiometabolic risk factor was simultaneously regressed on each of the observed health behavior variables and on each of the latent impulsivity factors. Given the complexity of the structural model, it was not expected to achieve conservative estimates of model fit (Hu & Bentler, 1999). Indeed, the chi-square test was significant ($\chi^2(2296) = 14942.82$, $p < 0.0001$), indicating incongruence between the theorized model and the empirical data from the sample. The CFI also indicated poor model fit (CFI = 0.71). However, the SRMR and RMSEA, which both account for model complexity (Cangur & Ercan, 2015), indicated acceptable model fit (SRMR = 0.06; RMSEA = 0.07, 90% CI [0.06, 0.07]).

4.2.2.1 Associations between the latent impulsivity factors and the latent cardiometabolic risk factor. The latent cardiometabolic risk factor was regressed on each of the latent impulsivity factors and the standardized parameter estimate for each direct effect was obtained (see Table 8).

Table 8. Regression parameters obtained from the structural analysis examining the effects of the latent impulsivity factors and observed health behavior variables on the latent cardiometabolic risk factor

Latent Impulsivity Factors	β	95% CI	p
DvC/C	0.04	-0.02, 0.11	0.20
E/PE	0.07	-0.004, 0.14	0.07
N/NE	0.09	0.01, 0.16	**0.02**
Inattention	-0.05	-0.11, 0.004	0.07
Inhibition	-0.002	-0.11, 0.10	0.97
Impulsive Decision-Making	-0.02	-0.11, 0.08	0.74
Set-Shifting	0.03	-0.04, 0.09	0.46
Observed Health Behavior Variables			
Cigarette Smoking (log)	-0.009	-0.07, 0.05	0.78
Alcohol Use (log)	-0.07	-0.13, -0.001	**0.05**
Physical Activity	-0.20	-0.24, -0.15	**<0.01**
Energy Intake	-0.03	-0.16, 0.10	0.67
Carbohydrate Intake	0.007	-0.19, 0.21	0.94
Protein Intake	0.13	0.01, 0.25	**0.04**
Saturated Fat Intake	0.21	0.09, 0.34	**<0.01**
Unsaturated Fat Intake	0.0001	-0.14, 0.14	0.99

Note. Standardized regression coefficients are displayed. The model was estimated using the maximum likelihood method with robust standard errors. All latent and observed study variables were allowed to freely covary and were conditioned on key demographic variables, including age, sex, race, and number of years of education. Boldface data indicate significance. CI = Confidence Interval; DvC/C = Disinhibition versus Constraint/Conscientiousness; E/PE = Extraversion/Positive Emotionality; N/NE = Neuroticism/Negative Emotionality.

As shown in Table 8, N/NE was the only latent impulsivity factor directly related to cardiometabolic risk. Specifically, individuals high on N/NE were at greater cardiometabolic risk than were individuals low on N/NE.

4.2.2.2 Associations between the latent impulsivity factors and the observed health behavior variables. Each of the observed health behavior variables were regressed on each of the latent impulsivity factors and the standardized parameter estimate for each association was obtained (see Table 9). As shown in Table 9, there were several significant associations among the latent impulsivity factors and the observed health behavior variables. Among the latent questionnaire factors, DvC/C positively related to cigarette smoking and alcohol use, E/PE positively related to

alcohol use and physical activity, and N/NE positively related to energy intake and negatively related to protein intake. These findings indicate that individuals high on DvC/C smoked more cigarettes per day and consumed more alcoholic beverages per week than did individuals low on DvC/C. Moreover, individuals high on E/PE consumed more alcoholic beverages per week and expended more calories from physical activity per week than did individuals low on E/PE. Finally, individuals high on N/NE consumed more overall calories per day and consumed proportionally fewer calories per day from protein than did individuals low on N/NE.

Among the latent behavioral task factors, Inhibition negatively related to carbohydrate intake and positively related to protein, saturated fat, and unsaturated fat intake, Impulsive Decision-Making negatively related to carbohydrate intake and positively related to saturated fat and unsaturated fat intake, and Set-Shifting negatively related to energy intake. Inattention did not significantly relate to any of the observed health behavior variables. These findings indicate that individuals low on Inhibition consumed proportionally more calories per day from carbohydrates and proportionally fewer calories per day from protein, saturated fat, and unsaturated fat than did individuals high on Inhibition. Moreover, individuals high on Impulsive Decision-Making consumed proportionally fewer calories per day from carbohydrates and proportionally more calories per day from saturated fat and unsaturated fat than did individuals low on Impulsive Decision-Making. Finally, individuals high on Set-Shifting consumed fewer overall calories per day than did individuals low on Set-Shifting.

Table 9. Regression parameters obtained from the structural analysis examining the effects of the latent impulsivity factors on the observed health behavior variables

	Cigarette Smoking (log)			Alcohol Use (log)		
Latent Impulsivity Factor	β	95% CI	p	β	95% CI	p
DvC/C	0.21	0.15, 0.27	**<0.01**	0.20	0.13, 0.26	**<0.01**
E/PE	-0.01	-0.07, 0.06	0.86	0.12	0.06, 0.18	**<0.01**
N/NE	-0.01	-0.07, 0.06	0.88	0.04	-0.03, 0.11	0.23
Inattention	0.004	-0.05, 0.06	0.87	0.02	-0.04, 0.08	0.50
Inhibition	0.02	-0.06, 0.10	0.61	0.001	-0.09, 0.09	0.98
Impulsive Decision-Making	0.04	-0.05, 0.12	0.39	-0.07	-0.16, 0.02	0.10
Set-Shifting	0.04	-0.03, 0.10	0.25	-0.01	-0.07, 0.06	0.88
	Physical Activity			Energy Intake		
Latent Impulsivity Factor	β	95% CI	p	β	95% CI	p
DvC/C	0.01	-0.06, 0.08	0.85	0.001	-0.09, 0.09	0.99
E/PE	0.19	0.13, 0.25	**<0.01**	0.05	-0.04, 0.15	0.29
N/NE	-0.02	-0.09, 0.04	0.52	0.16	0.06, 0.26	**<0.01**
Inattention	0.03	-0.03, 0.09	0.36	0.02	-0.06, 0.09	0.71
Inhibition	-0.03	-0.12, 0.05	0.41	0.05	-0.08, 0.17	0.46
Impulsive Decision-Making	0.01	-0.07, 0.08	0.9	0.03	-0.09, 0.14	0.68
Set-Shifting	-0.04	-0.09, 0.02	0.22	-0.09	-0.18, -0.01	**0.04**
	Carbohydrate Intake			Protein Intake		
Latent Impulsivity Factor	β	95% CI	p	β	95% CI	p
DvC/C	-0.11	-0.24, 0.01	0.08	-0.03	-0.13, 0.07	0.59
E/PE	-0.06	-0.18, 0.07	0.37	0.10	-0.01, 0.20	0.07
N/NE	0.09	-0.05, 0.24	0.21	-0.14	-0.25, -0.03	**0.01**
Inattention	-0.05	-0.16, 0.06	0.40	0.004	-0.09, 0.10	0.94
Inhibition	-0.34	-0.55, -0.13	**<0.01**	0.21	0.07, 0.35	**<0.01**
Impulsive Decision-Making	-0.27	-0.47, -0.07	**0.01**	0.10	-0.03, 0.24	0.14
Set-Shifting	0.04	-0.12, 0.20	0.61	0.01	-0.12, 0.14	0.87
	Saturated Fat Intake			Unsaturated Fat Intake		
Latent Impulsivity Factor	β	95% CI	p	β	95% CI	p
DvC/C	0.04	-0.08, 0.15	0.53	0.07	-0.05, 0.18	0.26
E/PE	-0.02	-0.13, 0.09	0.68	0.01	-0.10, 0.12	0.87
N/NE	-0.01	-0.14, 0.12	0.90	-0.05	-0.18, 0.07	0.41
Inattention	0.06	-0.05, 0.17	0.26	0.05	-0.05, 0.15	0.33
Inhibition	0.23	0.06, 0.41	**0.01**	0.29	0.11, 0.47	**<0.01**
Impulsive Decision-Making	0.23	0.06, 0.40	**0.01**	0.25	0.07, 0.42	**0.01**
Set-Shifting	-0.05	-0.17, 0.08	0.45	-0.07	-0.21, 0.06	0.29

Note. Standardized regression coefficients are displayed. The model was estimated using the maximum likelihood method with robust standard errors. All latent and observed study variables were allowed to freely covary and were conditioned on key demographic variables, including age, sex, race, and number of years of education. Boldface data indicate significance. CI = Confidence Interval; DvC/C = Disinhibition versus Constraint/Conscientiousness; E/PE = Extraversion/Positive Emotionality; N/NE = Neuroticism/Negative Emotionality.

4.2.2.3 Associations between the observed health behavior variables and the latent cardiometabolic risk factor. The latent cardiometabolic risk factor was regressed on each of the observed health behavior variables and the standardized parameter estimate for each association was obtained (see Table 8). As shown in Table 8, there were four significant associations between the observed health behavior variables and the latent cardiometabolic risk factor. Specifically, alcohol use and physical activity both negatively related to cardiometabolic risk whereas protein intake and saturated fat intake both positively related to cardiometabolic risk. Cigarette smoking, energy intake, carbohydrate intake, and unsaturated fat intake did not significantly relate to cardiometabolic risk. These findings indicate that individuals who consumed more alcoholic beverages per week were at lower cardiometabolic risk than were individuals who consumed fewer alcoholic beverages per week. In addition, individuals who expended more calories from physical activity per week were at lower cardiometabolic risk than were individuals who expended fewer calories from physical activity per week. Finally, individuals who consumed proportionally more calories per day from protein or saturated fat were at higher cardiometabolic risk than were individuals who consumed proportionally fewer calories per day from protein or saturated fat.

4.2.2.4 Indirect effects of the observed health behavior variables on the associations between the latent impulsivity factors and the latent cardiometabolic risk factor. Figure 3 summarizes each of the previously described significant associations found among the latent and observed study variables in the structural model. Indirect effects through the observed health behavior variables were also examined for each direct effect between the latent impulsivity factors and the latent cardiometabolic risk factor, regardless of significance. The standardized parameter estimates for each direct, indirect, and total effect are displayed in Table 10. As shown in Table 10, there

were no significant total effects. However, several significant indirect effects were documented for the effects of E/PE, Inhibition, and Impulsive Decision-Making on the latent cardiometabolic risk factor. Examination of the specific indirect effects showed that E/PE indirectly related to cardiometabolic risk through physical activity (β = -0.04, 95% CI [-0.06, -0.02], $p < 0.001$) and that both Inhibition (β = 0.02, 95% CI [0.001, 0.04], $p = 0.05$) and Impulsive Decision-Making (β = 0.08, 95% CI [0.001, 0.15], $p = 0.05$) indirectly related to cardiometabolic risk through saturated fat intake. The specific indirect effects for the remaining observed health behavior variables were not significant for the effects of E/PE, Inhibition, and Impulsive Decision-Making on the latent cardiometabolic risk factor (ps > 0.08). These findings indicate that higher E/PE was associated with a greater number of calories expended from physical activity per week, which in turn, related to lower cardiometabolic risk. Moreover, lower Inhibition was associated with the consumption of proportionally fewer calories per day from saturated fat, which in turn, related to lower cardiometabolic risk. Meanwhile, higher Impulsive Decision-Making was associated with the consumption of proportionally more calories per day from saturated fat, which in turn, related to greater cardiometabolic risk.

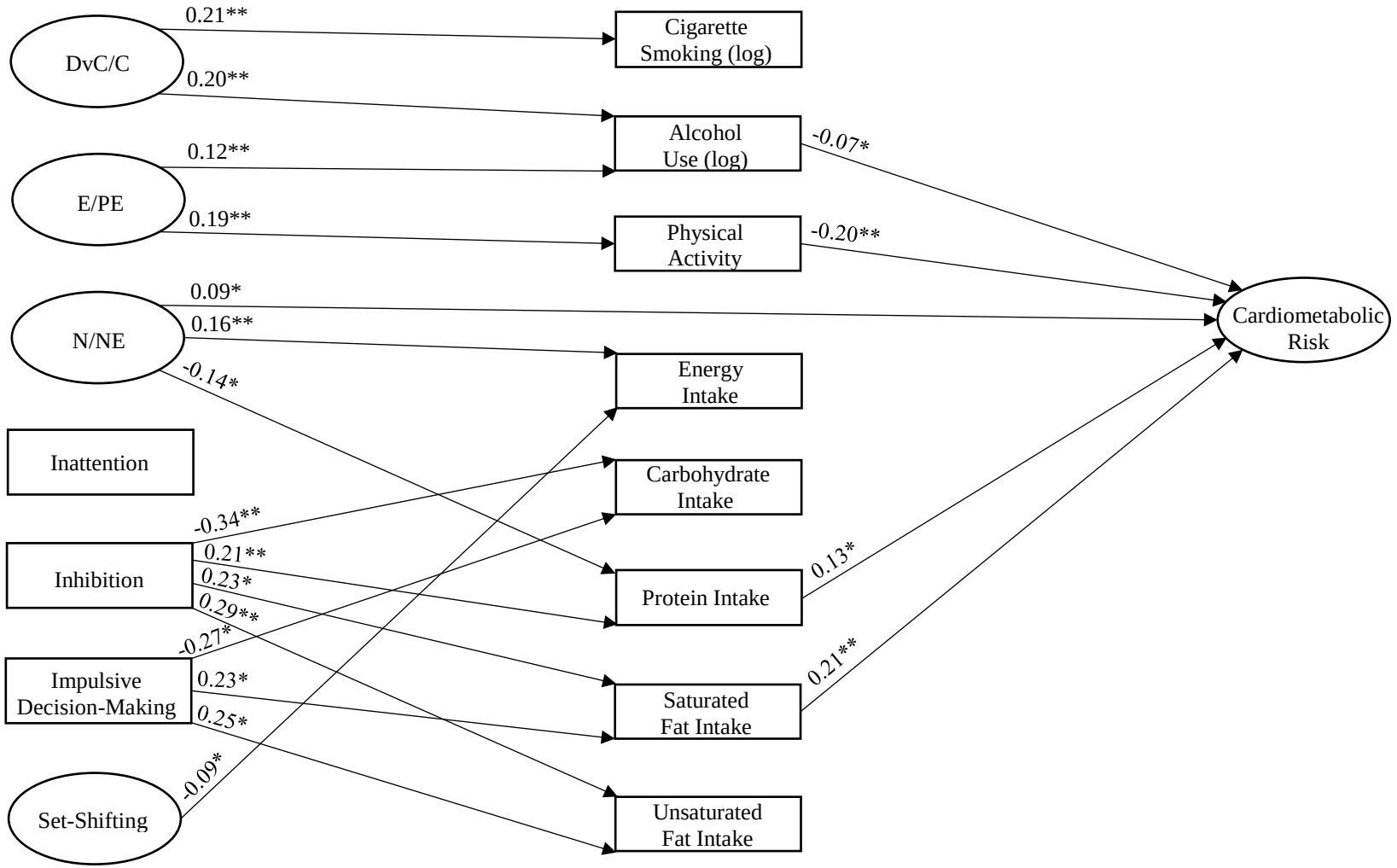

Figure 3. Significant pathways from the Structural analysis of the latent impulsivity factors, observed health behavior variables, and latent cardiometabolic risk factor. *Note.* Standardized regression coefficients are displayed. The model was estimated using the maximum likelihood method with robust standard errors. All latent and observed study variables were allowed to freely covary and were conditioned on key demographic variables, including age, sex, race, and number of years of education. Only significant pathways are included. Residual arrows for the observed variables and latent factors are omitted to simplify the figure. DvC/C = Disinhibition versus Constraint/Conscientiousness; E/PE = Extraversion/Positive Emotionality; N/NE = Neuroticism/Negative Emotionality.
*$p < 0.05$; **$p < 0.01$.

Table 10. Total, direct, and indirect effects obtained from the structural analysis of the latent impulsivity factors, observed health behavior variables, and latent cardiometabolic risk factor

Pathway	β	95% CI	p
DvC/C → Cardiometabolic Risk			
Total Effect	0.03	-0.03, 0.09	0.34
Direct Effect	0.04	-0.02, 0.11	0.20
Indirect Effect	-0.01	-0.05, 0.02	0.49
E/PE → Cardiometabolic Risk			
Total Effect	0.03	-0.04, 0.10	0.44
Direct Effect	0.07	-0.004, 0.14	0.07
Indirect Effect	-0.04	-0.07, -0.01	**0.02**
N/NE → Cardiometabolic Risk			
Total Effect	0.07	-0.004, 0.13	0.07
Direct Effect	0.09	0.01, 0.16	**0.02**
Indirect Effect	-0.02	-0.07, 0.02	0.32
Inattention → Cardiometabolic Risk			
Total Effect	-0.05	-0.10, 0.01	0.11
Direct Effect	-0.05	-0.11, 0.004	0.07
Indirect Effect	0.005	-0.02, 0.04	0.72
Inhibition → Cardiometabolic Risk			
Total Effect	0.08	-0.01, 0.16	0.07
Direct Effect	-0.002	-0.11, 0.10	0.97
Indirect Effect	0.08	0.01, 0.15	**0.03**
Impulsive Decision-Making → Cardiometabolic Risk			
Total Effect	0.05	-0.03, 0.13	0.25
Direct Effect	-0.02	-0.11, 0.08	0.74
Indirect Effect	0.06	0.001, 0.13	**0.05**
Set-Shifting → Cardiometabolic Risk			
Total Effect	0.03	-0.04, 0.09	0.41
Direct Effect	0.03	-0.04, 0.09	0.46
Indirect Effect	0.001	-0.04, 0.04	0.96

Note. Standardized regression coefficients are displayed. The model was estimated using the maximum likelihood method with robust standard errors. All latent and observed study variables were allowed to freely covary and were conditioned on key demographic variables, including age, sex, race, and number of years of education. Boldface data indicate significance. CI = Confidence Interval; DvC/C = Disinhibition versus Constraint/Conscientiousness; E/PE = Extraversion/Positive Emotionality; N/NE = Neuroticism/Negative Emotionality.

5.0 DISCUSSION

The primary aims of the present study were twofold. First, the present study aimed to assess the overall relationship between distinct impulsigenic traits and cardiometabolic risk. Second, the present study aimed to examine the extent to which varied health behaviors, including cigarette smoking, alcohol use, physical activity, energy intake, and dietary composition, accounted for the relationships between each impulsigenic trait and cardiometabolic risk. The primary aims were tested in a large, community sample of midlife adults using a structural equation modeling approach. As discussed more fully below, the present study contributes to the existing literature by using novel methodology to demonstrate that distinct impulsigenic traits differentially relate to cardiometabolic risk through varied behavioral pathways.

The present study is the first to ascertain whether and how distinct impulsigenic traits relate to cardiometabolic risk. The impulsigenic traits examined in the present study were conceptualized in accordance with an increasingly tested multidimensional framework of impulsivity established by Sharma and colleagues (2014). As in previous research using this study sample (Creswell et al., 2018), the latent factor structures for questionnaire and behavioral task measures of impulsivity were highly consistent with those found by Sharma and colleagues (2014). Specifically, questionnaire measures of impulsivity comprised three latent factors, labelled DvC/C, E/PE, and N/NE, whereas behavioral task measures of impulsivity comprised four latent factors, labelled Inattention, Inhibition, Impulsive Decision-Making, and Set-Shifting. Correlational analyses

indicated that the latent questionnaire factors were largely unrelated to the latent behavioral task factors and further revealed several modest associations among latent impulsivity factors derived from the same measurement modality. These findings replicate those reported by Sharma and colleagues (2014) and lend support to the growing consensus that commonly used questionnaire and behavioral task measures of impulsivity encompass several related but distinct impulsigenic traits (Cyders & Coskunpinar, 2011; Duckworth & Kern, 2011; Lane et al., 2003; Reynolds et al., 2006; Thamotharan, Lange, Zale, Huffhines, & Fields, 2013). By using this particular hierarchical structure to conceptualize questionnaire and behavioral task measures of impulsivity, the present study is uniquely suited to clarify the varied relationships between distinct impulsigenic traits and cardiometabolic risk.

The present study further conceptualized cardiometabolic risk in accordance with a commonly used hierarchical structure of MetS that was initially described by Shen and colleagues (2003) and has since been extensively reproduced (Dermody et al., 2015; Marsland et al., 2010; McCaffery et al., 2012; McCaffery et al., 2007). Shen and colleagues (2003) specifically found that continuously distributed components of MetS can be modeled as a single, superordinate cardiometabolic risk factor that underlies covariation in blood pressure, insulin resistance, adiposity, and blood lipids. The present study replicated this factor structure by documenting that the superordinate cardiometabolic risk factor was strongly associated with insulin resistance, adiposity, and blood lipids and modestly associated with blood pressure. This particular pattern of findings is similar to that of previous research using this study sample (Dermody et al., 2015; Marsland et al., 2010; McCaffery et al., 2012; McCaffery et al., 2007) and confirms the presence of a common disease process underlying the component parts of MetS. This hierarchical structure is therefore consistent with the currently accepted definition of MetS (Alberti et al., 2009) and

ultimately allows for a greater understanding of interindividual variability in cardiometabolic risk by providing a means to model MetS as a continuous outcome rather than relying on clinical cutoffs to simply indicate the absence or presence of the syndrome as a whole.

Using these innovative hierarchical conceptualizations of impulsivity and cardiometabolic risk, the primary aims of the present study were tested with a multiple mediation approach to simultaneously examine the independent behavioral pathways driving the relationships between each impulsigenic trait and cardiometabolic risk. Results from this structural model indicated the presence of several notable associations among the latent and observed study variables. Although bivariate analyses showed the DvC/C, Inattention, and Set-Shifting factors to be significantly correlated with cardiometabolic risk, none of these effects persisted after controlling for key demographic variables and accounting for shared variance among the latent and observed study variables in the structural model. Instead, N/NE was found to be the only impulsigenic trait significantly related to cardiometabolic risk in the structural model. Though the positive association between N/NE and cardiometabolic risk was not accounted for by any of the observed health behavior variables, several significant indirect effects were found linking E/PE, Inhibition, and Impulsive Decision-Making to cardiometabolic risk through unique behavioral pathways. Specifically, E/PE was indirectly associated with cardiometabolic risk through physical activity, such that individuals high on E/PE were at reduced cardiometabolic risk as a consequence of greater engagement in physical activity. Moreover, both Inhibition and Impulsive Decision-Making were indirectly associated with cardiometabolic risk through saturated fat intake, such that individuals low on Inhibition had reduced cardiometabolic risk as a consequence of less saturated fat intake and individuals high on Impulsive Decision-Making had heightened cardiometabolic risk as a consequence of greater saturated fat intake. Collectively, these findings contribute to the

current paucity of literature exploring the unique behavioral pathways linking distinct impulsigenic traits to cardiometabolic risk, and the implications of these findings are discussed within the context of the broader literature in the proceeding sections.

5.1 NEUROTICISM/NEGATIVE EMOTIONALITY AND CARDIOMETABOLIC RISK

N/NE was the only impulsigenic trait directly related to heightened cardiometabolic risk in the present study. This finding is consistent with the broader literature documenting that personality traits defined by a proneness towards aggression and negative affectivity are associated with an increased likelihood of being diagnosed with MetS (Cohen et al., 2010; Elovainio et al., 2011; Goldbacher & Matthews, 2007; Mommersteeg et al., 2010; Räikkönen et al., 2003; Räikkönen et al., 2004; Tziallas et al., 2011) and further lends support to the assertion made by Mommersteeg and Pouwer (2012) that the overall relationship between personality and MetS is best assessed using a "clustering" of multiple personality traits related to aggression and negative affectivity rather than using single, omnibus personality measures of such traits. The N/NE factor consisted of numerous questionnaire measures of impulsivity that assess both general personality features and specific impulsive characteristics reflecting a tendency to experience psychological distress and to avoid situations with aversive consequences. The N/NE factor therefore represents a broad personality trait that underlies a disposition to act rashly in the face of negative emotions (Whiteside & Lynam, 2001) and is suited to distinguish subsets of individuals at risk for problematic health outcomes (Mommersteeg & Pouwer, 2012; Sharma et al., 2014). Accordingly, the previously mixed results relating MetS to omnibus personality measures of Neuroticism

(Dermody et al., 2015; Phillips et al., 2010; Ross et al., 2011; Sutin, Costa, et al., 2010; van Reedt Dortland et al., 2012) may be clarified through the comprehensive use of questionnaire measures of impulsivity that provide a more multifaceted assessment of N/NE.

Contrary to expectations, the positive association between N/NE and cardiometabolic risk was not accounted for by any of the observed health behavior variables included in the present study. The lack of indirect behavioral effects linking N/NE to cardiometabolic risk is particularly surprising given that measures of N/NE are widely associated with a greater likelihood of engaging in maladaptive health behaviors that confer risk for cardiometabolic morbidity and mortality (Emery, King, Fischer, & Davis, 2013; Emery, King, & Levine, 2014; Kelly, Cotter, & Mazzeo, 2014; Munafo, Zetteler, & Clark, 2007; Terracciano, Löckenhoff, Crum, Bienvenu, & Costa, 2008; VanderVeen et al., 2016; Zvolensky, Taha, Bono, & Goodwin, 2015). Findings from neuroimaging and behavioral research demonstrate that measures of N/NE are strongly related to dysfunctional prefrontal cortical activity and increased insular and amygdalar activity (Cyders & Smith, 2007; Cyders & Smith, 2008b; Ormel et al., 2013; Williams et al., 2018), which together result in a predisposition to frequently experience intense negative emotions as well as a tendency to avoid aversive emotional stimuli by participating in negatively reinforcing behaviors (Carver, 2005; Gray, 1970). Individuals high on measures of N/NE are therefore especially likely to engage in alcohol and substance use (Owens, Amlung, Stojek, & MacKillop, 2018; Terracciano et al., 2008; VanderVeen et al., 2016), cigarette smoking (Munafo et al., 2007; Zvolensky et al., 2015), palatable food overconsumption (Emery et al., 2014), and binge eating (Emery et al., 2013; Fischer, Wonderlich, & Becker, 2018; Kelly et al., 2014) as a means to distract from or reduce the experience of emotional distress. Although N/NE was unrelated to cigarette smoking and alcohol use in the present study, it was shown to positively relate to energy intake and negatively relate to

protein intake, suggesting that individuals high on N/NE may be likely to overeat as a consequence of consuming less satiating food sources (Weigle et al., 2005). However, neither of these observed health behavior variables adequately explained the relationship between N/NE and heightened cardiometabolic risk, indicating that alternative mechanisms must be responsible for driving the effect between N/NE and cardiometabolic risk.

The relationship between N/NE and cardiometabolic risk may depend on several biological and psychological constructs that were not assessed in the present study. For example, systemic inflammation is a biological process that plays a fundamental role in the pathogenesis of MetS (Dermody et al., 2015; Grundy et al., 2005; Marsland et al., 2010). Although the causes of systemic inflammation are complex and multifactorial (Tracey, 2002), symptoms of depression (Howren, Lamkin, & Suls, 2009; Stewart, Rand, Muldoon, & Kamarck, 2009) and anxiety (Pitsavos et al., 2006) are known to trigger a cascade of metabolic effects that can augment risk for systemic inflammation. Given that N/NE is characteristically associated with a tendency to experience symptoms of depression and anxiety (Cyders & Smith, 2007; Cyders & Smith, 2008b; Ormel et al., 2013; Williams et al., 2018), individuals high on N/NE may be at heightened cardiometabolic risk largely as a consequence of systemic inflammation. In support of this notion, accumulating evidence has documented a positive relationship between measures of N/NE and inflammatory markers (Marsland, Sathanoori, Muldoon, & Manuck, 2007; Sutin, Terracciano, Deiana, Naitza, et al., 2010), with additional findings demonstrating that systemic inflammation partially mediates the relationship between personality and cardiometabolic risk, above and beyond the effects of health behaviors (Dermody et al., 2015). Accordingly, the relationship between N/NE and cardiometabolic risk may be better accounted for by underlying inflammatory processes rather than maladaptive health behaviors.

Alternatively, the interrelationships among N/NE, maladaptive health behaviors, and cardiometabolic risk may depend on the experience of emotional distress. Previous research indicates that individuals high on measures of N/NE are primarily motivated to engage in negatively reinforcing behaviors to avoid or quickly regulate aversive emotional stimuli and are consequently at highest risk of participating in maladaptive health behaviors during periods of acute emotional distress (Cyders & Smith, 2007; Cyders & Smith, 2008b; Fischer et al., 2018; Settles et al., 2012). Laboratory studies confirm these findings by demonstrating that individuals high on measures of N/NE are particularly susceptible to drink alcohol (Owens et al., 2018; VanderVeen et al., 2016), smoke cigarettes (Doran, Cook, McChargue, Myers, & Spring, 2008), and overconsume palatable foods (Becker, Fischer, Smith, & Miller, 2016; Emery et al., 2014) during negative mood or stressful conditions but not during neutral conditions. These findings suggest that N/NE may interact with the experience of emotional distress to more accurately predict who is likely to engage in the maladaptive health behaviors known to promote cardiometabolic risk. Specifically, the presence of an indirect effect of maladaptive health behaviors on the association between N/NE and cardiometabolic risk may vary across different levels of emotional distress, such that individuals high on N/NE may only be at heightened cardiometabolic risk as a consequence of maladaptive health behaviors if they also endorse high levels of emotional distress. Thus, understanding how maladaptive health behaviors contribute to the association between N/NE and cardiometabolic risk may require additional exploration of the synergistic effect between N/NE and measures of emotional distress.

5.2 EXTRAVERSION/POSITIVE EMOTIONALITY AND CARDIOMETABOLIC RISK

Similar to previous research (Dermody et al., 2015; Sutin, Costa, et al., 2010), the present study found that E/PE and cardiometabolic risk were not directly related. However, E/PE was shown to be indirectly associated with cardiometabolic risk through physical activity. Specifically, individuals high on E/PE were at reduced cardiometabolic risk as a consequence of greater engagement in physical activity. This particular finding highlights the importance of examining indirect effects linking distinct impulsigenic traits to cardiometabolic health outcomes despite the absence of significant direct effects (Meule, 2017). Numerous meta-analytic studies have indeed shown that measures of E/PE are the strongest personality predictors of physical activity (Artese, Ehley, Sutin, & Terracciano, 2017; Rhodes & Smith, 2006; Sutin et al., 2016), which itself has been identified as a potent protective factor against MetS (Santos et al., 2007; Zhu et al., 2004). The present study thus integrates and extends these previous lines of research by indicating that, although individuals high on E/PE are not at reduced cardiometabolic risk overall, they are partially protected from cardiometabolic risk due to their tendency to be physically active.

Research has begun to uncover how the unique characteristics of individuals high on measures of E/PE contribute to their increased likelihood of being physically active (Cyders & Smith, 2007; Cyders & Smith, 2008b). The E/PE factor estimated in the present study consisted of questionnaire measures of impulsivity that assess both general personality features and specific impulsive characteristics reflecting a tendency to experience positive emotions and to engage in sensation and novelty seeking behavior. The E/PE factor is therefore best conceptualized as a broad personality trait that underlies a disposition to act rashly in the face of positive emotions (Carver, 2005; Gray, 1970; Whiteside & Lynam, 2001). The propensity for individuals high on measures

of E/PE to engage in positively reinforcing behaviors has been largely attributed to dysfunctional neurocircuitry in frontostriatal systems that enhance reward processing and increase motivation to approach exciting and pleasurable stimuli (Cyders & Smith, 2007; Cyders & Smith, 2008b; Smillie, 2013; Williams et al., 2018). Given that physical activity can produce feelings of euphoria (Anderson & Shivakumar, 2013; Basso & Suzuki, 2017; Boecker et al., 2008; Dinas, Koutedakis, & Flouris, 2011) and activate neural reward networks (Herrera et al., 2016), individuals high on measures of E/PE are theorized to lead active lifestyles because they derive an inflated sense of enjoyment from physical activity (Sutin et al., 2016). In line with this proposition, Lewis and Sutten (2011) found the positive association between measures of E/PE and physical activity to be mediated by a heightened reinforcing value of physical activity. Related evidence shows that measures of E/PE are also negatively associated with sedentary behavior (Sutin et al., 2016), indicating that the proneness for individuals high on measures of E/PE to be physically active further translates into a less sedentary lifestyle. Although the present study did not examine the specific indirect effect of sedentary behavior on the association between E/PE and cardiometabolic risk, it is possible that individuals high on measures of E/PE display additional reductions in cardiometabolic risk as a consequence of their tendency to be less sedentary (Hamilton et al., 2008; Wittink et al., 2011). Taken together, these findings indicate that the preference of individuals high on measures of E/PE to participate in stimulating and rewarding behaviors can lead them to engage in a more physically active lifestyle, and this inclination to be physically active ultimately serves to mitigate their cardiometabolic risk.

Despite the beneficial health effects of physical activity (Santos et al., 2007; Zhu et al., 2004), it is important to acknowledge that measures of E/PE are also frequently associated with behaviors that have deleterious health consequences, including substance use (Moreno-López et

al., 2012; Munafo et al., 2007; Turiano, Whiteman, Hampson, Roberts, & Mroczek, 2012; Walther, Morgenstern, & Hanewinkel, 2012), risky sex (Zapolski, Cyders, & Smith, 2009; Zietsch, Verweij, Bailey, Wright, & Martin, 2009), and pathological gambling (Cyders & Smith, 2008a; Michalczuk, Bowden-Jones, Verdejo-Garcia, & Clark, 2011). The present study indeed found that E/PE was positively related to alcohol use, which itself has been implicated as a prominent behavioral contributor to cardiometabolic morbidity and mortality (Djousse et al., 2004; Sun et al., 2014; Yoon et al., 2004; Zhu et al., 2004). This finding is consistent with the broader literature linking measures of E/PE to heavy alcohol use (Coskunpinar et al., 2013; Hakulinen et al., 2015; Kuntsche, von Fischer, & Gmel, 2008) and lends further support to recent evidence documenting that individuals high on measures of E/PE are particularly likely to engage in both heavy alcohol use and heightened levels of physical activity (Leasure & Neighbors, 2014). Because heavy alcohol use and physical activity generally have opposing effects on cardiometabolic risk, the tendency for individuals high on measures of E/PE to participate in these particular health behaviors may ultimately help to explain why previous research has failed to establish a significant direct effect between measures of E/PE and cardiometabolic risk (Dermody et al., 2015; Sutin, Costa, et al., 2010). Specifically, the strength of the overall relationship between measures of E/PE and cardiometabolic risk may be weakened among individuals high on measures of E/PE because the health-promoting effects of physical activity are dampened by the health-limiting effects of heavy alcohol use. However, it is crucial to note that the present study did not find a specific indirect effect of alcohol use on the association between E/PE and cardiometabolic risk. The present study also did not replicate previous research documenting a curvilinear relationship between alcohol use and cardiometabolic risk (Djousse et al., 2004; Sun et al., 2014; Yoon et al., 2004; Zhu et al., 2004) and instead found alcohol use to be negatively associated with cardiometabolic risk in a

linear fashion. Although this latter finding is unsurprising given the discrepant associations between alcohol use and cardiometabolic outcomes (O'Keefe et al., 2007), these inconsistent results indicate that future work is needed to further clarify whether and how alcohol use contributes to the relationship between E/PE and cardiometabolic risk.

5.3 INHIBITION, IMPULSIVE DECISION-MAKING, AND CARDIOMETABOLIC RISK

The present study further documented that Inhibition and Impulsive Decision-Making were both indirectly associated with cardiometabolic risk through saturated fat intake, but in opposing directions. Specifically, individuals low on Inhibition were at reduced cardiometabolic risk as a consequence of less saturated fat intake whereas individuals high on Impulsive Decision-Making were at heightened cardiometabolic risk as a consequence of greater saturated fat intake. Although these findings confirm the importance of saturated fat intake as a meaningful behavioral indicator of cardiometabolic risk (Hu et al., 2001; Mozaffarian et al., 2010), the divergent patterns of saturated fat intake between individuals low on Inhibition and those high on Impulsive Decision-Making were ultimately inconsistent with expectations, as discussed more fully below.

The Inhibition and Impulsive Decision-Making factors estimated in the present study each comprised a single behavioral task measure of impulsivity. The Inhibition factor was indexed by the Iowa Gambling Task (IGT), which assesses decision-making under risk and uncertainty (Bechara, 2007). Meanwhile, the Impulsive Decision-Making factor was indexed by the Delay Discounting Task (DDT), which assesses a general inability to tolerate reinforcement delays (Mitchell, 1999). Importantly, lower scores on the IGT indicate worse performance and lower

levels inhibitory control whereas higher scores on the DDT indicate worse performance and higher levels of impulsive decision-making. Although these behavioral task measures of impulsivity were modestly correlated in the present study and are largely believed to measure distinct impulsigenic traits (Sharma et al., 2014), they both provide an assessment of decision-making impairments in the context of perceived monetary rewards. Individuals who score low on the IGT or high on the DDT consequently exhibit many overlapping qualities. For example, low IGT and high DDT scores are both associated with neurobiological mechanisms that underlie a vulnerability towards addiction, including heightened reward responsivity and reduced inhibitory control (Appelhans, 2009; Bickel, Jarmolowicz, Mueller, Koffarnus, & Gatchalian, 2012; Lawrence, Jollant, O'Daly, Zelaya, & Phillips, 2009; Li, Lu, D'Argembeau, Ng, & Bechara, 2010). Low IGT and high DDT scores are therefore considered important indicators of addictive behaviors and have been extensively linked to a higher likelihood of engaging in heavy alcohol use (Amlung, Vedelago, Acker, Balodis, & MacKillop, 2017; Kovacs, Richman, Janka, Maraz, & Ando, 2017; MacKillop et al., 2011), substance abuse (Amlung et al., 2017; Barry & Petry, 2008; Grant, Contoreggi, & London, 2000), cigarette smoking (Ert, Yechiam, & Arshavsky, 2013; MacKillop et al., 2011), and pathological gambling (Amlung et al., 2017; Kovacs et al., 2017).

The addictive tendencies of individuals who score low on the IGT or high on the DDT have further been shown to promote palatable food overconsumption (Appelhans, 2009; Appelhans et al., 2011). Palatable foods are defined as heavily processed, calorically dense foods that are high in saturated fat, carbohydrate, and salt content and are similar to other addictive substances in their ability to activate neural reward networks (Avena, 2007). Individuals who score low on the IGT or high on the DDT thus tend to display a general preference for and sensitivity to the rewarding properties of palatable foods (Horstmann et al., 2011; Rasmussen, Lawyer, & Reilly, 2010;

Rollins, Dearing, & Epstein, 2010) and often struggle to inhibit behavioral responses to palatable foods (Appelhans et al., 2011; Pignatti et al., 2006). The propensity for individuals who score low on the IGT or high on the DDT to overconsume palatable foods ultimately confers obesity risk by promoting excess weight (Rotge, Poitou, Fossati, Aron-Wisnewsky, & Oppert, 2017; Yang, Shields, Guo, & Liu, 2018) and inhibiting attempts at weight loss (Brockmeyer, Simon, Becker, & Friederich, 2017; Witbracht, Laugero, Van Loan, Adams, & Keim, 2012). Accordingly, both low IGT and high DDT scores have been identified as salient predictors of the development and persistence of obesity (Emery & Levine, 2017; Rotge et al., 2017; Wu et al., 2016).

Given these previous findings, it has been theorized that individuals who score low on the IGT or high on the DDT may be at heightened cardiometabolic risk as a consequence of habitual palatable food overconsumption that promotes excess adiposity (Chang et al., 2016; Eisenstein et al., 2015). The findings from the present study partially support this hypothesis. Specifically, body mass index was the only component part of cardiometabolic risk positively correlated with scores on the DDT, suggesting that the tendency for individuals who scored high on the DDT to report greater saturated fat intake may be indicative of a proneness towards palatable food overconsumption that enhances cardiometabolic risk largely as a consequence of elevated weight status. However, this pattern of findings was not replicated among individuals who scored low on the IGT. Individuals who scored low on the IGT were instead found to report less saturated fat intake, which indirectly reduced their cardiometabolic risk. IGT scores were also unrelated to both indicators of adiposity included in the present study. Although these particular cardiometabolic outcomes are consistent with what would be expected among individuals whose diets are characterized by low saturated fat intake (Hu et al., 2001; Mozaffarian et al., 2010), it remains

unclear why individuals who scored low on the IGT and those who scored high on the DDT differed in their saturated fat intake.

It is possible that the difference in saturated fat intake between individuals who scored low on the IGT and those who scored high on the DDT is reflective of a broader tendency for such individuals to consume different types of palatable foods. Findings from the present study revealed that individuals who scored low on the IGT and those who scored high on the DDT reported opposite patterns of overall dietary composition. Specifically, individuals who scored low on the IGT reported greater intake of carbohydrates relative to protein, saturated fat, and unsaturated fat. Meanwhile, individuals who scored high on the DDT reported greater intake of saturated fat and unsaturated fat relative to carbohydrates. These differences in dietary composition suggest that individuals who score low on the IGT may prefer palatable foods high in carbohydrates versus fats whereas individuals high on the DDT may prefer palatable foods high in fats versus carbohydrates. However, this proposition remains speculative as the present study did not include a more precise assessment of the type or amount of foods consumed and instead examined the percentage of total energy intake derived from varied macronutrient sources. Although this allows for a general understanding of the relative proportion of calories consumed from different macronutrient sources, future research is needed to replicate and extend these findings to better understand how IGT and DDT scores relate to palatable food selection.

It is also possible that the differences in dietary composition between individuals who scored low on the IGT and those who scored high on the DDT resulted from methodological limitations of the present study. The IGT and DDT were both added late in the parent study, and dietary composition was only assessed among a subset of individuals who participated in the second phase of the parent study. As such, each of these measures had a large percentage of

missing data. Scores for the IGT and the DDT were only available for 44% (n = 575) and 57% (n = 743) of the total sample, respectively, whereas information on dietary composition was only available for 34% (n = 440) of the total sample. Although these missing data were handled using the FIML approach, which is valid for use in structural equation modeling when missing data are present at rates as high as 73% (Schafer & Graham, 2002), the potential for biased estimates has also been shown to increase with increasing rates of missingness under the FIML approach (Enders & Bandalos, 2001). As such, these findings should be considered preliminary and interpreted cautiously until future replication efforts can be conducted.

5.4 IMPLICATIONS AND FUTURE DIRECTIONS

The findings from the present study have several important implications. First, the present study contributes to a growing body of literature documenting the utility of using distinct impulsigenic traits to identify subsets of the population at differential risk for problematic cardiometabolic outcomes (Emery & Levine, 2017; Jokela et al., 2014; Jokela et al., 2013; Shipley et al., 2007; Vainik, Dagher, Dube, & Fellows, 2013). Second, the present study provides foundational knowledge of the unique behavioral pathways through which distinct impulsigenic traits relate to cardiometabolic risk. Third, the present study confirms previous findings showing limited overlap between questionnaire and behavioral task measures of impulsivity (Creswell et al., 2018; Cyders & Coskunpinar, 2011; Duckworth & Kern, 2011; Lane et al., 2003; Reynolds et al., 2006; Sharma et al., 2014), and thereby underscores the importance of using a multidimensional approach when assessing the relationship between impulsivity and varied health outcomes and behaviors.

Although future work is needed to replicate and extend these findings, the present study ultimately helps to clarify both *who* is at cardiometabolic risk and *how* those individuals are at risk.

The findings from the present study are timely given the current interest in using personality traits (Boersma et al., 2011) and related neural characteristics (Appelhans, French, Pagoto, & Sherwood, 2016; Williams et al., 2018) to inform precision medicine interventions for cardiometabolic diseases. The present study indicates that the N/NE, E/PE, Inhibition, and Impulsive Decision-Making factors represent distinct impulsigenic traits that may be particularly relevant for developing personalized approaches to interventions aimed at treating and preventing MetS. Although additional research is needed to further characterize the unique behavioral patterns common to individuals high on these particular impulsigenic traits, the present findings identify physical activity and saturated fat intake as being particularly important behaviors to target when creating personalized intervention plans for such individuals. For example, individuals high on E/PE may be especially likely to respond to and comply with intervention efforts that include a physical activity component (Boersma et al., 2011) whereas individuals low on Inhibition or high on Impulsive Decision-Making may require intervention approaches that have a specific focus on improving dietary habits and limiting palatable food intake (Appelhans et al., 2016).

In addition to modifying intervention approaches to focus on the behavioral tendencies associated with the N/NE, E/PE, Inhibition, and Impulsive Decision-Making factors, research is also needed to determine how best to adjust intervention efforts to address the unique cognitive characteristics of individuals high on these particular impulsigenic traits. Although limited research has attempted to implement cognitive training approaches in the treatment of MetS, a growing body of research has documented initial efficacy of such interventions in the treatment of obesity (Jones, Hardman, Lawrence, & Field, 2017). The majority of this research has focused on

developing cognitive training paradigms to reduce impulsive eating behaviors, such as palatable food overconsumption, to encourage weight loss. Numerous cognitive training strategies have been developed for this purpose, including attention bias modification (Kakoschke, Kemps, & Tiggemann, 2014; Kemps, Tiggemann, & Elford, 2015), inhibition training (Blackburne, Rodriguez, & Johnstone, 2016; Houben & Jansen, 2015), temptation management (Appelhans et al., 2016), distress tolerance skills (Glisenti & Strodl, 2012; Safer, Robinson, & Jo, 2010), and episodic future thinking (Daniel, Stanton, & Epstein, 2013; Dassen, Jansen, Nederkoorn, & Houben, 2016; O'Neill, Daniel, & Epstein, 2016). For example, episodic future thinking involves the practice of vividly imagining a desired future event, such as successful weight loss, and has been shown to be a particularly effective cognitive training strategy for reducing impulsive decision-making around food and promoting dietary adherence among individuals high on delay discounting (Daniel et al., 2013; Dassen et al., 2016; O'Neill et al., 2016). Accordingly, episodic future thinking may be a particularly effective cognitive training strategy to reduce saturated fat intake and lower cardiometabolic risk among individuals high on Impulsive Decision-Making. However, future research is needed to determine whether the cognitive training strategies used in the treatment of obesity can be effectively translated to the treatment of MetS and to further identify the optimal cognitive training paradigms necessary to facilitate behavior change among individuals high on the N/NE, E/PE, Inhibition, or Impulsive Decision-Making factors.

To further clarify the relationship between impulsivity and MetS, it will be of additional importance to understand how the distinct impulsigenic traits examined in the present study interrelate to predict cardiometabolic risk. Emerging models of convergence and divergence between questionnaire and behavioral task measures of impulsivity have documented unique patterns of overlap across diverse constructs of impulsivity (Cyders & Coskunpinar, 2011;

Duckworth & Kern, 2011; Enticott, Ogloff, & Bradshaw, 2006; Hirsh, Morisano, & Peterson, 2008; Suhr & Tsanadis, 2007), and several recent efforts have started to disentangle how these interrelationships influence broader health outcomes and behaviors (Becker et al., 2016; Bongers et al., 2015; Claes et al., 2006; Grant & Langan-Fox, 2006; Meule & Platte, 2015; Naragon-Gainey & Simms, 2017; Sutin, Terracciano, Deiana, Naitza, et al., 2010; Terracciano & Costa, 2004; Terracciano et al., 2008; Turiano, Mroczek, Moynihan, & Chapman, 2013; Vollrath & Torgersen, 2002). For example, research using omnibus personality inventories have demonstrated that measures of Neuroticism interact with measures of Conscientiousness to differentially predict both physical (Grant & Langan-Fox, 2006; Sutin, Terracciano, Deiana, Naitza, et al., 2010; Terracciano & Costa, 2004; Terracciano et al., 2008; Turiano et al., 2013) and psychological (Claes et al., 2006; Naragon-Gainey & Simms, 2017; Vollrath & Torgersen, 2002) health outcomes. Indeed, high levels of Neuroticism are frequently associated with problematic health outcomes and accumulating evidence has found low levels of Conscientiousness to augment this effect (Costa & McCrae, 2008; Rosenström & Jokela, 2017). However, additional work has found this effect to reverse at increasing levels of Conscientiousness, such that individuals high on Neuroticism tend to demonstrate favorable health outcomes when they are also high on Conscientiousness (Turiano, et al., 2013). It has been theorized that high Conscientiousness, which is defined by a greater ability to maintain inhibitory control and engage in planned and thoughtful action, allows individuals high on Neuroticism to more effectively constrain their behaviors, ultimately resulting in more positive health outcomes (Friedman, 2000; Rosenström & Jokela, 2017). Accordingly, although there was no main effect of DvC/C on cardiometabolic risk in the present study, the DvC/C factor may interact with the N/NE factor to differentially predict cardiometabolic risk. In particular, the positive association found between N/NE and cardiometabolic risk may be enhanced at low levels

of DvC/C and mitigated or reversed at high levels of DvC/C. Future work is therefore needed to explore the interactive effects of the distinct impulsigenic traits examined in the present study to further clarify our understanding of the dynamic associations among these constructs and also to refine our knowledge of the complex and synergistic effects of distinct impulsigenic traits on cardiometabolic risk.

5.5 STRENGTHS AND LIMITATIONS

The present study has several notable strengths. First, the present study used data from a large, community sample of midlife adults and was adequately powered to examine whether the relationships between distinct impulsigenic traits and cardiometabolic risk were mediated by varied health behaviors. Second, because the present study included a multidimensional assessment of impulsivity, it was uniquely suited to test the predictive validity of the hierarchical structure of impulsivity identified by Sharma and colleagues (2014) in relation to cardiometabolic risk and varied health behaviors. Third, the primary aims of the present study were tested using exploratory structural equation modeling, which permits the simultaneous estimation of exploratory and confirmatory factors within the same model. This particular approach to testing the primary aims allowed the independent effects of each direct and indirect pathway linking the distinct impulsigenic traits to cardiometabolic risk through varied health behaviors to be established. Finally, the present findings focused on examining modifiable health behaviors that are particularly relevant for the treatment of MetS and thus demonstrate a high degree of clinical relevance.

Despite these strengths, the findings from the present study should be considered in the context of certain limitations. First, the present study was cross-sectional in nature and therefore cannot provide insight into the temporal associations among impulsivity, health behaviors, and cardiometabolic risk. Although structural equation modeling is considered a causal modeling technique (Kline, 2015), future work is needed to replicate these findings using a prospective approach to allow for causal inference among these constructs. Second, although the present study included an extensive number of commonly used questionnaire measures of impulsivity that largely overlapped with those examined by Sharma and colleagues (2014), there were a limited number of behavioral task measures of impulsivity available for analysis. As such, three of the four latent behavioral task factors comprised a single indicator and did not fully correspond with the latent behavioral task factors found by Sharma and colleagues (2014). Additional work is therefore needed to replicate these findings using a more comprehensive battery of behavioral task measures of impulsivity. Third, the health behavior variables examined in the present study were assessed through self-report, which is subject to demand effects that may have caused participants to underreport undesirable behavior and overreport desirable behavior (Grimm, 2010). For example, when compared to objective assessment, individuals have been found to underreport their overall energy intake (Scagliusi et al., 2009) and overreport their engagement in physical activity (Brenner & DeLamater, 2014). Accordingly, future work should aim to replicate these findings using objectively measured health behaviors. Fourth, the present study used a community sample of midlife adults, who were primarily Caucasian and relatively healthy. As such, these findings may not be generalizable to more diverse populations of varying health statuses. Finally, although the present study focused on examining the core health behaviors linked to MetS as mediating variables of the associations between distinct impulsigenic traits and cardiometabolic risk, it will

be important for future research to examine how these particular impulsigenic traits relate to more varied health behaviors as well as additional physical and psychological health outcomes.

6.0 CONCLUSION

The present study is the first to examine whether and how distinct impulsigenic traits relate to cardiometabolic risk. The primary aims of the present study were to provide a fuller understanding of the distinct impulsigenic traits most strongly related to midlife cardiometabolic risk and to identify the unique behavioral mechanisms through which these distinct impulsigenic traits promote midlife cardiometabolic risk. The findings from the present study indicate that N/NE, E/PE, Inhibition, and Impulsive Decision-Making represent distinct impulsigenic traits that can effectively identify subsets of the population at differential cardiometabolic risk. The findings from the present study further identify physical activity and saturated fat intake as being especially important health behaviors to target when tailoring treatment approaches to the unique behavioral characteristics of individuals high on these particular impulsigenic traits. Although future research will be needed to replicate these findings across more diverse samples and to determine the prospective effect of the relationship between these constructs, the present findings ultimately serve to inform personalized approaches to interventions aimed at reducing the prevalence of cardiometabolic diseases.

7.0 BIBLIOGRAPHY

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