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A Structural Equation Model Of Factor Two Psychopathy, Behavioral Activation, Positive Alcohol Expectancies, And Alcohol Use

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A STRUCTURAL EQUATION MODEL OF FACTOR TWO PSYCHOPATHY, BEHAVIORAL ACTIVATION, POSITIVE ALCOHOL EXPECTANCIES, AND ALCOHOL USE

by

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CHAPTER 1 INTRODUCTION

A large body of literature indicates that there is a relationship between psychopathy and heavy drinking. Very few studies, however, have examined potential mediators of this relationship. As a result, it is difficult to develop intervention strategies that target early steps in the psychopathy/alcohol use pathway. The present study tested a structural equation model linking secondary psychopathy to alcohol use through the influence of both behavioral activation and positive alcohol expectancies. The study was also one of the first to utilize both self-report and laboratory measures of behavioral activation.

The Construct of Psychopathy

The construct of psychopathy was first delineated by Cleckley in 1941. Cleckley defined psychopathy in terms of 16 "characteristic points," which include interpersonal (e.g., untruthfulness and insincerity), behavioral (e.g., poor judgment, failure to learn by experience, and inadequately motivated antisocial behavior), and affective (pathologic egocentricity and lack of remorse or shame) features. Moreover, Cleckley stated that unlike traditional psychoses (e.g., schizophrenia) which are characterized by noticeably distorted thought processes (e.g., delusions), psychopathy is characterized by a "mask of sanity," impenetrable to psychiatric inquiry. In other words, psychopaths display antisocial behavior and marked interpersonal/affective deficits while, at the same time, appearing sane and rational.

While descriptive, Cleckley's theory of psychopathy drew criticism for its failure to address differing etiological pathways. Specifically, critics noted that (1) there are multiple pathways to psychopathic behavior (e.g., abusive environments, genetic risk for

antisociality, etc) and (2) these different etiological pathways may predict different subtypes of psychopathy distinguished by affect, behavior and physiology.

One of the first theorists to address this issue was Karpman (1941, 1948b), who proposed that psychopaths could be divided into “primary” and “secondary” subtypes. Karpman (1941, 1948a, 1948b) described primary psychopathy as an innate condition, resulting from a “constitutional” or biological deficit. Primary psychopaths were thought to be ‘cold’ and calculating in their actions while, at the same time, lacking in core emotions, such as anxiety, guilt, and empathy (i.e., emotions which tend to inhibit antisocial behavior). In contrast, “secondary” psychopathy was conceptualized as an externalized reaction to a negative or hostile environment (e.g., one that includes abuse, neglect, trauma, rejection, etc). Unlike primary psychopaths, secondary psychopaths were thought to be impulsive (rather than cold and calculating) and susceptible to (rather than immune to) negative emotions such as anxiety and hostility.

Over the past 60 years, several lines of research have provided empirical support for Karpman’s primary/secondary theory of psychopathy (Lykken, 1957; Hare & Neumann, 2008; Lilienfeld & Andrews, 1996). In particular, studies suggest that primary, but not secondary, psychopaths show (1) a decreased skin conductance response to fear-related cues (e.g., warning signals for or countdowns to shock (Lykken, 1957; Benning, Patrick & Iacono, 2005), (2) a reduced startle eyeblink response in the presence of fear-related cues (i.e., a reduced fear potentiated startle (Patrick, Bradley & Lang, 1993), and (3) a reduced skin conductance response to pictures of other people in distress (Blair, 1999). These findings suggest that primary (but not secondary) psychopaths may have a biologically-based deficit that reduces

their reactivity to inhibitory or fear-related cues. Notably, however, other research suggests that there are certain deficits which are shared by both primary and secondary psychopaths. In particular, both types of psychopaths appear to have a dominant response set for reward. That is, they are excessively motivated by reward and unable to modify their behavior even when contingencies change or rewards become unavailable. For example, Newman, Patterson and Kosson (1987) enlisted both incarcerated psychopaths and nonpsychopaths to complete a card-playing task with monetary rewards and punishments. In total, 100 cards in blocks of 10 were presented to the subjects, who were told to turn over as many cards as they wished. Subjects won 5 cents for every face card turned over and lost 5 cents for every number card turned over. The first block of cards had the lowest probability of punishment (10%), with the probability of punishment increasing at a rate of 10% with each subsequent block of cards (second block of cards-20%, third block of cards-30%, etc.). Results showed that psychopaths turned over significantly more cards than non-psychopaths, suggesting that they were excessively motivated by reward and unable to modify their dominant response set when reward-based contingencies were altered. Similarly, Masui & Nomura (2011) used a three condition (no reward/punishment, low reward/punishment, high reward/punishment) stop-signal task to investigate response inhibition (i.e., the ability to withhold a dominant key-pressing response) in a non-forensic sample of psychopathic and non-psychopathic participants. They found that the presence of both high and low-level rewards and punishments led to greater response inhibition in non-psychopathic participants. In contrast, in participants high on psychopathy, low-levels of rewards and punishments increased response inhibition, while high levels of rewards

and punishment decreased response inhibition. These findings suggest that high levels of reward may activate a dominant approach response set in psychopaths that is inflexible and resistant to punishment.

Reinforcement Sensitivity Theory

The empirical findings described above have been explained by Fowles (1993; 2001) and others in terms of Gray and McNaughton's (2000) reinforcement sensitivity theory (RST). RST is a biologically based theory of personality characterized by three motivational systems: the behavioral activation system (BAS), the behavioral inhibition system (BIS) and the fight/flight/freeze system (FFFS). The BAS is thought to be sensitive to reward cues, and to play a role in regulating approach behavior to appetitive stimuli (e.g., individuals with a weak BAS are less motivated by rewards than those with a strong BAS). On the other hand, the FFFS is believed to be sensitive to punishment cues or aversive stimuli and is associated with escape behavior and fear (e.g., individuals with a weak FFFS are more likely to disregard potential punishment than those with a strong FFFS). The third RST system, the BIS, works to resolve conflicts between the BAS (approach motivations) and the FFFS (avoidance motivations). The BIS weighs the strength of rewards and punishments and activates the appropriate behavioral response (e.g., if rewards outweigh punishments, the BIS will inhibit the FFFS and activate the BAS, resulting in approach behavior).

Over the last 30 years, several researchers (Fowles, 1980; Lykken, 1995) have posited that primary psychopathy is related to a weak FFFS and a normal/high BAS, whereas secondary psychopathy is related to a normal FFFS but a strong BAS. This theory helps to explain the empirical literature cited above (on fear-potentiated startle,

countdown to shock, dominant reward-based response sets, etc) and provides a theoretical framework for future psychopathy research. Moreover, the theory has been supported by research showing that survey measures of BAS and FFFS map onto dimensions of psychopathy, with low FFFS scores generally predicting primary psychopathy and high BAS scores predicting both primary and secondary psychopathy (Wallace, Malterer, & Newman, 2009; Ross, Molto, Poy, Segarra, Pastor & Montanes, 2007).

Notably, most research on RST constructs has been conducted with self-report measures. This is problematic for several reasons. First, self-report measures of BAS, BIS, and FFFS require insight into one's approach and avoidance motivations, as well as insight into to how these motivations compare to norms. For example, Carver's BIS/BAS Scale (the most widely used self-report measure of RST constructs) is comprised largely of insight-oriented items such as, "I have very few fears compared to my friends," and "When I go after something I use a "no holds barred" approach." Second, self-report measures are often affected by social desirability biases. This may be particularly true for secondary psychopaths; i.e – individuals who engage in antisocial behavior, while also experiencing anxiety, and guilt. Finally, BAS, BIS and FFFS are generally conceptualized as fairly complex, multidimensional constructs. Nonetheless, they are almost always measured in a single, relatively narrow way (i.e. with self-report survey measures), In fact, most studies of BAS, BIS and FFFS report associations between self-report measures of RST constructs and self-report measures of other outcomes. As a result, it is not clear whether the results of these studies are affected by shared method variance, or whether different methods of assessing RST tap

into different components of the constructs. To date, only two behavioral measures of RST constructs have been tested multiple times; the Iowa Gambling Task (IGT; Bechara, Damasio, Tranel, & Anderson, 1994) and the Card Arranging Reward Responsiveness Objective Test (CARROT; Powell, Al-Adawi, Morgn, & Greenwood, 1996). Notably, however, studies examining relationships between RST constructs (usually the BAS) and these behavioral tasks have yielded inconsistent results (Goudriaan, Oosterlaan, de Beurs, and van den Brink, 2006; Suhr and Tsanadis, 2007; Davis, Patte, Tweed and Curtis, 2007; Kambouropoulos and Staiger, 2004; Kambouropoulos and Staiger, 2007; Loxton and Dawe, 2007) and there is a need for additional multi-method studies using alternative measures of RST constructs.

Psychopathy and Alcohol Use

One of the most consistent findings to emerge from the psychopathy literature is the fact that there is a strong association between psychopathy and heavy alcohol use. This association has been found in forensic (Smith & Newman, 1990; Walsh, Allen, & Kosson, 2007), community (Neumann & Hare, 2008), and college student (Sylvers, Landfield, & Lilienfeld, 2011) samples and appears to be specific to secondary (rather than primary) psychopathy. For example, Walsh et al (2007) conducted assessments on 399 prison inmates and found that alcohol dependence symptoms were associated with secondary psychopathy, even after controlling for primary psychopathy and persistent criminal behavior. Sylvers et al (2011) administered surveys to 159 college students and found significant relationships between heavy drinking and secondary psychopathy, even after controlling for ASPD symptoms. Neumann and Hare (2008)

assessed psychopathy and alcohol use in a community-based sample of 514 adults and found that a superordinate psychopathy factor predicted past-week frequency of alcohol use. Together, these studies suggest that there is a moderately strong relationship between psychopathy and alcohol use, that is specific to secondary psychopathy and robust to covariates, such as primary psychopathy, antisocial behavior, and criminality.

There are several factors that may account for the relationship between secondary psychopathy and alcohol use. First, the psychopathy/alcohol use relationship may be partially accounted for by shared personality or cognitive correlates, such as impulsivity (Hopley & Brunelle, 2012), negative emotionality (Gudonis, Derefinko, & Giancola, 2009) and short-term memory deficits (Endres, Rickert, Bogg, Lucas, and Finn, 2011). Second, there may be a causal relationship between psychopathy and alcohol use. For example, heavy alcohol use may cause individuals to act in ways that are reckless or impulsive (i.e., traits indicative of secondary psychopathy). Finally, it is possible that alcohol use and externalizing behavior share a common genetic vulnerability (Slutske, Heath, Dinwiddie, Madden, Bucholz, Dunne, Statham, & Martin, 1998). For example, Hicks, Krueger, Iacono, McGue, & Patrick, (2004) examined the familial transmission of externalizing pathology among 542 families participating in the Minnesota Twin Family Study and found both a broad genetic vulnerability to externalizing pathology, as well as a more specific genetic vulnerability to conduct disorder, alcohol dependence, and drug dependence.

Alcohol Use and the Behavioral Activation System

Like psychopathy, heavy alcohol use has been associated with a strong behavioral activation system (BAS) in both undergraduate (Kambouropoulos & Staiger,

2007; O'Conner & Colder, 2005; Pardo, Aguilar, Molinuevo, & Torrubia, 2007; Hundt, Kimbrel, Mitchell, & Nelson-Gray, 2008) and clinical (Franken, 2002) samples. For example, Kambouropoulos and Staiger (2007) found that problem drinking college students scored higher than non-problem drinking students on self-report (but not laboratory) measures of behavioral activation. Franken and Muris (2006) examined associations between substance use and three components of the BAS (fun seeking, drive, and reward responsiveness) and found that BAS fun seeking was associated with quantity of alcohol use, frequency of binge drinking, and number of illegal substances used. O'Conner & Colder (2005) used latent profile analysis to identify five classes of college student drinkers based on quantity of alcohol use, frequency of alcohol use and alcohol-related problems. The authors labeled two of the five classes as problem drinking groups and found that reward sensitivity (an indicator of the BAS) predicted membership in these groups. Notably, studies examining relationships between the FFFS and alcohol use have yielded largely null findings (Johnson, Turner & Iwata, 2003; O'Conner & Colder, 2005; Hundt et al, 2008), despite the hypothesis that a strong FFFS might be associated with negative affect, which might, in turn, lead to self-medication drinking.

Positive Alcohol Expectancies

While the relationship between alcohol use and the BAS is well established, few studies have examined factors that may account for this relationship. Positive alcohol expectancies (PAEs) are one construct that may explain the relationship between the BAS and heavy drinking (Carey, 1995; Leigh & Stacy, 1993; Goldman, Del Boca, & Darkes, 1999). PAEs are beliefs that alcohol use will accomplish a desired goal for the

user and include (1) tension reduction (e.g., “Drinking helps me relax”), (2) social lubrication (e.g., “Drinking makes me feel less shy”), (3) activity enhancement (e.g., “Drinking can be exciting”), and (4) performance enhancement (e.g., “Drinking makes me more creative”) (Goldman, Brown, & Christiansen, 1987; Brown, Goldman, Inn, & Anderson, 1980). PAEs can be acquired both directly (i.e., by drinking) and indirectly (i.e. by watching parents drink, watching alcohol use on TV, etc.). In fact, alcohol expectancies have been repeatedly documented in young children, even those who have never tried alcohol. For example, Dunn & Goldman (1996) investigated alcohol expectancies in elementary school children in grades two through five. They concluded that children, much like adults, form a network of beliefs in their memory about the positive and negative behavioral effects of alcohol, even before initiation of use occurs.

A large body of literature suggests that PAEs are both cross-sectionally and prospectively associated with heavy drinking. For example, Carey (1995) sampled 140 college students at two time points, one month apart, and found that positive alcohol expectancies longitudinally predicted maximum daily quantity of alcohol use, even after controlling for baseline use. More recently, Nicolai, Moshagen, & Demmel (2012) examined alcohol expectancies and alcohol use in relation to age and gender. They found that, among young adults, alcohol use was strongly associated with social assertiveness and sexual enhancement expectancies, whereas, among older adults, alcohol use was associated with tension reduction and impairment expectancies.

Positive Alcohol Expectancies as a Mediator of the BAS/Drinking Relationship

Positive alcohol expectancies may be one potential mediator of the relationship between the BAS and heavy alcohol use. More specifically, individuals with a strong

BAS, who are highly motivated by reward, may be particularly sensitive/attentive to alcohol's reinforcing properties. As a result, these individuals may easily develop positive alcohol expectancies, which may, in turn, lead to frequent heavy drinking (Jones, Corbin, & Fromme, 2001; Corbin, Iwamoto, & Fromme, 2011).

To date, however, only two studies have examined the degree to which alcohol expectancies mediate the relationship between the BAS and alcohol use. In the first study, Lopez-Vergara, Colder, Hawk, Wieczorek, Eiden, Lengua, and Read (2012) assessed 378 families at two time points, separated by one year. At time 1, caregivers rated their children (ages 10-12 years old) on BIS, FFFS, and 3 facets of BAS (drive, social approval, and impulsivity/ fun seeking). Moreover, at times 1 and 2, children completed surveys assessing both positive and negative alcohol expectancies, as well as past year alcohol use. Results revealed indirect paths between both BAS drive and FFFS and time 1 alcohol use, through positive alcohol expectancies. Contrary to predictions, there was also an indirect path between BAS drive and time 2 alcohol use through negative alcohol expectancies. Finally, there was a 3-step, indirect path from BAS drive to time 2 alcohol use, whereby BAS drive predicted positive alcohol expectancies, which predicted time 1 alcohol use, which predicted time 2 alcohol use. In total, these findings suggest that ideas about alcohol and subsequent use are strongly shaped by personality characteristics such as Drive, and Fear/Shyness.

In the second study, Wardell, Read, Colder, & Merrill (2012) analyzed relationships between three components of the BAS (fun seeking, drive and reward responsiveness), four types of PAEs (tension reduction, social lubrication, activity enhancement, and performance enhancement) and both quantity and frequency of

alcohol use in a longitudinal sample of college students. Results revealed that (1) BAS Fun-Seeking prospectively predicted both tension reduction and activity enhancement PAEs, (2) the activity enhancement PAE predicted both quantity and frequency of drinking and (3) there was an indirect path between BAS fun seeking and both alcohol quantity and alcohol frequency through the activity enhancement PAE.

While both studies demonstrate promise in determining paths to alcohol use, they have not been replicated and thus, the extent to which results will generalize to other populations is unclear. Moreover, these studies relied solely on self-report measures of BAS, which may be hampered by lack of insight or concerns about social desirability.

The Current Study

A growing number of studies suggest that secondary psychopathy is strongly associated with alcohol use; however, few studies have investigated mediators of this relationship. The current study examined two potential mediators of the secondary psychopathy/alcohol use relationship: 1) the behavioral activation system and 2) positive alcohol expectancies. Moreover, the current study expanded upon attempts to 11perationalized behavioral activation by utilizing both self-report and laboratory measures of the construct. More specifically, the current study used structural equation modeling to examine 1) the degree to which secondary psychopathy, behavioral activation, and positive alcohol expectancies directly predicted quantity and frequency of alcohol use and 2) the degree to which both behavioral activation and positive alcohol expectancies mediated the relationship between secondary psychopathy and alcohol

use. Models included gender, short-term memory, and primary psychopathy as covariates.

Based on theory and previous research, we hypothesized significant, direct paths from all three predictor variables (i.e. secondary psychopathy, behavioral activation and positive alcohol expectancies) to alcohol use and from secondary psychopathy to positive alcohol expectancies. Additionally, we hypothesized a significant indirect path from secondary psychopathy to alcohol use through both the behavioral activation system and positive alcohol expectancies. (See Figure 1).

CHAPTER 2 METHOD

Participants

Two hundred and two college undergraduates were recruited from an upper level psychology course at Wayne State University. Of these 202 participants, 196 completed all study measures. Participants ranged in age from 19-43 with a mean age of 23.74 (*SD* = 3.81). Participants were predominantly female (66.8%) and were ethnically diverse; 46.4% identified as Caucasian/White, 17.9% identified as African American/Black, 16.8% identified as Arab/Chaldean, 8.7% identified as South Asian, 2.6% identified as Hispanic/Latino, 1.5% identified as East Asian, 0.5% identified as American Indian, and 5.6% identified as 'Other.'

Measures

Demographic information. Participants were asked to report their age, gender, and ethnicity.

Psychopathy. The Psychopathic Personality Inventory-Revised (PPI-R; Lilienfeld & Widows, 2005) is a 154-item, self-report measure that can be used to assess the

continuum of psychopathic personality traits in student and community samples. Items are rated on a 4-point scale (1 = false, 2 = mostly false, 3 = mostly true, 4 = true) and cover both the behavioral and affective components of psychopathy. The PPI-R yields a total score and two factor scores (Fearless Dominance [which reflects primary psychopathy], and Self-centered Impulsivity [which reflects secondary psychopathy]). The PPI-R has demonstrated sufficient reliability and construct validity and is highly correlated with similar measures of psychopathy (Lilienfeld, & Widows, 2005; Marcus, Fulton, & Edens, 2012; Poythress et al., 2010; Ray, Weir, Poythress, & Rickelm, 2011). Internal reliabilities for Factor One Psychopathy (FOP) and Factor Two Psychopathy (FTP) in the current sample were excellent (α = .90).

Behavioral Activation. Behavioral activation was assessed using both a self-report measure and a laboratory measure. The BAS scale (Carver & White, 1994) was used as the self-report measure. The BAS scale is a 13-item scale consisting of 3 subscales: Drive (4 items; e.g., “When I go after something I use a “no holds barred” approach”), Fun-Seeking (4 items; e.g., “I will often do things for no other reason than that they might be fun”), and Reward Responsiveness (5 items; “When I see an opportunity for something I like I get excited right away”). These 13 items were rated on a 4-point scale (1 = very true for me, 2 = somewhat true for me, 3 = somewhat false for me, 4 = very false for me). The BAS scale has shown adequate test-retest reliability and has been found to be a valid indicator of sensitivity to appetitive stimuli (Carver & White, 1994). Internal reliability for the overall BAS score in the current sample was good (α = .84).

The Columbia Card Task (CCT; Figner, Mackinlay, Wilkening, & Weber, 2009) is a computerized laboratory task that has been associated with the BAS (Penolazzi, Gremigni, & Russo, 2012). The CCT consists of two versions, the hot version and the cold version. Only the hot version of the CCT was used in this study. The hot version of the CCT presents participants with a total of 32 cards that are face down. Cards are either gain cards (i.e., cards associated with gaining points) or loss cards (i.e., cards associated with losing points). Participants are instructed to turn over cards, one at time, with the goal of maximizing their point total (which is continuously displayed at the top of the computer screen). Additionally, at the beginning of each trial, participants are provided with three game parameters to facilitate their decision making: Gain Amount (the value of the winning cards), Loss Amount (the value of the losing cards) and Loss Probability (the number of losing cards). For example, participants might be told that (a) gain cards result in a gain of 30 points, (b) loss cards result in a loss of 250 points, and (c) 3 of the 32 cards are loss cards.

Each time participants select a gain card, the appropriate number of points is added to their total (i.e. if a 30 point card is selected, 30 points are added to the participant's total). However, as soon as participants select a loss card, the trial is automatically terminated and the loss amount associated with the card is subtracted from the participants' total. Participants can choose to stop turning over cards at any time during a trial and receive their payoff. In an attempt to accurately capture participants' voluntary stopping behavior, the task is rigged such that the losing cards are always placed at the end of the last row of cards (a spot participants do not generally select from at the beginning of a trial). Additionally, to keep participants from

noticing that the task is rigged, some trials are presented (but not included in analyses) in which the loss cards are placed in the first row of the display (a spot participants often select from at the beginning of a trial). These task modifications were utilized by both Figner et al (2009) and Penaolazzi et al (2012).

In total, participants were randomly administered 33 trials of the hot CCT in random order (27 experimental trials and 6 rigged trials). Each participant was administered 3 trials of all possible contingencies. That is, three gain amounts (10, 20, or 30 points per winning card), 3 loss amounts (250, 500, or 750 points per losing card), and three loss card probabilities (1, 2, or 3 loss cards). The outcome variable that was utilized in this study was the average number of cards turned over across the 27 experimental trials.

Positive Alcohol Expectancies. Positive Alcohol Expectancies (Kushner, Sher, Wood, & Wood, 1994) were assessed with 35 items rated on a 4-point scale (1 = Rarely, 2 = Sometimes, 3 = Often, 4 = A lot). These items make up four subscales: Tension reduction (9 items; e.g., “Drinking helps me to relax”), Social lubrication (8 items; e.g., “Drinking makes me feel less shy”), Activity enhancement (9 items; e.g., “Drinking can be exciting”), and Performance enhancement (9 items; e.g., “Drinking makes me more creative”). This scale was empirically derived by Kushner et al (1994) from longer alcohol outcome expectancy measures in order to briefly assess a wide range of positive alcohol expectancies. While Kushner et al (1994) initially identified 12 a priori domains, the four domains listed above were selected as a more parsimonious model with moderate goodness-of-fit. Overall, this scale demonstrates adequate

psychometric properties (Kusher et al., 1994). Internal reliability for the overall PAE scale in the current sample was excellent (α = .95).

Alcohol Use. Quantity and frequency of alcohol use (AU) was assessed with the following two questions: “In the past year, how often have you had some type of beverage containing alcohol?” and “In the past year, when you drank, how many drinks did you usually have on one occasion?” The bivariate correlation between these two items in the current sample was .64

Short-term Memory. The N-Back Task (Jaeggi, Buschkuehl, Perrig, & Meier, 2010) was used as a measure of short-term memory (STM). Participants were shown stimuli (8 shapes in total) and asked to respond (i.e. press a key) if the current stimulus matched the stimulus seen *n* times back. *N* times back refers to zero, one, two, or three stimuli back in the sequence of stimuli presented to a participant. For example, if it is a 2-back trial, the participant would be shown a sequence of shapes and would only respond if the current stimulus was seen two stimuli back in the sequence. Overall, participants were administered 3 blocks of the four trial types (0-back, 1-back, 2-back, 3-back) for a total of 12 trials, which were presented in random order. Participants were given practice trials for each type of trial prior to completing the actual task. The variable yielded from this task is the total number of false hits subtracted from the total number of correct hits divided by the total number of trials ([CH – FH]/12).

Procedure

After gaining approval from the Wayne State University Institutional Review Board (IRB), the principal investigator (PI) went to 17 sections of an upper level psychology course one week before study administration and informed students of the

opportunity to participate in a study on personality factors related to alcohol use. Students were told that the study would be administered in group format and would take place in their classroom, immediately after their regularly scheduled class. Students were also told that they would be given psychology credit for participating.

The study was administered to students in groups of 5-14. There were at least two researchers present at each group administration to monitor study procedures and answer questions. Immediately after class on the day of the study, students were reminded about the study, told their participation was voluntary and anonymous, and given five minutes to decide whether or not they wanted to participate (the PI and his research assistants left the room so as not to coerce students into participation with their presence). After five minutes, the PI and his research assistants reentered the classroom and verified that the individuals present were interested in participating in the study. After verification, students were sent a hyperlink containing the study's self-report measures. When the surveys were completed, participants were instructed to complete both computerized tasks (the CCT and the N-Back Task). Before beginning the CCT (the last task completed for the study), participants were informed that they could win money for their participation. Once all study measures were completed, students were assigned psychology course credit for their participation and paid between one and three dollars for their performance on the CCT.

Analytic Strategy

To test our hypotheses two structural equation models were specified. Both models contained four latent variables. Model 1 contained FTP (factor two psychopathy), BAS (survey assessment of the behavioral activation system), PAE

(positive alcohol expectanices), and AU (alcohol use). Model 2 contained FTP, CCT (Columbia Card Task), PAE, and AU. In each model, FOP (factor one psychopathy), STM (short-term memory), and GEN (gender) were included as manifest control variables. Because FTP, BAS, and PAE are multidimensional scales, domain representative parceling (DRP) was used (Little, Rhemtulla, Gibson, & Schoemann, 2013). This technique allows a multidimensional scale to be transformed into a single latent variable via parcels. Parcels are composed of items from each subscale such that each parcel represents the entire domain of the multidimensional scale. Little (2013) has argued that it is best to use three parcels per latent variable. Therefore, in the current study, FTP, BAS, and PAE were indicated with three parcels apiece, each parcel representing the entirety of the construct it was meant to measure (e.g., FTP parcels contained items from each of its subscales as did parcels indicating BAS and PAE). AU was indicated with two items (quantity of alcohol use and frequency of alcohol use). CCT was indicated with only one item (average number of cards turned over across the 27 experimental trials), thus the error variance was set to zero.

Before proceeding with the structural models, we first verified our measurement models through confirmatory factor analyses (CFA) and checked their factor correlations and factor loadings (see Figures 1 and 2). Once measurement model fit was determined to be satisfactory, model 1 was tested with BAS and PAE as mediators of FTP and AU. This model contained direct paths from all three predictor variables to AU, and a direct path from FTP to PAE (See Figure 3). Model 2 was then tested with CCT and PAE as mediators of FTP and AU with direct paths from all three predictor variables to AU, and a direct path from FTP to PAE (See Figure 4).

Both models were specified with Lisrel 8.80 (Jöreskog and Sörbom, 2006). Covariance matrices were used to evaluate the models. While missing data were present, each item (after deleting six cases; discussed below in data screening) was only missing between 0%-1.0% of responses. Thus, mean imputation was used and missing data were assumed to be random. Model fit was assessed such that an RMSEA < .06, a CFI > .95, and an NNFI > .95 indicate a good model fit as recommended by Hu and Bentler (1999) and Browne and Cudeck (1992).

CHAPTER 3 RESULTS

Data Screening

After obtaining an overall sample of 202 participants, six were deleted due to incomplete study measures (i.e., missing more than 10% of responses). Data screening was then conducted on the 196 remaining participants. First, all variables were examined for out of range values. No values were out of range and all means and standard deviations were plausible. Additionally, the coefficient of variation (*SD*/*M*) for each variable was well above .001, which suggests adequate variance in responses for each item (Tabachnick & Fidell, 2007). Next, skewness and kurtosis were evaluated in order to assess normality of the primary variables. With the exception of the CCT and one subscale of PAE (performance enhancement), all variables fell within acceptable skew and kurtosis ranges (i.e., between -1 and +1; see Table 1). CCT was highly negatively skewed (-2.05) and leptokurtic (3.95). Per Tabachnick & Fidell (2007), CCT was reflected and logarithmically transformed such that skew and kurtosis improved and fell into acceptable ranges (.625 and -.441, respectively). Performance enhancement was highly positively skewed (2.98) and leptokurtic (9.30). Per Tabachnick & Fidell

(2007), performance enhancement was logarithmically transformed such that skew and kurtosis were somewhat improved (2.34 and 5.07, respectively). Univariate outliers were examined by standardizing primary variables into z-scores. Per Tabachnick & Fidell (2007), scores falling three standard deviations above or below the mean were labeled as outliers. PAE was the only variable to contain univariate outliers. In total, four cases fell more than three standard deviations outside of the mean. However, these cases were retained because they did not appear to influence any analyses. Multivariate outliers were subsequently examined by entering all relevant variables into a multiple regression and using subject identification number as the dependent variable. Mahalanobis Distance was then evaluated to determine the presence of multivariate outliers. Two cases appeared to be multivariate outliers but were retained because they did not affect results.

Descriptives and Bivariate Associations

Table 1 shows the descriptive and bivariate associations between all study variables overall. Tables 2 and 3 show descriptive statistics and bivariate correlations among all primary study variables before and after parceling. Overall, the correlations between variables were as expected. Strong positive correlations were seen among the subscales comprising the variable total score and the indicators comprising the latent variables (i.e., the three parcels that make FTP correlated highly with one another as did the three parcels making up BAS and PAE). AU showed strong positive correlations with PAE but only weak to moderate positive relationships with FTP and BAS. FTP and BAS were moderately correlated. The CCT was unrelated to nearly every variable.

Latent Variable Analysis

Model 1: Factor Two Psychopathy, Behavioral Activation System, Positive Alcohol Expectancies, and Alcohol Use

Before testing the structural paths of Model 1, a 4-factor confirmatory factor analysis was run with FTP, BAS, PAE, and AU (See Figure 5). Model identification was achieved by setting factor variances to 1.0. The model showed good overall fit: χ^2 (38) = 65.08, p = 0.004, RMSEA = .057 (90% CI = .03 - .082), CFI = .99, NNFI = .98). All indicators loaded significantly onto their respective factors (see Table 2). Squared multiple correlations ranged from .49 - .90 suggesting that nearly all indicators reliably assessed their respective construct. Factor correlations ranged from .23 to .61 indicating sufficient discriminant validity such that each factor appeared to be related to the other factors, while still assessing a distinct domain. Because model fit was good, all factor loadings were significant and reliable, and construct validity was demonstrated, the structural model was then assessed.

The structural equation model specified (1) direct paths from all three predictor variables (FTP, BAS and PAE) to AU, (2) a direct path from FTP to PAE, (3) an indirect path from FTP to AU through BAS and PAE and (4) an indirect path from BAS to AU through PAE. Additionally, factor one psychopathy (FOP), short term memory (STM), and gender (GEN) were used as control variables (see Figure 6). Model identification was achieved by setting a referent factor loading to 1.0 for each endogenous variable.

Overall, the sample data fit the model well: χ^2 (59) = 101.73, p < .001, RMSEA = .057 (90% CI = .035 -. 077), CFI = .98, NNFI = .97). All of the estimated indicators loaded significantly onto their respective factors (see Table 3). In total, 39% of the variance in AU was explained by FTP, BAS, and PAE. Factor one psychopathy was

positively related to FTP and BAS, whereas short term memory was negatively related to FTP. All other relationships between latent and control variables were found to be non-significant. There were significant direct paths from FTP to both BAS (β = .23, p < .001) and PAE (β = .28, p < .001). There were also significant direct paths from BAS to PAE (β = .29, p < .001) and from PAE to AU (β = .61, p < .001). Additionally, there were significant indirect paths from FTP to both PAE (β = .06, p < .05) and AU (β = .21, p < .001) and from BAS to AU (β = .17, p < .01). Overall, the indirect effect of FTP on AU was significant (η = .21, p < .001), and accounted for 100% of the variance in the FTP/AU relationship.

Model 2: Factor Two Psychopathy, Columbia Card Task, Positive Alcohol Expectancies, and Alcohol Use

Before testing the structural paths of Model 2, a 4-factor CFA was run with FTP, CCT, PAE, and AU (See Figure 7). Model identification was achieved by setting factor variances to 1.0 as well as setting CCT error variance to 0 (because CCT only had one indicator). The model showed good overall fit: χ^2 (22) = 28.71, p = 0.15, RMSEA = .035 (90% CI = 0.0 - .73), CFI = .99, NNFI = .99). All indicators loaded significantly onto their respective factors (see Tables 4). Squared multiple correlations ranged from .48 - .90 suggesting that nearly all indicators reliably assessed their respective construct. Factor correlations among FTP, PAE, and AU ranged from .22 to .61, which again indicated sufficient discriminant validity among these constructs. However, CCT showed non-significant factor correlations with FTP, PAE, and AU, suggesting that it is largely unrelated to those constructs. Because model fit was good, all factor loadings were

significant and reliable, and construct validity was sufficient, the structural model was then assessed.

The second structural equation model specified (1) direct paths from all three predictor variables to AU, (2) a direct path from FTP to PAE, (3) an indirect path from FTP to AU through CCT and PAE and (4) an indirect path from CCT to AU through PAE. Additionally, factor one psychopathy (FOP), short term memory (STM), and gender (GEN) were used as control variables (see Figure 8). Model identification was achieved by setting a referent factor loading to 1.0 for each endogenous variable. Overall, the sample data fit the model well: χ^2 (37) = 58.37, p = .014, RMSEA = .049 (90% CI = .015 - .076), CFI = .99, NNFI = .97). All of the estimated indicators loaded significantly onto their respective factors (see Table 5). In total, 39% of the variance in AU was explained by FTP, CCT, and PAE.

Factor one psychopathy was positively related to FTP, whereas short term memory was negatively related to FTP and positively related to CCT. All other relationships between latent and control variables were found to be non-significant. There were significant direct paths from FTP to PAE (β = .35, p < .001) and from PAE to AU (β = .60, p < .001). Additionally, there was a significant indirect path from FTP to AU (β = .20, p < .001) but not from FTP to PAE (β = .00, p > .001) or CCT to AU (β = -.01, p > .05). Overall, the indirect effect of FTP on AU was significant (η = .20, p < .001), and accounted for 100% of the variance in the FTP/AU relationship.

CHAPTER 4 DISCUSSION

Results from this study indicate that both behavioral activation (as measured by self-report) and positive alcohol expectancies fully mediate the relationship between

factor two psychopathy and alcohol use. This is one of the first studies to examine pathways through which secondary psychopathy can increase heavy drinking. Additionally, this is the first study to examine both personality characteristics (i.e., BAS) and a specific cognitive set (i.e., PAE) as potential mediators of the factor two psychopathy/alcohol use relationship. Results suggest that individuals with secondary psychopathy may be highly motivated by reward and more attentive to the rewarding properties of alcohol. This heightened reward responsiveness may, in turn, facilitate the development of positive alcohol expectancies which may lead to increased alcohol use.

Results of this study have important implications for clinical intervention. Psychopathy (and associated problem behaviors) has traditionally been conceptualized as an intractable disorder, characterized by stable, unmodifiable personality characteristics (e.g. impulsivity, aggressiveness). This widely held view dates back to Cleckley who, in his classic book, "The Mask of Sanity" stated:

> I have had the opportunity to see patients of this sort who were treated by psychoanalysis, by psychoanalytically oriented psychotherapy, by group and by milieu therapy, and by many other variations of dynamic method…some…were treated for years…None of these measures impressed me as achieving successful results…we do not at present have any kind of psychotherapy that can be relied upon to change the psychopath fundamentally. (pp. 438-439).

While recent empirical evidence has yielded more hopeful results regarding the efficacy of psychopathy treatment (Salekin, 2002; Wong & Hare, 2005; Harris & Rice, 2006; Polaschek & Daly, 2013), research in this area has been extremely limited and

there remains a widespread belief that attempts to initiate behavior change within psychopathic populations will be unsuccessful. Results of the current study stand in contrast to this belief and highlight an important motivational/cognitive pathway that may be a target for intervention in heavy drinking psychopaths. More specifically, results suggest that modifying positive beliefs about alcohol in reward-motivated psychopaths may lead to meaningful reductions in drinking.

Notably, there have been a number of recent attempts to develop alcohol expectancy interventions or laboratory experiences that challenge participants to differentiate between expected and actual (pharmacological) effects of alcohol. In a typical 'expectancy challenge,' participants are (1) put into small groups, (2) given alcohol or placebo, (3) told to interact with the other members of their group, and (4) asked to guess who in the group had consumed alcohol. Participants' 'guesses' are often incorrect and form the basis of a group discussion about the pharmacological versus expected effects of alcohol. Notably, existing literature suggests that these expectancy interventions are effective in both changing beliefs about alcohol and in reducing alcohol consumption. For example Lau-Barraco & Dunn (2008) compared a two-hour expectancy challenge (EC) session with two control conditions (alcohol education and assessment only) in a sample of moderate to heavy drinking college students. Results indicated that students in the EC condition decreased their positive alcohol expectancies and their alcohol consumption significantly more than students in the control condition. Similarly Fried & Dunn (2012) randomly assigned fraternity members to an EC or a control condition. Participants in the EC condition watched a scientific video about the pharmacological effects of alcohol along with four alcohol

advertisements. The participants then discussed the contradictions between the two videos as a means of challenging alcohol expectancies. Participants in the control condition watched a presentation that deconstructed advertisements about personal appearance. Results indicated that compared with the control group, the EC group showed significant reductions in average drinking days during the week, number of drinks consumed per sitting, and amount of binge drinking days at a one-month follow up. In sum, alcohol expectancy interventions appear to be effective in reducing drinking and adapting this type of intervention for individuals with psychopathy may be useful in reducing alcohol use and alcohol-related consequences.

One unexpected finding from the current study involved the non-significant relationships between the Columbia Card Task and all other study variables. There are several possible explanations for these null findings. First, the CCT and the BAS could be tapping into different types of reward responsiveness. The BAS questionnaire consists of 13 questions related to perceptions of personality and desire for stimulation across a wide range of situations. In contrast, the CCT assesses behavior in a narrow, time-limited situation. Thus, individuals who are excited and energized by rewards in some situations (e.g. winning a sports contest) may not feel the same way when given the opportunity to win money in a constrained laboratory task. Second, methodological problems may have distorted results. Specifically, the CCT was administered in group format. Although precautions were taken to ensure adequate participant engagement in the task, it is possible that less than optimal effort played a role in the non-significant findings. Third, the type of statistical analysis used to examine the CCT may not have been appropriate. The CCT was treated as a latent variable despite it being indicated

by only one observed variable. This was taken into account by setting the error variance to 0, however, by doing this, the main advantage of utilizing SEM (accounting for error variance) was lost. Moreover, analyses did not examine interactions between CCT contingencies (e.g. gain amount, number of loss trials, etc) and psychopathy status. Thus, it is possible that certain CCT contingencies had different effects on people high versus low in psychopathy. For example, an increase in card 'gain amount' may have affected people high in psychopathy more than people low in psychopathy. Lastly, due to time constraints, the version of the task administered in this study utilized only half the number of trials originally administered by Figner et al (2009). The reduced number of rigged trials may have led the participants to recognize a pattern in the task, allowing them to choose more cards than they would have otherwise. While Figner et al (2009) accounted for this in their experiment, we did not and it may have adversely impacted our findings. Overall, the literature on laboratory tasks of behavioral activation has been inconsistent and more work needs to be done to examine patterns of concurrent and predictive validity among these tasks.

Other Findings

In addition to the main findings, results of note were found with two covariates, factor one psychopathy (FOP) and short-term memory (STM). In model 1, FOP was positively related to FTP and BAS but unrelated to AU. These findings are consistent with the literature on psychopathy, reinforcement sensitivity theory, and alcohol use. FOP and FTP are widely regarded as related constructs (Hare & Nuemann, 2008) and FOP is thought be characterized by normal to high BAS (Fowles, 1980; Lykken, 1995). Additionally, the non-significant relationship between FOP and AU replicates the finding

that FTP is more related to AU than FOP (Walsh et al., 2007; Sylvers et al., 2011). These findings were replicated in model 2 with the exception of the BAS finding because BAS was not included in model 2.

It should also be noted that STM was negatively related to FTP in both model 1 and 2. This is consistent with previous studies that have found working memory deficits in individuals exhibiting externalizing psychopathology (Endres et al., 2011). Specific to model 2, STM was positively related to the CCT, suggesting that working memory may play a role in task performance (and should be treated as a covariate in future CCT studies). Overall, the findings related to FOP and STM are consistent with the literature and add to the validity of our model.

Limitations

Despite the strengths of this study, there are some limitations that need to be addressed. First, data are cross-sectional and, therefore, cannot provide information about causality. Moreover, it is likely that (at least some) relationships between our constructs are bi-directional. For example, while beliefs about alcohol may influence consumption patterns, engaging in heavy drinking may in turn influence alcohol expectancies. Second, testing was conducted in group format. While every precaution was taken to ensure adequate engagement in testing (e.g., at least three researchers for every group), it is not as ideal as individual testing. Third, we only assessed quantity and frequency of alcohol use. In the future, variables such as heavy alcohol use or alcohol-related problems would be useful to include in order to examine whether this model is relevant to more pathological forms of drinking. Fourth, we did not assess negative alcohol expectancies (NAE). It may be useful to do so in the future to examine

whether or not NAE can serve as protective factors against use in this population.

Conclusion and Future Directions

In conclusion, this study contributes to the literature on psychopathy and alcohol use by elucidating a pathway (i.e., BAS and PAE) to alcohol use for people high on the trait of FTP. Insight into this pathway may provide guidance in the treatment of alcohol use problems in individuals with FTP, which is vital for many reasons. First, targeting FTP for change is a daunting task that has been met with limited success (though recent studies have been somewhat more promising; e.g. Conrod et al, 2006). Therefore, targeting cognitive sets like PAE, which have been shown to be amenable to change in other populations, may prove more fruitful. To date, no studies have tested alcohol expectancy challenges in psychopathic populations and research is needed to investigate this potential intervention strategy. All in all, future research should 1) focus on the replication of this study's model, 2) include negative alcohol expectancies and pathological drinking outcomes, and 3) investigate potential interventions.

Table 1

Descriptive statistics and bivariate correlations for FTP, BAS, CCT, PAE, and AU total scores

	1	2	3	4	5	6
1. FTP	1.00					
2. BAS	.30**	1.00				
3. CCT	-.01	-0.05	1.00			
4. PAE	.32**	.32**	0.06	1.00		
5. AU1	.18*	.23**	0.13	.55**	1.00	
6. AU2	.19**	.14*	0.00	.42**	.64**	1.00
Mean	135.18	42.11	0.55	45.98	4.28	3.67
SD	22.00	5.62	0.33	16.61	2.38	2.36
Skewness	0.30	-0.10	0.63	0.82	0.07	0.87
Kurtosis	-0.54	-0.62	-0.44	0.07	-1.20	0.26

Note. $N = 196$. FTP = Factor two total score; BAS = Behavioral activation total score; CCT = Columbia card task score; PAE = Positive alcohol expectancy total score; AU1 and AU2 = Alcohol use frequency and quantity

* $p < .05$, ** $p < .01$

Table 2

Descriptive statistics and bivariate correlations for subscales of primary study variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. FTPME	1.00													
2. FTPRN	.46**	1.00												
3. FTPBE	.40**	.27**	1.00											
4. FTPCN	.35**	.31**	0.07	1.00										
5. BASD	.44**	.26**	.24**	-0.14	1.00									
6. BASFS	.38**	.50**	.23**	0.11	.60**	1.00								
7. BASRR	.14*	0.04	0.13	-.30**	.59**	.47**	1.00							
8. CCT	0.05	-0.03	-.15*	0.09	-0.03	-0.05	-0.06	1.00						
9. PAETR	.35**	.23**	0.09	0.06	.20**	.28**	.26**	0.07	1.00					
10. PAESL	.38**	.27**	0.12	0.12	.14*	.23**	.22**	0.08	.78**	1.00				
11. PAEAE	.33*	.28**	0.08	0.02	.23**	.28**	.25**	0.02	.78**	.73**	1.00			
12. PAEPE	.40**	.30**	.32**	0.13	.21**	.26**	0.06	-0.03	.44**	.53**	.46**	1.00		
13. AU1	.19**	.25**	-0.04	0.09	.17*	.28**	0.09	0.13	.51**	.42**	.57**	.30**	1.00	
14. AU2	.21**	.20**	0.08	0.02	.14*	.18*	0.00	0.00	.38**	.34**	.44**	.26**	.64**	1.00
Mean	41.08	32.98	31.08	32.35	11.93	12.10	17.62	0.55	15.32	13.32	16.33	1.01	4.28	3.67
SD	9.03	7.89	7.43	7.50	2.52	2.51	2.01	0.33	6.42	5.44	6.15	0.10	2.38	2.26
Skewness	0.38	0.42	0.43	0.44	-0.32	-0.27	-0.59	0.63	1.03	0.88	0.62	2.34	0.07	0.87
Kurtosis	-0.47	-0.21	-0.50	-0.37	-0.50	-0.38	-0.20	-0.44	0.38	-0.22	-0.47	5.06	-1.20	0.26

Note. N = 196. FTME, FTRN, FTBE, and FTCN = Factor two Machiavellian egocentricity, Rebelious nonconformity, Blame externalization, and Carefree nonplanfulness; BASD, BASFS, BASRR = Behavioral activation system Drive, Fun seeking, and Reward Responsiveness; CCT = Columbia card task; PAETR, PAE SL, PAEAE, and PAEPE = Positive alcohol expectancy Tension reduction, Social lubrication, Activity Enhancement and Performance enhancement; AU1 and AU 2 = Alcohol use frequency and quantity; * $p < .05$, ** $p < .01$

Table 3

Descriptive statistics and bivariate correlations for primary study variables after parceling

	1	2	3	4	5	6	7	8	9	10	11	12
1. FTP1	1.00											
2. FTP2	.75**	1.00										
3. FTP3	.75**	.74*	1.00									
4. BASP1	.24**	.15*	0.13	1.00								
5. BASP2	.41**	.29**	.28**	.73**	1.00							
6. BASP3	.32**	.21**	.21*	.73**	.68**	1.00						
7. CCT	-0.01	-0.02	0.00	-0.08	0.00	-0.06	1.00					
8. PAEP1	.35**	.26**	.34**	.30**	.29**	.23**	0.06	1.00				
9. PAEP2	.29**	.23**	.32**	.32**	.27**	.24**	0.04	.88**	1.00			
10. PAEP3	.34**	.26**	.33*	.31**	.28**	.27**	0.04	.89**	.90**	1.00		
11. AU1	.19**	0.12	.19**	.19**	.23**	.20**	0.13	.54**	.55**	.48**	1.00	
12. AU2	.18*	0.14	.19**	0.09	.17*	0.12	0.01	.41**	.45**	.37**	.64**	1.00
Mean	44.25	45.83	47.41	13.39	12.54	16.18	0.55	17.41	19.14	18.39	4.48	3.67
SD	8.89	7.47	8.11	1.87	2.04	2.34	0.33	6.25	6.82	6.00	2.38	2.26
Skewness	0.30	0.24	0.21	-0.37	.68**	-0.21	0.63	0.98	0.73	1.03	0.07	0.87
Kurtosis	-0.89	-0.10	-0.24	-0.28	-0.80	-0.56	-0.44	0.21	-0.03	0.54	-1.20	0.26

Note. *N* = 196. FTP1-FTP3 = Factor two psychopathy parcels 1, 2, and 3; BASP1-BASP3 = Behavioral activation system parcels 1, 2, and 3; CCT = Columbia card task; PAEP1-PAEP4 = Positive alcohol expectancies parcels 1, 2, and 3; AU1 = Alcohol frequency and AU2 = Alcohol quantity

* p < .05, ** p < .01

Table 4
Factor Loadings and Factor Correlations in Model 1 CFA (Standard Errors in Parentheses; N=196)

Parameter Estimate	Unstandardized	Standardized	*p* value
Factor Loadings			
FTP --> P1	7.84 (.52)	0.88	$p < .001$
FTP --> P2	6.37 (.45)	0.85	$p < .001$
FTP --> P3	6.98 (.48)	0.86	$p < .001$
BAS --> P1	1.63 (.11)	0.87	$p < .001$
BAS --> P2	1.69 (.12)	0.83	$p < .001$
BAS --> P3	1.94 (.14)	0.83	$p < .001$
PAE --> P1	5.87 (.34)	0.94	$p < .001$
PAE --> P2	6.46 (.37)	0.95	$p < .001$
PAE --> P3	5.70 (.32)	0.95	$p < .001$
AU --> Fr	2.16 (.18)	0.91	$p < .001$
AU --> Qu	1.59 (.17)	0.70	$p < .001$
Factor Correlations			
FTP --> BAS	.34 (.07)	0.34	$p < .001$
FTP --> PAE	.37 (.07)	0.37	$p < .001$
FTP --> AU	.23 (.08)	0.23	$p < .01$
BAS --> PAE	.35 (.07)	0.35	$p < .001$
BAS --> AU	.26 (.08)	0.26	$p < .001$
PAE --> AU	.61 (.06)	0.61	$p < .001$

Note. FTP = Factor Two Psychopathy, BAS = Behavioral Activation System, PAE = Positive Alcohol Expectancies, AU = Alcohol Use (Fr = Frequency, Qu = Quantity), P 1, 2, and 3 = Parcel 1, 2, and 3 for each multidimensional construct

Table 5
Factor Loadings, Path Coefficients, Total Effects and Indirect Effects in Structural Model 1 (Standard Errors in Parentheses; N=196.)

Parameter Estimate	Unstandardized	Standardized	*p* value
Factor Loadings			
FTP --> P1	1.00 (N/A)	0.88	N/A
FTP --> P2	6.38 (.42)	0.85	$p < .001$
FTP --> P3	6.94 (.46)	0.86	$p < .001$
BAS --> P1	1.00 (N/A)	0.87	N/A
BAS --> P2	1.04 (.08)	0.83	$p < .001$
BAS --> P3	1.21 (.09)	0.84	$p < .001$
PAE --> P1	1.00 (N/A)	0.94	N/A
PAE --> P2	1.10 (.04)	0.95	$p < .001$
PAE --> P3	.97 (.04)	0.95	$p < .001$
AU --> Fr	1.00 (N/A)	0.89	N/A
AU --> Qu	.76 (.10)	0.71	$p < .001$
Path Coefficients			
FTP --> BAS	.37 (.12)	0.23	$p < .01$
FTP --> PAE	1.66 (.47)	0.28	$p < .001$
FTP --> AU	-.08 (.17)	-0.04	$p > .05$
BAS --> PAE	1.04 (.32)	0.29	$p < .001$
BAS --> AU	.02 (.12)	0.01	$p > .05$
PAE --> AU	.22 (.03)	0.61	$p < .001$
Total Effects			
FTP --> BAS	.37 (.12)	0.23	$p < .01$
FTP --> PAE	2.05 (.47)	0.35	$p < .001$
FTP --> AU	.37 (.18)	0.17	$p < .05$
BAS --> PAE	1.04 (.32)	0.29	$p < .001$
BAS --> AU	.24 (.13)	0.19	$p > .05$
PAE --> AU	.22 (.03)	0.61	$p < .001$
Indirect Effects			
FTP --> PAE	.38 (.17)	0.06	$p < .05$
FTP --> AU	.45 (.12)	0.21	$p < .001$
BAS --> AU	.23 (.07)	0.17	$p < .01$

Note. FTP = Factor Two Psychopathy, BAS = Behavioral Activation System, PAE = Positive Alcohol Expectancies, AU = Alcohol Use (Fr = Frequency, Qu = Quantity), P 1, 2, and 3 = Parcel 1, 2, and 3 for each multidimensional construct

Table 6
Factor Loadings and Factor Correlations in Model 2 CFA (Standard Errors in Parentheses; N=196)

Parameter Estimate	Unstandardized	Standardized	*p* value
Factor Loadings			
FTP --> P1	7.78 (.52)	0.87	$p < .001$
FTP --> P2	6.39 (.44)	0.86	$p < .001$
FTP --> P3	7.03 (.48)	0.87	$p < .001$
CCT --> CT	.12 (.01)	1.00	$p < .001$
PAE --> P1	5.88 (.34)	0.94	$p < .001$
PAE --> P2	6.46 (.37)	0.95	$p < .001$
PAE --> P3	5.69 (.32)	0.95	$p < .001$
AU --> Fr	2.19 (.18)	0.92	$p < .001$
AU --> Qu	1.57 (.17)	0.69	$p < .001$
Factor Correlations			
FTP --> CCT	-.03 (.08)	-0.03	$p > .05$
FTP --> PAE	.37 (.07)	0.37	$p < .001$
FTP --> AU	.22 (.08)	0.22	$p < .001$
CCT --> PAE	-.04 (.07)	-0.04	$p > .05$
CCT --> AU	-.12 (.08)	-0.12	$p > .05$
PAE --> AU	.61 (.06)	0.61	$p < .001$

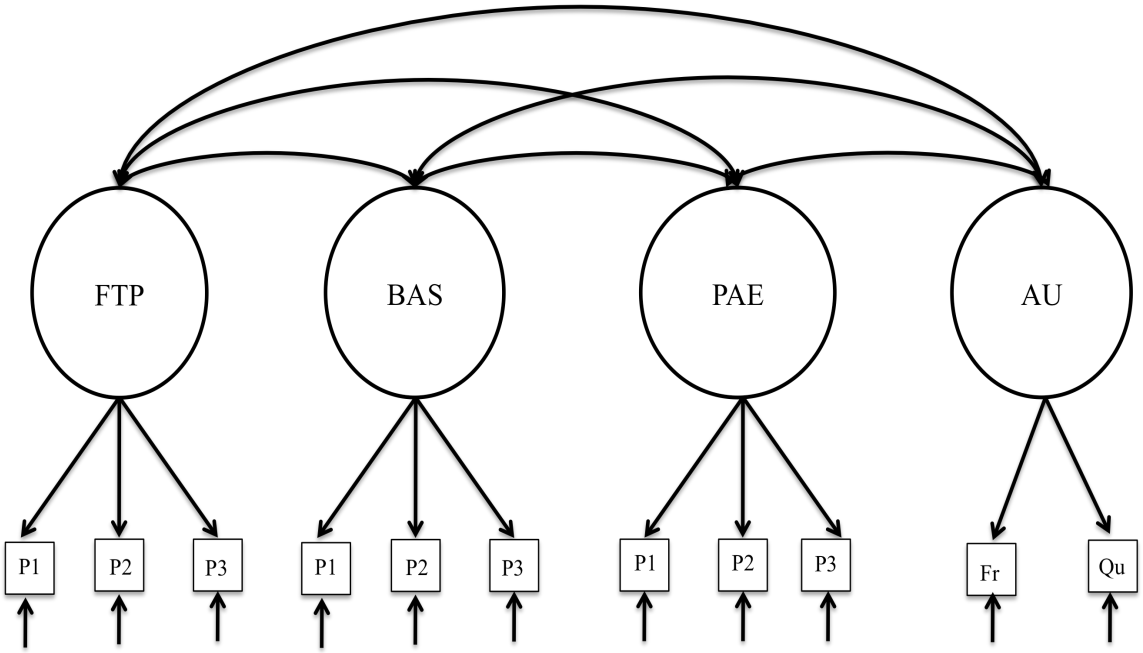
Note. FTP = Factor Two Psychopathy, CCT = Columbia Card Task, PAE = Positive Alcohol Expectancies, AU = Alcohol Use (Fr = Frequency, Qu = Quantity, P 1, 2, and 3 = Parcel 1, 2, and 3 for each multidimensional construct

Table 7
Factor Loadings, Path Coefficients, Total Effects and Indirect Effects in Structural Model 2 (Standard Errors in Parentheses; N=196.)

Parameter Estimate	Unstandardized	Standardized	*p* value
Factor Loadings			
FTP --> P1	1.00 (N/A)	0.88	N/A
FTP --> P2	6.41 (.43)	0.86	$p < .001$
FTP --> P3	6.96 (.46)	0.86	$p < .001$
CCT --> CT	1.00 (N/A)	1.00	N/A
PAE --> P1	1.00 (N/A)	0.94	N/A
PAE --> P2	1.10 (.04)	0.95	$p < .001$
PAE --> P3	.97 (.04)	0.95	$p < .001$
AU --> Fr	1.00 (N/A)	0.92	N/A
AU --> Qu	.72 (.10)	0.69	$p < .001$
Path Coefficients			
FTP --> CCT	.05 (.08)	0.05	$p > .05$
FTP --> PAE	2.05 (.47)	0.35	$p < .001$
FTP --> AU	-.08 (.17)	-0.04	$p > .05$
CCT --> PAE	-.13 (.42)	-0.02	$p > .05$
CCT --> AU	-.18 (.15)	-0.08	$p > .05$
PAE --> AU	.22 (.03)	0.60	$p < .001$
Total Effects			
FTP --> CCT	.05 (.08)	0.05	$p > .05$
FTP --> PAE	2.04 (.47)	0.35	$p < .001$
FTP --> AU	.37 (.19)	0.17	$p < .05$
CCT --> PAE	-.13 (.42)	-0.02	$p > .05$
CCT --> AU	-.21 (.17)	-0.10	$p > .05$
PAE --> AU	.22 (.03)	0.60	$p < .001$
Indirect Effects			
FTP --> PAE	-.01 (.02)	0.00	$p > .05$
FTP --> AU	.44 (.12)	0.20	$p < .001$
CCT --> AU	-.03 (.09)	-0.01	$p > .05$

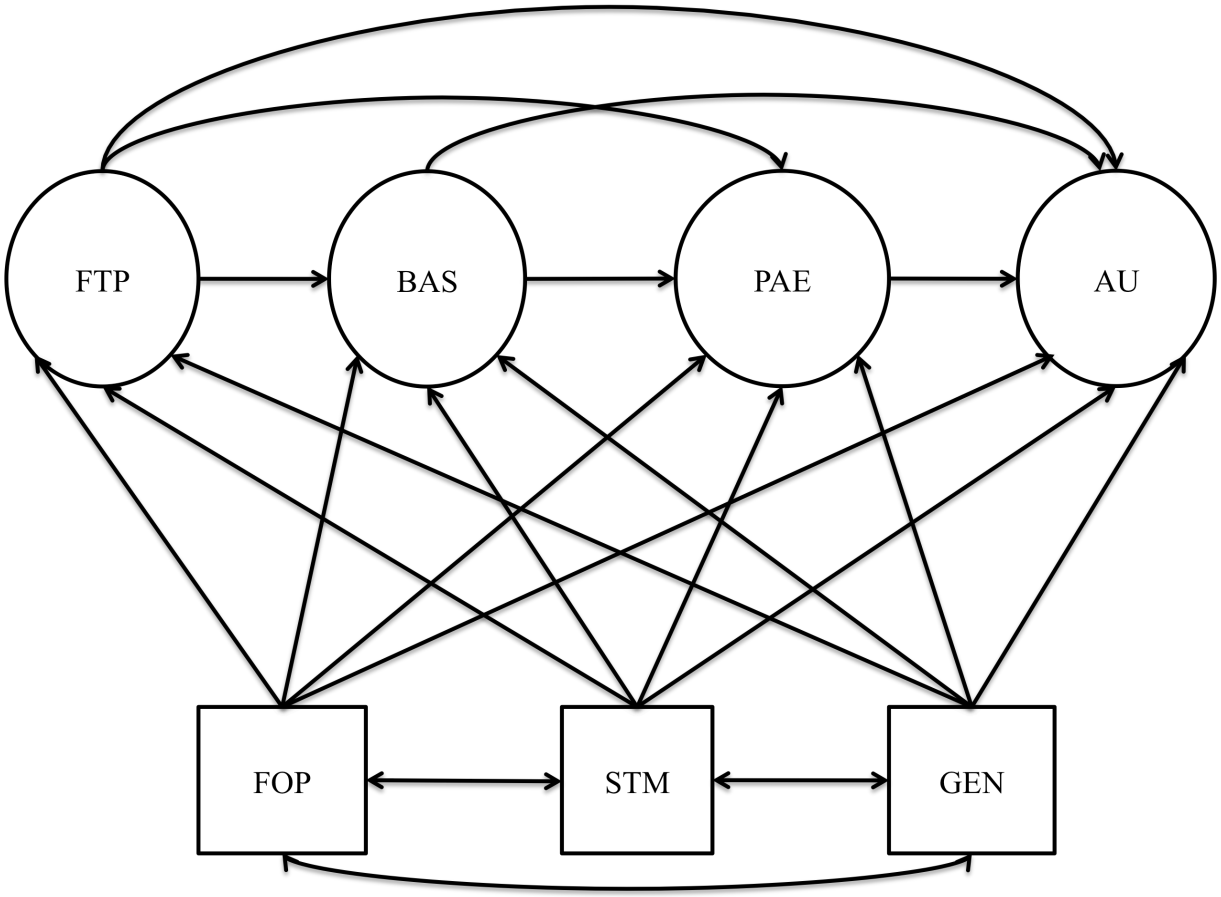
Note. FTP = Factor Two Psychopathy, CCT = Columbia Card Task, PAE = Positive Alcohol Expectancies, AU = Alcohol Use (Fr = Frequency, Qu = Quantity, P 1, 2, and 3 = Parcel 1, 2, and 3 for each multidimensional construct

Figure 1. Proposed 4-factor CFA to test model 1 fit of FTP, BAS, PAE, and AU



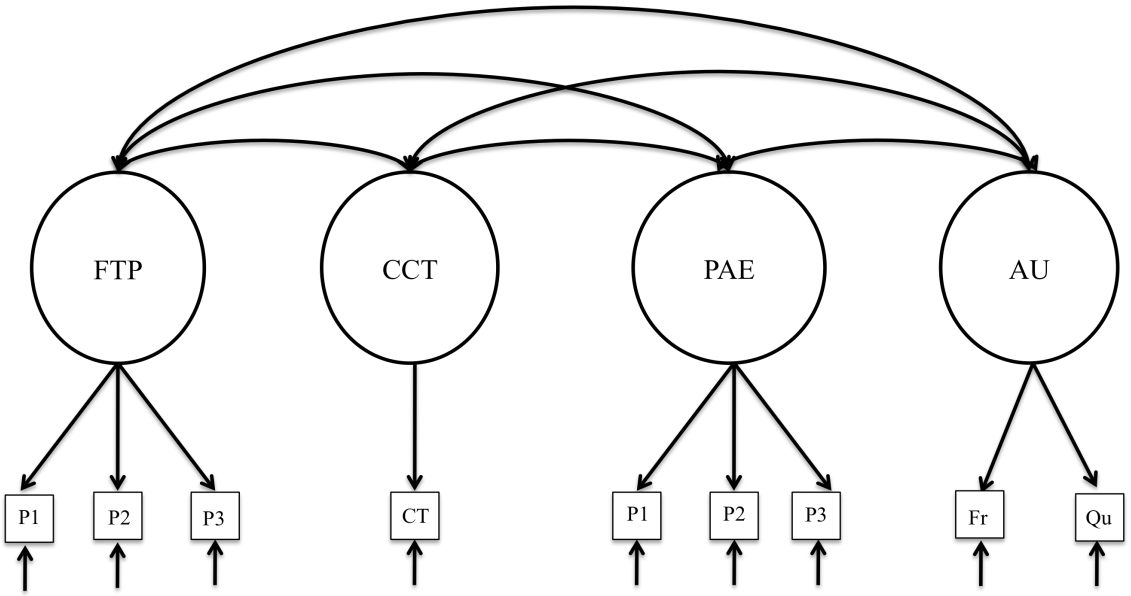
Note. FTP = Factor Two Psychopathy, BAS = Behavioral Activation System, PAE = Positive Alcohol Expectancies, AU = Alcohol Use (Fr = Frequency, Qu = Quantity), and P 1, 2, and 3 = Parcels 1, 2, and 3, for each multidimensional construct

Figure 2. Proposed structural equation model 1 with BAS and PAE as mediators of FTP and AU; direct paths will be specified from all three predictor variables to AU, and from FTP to PAE.



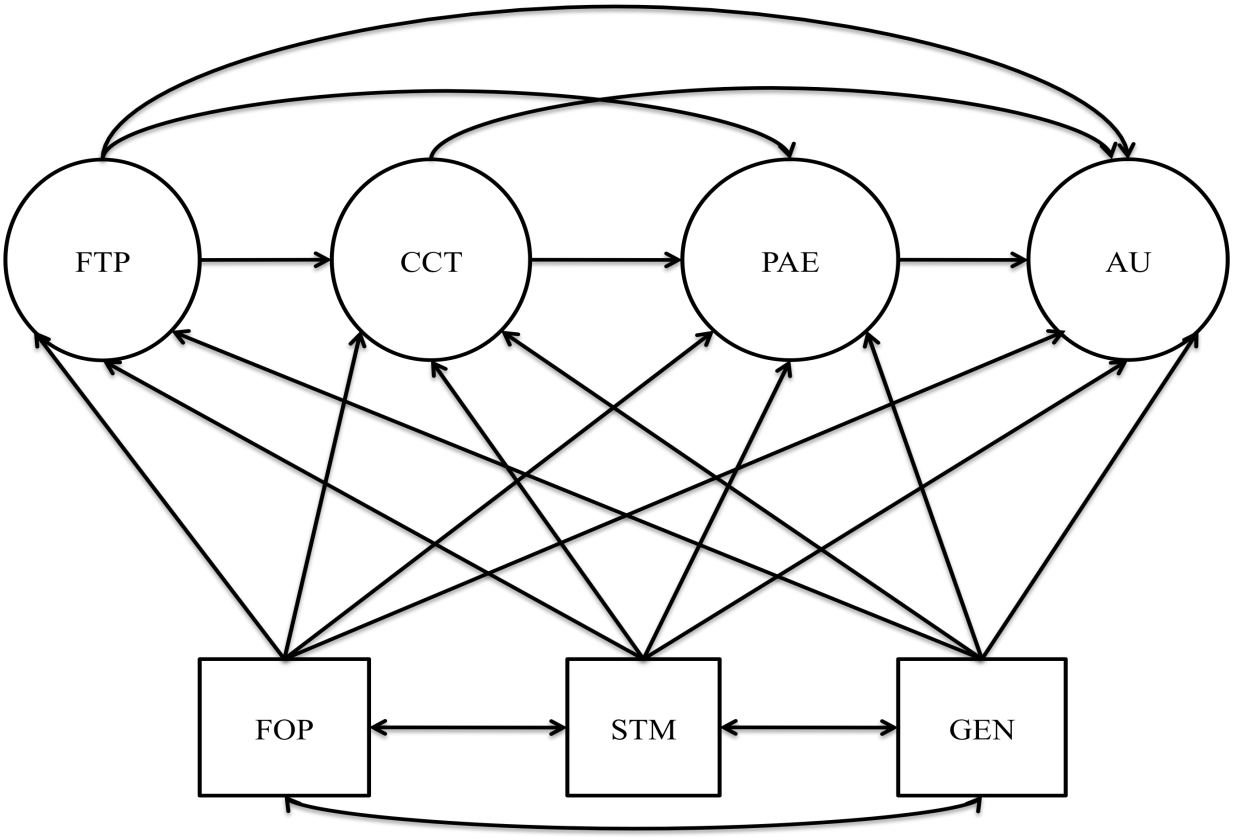
Note. FTP = Factor Two Psychopathy, BAS = Behavioral Activation System, PAE = Positive Alcohol Expectancies, AU = Alcohol Use, FOP = Factor One Psychopathy, STM = Short-term Memory, and GEN = Gender. Proposed 4-factor CFA to test model fit of FTP, CCT, PAE, and AU

Figure 3. Proposed 4-factor CFA to test model 2 fit of FTP, CCT, PAE, and AU



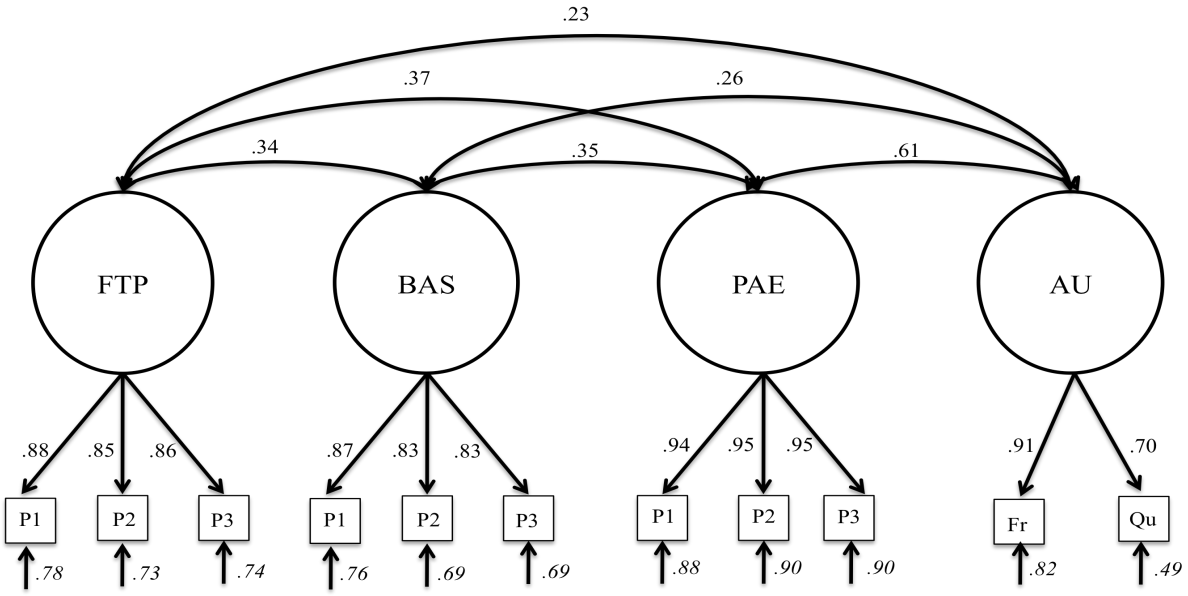
Note. FTP = Factor Two Psychopathy, CCT = Columbia Card Task, PAE = Positive Alcohol Expectancies, AU = Alcohol Use (Fr = Frequency, Qu = Quantity), and P 1, 2, and 3 = Parcels 1, 2, and 3, for each multidimensional construct

Figure 4. Proposed structural equation model 2 with CCT and PAE as mediators of FTP and AU; direct paths will be specified from all three predictor variables to AU, and from FTP to PAE



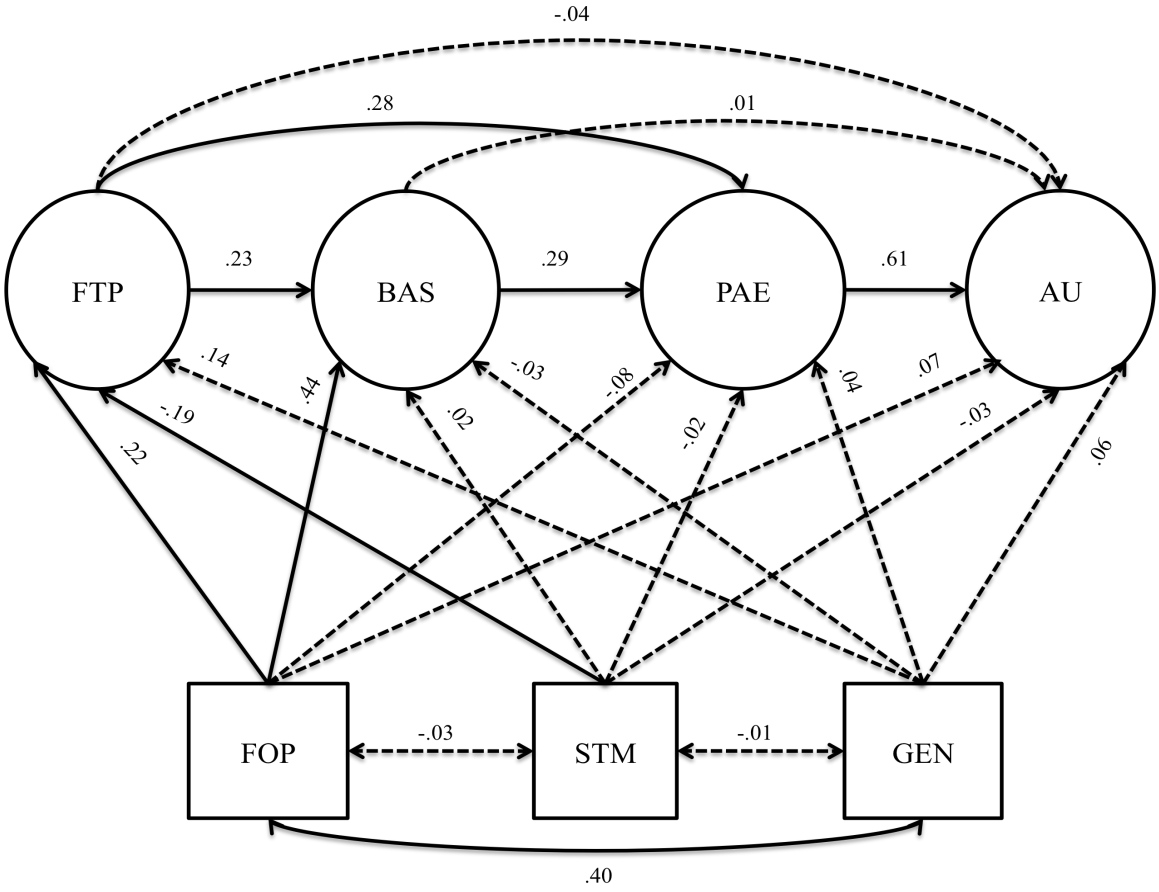
Note. FTP = Factor Two Psychopathy, CCT = Columbia Card Task, PAE = Positive Alcohol Expectancies, AU = Alcohol Use, FOP = Factor One Psychopathy, STM = Short-term Memory, and GEN = Gender

Figure 5. A 4-factor CFA testing model 1 fit with FTP, BAS, PAE, and AU



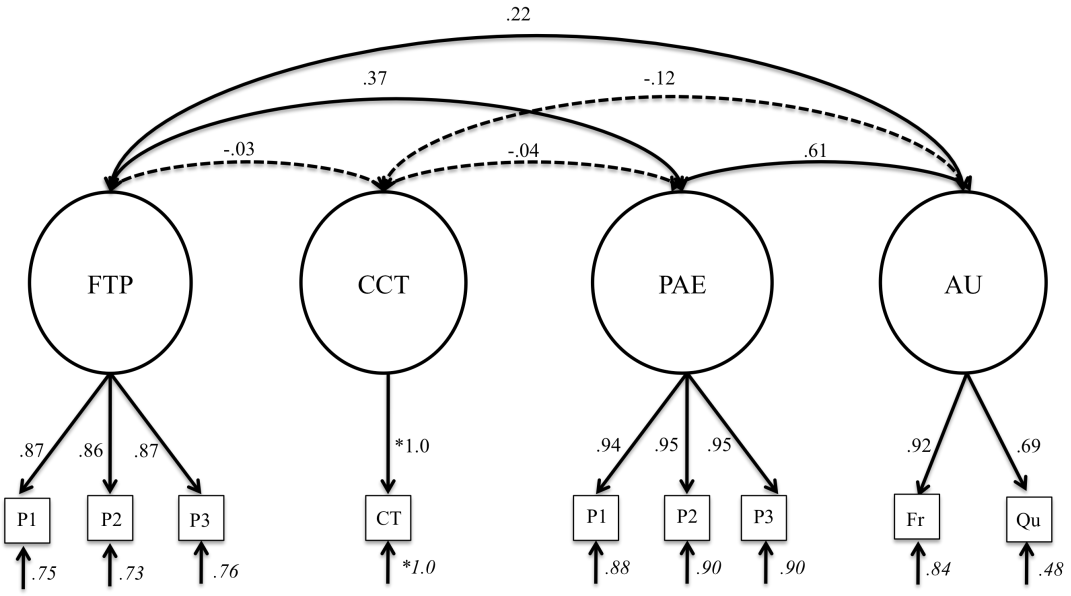
Note. FTP = Factor Two Psychopathy, BAS = Behavioral Activation System, PAE = Positive Alcohol Expectancies, AU = Alcohol Use (Fr = Frequency, Qu = Quantity), and P 1, 2, and 3 = Parcels 1, 2, and 3, for each multidimensional construct. RMSEA = .057, CFI = .99, NNFI = .98. Solid arrows denote significant factor correlations and factor loadings at $p < .001$. Factor loadings appear as standardized beta weights. Squared multiple correlations are in italics behind the indicators.

Figure 6. Structural equation model 1 with BAS and PAE as mediators of FTP and AU; direct paths were also specified from all three predictor variables to AU; FOP, STM, and GEN are all covariates



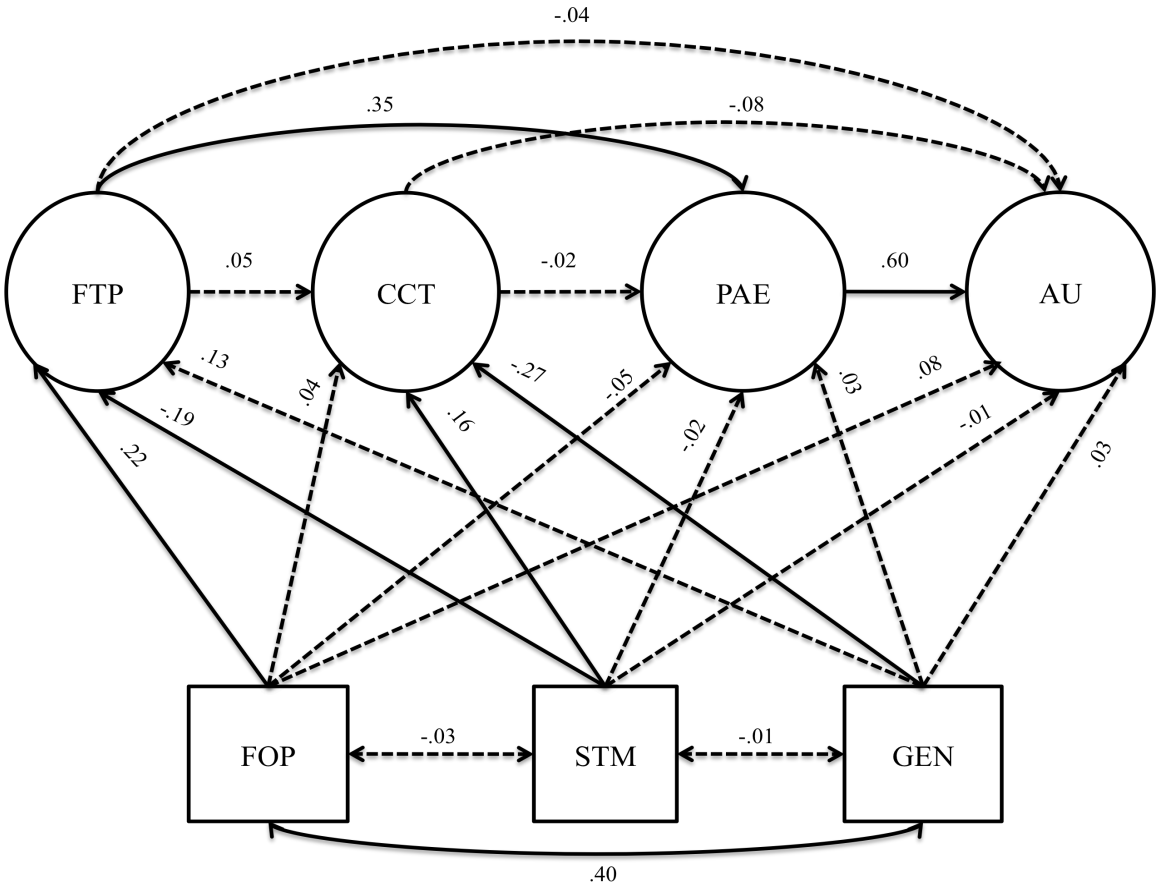
Note. FTP = Factor Two Psychopathy, CCT = Columbia Card Task, PAE = Positive Alcohol Expectancies, AU = Alcohol Use (Fr = Frequency, Qu = Quantity), and P 1, 2, and 3 = Parcels 1, 2, and 3, for each multidimensional construct. RMSEA = .057, CFI = .98, NNFI = .97. Solid arrows denote significance at the $p < .001$ level (dashed lines are non-significant). Path coefficients appear as standardized beta weights

Figure 7. A 4-factor CFA testing model 2 fit with FTP, CCT, PAE, and AU



Note. FTP = Factor Two Psychopathy, CCT = Columbia Card Task, PAE = Positive Alcohol Expectancies, AU = Alcohol Use (Fr = Frequency, Qu = Quantity), and P 1, 2, and 3 = Parcels 1, 2, and 3, for each multidimensional construct. RMSEA = .035, CFI = .99, NNFI = .99. Solid arrows denote significant factor correlations and factor loadings at $p < .001$. Factor loadings appear as standardized beta weights. Squared multiple correlations are in italics behind the indicators. CT error variance was set to 0.

Figure 8. Structural equation model 2 with CCT and PAE as mediators of FTP and AU; direct paths were also specified from all three predictor variables to AU; FOP, STM, and GEN were all covariates



Note. FTP = Factor Two Psychopathy, CCT = Columbia Card Task, PAE = Positive Alcohol Expectancies, AU = Alcohol Use, FOP = Factor One Psychopathy, STM = Short-term Memory, and GEN = Gender RMSEA = .049, CFI = .99, NNFI = .97. Solid arrows denote significance at the $p < .001$ level (dashed lines are non-significant). Path coefficients appear as standardized beta weights. References

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ABSTRACT

A STRUCTRUAL EQUATION MODEL OF FACTOR TWO PSYCHOPATHY, BEHAVIORAL ACTIVATION, POSITIVE ALCOHOL EXPECTANCIES, AND ALCOHOL USE

by

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MAY 2015

Advisor: Dr. Emily Grekin

Major: Psychology (Clinical)

Degree: Master of Arts

A large body of literature indicates that there is a relationship between psychopathy and heavy drinking. Very few studies, however, have examined potential mediators of this relationship. As a result, it is difficult to develop intervention strategies that target early steps in the psychopathy/alcohol use pathway. The current study tested a structural equation model linking secondary psychopathy to heavy drinking through the influence of both behavioral activation and positive alcohol expectancies. The study was also one of the first to utilize both self-report and laboratory measures of behavioral activation.

AUTOBIOGRAPHICAL STATEMENT

Prior to entering graduate school in 2012, I earned a bachelor's degree in psychology from Wayne State University. It was during this time that I developed my passion for clinical psychology through both clinical work and psychological research. Now, as a third year student in Wayne State's clinical psychology PhD program, I am focused on conducting research centering on alcohol use and comorbid personality disorders such as psychopathy and borderline personality disorder. It is my hope that through this line of research, a better understanding of the interplay between these disorders will be gained and, ultimately, efficacious interventions will be developed.