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The Influence of Resting State Functional Connectivity on Personality Traits in Adolescents and Young Adults

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Abstract

The neurobiological basis of individual differences in personality is still poorly understood. Previous research has previously linked personality traits to risk factors for developing mental illness or engaging in high-risk behavior. Previous research has also linked personality traits to individual differences in resting state functional connectivity, which describes regional interactions in the brain when a person is not engaged in an active task. This study aimed to apply a theory of personality derived from behavioral neuroscience and ethology known as Reinforcement Sensitivity Theory (RST) to the characterization of brain networks underlying personality. RST defines four motivational systems in the brain, the Behavioral Approach System (BAS), Behavioral Inhibition System (BIS), Fight-Flight-Freeze System (FFFS), and Executive Control System (ECS) which enable learning from and responding to current environmental demands. Individual variation in the functioning of these motivational systems creates different propensities to seek out rewarding stimuli and avoid potentially threatening ones, corresponding to the ubiquitous presence of reward and punishment sensitivity related traits across personality models. However, RST has not been applied to characterizing the relationship of brain development to the maturation of personality and the relationship of the RST systems to resting state networks. Understanding how these circuits change developmentally from adolescence to adulthood is vital to identify how high-risk personality traits become stable parts of adult personality. In this study, 25 adolescents and 52 adults each completed a battery of personality assessments and a six minute resting state fMRI scan. It was hypothesized that a factor analysis of each personality trait would reveal factors corresponding to the RST motivational systems. For each participant factor scores were computed corresponding to these traits. Network analysis was completed using a graph theory approach to characterize important nodes in the brain. Following this, an a priori set of brain regions derived from RST was used for a regression analysis to test the hypothesis that nodes within the RST systems would predict the corresponding personality trait. The results support the notion that important resting state network nodes within the RST systems correspond to personality traits. The results also suggest that these networks are established during adolescence and change little into young adulthood, with the exception of the ECS. Therefore, early intervention to teach adaptive coping skills and behaviors is desirable to ameliorate the potential life outcomes of high-risk personality traits for drug abuse or mood disorders.

1 Introduction

The neurobiological basis of individual differences in personality is still poorly understood. Previous research has linked personality traits such as neuroticism, extraversion, and conscientiousness with neuropsychiatric conditions such as anxiety disorders, substance use disorders, and attention-deficit hyperactivity disorder (Terracciano, Löckenhoff, Zonderman, Ferrucci, & Costa, 2008; Valero et al., 2012; Jeronimus, Riese, Sanderman, & Ormel, 2014). Understanding the neurobiological basis of personality is important for two reasons. First, understanding what combination of traits puts one at risk for developing neuropsychiatric problems or poor treatment outcomes can allow clinicians to identify high-risk groups for preventative care. Second, understanding the neurobiological basis of personality traits can assist in personalized treatments. It has been documented that personality traits can modulate responsiveness to psychiatric medications (Mizuki, Suetsugi, Ushijima, & Yamada, 1996, 1997; Bagby, Levitan, Kennedy, Levitt, & Joffe, 1999; Quilty, Meusel, & Bagby, 2008; Quilty, De Fruyt, et al., 2008). However, research on the interface between personality and neuroscience often utilizes a variety of different approaches to measuring personality.

1.1 Current Theories of Personality

There are a large number of personality theories which purport to characterize the basic traits forming human personality. The dominant theory in personality psychology is the five factor model, which includes Openness to Experience/Intellect, Conscientiousness, Extraversion, Agreeableness, and Neuroticism (McCrae & Costa, 1987; Digman, 1989; Tupes & Christal, 1992; McCrae & John, 1992; Goldberg, 1993). However, it is without biological basis due to the athe-

oretical approach to its development. This presents a difficult problem in neuroscientific studies of personality because it can be difficult to link broadly defined traits to specific neural processes. Other approaches have taken into account that the basic human traits must have evolutionary or neurobiological basis. For example, there exists an alternative Big Five model based on phylogenetic considerations of what kind of traits should be regarded as basic (Zuckerman, Michael Kuhlman, Thornquist, & Kiers, 1991; Zuckerman, 1992; Mizuki, Suetsugi, Ushijima, & Yamada, 1994; Zuckerman & Cloninger, 1996). The traits the alternative Big Five contains are Neuroticism-Anxiety, Sociability, Aggression-Hostility, Activity Level, and Impulsive-Sensation Seeking. Historically preceding this is Eysenck's three factor model based in learning theory and what was known about neurobiology at the time. Eysenck's three factor model includes Neuroticism, Extroversion, and Psychoticism in later versions of the theory (Eysenck, Eysenck, & Barrett, 1985). Cloninger's similar model proposes Harm Avoidance, Novelty Seeking, and Reward Dependence as fundamental features of personality (Cloninger, 1986). However, these biologically based theories are theoretically difficult to apply to the rich information neuroscience can provide due to vague connections to current neuroscientific knowledge. Nevertheless, the conceptual overlap provides clues as to what one might expect from a fully developed theory of personality. All agree on traits tied to reward seeking behavior, negative mood and harm avoidance, and impulsivity.

1.2 Reinforcement Sensitivity Theory

A model of personality more closely tied to learning theory, ethology, and behavioral neuroscience was proposed by a former student of Hans Ensyeck named

Jeffrey Alan Gray (Gray, 1970, 1982). Gray's theory is known as Reinforcement Sensitivity Theory (RST). RST posits three major behavioral systems that underlie individual differences in personality: the Behavioral Approach System (BAS), Fight-Flight-Freeze System (FFFS), and Behavioral Inhibition System (BIS). Conceptually, the BAS corresponds to Eysenck's Extraversion and Cloninger's Reward Dependence and Novelty Seeking. The BIS and FFFS correspond to Neuroticism and Harm Avoidance.

The BAS responds to both unconditioned and conditioned rewarding stimuli. The BAS consists primarily of dopaminergic striatal structures and their connections in the medial frontal and orbital frontal cortex. The BAS also contains certain amygdala circuits and the hypothalamus. Importantly, the BAS also contributes to the persistence of behavior in the face of non-reward. When an organism no longer receives an expected reinforcer, it may perseverate and continue to try. A rat may keep pushing a lever or display response variability as it tries new things to get the reward. Such scenarios may also elicit emotional reactions from the FFFS known as frustrative non-reward (Amsel, 1958). Originally such responses were thought to be driven entirely by the FFFS, but more recent evidence from studies using human subjects suggests individuals sensitive to reward tend to experience frustrative non-reward(Corr, 2002; Carver, 2004, 2006).

The FFFS and BIS were once considered as a single system due to the factor analytic origins of the theory and the then-current state of knowledge (Gray, 1970). As a better understanding of the limbic system developed the theory was revised. It was proposed that the BIS was responsible for learning conditioned stimuli predicting aversive situations and initiating inhibitory behaviors (such as avoidance or freezing) in order to avoid the aversive outcome, while the then-named FFS initiated fight or flight responses to (innate) aversive stimuli (Gray, 1982). This

was a substantial improvement to the theory. In 2000 the theory received another major revision. Hippocampus researcher Neil McNaughton contributed to the third and most complete statement of the theory (Gray & MacNaughton, 2000). The FFS was renamed the FFFS, so that this system was completely responsible for defensive behaviors. The FFFS consists of neural circuits in the amygdala and projections to the periaqueductal gray for the identification of threats and execution of defensive responses. The BIS is comprised primarily of the anterior cingulate and hippocampal structures.

The primary purpose of the BIS is to detect goal conflicts in situations where the conflict is severe enough to result in equivalent activation of the FFFS and BAS (approach-avoidance conflict). Such equivalent activation will produce incompatible motor programs where one must be inhibited. In this situation the function of the BIS is to increase arousal and bias decision making towards the FFFS. Hence, the "inhibition" in BIS now refers to the termination of a motor program, and no longer to inhibition responses such as freezing (Gray & MacNaughton, 2000). This may sound strange, but consider that evolutionarily speaking, an organism with only exploratory reward responses and defensive responses would have no means of properly adapting to its environment. Were its BAS the most sensitive, it would always opt to explore and obtain rewards in spite of aversive consequences or danger. Were its FFFS the most sensitive, it would live in fear and be unable to properly develop within its ecological niche. By acting as a mediating third party to nudge responses towards the FFFS when there is equivalent activation between the BAS and FFFS, the BIS allows an organism to engage in the adaptive response and survive.

The BIS also increases arousal, which makes an organism alert and attend to a potential threat if it is uncertain or distant. The BIS may allow the current behav-

ior to remain ongoing but constantly assess the likelihood or imminence of threat. This is known as defensive distance (Gray & MacNaughton, 2000; McNaughton & Corr, 2004; McNaughton, DeYoung, & Corr, 2016). The arousal increasing nature of the BIS can lend to the development of compulsive checking for danger or suffering intrusive worrisome thoughts or feelings of danger. Such behaviors are a major part of human clinical anxiety disorders as obsessive-compulsive disorder and generalized anxiety disorder. These disorders can occur due to insufficient inhibition of the FFFS by the BIS, allowing spatiotemporaly distant aversive stimuli to induce unwarranted degrees of psychological and physiological discomfort and maladaptive behaviors. In light of these functions, a better name for the BIS in current theory might be "Conflict Monitoring System", but keeping with current tradition the name BIS will be used.

One limitation is that RST does not account for variation in executive functions in humans as a personality trait. This is likely the result of the behavioral and biological support for the theory being largely drawn from behavior analytic and ethological studies. Attempts have been made to relate the components of RST to executive function. Higher scores on the flight facet of a FFFS scale was negatively correlated with faster scores on the stroop task and greater speed at accomplishing tasks (Jackson, Loxton, Harnett, Ciarrochi, & Gullo, 2014). The authors propose when resources are directed towards escaping aversive situations resources are taken away from top-down executive functions (Jackson et al., 2014). This is consistent with the activation of limbic alpha-1 adrenergic receptors in the presence of a threat reducing prefrontal activity, as well as the reduction of noradrenergic tone in limbic areas and additional enhancement of prefrontal cortical activity by alpha-2A receptors in the presence of lower noradrenaline levels (Arnsten, 2011; Sallee, Connor, & Newcorn, 2013). High scores on FFFS and

BIS scales have also been associated with increased severity of symptoms as well as tic disorders among those diagnosed with ADHD (Heym, Kantini, Checkley, & Cassaday, 2014). However, while FFFS and BIS activity may reduce executive functions, they are not responsible for maintaining executive functions such as voluntary behavioral activation and inhibition, working memory, or sustained attention.

In order to deal with this shortcoming of RST a proposal was made to add the Executive Control System (ECS) to RST (Kennis, Rademaker, & Geuze, 2013). The ECS consists of neural circuits in the frontal lobe and parietal lobe for the top-down control of behavior and emotion. While the ECS is associated with behavioral inhibition, its function is to enable voluntary behavioral control and regulate neural processes such that an individual is able to successfully interact with their world. Breakdown of the ECS can lead to attention and working memory deficits, behavioral disinhibition, as well as disorganized and even delusional thinking (Kennis et al., 2013; Jackson et al., 2014).

1.3 Evidence for the Hippocampus as Part of the BIS

The most important aspect of reinforcement sensitivity theory is the unique prediction that the hippocampus is an important part of the neural basis of anxiety. The functions of the hippocampus are often considered to relate to memory and spatial reasoning (Scoville & Milner, 1957; Nadel, O'Keefe, & Black, 1975; Morris, 2007). However, hippocampectomized rodents struggle to extinguish learned aversive associations and also display reward learning deficits (Altman, Brunner, & Bayer, 1973). Although supporters of the memory and spatial learning view have criticized the behavioral inhibition view, a purely memory oriented per-

spective fails to address the fact that hippocampectomized rodents also display hyperactive behavior (Nadel et al., 1975; Altman et al., 1973). This suggests the hippocampus plays a role in executive functions as well as approach-avoidance conflict in smaller mammals. Other evidence for linking the hippocampus to mood related processes comes from the fact that increasing neurogenesis in the hippocampus appears to be how antidepressant therapies mediate their effects (Anacker et al., 2011). The hippocampus is rich with GABA-A receptors containing benzodiazapene receptor subunits as well as serotonin binding 5-HT1A postsynaptic receptors. Both of these receptors are targets of clinically used anxiolytic drugs such as the benzodiazapenes and 5-HT1A agonists (Lader, 1988; Sanger, Perrault, Morel, Joly, & Zivkovic, 1991; Blanchard, Yudko, Rodgers, & Blanchard, 1993).

Magnetic resonance spectroscopy has yielded evidence that lower levels of GABA in the hippocampal-medial frontal pathway are associated with increased vulnerability to intrusive thoughts, which is a major characteristic of the subjective human experience of anxiety (Schmitz, Correia, Ferreira, Prescot, & Anderson, 2017). This both explains the efficacy of anxiolytic drugs and confirms hippocampal involvement in anxiety. Indeed, theta oscillations between the septal-hippocampal and medial frontal regions (midline frontal theta) have been linked to anxiolysis in MEG and EEG studies of anxiolytic drugs (Mizuki et al., 1994; Suetsugi, Mizuki, Ushijima, Yamada, & Imaizumi, 1998; Mitchell, McNaughton, Flanagan, & Kirk, 2008; Cavanagh & Frank, 2014; Cavanagh & Shackman, 2015). There are also several neuroimaging studies which offer evidence from task-based studies that the hippocampus is involved in approach-avoidance conflict monitoring and anxiety (Frodl et al., 2006; Bach et al., 2014; Strange, Witter, Lein, & Moser, 2014; O'Neil et al., 2015; Oehrn et al., 2015; Yu & Frank, 2015; Ito & Lee,

2016; Khemka, Barnes, Dolan, & Bach, 2017). Other evidence has continued to accumulate linking the hippocampus to behavioral inhibition and anxiety from both animal and human studies, suggesting the hypotheses of Altman, Gray, and McNaughton have more credibility than critics have historically granted (Satpute, Mumford, Naliboff, & Poldrack, 2012; Cominski, Jiao, Catuzzi, Stewart, & Pang, 2014; Ito & Lee, 2016).

1.4 Resting State Functional Connectivity

Although support for RST is strong from animal behavior, psychopharmacology, and neuroimaging studies, little is said concerning how the sensitivities of the RST systems underlying personality are reflected in the brain in the absence of active behavior. One way to study the brain while a person is not actively interacting with the world is the resting state functional connectivity approach. Resting state functional connectivity is defined by the correlation of brain activity across different parts of the brain. It can be characterized using a variety of brain imaging techniques including EEG and fMRI. fMRI based functional connectivity calculates the correlation between the resting state BOLD signal. It can then be sorted into different networks using a variety of different techniques including independent components analysis or network theoretic approaches (Fransson, 2005).

Previous research has linked personality with resting state functional connectivity (Gao et al., 2013; Dubois, Galdi, Han, Paul, & Adolphs, 2018). However, no research has yet characterized the relationship of brain regions specified by RST. The goal of this project is to characterize key regions within each of the RST systems that predict RST-derived personality traits. In order to understand how personality development may unfold an additional research goal is to investigate

the changes in brain networks associated with maturation from adolescence to young adulthood.

1.5 Hypotheses

In this study, it is hypothesized that traits corresponding to the Behavioral Approach System, Behavioral Inhibition System, Fight-Flight-Freeze System, and Executive Control System (BAS, BIS, FFFS, and ECS) will be revealed through factor analysis of the measures collected. It is expected, however, that the BIS and FFFS will be reflected by a single factor because BIS engagement dictates that the FFFS and BAS are co-activated, therefore the influences of the FFFS and BIS to trait-anxiety behavior are be difficult to disentangle through questionnaires (McNaughton & Corr, 2008).

For each corresponding trait, specific brain regions are hypothesized to bear relationships to the traits. The BIS-FFFS trait is expected to be predicted by the resting state functional connectivity of the hippocampus, (dorsal) amygdala, and anterior cingulate.The BAS trait is expected to be predicted by the caudate, putamen, nucleus accumbens, and (medial) amygdala. The ECS trait is expected to be predicted by the the dorsolateral frontal cortex and medial frontal cortex. Nodes for both the left and ride sides will be selected located at coordinates mirroring one another to the closest extent possible. Because many of these regions cover large swathes of brain tissue multiple nodes may contain the region. Since they are likely to be correlated only one node (per hemisphere) for a region will be selected based on a literature review. The literature review was conducted using Neurosynth (Yarkoni, Poldrack, Nichols, Van Essen, & Wager, 2011). A list of node coordinates is provided in Table 1. A visualization of these coordinates can

be found in Figure 1.

The nodes in the dorsolateral frontal cortex were chosen on the basis of experiments showing these particular coordinates are involved in response inhibition (Booth et al., 2003; Stevens, Kiehl, Pearlson, & Calhoun, 2007; Jamadar, Hughes, Fulham, Michie, & Karayanidis, 2010). The nodes in the medial frontal cortex were chosen based on studies showing involvement in working memory, voluntary movement inhibition, as well as trait-impulsivity modulated activity in reward learning tasks (Jaffard et al., 2008; Sripada, Gonzalez, Phan, & Liberzon, 2011; Wager, Spicer, Insler, & Smith, 2014).

The nodes in the anterior cingulate were chosen based on their connection to trait anxiety constructs relevant to the BIS (Xu et al., 2013). The nodes in the hippocampus were selected based on studies showing that the nodes are active during approach-avoidance tasks, consistent with the predictions of reinforcement sensitivity theory (Ploghaus, 2001; L. M. Williams et al., 2009; Schlund, Magee, & Hudgins, 2011; Oehrn et al., 2015). It was specifically hypothesized that the posterior hippocampus will bear the greatest relation to a BIS trait. There is evidence that the posterior hippocampus decreases general functional connectivity with age relative to the anterior hippocampus in a normal sample (Damoiseaux, Viviano, Yuan, & Raz, 2016). Hence, if trait anxiety is related to the posterior hippocampus, and the posterior hippocampus has a relative trend towards less functional connectivity with age, then it may be that trait anxiety maintains its connectivity. Therefore, the relative effect in adolescents (the interaction) of the posterior hippocampus is hypothesized to be negative.

The dorsal amygdala coordinates were chosen on the basis that the dorsal amygdala has been linked to both state and trait anxiety as well as to social anxiety (Choi, Padmala, & Pessoa, 2012; Xu et al., 2013; Schlund et al., 2011; Bickart,

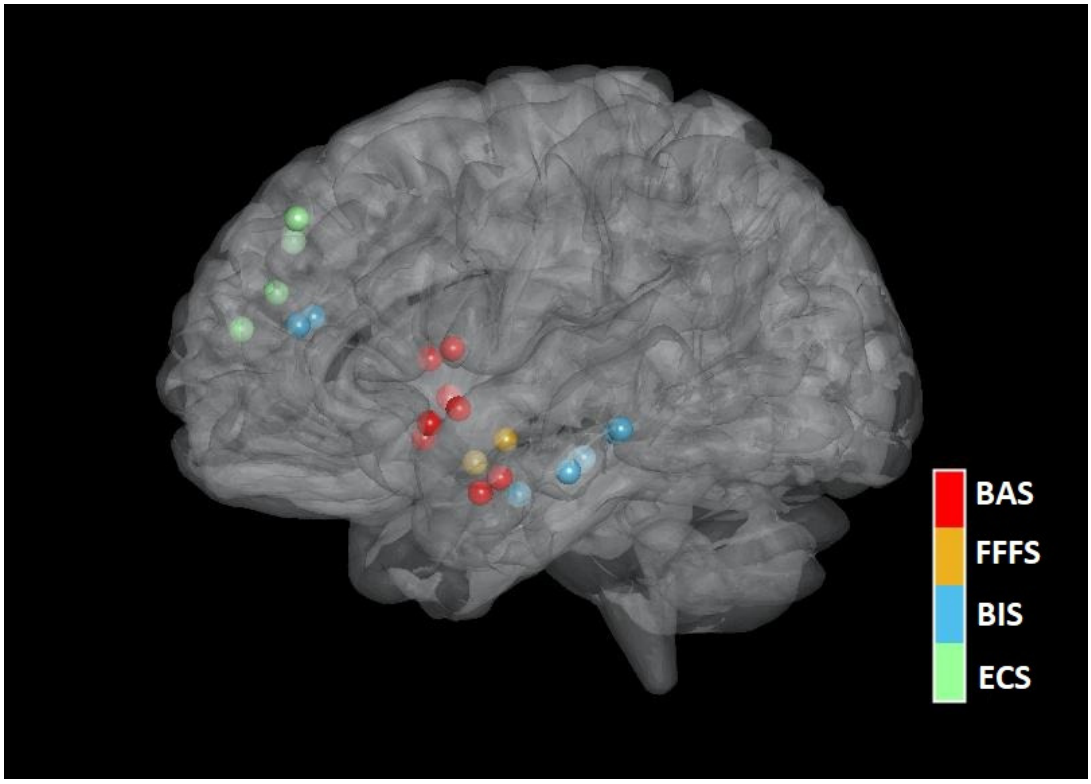

Figure 1: Visualization of nodes hypothesized to predict personality traits corresponding to the RST systems. See Table 1 for the MNI coordinates and labels for each node.

Hollenbeck, Barrett, & Dickerson, 2012; Bickart, Dickerson, & Feldman Barrett, 2014).

The caudate, putamen, and nucleus accumbens are well known to be involved in reward learning and positive affect. However, to help assure that the nodes chosen have construct validity to reward learning, the coordinates used for the caudate have been shown to be involved in reward learning (Staudinger, Erk, & Walter, 2011). The medial amygdala has been shown to be involved in social reward/affiliation networks (Bickart et al., 2012, 2014). Nodes used for the putamen have been shown to relate to reward learning (Kirsch et al., 2003).

Table 1: Nodes and corresponding hypotheses selected for analysis.

MNI	Anatomical	BA	Label	System	Predicted Effect
22, 39, 39	R Frontal Pole	8	R Dorsal Lateral Frontal Cortex	ECS	-
-20, 45, 40	L Frontal Pole	8	L Dorsal Lateral Frontal Cortex	ECS	-
6, 54, 16	R Superior Frontal Gyrus	9	R Medial Frontal Cortex	ECS	-
-8, 48, 15	L Superior Frontal Gyrus	9	L Medial frontal Cortex	ECS	-
-3, 42, 16	L Anterior Cingulate	32	L Dorsal Anterior Cingulate	BIS/FFFS	+
12, 36, 20	R Anterior Cingulate	32	R Dorsal Anterior Cingulate	BIS/FFFS	+
-20, -18, -20	L Hippocampus	54	L Anterior Hippocampus	BIS/FFFS	∅ (Adults), + (Teens)
-24, -30, -10	L Hippocampus	54	L Posterior Hippocampus	BIS/FFFS	+ (Adults), (Teens)
32, -16, -18	R Hippocampus	54	R Anterior Hippocampus	BIS/FFFS	∅ (Adults), + (Teens)
24, -30, -10	R Hippocampus	54	R Posterior Hippocampus	BIS/FFFS	+ (Adults), - (Teens)
-24, -4, -12	L Amygdala	53	L Dorsal Amygdala	BIS/FFFS	+
24, 4, -12	R Amygdala	53	R Dorsal Amygdala	BIS/FFFS	+
-22, 7, -5	L Putamen	49	L Putamen	BAS	+
31, -14, 2	R Putamen	49	R Putamen	BAS	+
-14, -4, -20	L Amygdala	53	L Medial Amygdala	BAS	+
14, -4, -20	R Amygdala	53	R Medial Amygdala	BAS	+
-13, 7, 10	L Caudate	48	L Caudate	BAS	+
14, 8, 11	R Caudate	48	R Caudate	BAS	+
-10, 12, -7	L Nucleus Accumbens	52	L Nucleus Accumbens	BAS	+
10, 10, -8	R Nucleus Accumbens	52	R Nucleus Accumbens	BAS	+

2 Procedures

2.1 Participants

25 adolescents (14 female, 11 male) and 52 adults (32 female, 20 male) were recruited to complete a resting state fMRI scan and complete a battery of personality questionnaires. 15 of the adults were recruited at the University of Kentucky. All remaining subjects were recruited at the Medical University of South Carolina. Prospective participants completed the Brief Sensation-Seeking Scale (BSSS) on the website RedCap (Hoyle, Stephenson, Palmgreen, Lorch, & Donohew, 2002; Harris et al., 2009). Survey responders with scores in the top and bottom quartiles were contacted and invited to participate in the study. Some subjects had to be excluded due to excessive head motion in the fMRI scan. The remaining sample consisted of 23 adolescents (13 female, 10 male) and 51 adults (31 female, 20 male). The average adolescent age is 13.17 adult age is 22.9. The average education level of the adolescents is 7 years of education, and 16 years of education for young adults.

2.2 Personality Assessments

The personality assessments used in present study are listed in Table 2.2. Because these data are from a previous study, some assessments and measures collected but not used in the present study include the Pubertal Developmental Scale (PDS) (Petersen, Crockett, Richards, & Boxer, 1988), forward and backward digit span (Rosenthal, Riccio, Gsanger, & Jarratt, 2006) , Peabody Picture Vocabulary Test, Fourth Edition (PPVT) (Dunn & Dunn, 2007) and Ruff's 2-7 selective attention test (Ruff, Niemann, Allen, Farrow, & Wylie, 1992).

Personality Assessment
Big-Five Inventory (BFI; John & Srivastava, 1999)
Urgency, Premeditation, Perseverance, and Sensation Seeking Scale (UPPS; Whiteside & Lynam, 2001)
BIS/BAS Scales (Carver & White, 1994)
Eysenck Personality Questionnaire (EPQ, Eysenck & Eysenck, 1975)
Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ, Torrubia, Avila, Molto, & Caseras, 2001)
Impulsive-Sensation Seeking; Zukerman-Kuhlman Personality Questionnaire (ZKPQ; Aluja, Rossier, García, Angleitner, Kuhlman, & Zuckerman, 2006)
Sensation Seeking Scales Form V (SSSV; Zuckerman, Eysenck, & Eysenck, 1978)

Table 2: Personality assessments utilized in the factor analysis.

2.3 fMRI Data Acquisition

25 adolescents (14 female, 11 male) and 52 adults (32 female, 20 male) completed a resting state fMRI (rs-fMRI) scan in a Siemens TIM Trio 3T scanner using a 12-channel head coil and single-shot gradient echo echo-planar imaging (repetition time = 3.4 seconds, echo time = 30 ms, flip angle = 85° , field of view = 210 mm, 51 2.5-mm thick AC-PC parallel). Each scan lasted six minutes and yielded 178 volumes. T1-weighted structural scans (TR = 2530 ms, TE = 2.77 ms, flip angle = 7°, field of view = 256 mm^2 ,1.0-mm thick slices) were also acquired.

2.4 Analytic Approach

2.4.1 Factor Analysis Approach

The data have a smaller amount of participants than are usually used for factor analysis. For these reasons, some would warn against the use of factor analysis and instead recommend principal components analysis. However, principal components analysis is a data reduction method meant for the reduction of a priori known correlated variables into a smaller meaningful set that is easier to work with. Certainly it is known a priori that many of the traits in the present data are correlated, so this does not seem entirely unreasonable. However, factor analysis is the appropriate method to identify what theoretical variables might be driving these correlations (Bandalos & Boehm-Kaufman, 2009). Investigations of various sample size recommendations have found them to be largely unfounded. For example, the recommendation that the number of observations per variable should be greater than the number of variables has born out to be of less importance than the number of variables per latent factor. It has been shown that increasing the number of variables relevant to the latent factors of interest results in better estimates of the latent factors for the same sample size (Velicer & Fava, 1999).

Alpha factoring, a method of factor analysis that fits factors based on maximizing Cronbach's alpha, is used here (Kaiser & Caffrey, 1965). Alpha factoring shares in common with least squares methods the lack of distributional assumptions, while maximum likelihood assumes multivariate normality (Kaiser & Derflinger, 1990; MacCallum & Browne, 2007). Alpha factoring differs from least squares and maximum likelihood in its treatment of error. Instead of treating error as originating from individual measurement error, error is treated as originating from the fact that there is a theoretical pool of all possible relevant vari-

ables and any given factor analysis only has sampled a portion of these variables (MacCallum & Browne, 2007).

Any factor analysis requires a covariance or correlation matrix. However, the Pearson correlation is highly non-robust and vulnerable to small changes in the marginal distributions (Wilcox, 1994). Outliers frequently cause Pearson's method to overestimate ρ. The percentage-bend correlation coefficient, ρ_{pbcor} is less vulnerable to errors of magnitude and has a minimal loss of power under conditions of normality (Wilcox, 1994; Pernet, Wilcox, & Rousselet, 2012). The percentage-bend correlation is correlated by down-weighting a specified percentage of observations deviating from the median and computing Pearson's ρ on the weighted data (Pernet et al., 2012). Given the relatively small sample size of the current study compared to that traditionally used in factor analysis individual measurement error is still of concern to the author. For this reason, the correlation matrix of personality traits is estimated using ρ_{pbcor}.

The next methodological decision concerns the method of factor rotation. Orthogonal rotations are the most commonly used methods, which rotates recovered factors such that they have minimal correlation. However, there are theoretical reasons to suspect that RST related factors are not uncorrelated. Heightened sensitivity of the BIS to goal conflict would produce a tendency towards fearful behavior through FFFS activation even when direct FFFS sensitivity is not the underlying cause of fearful behavior (McNaughton & Corr, 2004; McNaughton et al., 2016). Therefore, it is unlikely that separable factors for the BIS and FFFS would be recovered from a factor analysis of existing trait measures not designed to measure them separately. However, even if this turns out to be the case it is expected that FFFS and BIS factors would be correlated rather than orthogonal. It is however possible that traits related to functions of the FFFS such as (dis)agreeableness

and urgency may comprise a factor related to the FFFS separately measurable from the BIS.

Another reason to expect obliqueness in factors is that BIS sensitivity would bias an individual towards passive avoidance behavior and consequently less behavioral approach. For this reason Gray originally postulated the BIS factor as being at a 45 to 30 degree rotation from Neuroticism, corresponding to Neurotic-Introversion. Therefore, one might expect a BAS trait to be negatively correlated with measures of the BIS & FFFS. Furthermore, if the data suggest that the latent factors are orthogonal, an oblique rotation will result in orthogonal results. Oblique rotations permit, but do not force, correlated factors (Bandalos & Boehm-Kaufman, 2009).

2.4.2 Regression Modeling

One initially considered strategy was to utilize stepwise regression to test the hypotheses. However, stepwise regression is prone to false positives. Another considered strategy was to utilize a penalized regression method such as LASSO or ridge regression on all 294 nodes. This was decided against because the expected effect sizes for the hypothesized predictors were expected to be small and potentially missed due to the strong shrinkage of each coefficient towards zero. Instead, a Bayesian approach was taken.

2.5 Graph Theory

A graph is a set of nodes connected by edges. The nodes are vertices, which represent entities capable of bearing relationship to other vertices in the graph, and the edges representations of their relationships. In functional brain imaging

these nodes can be defined as voxels or regions which correlate with one another by virtue of their BOLD signal. This can be used to characterize which brain regions communicate with one another. Low frequency fluctuations in the range 0.01–0.1 Hz characterize resting state networks. BOLD signal fluctuations in this range are negatively correlated with the brain when engaged in a task (Fox et al., 2005; Fransson, 2005). These resting state networks have been characterized through other methods such as independent components analysis or seed-based networks. While these approaches can characterize which functional networks exist or the correlation of a seed region with other network components graph theory can provide richer information about the network as a whole and the individual nodes in a network.

The most basic graph theoretic measures are degree and strength. Degree characterizes the number of connections a node has in a binary network. The counterpart in a weighted network is strength, which adds the connection weights (correlations in the case of an fMRI network). Strength has two components, positive strength and negative strength. Positive strength adds the positive connection weights together and negative strength adds the negative connection weights. These can be combined into strength*. The formula from this is given in Equation 1 (Rubinov & Sporns, 2011). The formula calculates a weighted sum of both positive and negative edges, giving the positive edges more weight because they are typically of more interest.

$$S^* = S^+ - \left(\frac{S^-}{S^- + S^+}\right) S^- \tag{1}$$

While strength* offers a straightforward measure of whole-brain network integration for a given node, graph theory can also provide higher order information

about the connectivity of a network. Consider for instance the concept of centrality. One measure of how central a node is to the network is eigenvector centrality. Eigenvector centrality accounts for both the number and strength of a node's connections. It also considers both the strength of a given node and the strength of its neighbors. An eigenvector is a vector x that when multiplied by a connectivity matrix M is equal to $M \cdot x = \lambda \cdot x$. λ is the eigenvalue that can be multiplied by x to obtain $M \cdot x$. The eigenvector centrality for $node_i$ sums together the values of the multiplier x (also called the individual centrality x for each other node in the network. The equation for this is given in Equation 2 (Newman, 2010; Fornito, Zalesky, & Bullmore, 2016).

$$C_{Eigen} = \frac{1}{\lambda_1} \sum_{j=1}^{n} M_{ij} x_j \tag{2}$$

What does Eigenvector centrality offer that strength* does not? Practically, a node with a high eigenvector centrality is important to the overall network. This is either because it is highly connected to many nodes or it shares connections that are themselves highly connected. Consider that the CEO of a company may only frequently communicate with a few higher-up workers in the company. Despite the small number of direct connections the CEO has, the CEO is highly central to the company. If you only consider the people with whom the CEO directly communicates with on a regular basis the strength of his or her connections to the company might appear weak, but if you consider that those individuals can quickly spread any decisions the CEO has made to the company then the CEO appears highly connected. Eigenvector centrality does the latter.

2.5.1 fMRI Preprocessing

The data were pre-processed using FMRIB's FSL program. (Jenkinson & Smith, 2001; Jenkinson et al., 2002) Pre-processing geometric distortion correction and rigid body head motion correction using MCFLIRT. 3 subjects were dropped from the analysis due to excessive head motion. The remaining sample consisted of 23 adolescents (13 female, 10 male) and 51 adults (31 female, 20 male).

Following this, the time series for each subject was extracted for 294 regions. These regions consist of the 264 regions in the Power atlas (2011) and 30 additional nodes added for limbic and paralimbic regions based on potential theoretical interest.

2.5.2 Graph Theory Processing

Each subject's resting state time series and confound matrix was processed in MATLAB. (MathWorks, 2012) This removes points in the time series where excessive head motion is present. Following this the covariance for each subject was calculated. Shrinkage was used due to the fact that the number of nodes outweighs the number of subjects. Maximum likelihood estimation is known to be inefficient in these situations, so a shrinkage estimator based on the Ledoit-Wolf lemma was used. Partial correlations were then obtained for each subject's partial covariance matrix. The Brain Connectivity Toolbox (BCT) was then used to calculate strength and Eigenvector centrality for each node.

2.6 A Crash Course in Bayesian Methods

The majority of model fitting in this paper will utilize Bayesian methods. Bayesian methods have traditionally been utilized far less than classical frequentist meth-

ods. For the reader unfamiliar with the contrast between frequentist and Bayesian methods, a relatively brief account is given here. Central to all statistical inference is the concept of likelihood. The likelihood describes a value proportional to the probability of the data given certain parameter values. The likelihood can be thought of as equal to the probability of the data given the model multiplied by an imaginary arbitrary constant $\boldsymbol{K}$, as shown in Equation 3 (Etz, 2017).

$$L(\theta|\mathbf{D}) = P(\mathbf{D}|\theta) * \boldsymbol{K} \tag{3}$$

Consider that the conditional probability of the data given the model has multiple mathematically equivalent statements, given in Equation 4. The $P(\mathbf{D}|\theta)$ ends up being equivalent to the conditional probability of the model itself multiplied by the proportion $\frac{P(\mathbf{D})}{P(\theta)}$. The imaginary constant $\boldsymbol{K}$ removes this multiplier leaving the conditional probability of the model, although it is no longer normalized to be a probability and is now a likelihood.

$$P(\mathbf{D}|\theta) = \frac{P(\mathbf{D} \cap \theta)}{P(\theta)} = \frac{P(\theta|\mathbf{D})P(\mathbf{D})}{P(\theta)} \tag{4}$$

Conceptually, the likelihood of a model or parameter given the data means little without comparison. This comparison is called a likelihood ratio. This results in very different uses for likelihoods and probabilities; likelihoods do not inform about absolute chance because the scale is arbitrary. By contrast, a probability statement stands on its own. In frequentist inference probability is used to describe the behavior of systems which generate random frequencies. For example, die roll, coin flips, or a test statistic under the null hypothesis are random types of data and can be described with probability.

Bayesians treat probability with a different perspective. Bayesians believe

probability describes the uncertainty one has in a model over the range of possible values. The data are treated as fixed values, while the possible parameter values are treated as random. This perspective was founded when Thomas Bayes and Pierre-Simon Laplace realized the probability of the model given the data can be obtained by solving the furthermost right form of Equation 4 for $P(\theta|\mathbf{D})$ (Bayes & Price, 1763; Laplace, 1774, 1810, 1814). This results in Bayes' Theorem, given in Equation 5. To do this, assumptions must be made by defining a range of possible which cover what is presumed to be the entire parameter space (so that mathematically it integrates to 1). This is called the prior and is represented in Equation 5 by $P(\theta)$. The conditional probability of the model given the data is called the posterior probability $P(\theta|\mathbf{D})$. The posterior is the product of the prior and the conditional probability of the data given the model, normalized by $P(\mathbf{D})$ which defines the probability of the data at random. In essence, Bayesian methods begin with initial assumptions and learn from the data.

$$P(\theta|\mathbf{D}) = \frac{P(\mathbf{D}|\theta)P(\theta)}{P(\mathbf{D})} \tag{5}$$

An Applied Example

The best way to understand Bayesian inference is by example. This example will use the data provided by The data for this analysis are provided from the machine learning repository (Dheeru & Karra Taniskidou, 2017). For now, consider the case where one wishes to estimate the average alcohol content of wine with red and white wines pooled together. The model will assume the data are normally distributed and utilize a wide prior to describe the range of possible parameter values for the mean. A half Student-t(df=3, location=0, scale=10) distribution will be used to describe the range of possible standard deviations in the data, and a

normal(0,100) distribution used for the intercept and coefficient intercept.

After setting up the model and updating it on the data, you find the following results:

			95 % Credible Interval	
	Estimate	**Estimate Error**	**Lower**	**Upper**
$\hat{\mu}$	10.49	0.01	10.46	10.52
$\hat{\sigma}$	1.19	0.01	1.17	1.21

The column labeled **Estimate** gives the mean of the posterior distribution, which is the most probable parameter value. The standard deviation of the posterior distribution gives the **Estimate Error**, which describes the uncertainty in the estimate. This is analogous to the standard error of classical statistics, and is often just called standard error. However, the posterior estimate error reflects not only sampling error but also uncertainty in the prior.

The 95% credible interval defines the region centered around the estimate and contains values which collectively make up 95% of the posterior distribution's mass. (see Figure 2) For any credible region you can state that the true parameter lies within the interval with 1-$\alpha \cdot 100\%$ probability. This is in contrast to the frequentist confidence interval's interpretation under the repeated sampling principle, which does not permit a probability statement about the numbers within the interval, only the interval itself. A confidence interval is like a flip of a coin, either succeeding or failing to cover the true value with some probability.

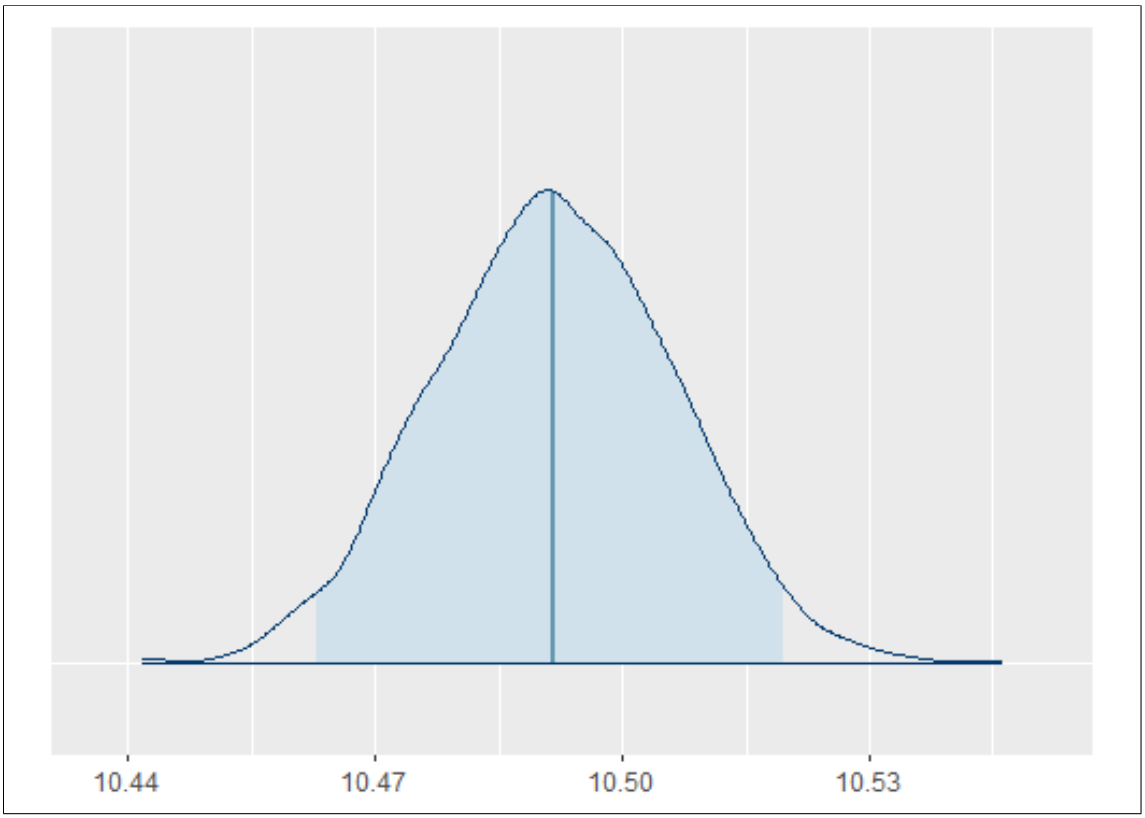

Figure 2: Posterior Distribution with shaded 95% credible region. The Bayesian counterpart to the confidence interval is an area of the posterior probability distribution. Intuitively, there is a 95% probability that the true parameter value lies in this region.

Linear Regression

A model estimating the mean is easily expanded to the case of linear regression. The R package brms (Bayesian Regression Models using Stan) provides a standard linear model syntax interface to the Stan program, which uses a type of monte carlo method called No U-Turn Sampling (NUTS) to fit Bayesian models (Bürkner, 2017; Stan Development Team, 2018). In the syntax of linear models in R, estimating the mean of a dependent variable is accomplished using the syntax DV $\sim$ 1. This defines a linear model with only an intercept, which is the mean of

the dependent variable when all predictors are zero. Since there are no predictors, the intercept is just the mean. To create a linear regression model an independent variable is added. Suppose we hypothesize that the amount of residual sugar has a negative relationship with the amount of alcohol due to the fact that ethanol is created from microorganisms fermenting sugar. The variable is log-normally distributed, so the natural log of the predictor is entered into the linear model. The results are given below.

			95 % Credible Interval	
	Estimate	**Estimate Error**	**Lower**	**Upper**
Intercept	11.06	0.03	11.01	11.11
log(sugar)	-0.43	0.02	-0.46	-0.39

Hypothesis Testing

If one wishes to test the hypothesis that the relationship of the logarithm of residual sugar is zero the simplest way is to check the interval for zero. This procedure is the same as the Neyman-Pearson hypothesis testing procedure with confidence intervals. When the interval excludes the null value, we reject the null hypothesis at confidence level 1-α. This is equivalent to having a p-value smaller than α.

Another way to test hypotheses is with the Bayes Factor. The Bayes Factor is a likelihood ratio that quantifies the level of evidence for or against a hypothesis. They can be calculated in a couple ways depending on the context. In model comparison the Bayes Factor compares the likelihood functions for two different models. One would fit each model separately and compare them to find out under which model the data are more probable. The mathematical statement is given below in Equation 6.

$$BF_{H_1} = \frac{P(\mathbf{D}|\theta, H_1)}{P(\mathbf{D}|\theta, H_2)} \tag{6}$$

Another way to calculate a Bayes Factor is to treat the prior distribution as one model and the posterior distribution as the second model. For a given parameter value the degree of change in probability between the prior and posterior defines the Bayes Factor. This is called the Savage-Dickey Bayes Factor.

$$BF_{H_1} = \frac{P(\theta|\mathbf{D})}{P(\theta)} \tag{7}$$

A one sided Bayes Factor is also attainable. The method used by brms to calculated one sided Bayes Factor is given in Equation 8. It is equivalent to the ratio of the posterior density above (below) the hypothesized value to the density below (above) the hypothesized value.

$$\frac{\int_0^\infty \frac{p(\mathbf{D}|\theta)p(\theta)}{\int_{-\infty}^\infty p(\mathbf{D}|\theta)p(\theta)}}{\int_{-\infty}^0 \frac{p(\mathbf{D}|\theta)p(\theta)}{\int_{-\infty}^\infty p(\mathbf{D}|\theta)p(\theta)}} = \frac{\int_0^\infty p(\mathbf{D}|\theta)p(\theta)}{\int_{-\infty}^0 p(\mathbf{D}|\theta)p(\theta)} = BF_{\theta>0 \text{ vs. } \theta<0} \tag{8}$$

Bayes Factors are interpreted along a continuum, and are not described by a significance level such as α for p-values and confidence intervals. Nevertheless, interpretative guidelines are given in Table 3 based on the ones provided by Jeffreys (1939).

In the regression example, the (Savage-Dickey) Bayes Factor for the null hypothesis that the relationship between log(sugar) and alcohol is zero is $BF_{null} = 1.3754^{-16}$. This indicates decisive evidence against the null hypothesis. The Bayes Factor for the existence of an effect is simply $1/BF_{null}$. Hence, $BF_{effect} = 7.270612^{15}$. The one sided Bayes Factor corresponding to the hypothesis that the effect is less than zero is returned as infinity because the entire posterior distribution lies be-

$\mathbf{BF_H}$	Strength of Evidence
BF <.01	Conclusive Against
.03 - .01	Very Strong Against
.10 - .03	Strong Against
.33 - .10	Notable Against
1 - .33	Anecdotal Against
1	Inconclusive
1 - 3	Anecdotal
3-10	Notable
10-30	Strong Evidence
30-100	Very Strong
100 <BF	Conclusive

Table 3: Bayes Factor Interpretive Guidelines

low zero.

The 'Problem' of Multiple Comparisons

When considering type I error rates, the probability of making at least one type I error increases dramatically with each additional statistical test. Assuming the independence of each test, if 20 tests are conducted at $\alpha = .05$ the probability of making at least one type I error is $1 - (1 - \alpha = .05)^{m=20} \approx 64\%$ This is known as the family wise error rate (FWER). The prototypical methods of FWER correction include the Bonferroni and Sidák procedures (Equations 9). These formulas adjust α such that the overall type I error rate is controlled. Confidence/Credibility levels are represented as "$1 - \alpha$" and the number of hypotheses being tested as $\mathbf{m}$.

$$\text{Bonferroni's Correction:} 1 - \alpha_{adjusted} = 1 - \frac{\alpha}{\mathbf{m}}$$
$$\text{Sidák's Correction:} 1 - \alpha_{adjusted} = (1 - \alpha)^{\frac{1}{\mathbf{m}}} \tag{9}$$

These adjustments are but two of the many approaches to multiple comparisons corrections, but are the most straightforward to apply to confidence intervals. When correcting intervals for multiplicity these adjustments widen the intervals such that the false coverage rate (Type I error) is the nominal α. This occurs because the interval is more likely to include the null value when it is widened.

Considered from a Bayesian perspective there are downsides to this approach. Since a credibility interval is a density, increasing the width of a credibility interval from $1-\alpha\%$ to $1-\alpha_{adjusted}\%$ means asymptotically describing the minimum and maximum values in posterior distribution (a 100% Confidence Level). To this end there is little point in constructing a credible interval. There are also computational reasons why such adjusted intervals are ill-advised in the Bayesian approach. Virtually all model fitting is done using Markov-Chain Monte Carlo or other methods that result in samples from the posterior distribution. If constructing a 95% Credible Interval, there are $.025n$ samples outside the center 95% density from which to estimate the credible limits. However, if using an adjusted confidence level of 99.75%, $.00125n$ samples are available for estimating the credible limits. If the Markov chain has even a generous 20,000 samples only 25 are available to estimate the lower and upper credible limits for the adjusted interval. 20,000 samples for many models would require a large amount of time and processing power and the sparseness of the posterior tails would result in unreliable credible intervals.

Another issue with multiple comparisons corrections from a Bayesian perspective the number of intervals constructed in a family of analyses doesn't change what the probability of each of the estimates in the interval are. The only sensible adjustment to make to a credible interval is to switch from an equal tailed interval

to a highest density interval in the case of a skewed posterior distribution to ensure the most probable parameter values are summarized. This is an issue quite separate from multiplicity.

Another issue to consider is the motivation behind the concept multiple comparisons correction. The philosophy of multiple comparisons corrections aims to solve the problem of making at least one type I error increasing with each test. The majority of approaches consider the "family wise error rate", but the concept of a family of tests is vaguely defined. If the probability of a type 1 error increases with each hypothesis test, should the same correction be applied to every test in a paper (Perneger, 1998)? Should it be done by the number of tests conducted in each published journal issue? One can make this argumentum 8ad absurdum, but a more important issue is that errors are made because the data have mislead, and there are two ways the data can mislead you. These are exaggerated effect sizes, which are errors of magnitude, and effect size estimates with the wrong sign. These are called type M and type S errors respectively (Gelman & Tuerlinckx, 2000; Gelman, Hill, & Yajima, 2012; Gelman & Carlin, 2014). Inflated intervals do not change the fact that if it fails to cover the true θ it has done so because the estimate is of the wrong magnitude or wrong sign. Furthermore, the multiplicity adjusted confidence intervals depend on the intentions of the researcher or analyst. Entirely different decisions can be made for the same estimate after an analysis depending on how many tests are run. Simulation studies have also found that given a statistically significant result, the Bayesian credible intervals on average produce fewer type M and type S errors compared to the standard counterpart frequentist estimator (Gelman & Tuerlinckx, 2000; Gelman et al., 2012; Gelman & Carlin, 2014). By necessity, this means the type I error is also controlled to a greater extent.

In a frequentist framework, the false discovery rate methods of correction offer conceptual benefits over family-wise error rate control by controlling for the estimated number of false discoveries rather than controlling for the overall probability of making at least one type I error (Benjamini & Hochberg, 1995; Benjamini, 2010). False discovery rate control also has connections to certain Bayesian approaches such as the empirical Bayes approach. The empirical Bayes approach is related to penalized maximum likelihood approaches and works by estimating the optimal prior distribution from the data (Efron & Tibshirani, 2002; Storey, 2003). However, Bayesian methods provide incredible flexibility in estimator construction beyond empirical Bayes. By constructing estimators that address the possibility of aberrant estimates caused by outliers the likelihood of making a false claim is reduced without the need for post-hoc comparisons that fail to address the underlying cause of error. Addressing such concerns in the prior distribution itself is a sensible way to address the possibility of inflated type I errors from a Bayesian perspective (Westfall & Utts, 1997; Berry & Hochberg, 1999).

2.6.1 Regression Models

Values for Strength* and Eigencentrality were both converted to z-scores in order to be on the same scale as the factor scores. This makes formulating prior distributions simpler and interpretation also easier. This is important because the untransformed values for graph theory metrics could be different depending on the particular methods used.

Each model is coded such that the young adults are used as the reference level ("dummy coding"). Interaction terms contain the difference in the effect of an independent variable from the reference level. The actual effect in of the strength of a particular node in adolescents is For ease of interpretation the main regression

tables report the total effect of a given node $\beta_{roi|Teen} = \beta_{roi} + \beta_{Teen \cdot roi}$. The interactions, which represent the difference from the young adult reference group, are reported in the hypothesis testing tables. Models are fit using the R package brms package, an interface to rstan (Bürkner, 2017; Stan Development Team, 2018).

Highest density intervals (HDIs) are also reported. Since brms only reports equal tailed intervals (ETIs), the broom package is used to obtain the HDIs. HDIs in the case of a normally distributed posterior distribution are equivalent to ETIs. In the case of a skewed or otherwise non-gaussian posterior the HDI better represents the values which are most probable. In text, one sided hypothesis tests are presented as 90% HDIs. The reason for this is that for a one sided hypothesis test the lower (upper) bound of the 90% credible interval defines the limit of the one sided credible limit. For example, the lower bound of the 90% credible interval defines the 95% credible limit for $H : \beta > 0$. Bayes Factors for the hypothesis tests are obtained with brms.

3 Results

3.1 Factor Analysis Results

25 adolescents (14 female, 11 male) and 52 adults (32 female, 20 male) completed a battery of personality tests. Of these factor scores were obtained for 23 adolescents (13 female, 10 male) and 51 adults (31 female, 20 male) due to three subjects being excluded from analysis due to excessive head motion. Missing values for any traits entered into the factor analysis were imputed using the means of the remaining scores.

The number of factors that could be extracted was determined with parallel

analysis. Parallel analysis compares the eigenvalues of extracted factors against the eigenvalues of re-sampled data (Bandalos & Boehm-Kaufman, 2009). Factors with eigenvalues that lie outside the error bars of the re-sampled data are used to determine the cutoff. The scree plot indicates that four factors can be extracted from the current data (see Figure 3). Parallel analysis and alpha factor analysis were conducted in R using the psych package (Revelle, 2017).

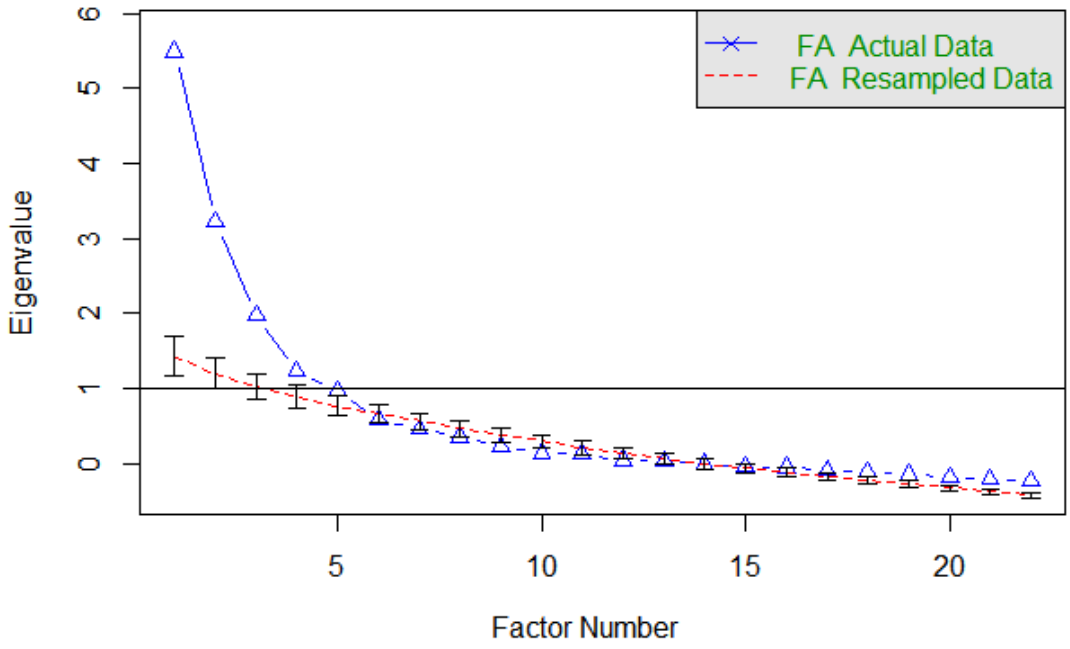

Figure 3: Parallel analysis scree plot showing the number of factors which can be extracted. The decision method compares the eigenvalues < 1 against eigenvalues from re-sampled data and counts those which lie outside of the bootstrapped error bars. This ensures greater reliability than a standard scree plot.

Results of the factor analysis are summarized in Table 4. Factor scores were extracted with the Ten Berge method. Names were given on the basis of the strongest factor loadings to relate them to RST systems believed to underly individual differences in personality. Threat aversion is the name given to the trait corresponding to the BIS and FFFS. Impulsivity is the name given to the trait

corresponding to the ECS. Two BAS traits emerged which are given the names Novelty Seeking and Reward Dependence. Conceptually, these correspond to the seeking out of novel stimuli and tendency to work towards known rewards, corresponding to the two reward traits in Cloninger's personality model (Cloninger, 1986).

Descriptive statistics in Table 5. Accompanying boxplots give a graphical summary in Figure 4. Because the data came from a previous study where subjects were recruited based on being higher or low sensation seekers there was a possibility that the BAS trait factor scores had a bimodal distribution. The mean and standard deviations of the novelty seeking scores for the high sensation seeking condition are M = 0.53 and SD = 0.72; the mean and standard deviation for the low sensation seeking condition M = -0.53 and SD = 0.97. The mean and standard deviations of the reward dependence scores for the high sensation seeking condition are M = 0.41 and SD = 0.95; the mean and standard deviation for the low sensation seeking condition M = -0.41 and SD = 0.88.

	Factor a	Factor b	Factor c	Factor d
1. BIS	-0.035	-0.164	**0.613**	0.104
2. REWARD (BAS)	0.134	-0.360	0.175	**0.494**
3. DRIVE (BAS)	0.240	-0.116	0.092	**0.505**
4. FUN (BAS)	**0.641**	-0.135	-0.106	0.169
5. EXTRAVERSION (EPI)	0.295	0.384	-0.076	**0.587**
6. NEUROTICISM (EPI)	0.133	0.111	**0.768**	-0.049
7. DISINHIBTION (SSSV)	**0.815**	0.032	0.095	-0.186
8. BOREDOM SUSCEPTIBILITY (SSSV)	**0.463**	0.182	-0.102	-0.014
9. THRILL SEEKING (SSSV)	**0.698**	0.041	-0.119	0.071
10. EXPERIENCE SEEKING (SSSV)	**0.729**	-0.069	0.014	-0.216
11. OPEN-MINDEDNESS (BIG5)	0.455	**-0.540**	0.024	-0.167
13. CONSCIENTIOUS (BIG5)	-0.026	**-0.709**	-0.106	0.279
14. EXTRAVERSION (BIG5)	-0.207	-0.050	-0.215	**0.729**
15. AGREEABLENESS (BIG5)	0.018	**-0.431**	-0.190	-0.046
16. NEUROTICISM (BIG5)	-0.211	0.134	**0.764**	-0.028
17. URGENCY (UPPS)	0.089	**0.595**	0.397	0.249
18. LACK OF PREMEDITATION (UPPS)	0.149	**0.592**	-0.297	0.181
19. LACK OF PERSEVERANCE (UPPS)	0.023	**0.595**	0.015	-0.199
20. SENSATION SEEKING (UPPS)	**0.794**	-0.080	-0.084	0.109
21. SENSATION SEEKING (ZKPQ)	**0.902**	0.030	-0.016	0.020
22. PUNISHMENT SENSITIVITY (SPSRQ)	-0.049	0.113	**0.660**	-0.288
23. REWARD SENSITIVITY (SPSRQ)	**0.498**	0.207	0.240	0.252
SS loadings	4.63	2.551	2.53	2.042
Factor Correlations				
a Novelty Seeking (BAS)	1.000	0.026	0.033	0.487
b Impulsivity (ECS)	0.026	1.000	0.121	-0.003
c Threat Aversion (BIS + FFFS)	0.033	0.121	1.000	-0.062
d Reward Dependence (BAS)	0.487	-0.003	-0.062	1.000
Tucker Lewis Index of factoring reliability = 0.742 ; BIC = -382.5				

Table 4: Factor loadings for alpha factor analysis with accompanying factor correlations. In bold are the largest factor loadings for a given variable.

	Adults				Adolescents			
	Mean	SD	Median	MAD	Mean	SD	Median	MAD
Novelty Seeking	0.019	1.012	0.175	1.133	-0.043	0.992	0.152	0.763
Impulsivity	-0.181	0.921	-0.351	0.970	0.403	1.069	0.507	1.273
Aversion	-0.059	1.059	-0.316	1.112	0.130	0.860	-0.095	0.952
Reward Dependence	-0.077	1.027	0.021	1.174	0.171	0.934	0.178	0.823

Table 5: Descriptive statistics by age group for the personality scores of each extracted factor.

Figure 4: Boxplots of factor scores by age group. The boxplots suggest that adolescents are more impulsive than young adults.

3.2 Regression Results

3.2.1 Impulsivity

Given that the distribution of the Impulsivity factor scores appears normal the scores from individual subjects are assumed to come from a normal distribution. The mean of this distribution is defined by the regression formula. The inter-

cept β_0 and each coefficient β_n are assumed to come from a t-distribution with 3 degrees of freedom, a mean of zero, and standard deviation of .5. This prior is chosen to regularize estimates without apply excess shrinkage. The effect sizes are expected to be fairly small, so excessive shrinkage would result in a loss of power, but some regularization is desired to avoid type M errors. The distribution for the effect of age is a t-distribution with 3 degrees of freedom, a mean of 1, and standard deviation of .25. This is to reflect that adolescents are expected to be more impulsive than young adults. Finally, the standard deviation of Impulsivity is assumed to come from a gamma distribution with shape and rate parameters equal to 2. This results in a prior distribution that places the bulk of probability over values between about .5 and 2 with larger values being increasingly less probable. This is well justified because it is known that the standard deviation of the sample is 1.

$$y_i \sim normal(\mu, \sigma)$$

$$\mu = \beta_0 + Age_0 + \sum_n \beta_n x_n + \sum_{n \cdot Age} \beta_n x_n \cdot Age$$

$$\beta_0 \sim t\ distribution(\nu = 3, \mu = 0, \sigma = .5)$$

$$Age_0 \sim t\ distribution(\nu = 3, \mu = 0, \sigma = .25)$$

$$\beta_n \sim t\ distribution(\nu = 3, \mu = 0, \sigma = .5)$$

$$\beta_n \cdot Age \sim t\ distribution(\nu = 3, \mu = 1, \sigma = .25)$$

$$\sigma \sim gamma(s = 2, r = 2)$$

Impulsivity - Strength* Results

Multiple regression analysis was used to test if the Impulsivity trait was predicted by the strength of the functional connectivity of brain regions outlined in Table

1. The results of the regression indicated the predictors explained 32.9% of the variance (R2 =.329, LOO-IC=202.22). In young adults a node in the left dorsal frontal cortex predicted lower impulsivity ($\hat{\beta}$ = -0.362, 90% HDI= [-0.576, -0.152], $BF_{\beta<0}$ = 387.35). There was also a significant age group by node interaction in the right medial frontal cortex indicating the node predicted significantly less impulsivity compared to adults ($\hat{\beta}$ = -0.629, 90% HDI= [-1.026, -0.629], $BF_{\beta<0}$ = 257.065). The effect estimate of the effect in teenagers is significant, indicating that this region is not related in adults but is related in adolescents ($\hat{\beta}_{teen}$ = -0.583, 90% HDI= [-.916, -0.233]). This is an important point because it is feasible for the estimate of the interaction (which is the difference in effect compared to adults) to yield an effect estimate that is not significant. Results are summarized in Table 6 and Table 7.

	$\hat{\beta}$	**Est.Error**	90 % HDI		
Young Adults					
Intercept	-0.223	0.117	-0.416	-0.033	
R Dorsal Frontal Cortex	0.063	0.130	-0.151	0.278	
L Dorsal Frontal Cortex	-0.362	0.129	-0.576	-0.152	*
R Medial Frontal Cortex	0.046	0.124	-0.159	0.246	
L Medial Frontal Cortex	0.139	0.116	-0.053	0.330	
Adolescents					
TEEN	0.841	0.183	0.549	1.149	
R Dorsal Frontal Cortex	-0.046	0.185	-0.347	0.259	
L Dorsal Frontal Cortex	-0.128	0.173	-0.414	0.153	
R Medial Frontal Cortex	-0.583	0.207	-0.916	-0.233	*
L Medial Frontal Cortex	0.073	0.216	-0.283	0.426	
Family Parameters					
$\hat{\sigma}$	0.887	0.079	0.758	1.014	
LOO-IC = 202.22; WAIC=201.365; R2=0.329					
'*': Estimate is credibly non-zero with 90% probability					

Table 6: Parameter estimates for the relationship of each node's Strength* to the Impulsivity trait in each age group. Note that the adolescents shows the overall parameter estimate, rather than the deflection from the young adults.

	$\hat{\beta}$	**Est.Error**	**BF**	
R Dorsal Frontal Cortex <0	0.063	0.130	0.451	
L Dorsal Frontal Cortex <0	-0.362	0.129	387.35	⋆
R Medial Frontal Cortex <0	0.046	0.124	0.551	
L Medial Frontal Cortex <0	0.139	0.116	0.130	
TEEN:R Dorsal Frontal Cortex <0	-0.110	0.213	2.327	
TEEN:L Dorsal Frontal Cortex <0	0.234	0.206	0.146	
TEEN:R Medial Frontal Cortex <0	-0.629	0.238	257.07	⋆
TEEN:L Medial Frontal Cortex <0	-0.067	0.234	1.587	
⋆: The evidence is in favor of the hypothesis by a factor of at least 10				
†: The evidence is against of the hypothesis by a factor of at least 10				

Table 7: Bayes Factor tests for the relationship of each node's Strength* to Impulsivity. Note that the tests shown here are for the interactions, which represent the deflection from the young adult group.

Impulsivity - Eigenvector Centrality

Multiple regression analysis was used to test if the Impulsivity trait was predicted by the eigenvector centrality of brain regions outlined in Table 1. However, none of the eigenvector centrality of the nodes significantly predicted Impulsivity. Results are summarized in Table 8 and Table 9.

	$\hat{\beta}$	**Est.Error**	90 % HDI		
Young Adults					
Intercept	-0.239	0.128	-0.450	-0.029	*
R Dorsal Frontal Cortex	-0.071	0.131	-0.288	0.145	
L Dorsal Frontal Cortex	0.146	0.123	-0.055	0.350	
R Medial Frontal Cortex	-0.112	0.135	-0.329	0.113	
L Medial Frontal Cortex	0.014	0.130	-0.203	0.222	
Adolescents					
TEEN	0.804	0.202	0.479	1.136	*
R Dorsal Frontal Cortex	-0.179	0.225	-0.551	0.191	
L Dorsal Frontal Cortex	0.014	0.250	-0.401	0.421	
R Medial Frontal Cortex	0.158	0.195	-0.162	0.482	
L Medial Frontal Cortex	0.300	0.261	-0.125	0.734	
Family Parameters					
$\hat{\sigma}$	0.989	0.087	0.859	1.143	
LOO-IC = 218.343; WAIC=217.655; R2=0.212					
'*': Estimate is credibly non-zero with 90% probability					

Table 8: Parameter estimates for the relationship of each node's eigenvector centrality to the Impulsivity trait in each age group. Note that the adolescents shows the overall parameter estimate, rather than the deflection from the young adults.

	$\hat{\beta}$	**Est.Error**	**BF**
R Dorsal Frontal Cortex <0	-0.071	0.131	2.413
L Dorsal Frontal Cortex <0	0.146	0.123	0.131
R Medial Frontal Cortex <0	-0.112	0.135	3.986
L Medial Frontal Cortex <0	0.014	0.130	0.837
TEEN:R Dorsal Frontal Cortex <0	-0.108	0.246	2.023
TEEN:L Dorsal Frontal Cortex <0	-0.132	0.264	2.259
TEEN:R Medial Frontal Cortex <0	0.270	0.227	0.128
TEEN:L Dorsal Frontal Cortex <0	0.286	0.280	0.177
⋆: The evidence is in favor of the hypothesis by a factor of at least 10			
†: The evidence is against of the hypothesis by a factor of at least 10			

Table 9: Bayes Factor tests for the relationship of each node's eigenvector centrality to Impulsivity. Note that the tests shown here are for the interactions, which represent the deflection from the young adult group.

3.2.2 Threat Aversion

Given that the distribution of the Threat Aversion factor scores appear skewed the scores from individual subjects are modeled here to come from a skewed normal distribution. The mean of this distribution is defined by the regression formula. The intercept β_0 and each coefficient β_n are assumed to come from a t-distribution with 3 degrees of freedom, a mean of zero, and standard deviation of .5. This prior is chosen to regularize estimates without apply excess shrinkage. The distribution for the effect of age is a t-distribution with 3 degrees of freedom, a mean of .25, and standard deviation of .25. This is to reflect that adolescents are expected to be weakly more threat averse than adults. This is supported by a large scale study of personality over the lifespan (Donnellan & Lucas, 2008). The standard deviation of Threat Aversion is assumed to come from a gamma distribution with shape and rate parameters equal to 2. Finally, the distribution of the skewness parameter α is set at the brms default of a normal distribution with mean 0 and standard deviation of 4.

$$y_i \sim skewed\ normal(\mu, \sigma, \alpha)$$

$$\mu = \beta_0 + Age_0 + \sum_n \beta_n x_n + \sum_{n \cdot Age} \beta_n x_n \cdot Age$$

$$\beta_0 \sim t\ distribution(\nu = 3, \mu = 0, \sigma = .5)$$

$$Age_0 \sim t\ distribution(\nu = 3, \mu = 0, \sigma = .25)$$

$$\beta_n \sim t\ distribution(\nu = 3, \mu = 0, \sigma = .5)$$

$$\beta_n \cdot Age \sim t\ distribution(\nu = 3, \mu = .25, \sigma = .25)$$

$$\sigma \sim gamma(s = 2, r = 2)$$

$$\alpha \sim normal(\mu = 2, \sigma = 4)$$

Threat Aversion - Strength*

Multiple regression analysis was used to test if the Threat Aversion trait was predicted by the strength of the functional connectivity of brain regions outlined in Table 1. The results of the regression indicated the predictors explained 25.9% of the variance (R2 =.259, LOO-IC=210.455). In young adults a node in the left dorsal amygdala significantly predicted lower threat aversion. The direction of prediction was opposite of the hypothesis that the strength of the node would predict higher threat aversion ($\hat{\beta}$ = -0.221, 90% HDI= [-0.418, -0.019], $BF_{\beta>0 vs \beta<0} = 0.034$). A node in the right dorsal amygdala significantly predicted higher threat aversion ($\hat{\beta}$ = 0.256, 90% HDI= [0.045, 0.457], $BF_{\beta>0 vs \beta<0} = 40.609$). The right posterior hippocampus predicted higher threat aversion in young adult ($\hat{\beta}$ = 0.252, 90% HDI= [0.081, 0.414], $BF_{\beta>0} = 155.250$). There was also a significant age group by node interaction in the right dorsal amygdala indicating the right dorsal amygdala predicts significantly less threat aversion in adolescents compared to adults. The direction of prediction was opposite of the hypothesis that the strength of the node would predict higher threat aversion in adolescents compared to adults ($\hat{\beta}$ = -0.442, 90% HDI= [-0.881, 0.003], $BF_{\beta>0 vs \beta<0} = 0.050$). This results in the estimate of the overall effect in adolescents being not significant ($\hat{\beta}_{teen}$ = -0.186, 90% HDI= [-0.586, 0.209]). Results are summarized in Table 10 and Table 11.

	$\hat{\beta}$	**Est.Error**	90 % HDI		
Young Adults					
Intercept	-0.071	0.119	-0.270	0.122	
L Ant. Dorsal Cingulate	-0.057	0.115	-0.250	0.126	
R Ant. Dorsal Cingulate	0.128	0.106	-0.047	0.301	
L Dorsal Amygdala	-0.221	0.121	-0.418	-0.019	*
R Dorsal Amygdala	0.256	0.125	0.045	0.457	*
L Anterior Hippocampus	0.160	0.122	-0.040	0.359	
L Posterior Hippocampus	0.028	0.118	-0.165	0.220	
R Anterior Hippocampus	0.025	0.095	-0.125	0.183	
R Posterior Hippocampus	0.252	0.102	0.081	0.414	*
Adolescents					
TEEN	0.266	0.201	-0.059	0.595	
L Ant. Dorsal Cingulate	-0.249	0.243	-0.637	0.159	
R Ant. Dorsal Cingulate	0.060	0.231	-0.313	0.442	
L Dorsal Amygdala	-0.058	0.176	-0.344	0.231	
R Dorsal Amygdala	-0.186	0.243	-0.586	0.209	
L Anterior Hippocampus	0.118	0.241	-0.285	0.502	
L Posterior Hippocampus	-0.215	0.258	-0.637	0.205	
R Anterior Hippocampus	0.072	0.269	-0.364	0.515	
R Posterior Hippocampus	0.170	0.253	-0.251	0.577	
Family Parameters					
$\hat{\sigma}$	0.928	0.085	0.767	1.063	
$\hat{\alpha}$ (Skewness)	4.815	2.539	0.886	9.187	
LOO-IC = 210.455; WAIC=206.725; R2= 0.259					
'*': Estimate is credibly non-zero with 90% probability					

Table 10: Parameter estimates for the relationship of each node's Strength* to the Threat Aversion trait in each age group. Note that the adolescents shows the overall parameter estimate, rather than the deflection from the young adults.

	$\hat{\beta}$	Est.Error	BF	
L Ant. Dorsal Cingulate >0	-0.057	0.115	0.406	
R Ant. Dorsal Cingulate >0	0.128	0.106	8.217	
L Dorsal Amygdala >0	-0.221	0.121	0.034	†
R Dorsal Amygdala >0	0.256	0.125	40.609	⋆
L Anterior Hippocampus = 0	0.160	0.122	1.964	
L Posterior Hippocampus >0	0.028	0.118	1.462	
R Anterior Hippocampus = 0	0.025	0.095	5.658	
R Posterior Hippocampus >0	0.252	0.102	155.250	⋆
TEEN: L Ant. Dorsal Cingulate >0	-0.193	0.260	0.313	
TEEN: R Ant. Dorsal Cingulate >0	-0.068	0.243	0.634	
TEEN: L Dorsal Amygdala >0	0.163	0.207	3.706	
TEEN: R Dorsal Amygdala >0	-0.442	0.270	0.050	†
TEEN: L Anterior Hippocampus >0	-0.042	0.253	0.749	
TEEN: L Anterior Hippocampus <0	-0.243	0.272	4.474	
TEEN: R Anterior Hippocampus >0	0.048	0.269	1.414	
TEEN: R Posterior Hippocampus <0	-0.082	0.264	1.601	
⋆: The evidence is in favor of the hypothesis by a factor of at least 10				
†: The evidence is against of the hypothesis by a factor of at least 10				

Table 11: Bayes Factor tests for the relationship of each node's Strength* to Threat Aversion. Note that the tests shown here are for the interactions, which represent the deflection from the young adult group.

Threat Aversion - Eigenvector Centrality

Multiple regression analysis was used to test if the threat aversion trait was predicted by the eigenvector centrality of brain regions outlined in Table 1. A node in the right dorsal amygdala significantly predicted higher threat aversion ($\hat{\beta}$ = 0.248, 90% HDI= [0.040, 0.454], $BF_{\beta>0 vs \beta<0} = 35.541$). The left posterior hippocampus predicted higher threat aversion in young adults ($\hat{\beta}$ = 0.252, 90% HDI= [0.001, 0.383], $BF_{\beta>0 vs \beta<0} = 18.724$). Results are summarized in Table 12 and Table 13.

	$\hat{\beta}$	**Est.Error**	90 % HDI		
Young Adults					
Intercept	-0.044	0.125	-0.248	0.160	
L Ant. Dorsal Cingulate	0.164	0.141	-0.065	0.396	
R Ant. Dorsal Cingulate	0.106	0.132	-0.109	0.324	
L Dorsal Amygdala	-0.052	0.125	-0.025	0.155	
R Dorsal Amygdala	0.248	0.127	0.040	0.454	*
L Anterior Hippocampus	0.209	0.134	-0.008	0.429	
L Posterior Hippocampus	0.189	0.116	0.001	0.383	*
R Anterior Hippocampus	-0.132	0.119	-0.333	0.060	
R Posterior Hippocampus	0.150	0.132	-0.067	0.369	
Adolescents					
TEEN	0.279	0.186	-0.023	0.591	
L Ant. Dorsal Cingulate	-0.005	0.168	-0.277	0.275	
R Ant. Dorsal Cingulate	-0.147	0.166	-0.416	0.130	
L Dorsal Amygdala	-0.153	0.216	-0.506	0.200	
R Dorsal Amygdala	0.205	0.186	-0.105	0.503	
L Anterior Hippocampus	-0.068	0.191	-0.375	0.257	
L Posterior Hippocampus	0.176	0.278	-0.289	0.620	
R Anterior Hippocampus	-0.105	0.265	-0.536	0.337	
R Posterior Hippocampus	-0.113	0.165	-0.382	0.162	
Family Parameters					
$\hat{\sigma}$	0.932	0.087	0.788	1.068	
$\hat{\alpha}$ (Skewness)	2.168	1.795	-0.989	4.787	
LOO-IC = 218.500; WAIC=215.208; R2= 0.272					
'*': Estimate is credibly non-zero with 90% probability					

Table 12: Parameter estimates for the relationship of each node's eigenvector centrality to the Threat Aversion trait in each age group. Note that the adolescents shows the overall parameter estimate, rather than the deflection from the young adults.

	$\hat{\beta}$	Est.Error	BF	
L Ant. Dorsal Cingulate >0	0.164	0.141	7.850	
R Ant. Dorsal Cingulate >0	0.106	0.132	3.861	
L Dorsal Amygdala >0	-0.052	0.125	0.507	
R Dorsal Amygdala >0	0.248	0.127	35.541	$\star$
L Anterior Hippocampus = 0	0.209	0.134	1.183	
L Posterior Hippocampus >0	0.189	0.116	18.724	$\star$
R Anterior Hippocampus = 0	-0.132	0.119	2.541	
R Posterior Hippocampus >0	0.150	0.132	6.786	
TEEN:L Ant. Dorsal Cingulate >0	-0.170	0.216	0.278	
TEEN:R Ant. Dorsal Cingulate >0	-0.253	0.205	0.119	
TEEN:L Dorsal Amygdala >0	-0.101	0.238	0.502	
TEEN:R Dorsal Amygdala >0	-0.043	0.213	0.728	
TEEN:L Anterior Hippocampus >0	-0.278	0.224	0.118	
TEEN:L Posterior Hippocampus <0	-0.013	0.287	1.066	
TEEN:R Anterior Hippocampus >0	0.027	0.277	1.154	
TEEN:R Posterior Hippocampus <0	-0.263	0.203	9.431	

$\star$: The evidence is in favor of the hypothesis by a factor of at least 10
†: The evidence is against of the hypothesis by a factor of at least 10

Table 13: Bayes Factor tests for the relationship of each node's eigenvector centrality to Threat Aversion. Note that the tests shown here are for the interactions, which represent the deflection from the young adult group.

3.2.3 Novelty Seeking and Reward Dependence

Novelty Seeking and reward dependence emerged from the factor analysis as two correlated, but ostensibly distinct, factors. To test the hypothesis that these factors are determined by distinct neural circuits a multivariate model was fit. The model utilizes a skew normal distribution to account for skew in the factor scores. Because the correlation between response variables is not estimated the model essentially consists of two generalized linear models run side by side (Bürkner, 2017).

Each model was set up similarly to what came before, with the addition of parameter estimates for the means of the two sensation seeking conditions the

original data collection utilized are also given. Parameter estimates were derived from the estimated variance component for between-condition variability using brms (Bürkner, 2017). Model formulas are given below:

$$y_i \sim skewed\ normal(\mu, \sigma, \alpha)$$

$$\mu = \beta_0 + Age_0 + \sum_n \beta_n x_n + \sum_{n \cdot Age} \beta_n x_n \cdot Age + u_{group}$$

$$\beta_0 \sim t\ distribution(\nu = 3, \mu = 0, \sigma = .5)$$

$$Age_0 \sim t\ distribution(\nu = 3, \mu = 0, \sigma = .25)$$

$$\beta_n \sim t\ distribution(\nu = 3, \mu = 0, \sigma = .5)$$

$$\beta_n \cdot Age \sim t\ distribution(\nu = 3, \mu = .25, \sigma = .25)$$

$$\sigma \sim gamma(s = 2, r = 2)$$

$$\alpha \sim normal(\mu = 2, \sigma = 4)$$

BAS Traits - Strength*

The model of strength* showed that the left and right medial amygdala predict reward dependence in young adults, but in opposite directions. The left medial amygdala predicts higher reward dependence ($\hat{\beta}$=.250, 90% HDI=[0.053,0.459]). The right medial amygdala predicts lower reward dependence ($\hat{\beta}$=-0.364, 90% HDI=[-0.561,-0.171]). While falling just short of significance, the estimate for the effect of the left amygdala on reward dependence in adolescents is consistent in both magnitude and direction with the estimate in young adults ($\hat{\beta}$=0.318, 90% HDI=[-0.017,0.684]).

The left putamen and right nucleus accumbens emerged as significant predictors for novelty seeking in young adults. The left putamen predicts higher novelty seeking ($\hat{\beta}$=0.324, 90% HDI=[0.132,0.525]). The right nucleus accumbens predicts

lower novelty seeking ($\hat{\beta}$=-0.180, 90% HDI=[-0.357,-0.001]).

While falling just short of significance, the estimate for the effect of the left putamen on novelty in adolescents is consistent in both magnitude and direction with the estimate in young adults ($\hat{\beta}$=0.208, 90% HDI=[-0.093,0.528]). The estimate for the effect of the right nucleus accumbens on novelty seeking in adolescents is consistent in both magnitude and direction with the estimate for young adults, but the credible interval is substantially wide and extends well past zero ($\hat{\beta}$=-0.104, 90% HDI=[-0.505,0.307]).

BAS Traits - Eigenvector Centrality

The model of eigenvector centrality revealed two significant predictors in young adults for reward dependence. The eigenvector centrality of the left putamen predicts higher reward dependence ($\hat{\beta}$=0.217, 90% HDI=[0.007, 0.459]). The eigenvector centrality of the right medial amygdala predicted lower reward dependence ($\hat{\beta}$=-0.217, 90% HDI=[-0.430,-0.010]). Only one significant predictor emerged for novelty seeking. The left nucleus accumbens predicted higher novelty seeking ($\hat{\beta}$=0.212, 90% HDI=[0.002,0.417]). Results are summarized in Table 14 and Table 15.

	REWARD DEPENDENCE					NOVELTY SEEKING				
	$\hat{\beta}$	Est.Error	90 % HDI			$\hat{\beta}$	Est.Error	90 % HDI		
Population Effects										
Young Adults										
Intercept	-0.051	0.361	-0.633	0.559		0.144	0.381	-0.496	0.767	
L Putamen	0.103	0.140	-0.136	0.322		0.324	0.122	0.132	0.525	*
R Putamen	-0.002	0.131	-0.213	0.217		-0.062	0.124	-0.257	0.151	
L Medial Amygdala	0.250	0.123	0.053	0.459	*	0.142	0.107	-0.051	0.303	
R Medial Amygdala	-0.364	0.121	-0.561	-0.171	*	-0.095	0.110	-0.273	0.090	
L Caudate	0.137	0.155	-0.118	0.389		0.073	0.125	-0.137	0.272	
R Caudate	0.061	0.137	-0.177	0.270		0.117	0.133	-0.110	0.331	
L Accumbens	0.115	0.139	-0.110	0.338		-0.196	0.125	-0.399	0.004	
R Accumbens	-0.166	0.125	-0.379	0.025		-0.180	0.110	-0.357	-0.001	*
Adolescents										
TEEN	0.119	0.246	-0.276	0.519		-0.398	0.240	-0.791	0.002	
L Putamen	-0.028	0.195	-0.334	0.305		0.208	0.188	-0.093	0.528	
R Putamen	0.240	0.209	-0.094	0.578		0.209	0.204	-0.113	0.543	
L Medial Amygdala	0.318	0.215	-0.017	0.684		0.183	0.190	-0.117	0.502	
R Medial Amygdala	-0.091	0.239	-0.490	0.291		0.216	0.234	-0.191	0.574	
L Caudate	0.074	0.246	-0.319	0.483		0.251	0.226	-0.122	0.614	
R Caudate	0.054	0.176	-0.214	0.358		0.092	0.162	-0.169	0.363	
L Accumbens	0.055	0.220	-0.320	0.400		-0.202	0.205	-0.532	0.136	
R Accumbens	-0.166	0.245	-0.567	0.238		-0.104	0.248	-0.505	0.307	
Group Effects										
HSS	0.420	0.376	-0.195	1.032		0.504	0.399	-0.143	1.161	
LSS	-0.435	0.369	-1.037	0.174		-0.502	0.393	-1.124	0.162	
Family Parameters										
$\hat{\sigma}$	0.891	0.087	0.759	1.040		0.858	0.089	0.716	1.040	
$\hat{\alpha}$ (Skewness)	-1.462	2.225	-4.816	1.874		-3.848	3.959	-10.552	2.048	
LOO-IC = 410.354; WAIC = 404.208; R2 = 0.411										
'*': Estimate is credibly non-zero with 90% probability										

Table 14: Regression Results for the relationship of each node's Strength* with BAS traits.

	REWARD DEPENDENCE					NOVELTY SEEKING				
	$\hat{\beta}$	Est.Error	90 % HDI			$\hat{\beta}$	Est.Error	90 % HDI		
Population Effects										
Young Adults										
Intercept	0.006	0.334	-0.052	0.574		0.020	0.377	-0.062	0.604	
L Putamen	0.217	0.139	0.007	0.459	*	-0.128	0.137	-0.348	0.100	
R Putamen	0.145	0.133	-0.071	0.358		-0.058	0.124	-0.259	0.147	
L Medial Amygdala	0.087	0.135	-0.140	0.302		0.085	0.130	-0.120	0.310	
R Medial Amygdala	-0.217	0.129	-0.430	-0.010	*	0.030	0.125	-0.170	0.243	
L Caudate	-0.014	0.131	-0.230	0.193		0.124	0.126	-0.083	0.329	
R Caudate	0.118	0.156	-0.140	0.376		0.039	0.143	-0.180	0.283	
L Accumbens	-0.173	0.132	-0.390	0.044		0.212	0.127	0.002	0.417	*
R Accumbens	0.147	0.122	-0.048	0.352		-0.078	0.124	-0.280	0.131	
Adolescents										
TEEN	-0.041	0.238	-0.437	0.344		-0.189	0.230	-0.550	0.198	
L Putamen	0.275	0.209	-0.0595	0.619		0.196	0.195	-0.109	0.536	
R Putamen	0.281	0.204	-0.0396	0.627		0.176	0.195	-0.137	0.499	
L Medial Amygdala	0.0315	0.315	-0.476	0.548		-0.0579	0.302	-0.539	0.440	
R Medial Amygdala	-0.107	0.259	-0.559	0.310		-0.0622	0.249	-0.468	0.351	
L Caudate	0.035	0.241	-0.389	0.403		0.0337	0.235	-0.341	0.429	
R Caudate	0.17	0.226	-0.198	0.543		-0.127	0.211	-0.459	0.240	
L Accumbens	0.143	0.204	-0.199	0.473		0.0972	0.192	-0.222	0.401	
R Accumbens	-0.258	0.263	-0.685	0.174		-0.0126	0.237	-0.395	0.372	
Group Effects										
HSS	0.303	0.352	-0.262	0.894		0.515	0.393	-0.124	1.152	
LSS	-0.305	0.348	-0.884	0.245		-0.502	0.394	-1.133	0.155	
Family Parameters										
$\hat{\sigma}$	.926	0.092	0.773	1.070		0.883	0.089	0.742	1.027	
$\hat{\alpha}$ (Skewness)	0.932	1.801	-1.847	3.604		-0.041	2.649	-4.168	4.174	
LOO-IC = 426.497; WAIC = 419.115; R2 = 0.385										
'*': Estimate is credibly non-zero with 90% probability										

Table 15: Regression Results for the relationship of each node's Strength$*$ with BAS traits.

3.3 Network Visualization

Important nodes in the network identified by strength* and eigenvector centrality indicate which nodes are highly connected and influential on the whole-brain network. Identifying the brain areas to which these nodes are connected can identify through which circuits they exert their influence. To investigate this approach the top 5 positive and top 5 negative connections the significant nodes were identified through inspection of the partial correlation matrix. All listed connections are in order from strongest to weakest. Figures visualizing these connections were created in BrainNet viewer (Xia, Wang, & He, 2013). Connections were set as 1 for a positive correlation and as -1 for a negative correlation for ease of visualization.

ECS Network

Impulsivity was found to be predicted best by the left dorsal lateral frontal cortex in young adults and right medial frontal cortex in adolescents. This network was the only one that showed a major difference between adolescents and young adults, consistent with the idea that the executive networks are still developing in adolescents. A visualization of the connectivity of this network can be seen in Figure 5 and Figure 6.

The left dorsal lateral frontal cortex is positively connected to a location in the left frontal pole, two locations in the left superior frontal gyrus, and two locations in the right frontal pole. It is negatively connected to the a different location in the right frontal pole, right thalamus, right supramarginal gyrus, right angular gyrus, and right cerebellar crus I.

The right medial frontal cortex is positively connected to the right frontal pole, another location in the right medial frontal cortex,two locations in the left anterior

cingulate gyrus, and a location in the left supramarginal gyrus. It is negatively connected to two locations in the left supramarginal gyrus, left lateral occipital cortex, right supramarginal gyrus, and right superior lateral occipital cortex.

The overall pattern is for frontal regions to be connected to one another. This is evident in the intrahemispheric and interhemispheric positive connections of the frontal lobe in adults. Negative connections are dispersed without a clear pattern. The same is true for the connectivity of the medial frontal cortex in adolescents. It appears to be heavily connected with both the frontal lobe and cingulate gyrus, with dispersed negative connections.

The shift in relationship between impulse control and the medial frontal cortex in adolescence to the dorsal lateral cortex in young adulthood likely reflects maturational effects.The brain is known to develop from a posterior to anterior direction (Gogtay et al., 2004). Frontal lobe maturation happens from the medial to lateral direction (Fuster, 2002). Furthermore, limbic regions are known to develop prior to cortical regions, although the uncinate fasciculus, which connects the ventral medial frontal lobe to the limbic regions, is still maturing through the second decade of life (Rothmond, Weickert, & Webster, 2012; Lebel, Walker, Leemans, Phillips, & Beaulieu, 2008). Hence, the inverse relationship of Impulsivity in adolescents likely relies on inhibitory circuits that are still developing. As experience and time shape development of the frontal lobe, more abstract social rules to guide culturally acceptable behavior are learned.

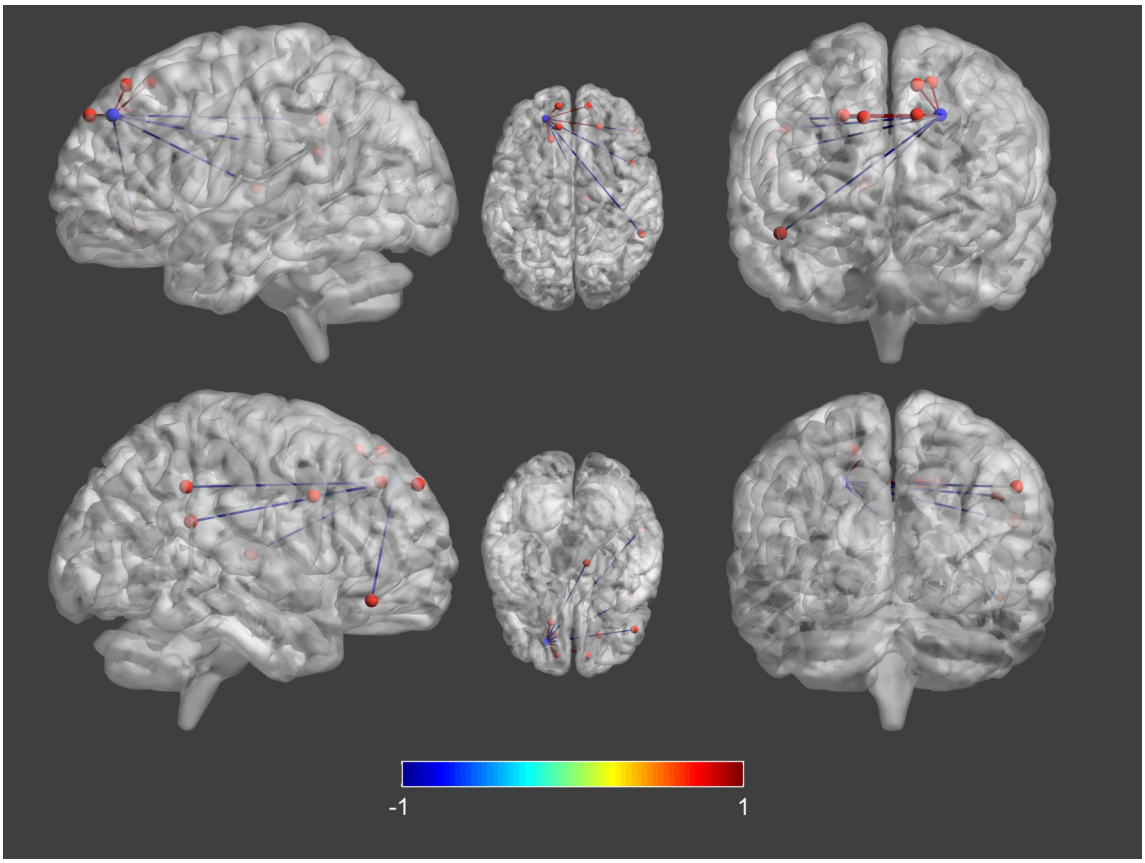

Figure 5: Strongest positive and negative connections of the left dorsal lateral frontal cortex, which predicted lower Impulsivity in adults. The connections are partial correlations scaled to -1 to 1 range.

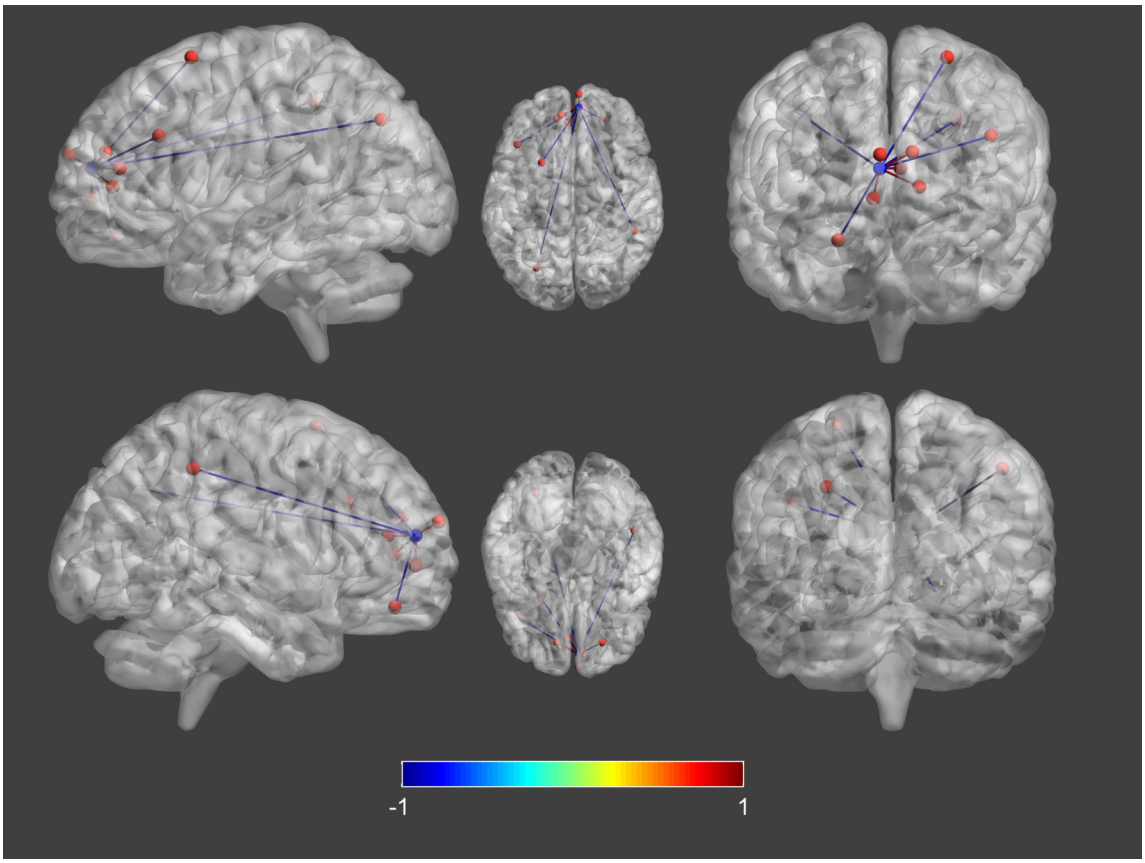

Figure 6: Strongest positive and negative connections of the right medial frontal cortex, which predicted lower Impulsivity in adolescents. The connections are partial correlations scaled to -1 to 1 range.

BIS and FFFS Network

Threat Aversion was found to be predicted by the bilateral dorsal amygdala and bilateral posterior hippocampus. The bilateral amygdala is presumed to represent the FFFS, and the bilateral posterior hippocampus to the BIS. A visualization of the connectivity of this network can be seen in Figure 7.

The left dorsal amygdala was found to be positively connected to the right dorsal amygdala, left putamen, left ventral-medial amygdala, left medial amygdala, and a second location in the left putamen. It is negatively connected to the

left precuneus, left occipital pole, right superior occipital cortex, left frontal pole, and right inferior temporal gyrus.

The right dorsal amygdala was found to be positively connected to the left dorsal amygdala, left putamen, right medial amygdala, right ventral-lateral amygdala, and right putamen. It is negatively connected to the right medial frontal cortex, right inferior temporal gyrus, right orbital frontal cortex, right cingulate gyrus, and left hypothalamus.

The left posterior hippocampus was found to be positively connected to the left anterior hippocampus, two locations in the left parahippocampal gyrus, and two locations in the right posterior hippocampus. It is negatively connected to the right posterior hippocampus, right anterior inferior temporal gyrus, right orbital frontal cortex, and two locations in the right frontal pole.

The right posterior hippocampus was found to be positively connected to the another location in the right posterior hippocampus, two locations in the right parahippocampal gyrus, the right subiculum, and the left posterior hippocampus. It is negatively connected to the left anterior inferior temporal gyrus, the right putamen, the left occipital pole, the right posterior inferior temporal gyrus,and right cerebellar crus II.

The overall pattern of connectivity is the bilateral amygdala is heavily interconnected and with strong connections to the putamen. There is also a trend for it to be negatively connected with both frontal and occipital regions. The bilateral posterior hippocampus shows a pattern of positive connectivity with other parts of the hippocampus and parahippocampal gyrus. There is also a trend trend towards negative connectivity with the inferior temporal gyrus and both occipital and frontal regions. This may be interpreted as an oppositional relationship to the BAS on the part of the hippocampus, and a positive one on the part of the FFFS. It

is sensible for the amygdala to be positively connected to the BAS due to the fact that the FFFS must quickly initialize defensive movements without conscious input. Amygdalostriatal structural connections are also established in the primate brain (Cho, Ernst, & Fudge, 2013).

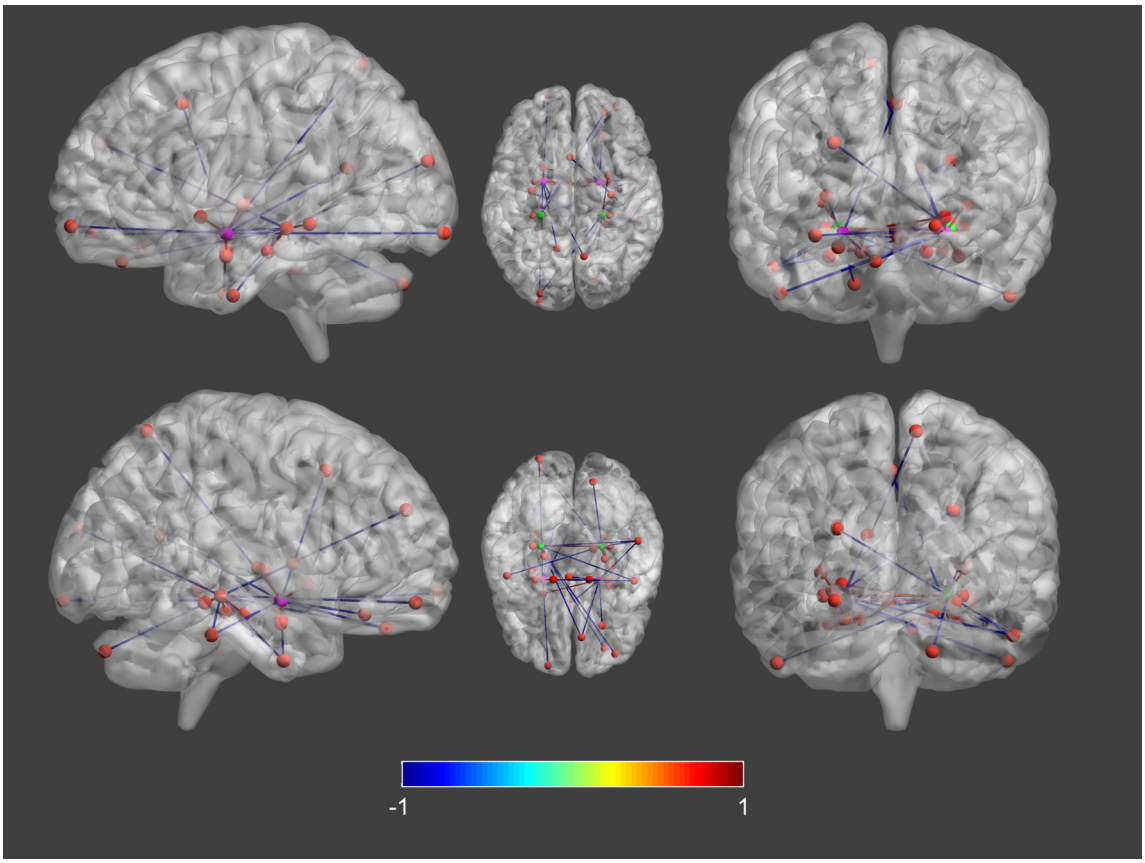

Figure 7: The bilateral posterior hippocampus (BIS) is represented in Green and bilateral dorsal amygdala (FFFS) in purple. Each contributed to predicting the Threat Aversion trait in adults. The connections are partial correlations scaled to -1 to 1 range.

BAS Network - Reward Dependence and Novelty Seeking

Reward Dependence was found to be predicted by the left putamen, as well as the left and right medial amygdala. A visualization of the connectivity of this network can be seen in Figure 8.

The left putamen is positively connected to two regions in the right putamen, a region lying between the caudate and putamen, the right nucleus accumbens, and the left dorsal amygdala. The left putamen is negatively connected to the left parahippocampal gyrus (entorhinal cortex), the left paracingulate gyrus, left posterior cingulate gyrus, right inferior frontal gyrus, and right insular cortex.

The left medial amygdala is positively connected to the right medial amygdala, left hippocampus-amygdala transition, right and left hypothalamus, and left dorsal amygdala. It is negatively connected to the right cerebellar crus V, left superior frontal gyrus, a region lying between the caudate and putamen, left cerebellar crus VI, and a region in the right frontal pole.

The right medial amygdala is positively connected to the right hypothalamus, left medial amygdala, right hippocampus-amygdala transition, right dorsal amygdala, and left hippocampus-amygdala transition. It is negatively connected to the left superior frontal gyrus, right insular cortex, right cerebellar crus V, right anterior cingulate gyrus, and left superior frontal gyrus.

Novelty Seeking was found to be predicted by the left putamen, as well as the left and right nucleus accumbens. A visualization of the connectivity of this network can be seen in Figure 9. The left putamen is a shared feature predicting both Novelty Seeking and Reward Dependence, so its connectivity profile will not be reiterated here.

The left nucleus accumbens is positively connected to the left caudate, a region lying between the caudate and putamen, the left and right thalamus, and the right caudate. It is negatively connected to the left lingual gyrus, left inferior lateral occipital cortex, right putamen, right insular cortex, and left cingulate gyrus.

The right nucleus accumbens is positively connected to the left putamen, right hypothalamus, right orbital frontal cortex, right insular cortex, and right puta-

men. It is negatively connected to the left central opercular cortex, left lateral occipital cortex, right inferior frontal gyrus, left superior lateral occipital cortex, and left subiculum.

A pattern emerged here that the nodes predicting the Reward Dependence trait are well connected with other regions known to relate to reward learning and motivational processes. Of note are positive connections with the nucleus accumbens and hypothalamus. The nucleus accumbens and hypothalamus are both well known to have roles in reward learning (Day & Carelli, 2007; R. A. Wise, 2005). The hypothalamus controls a wide host of biological processes, among them sexual behavior, feeding, and the activation of the sympathetic nervous system (Olds & Milner, 1954; Stuber & Wise, 2016; Tyree & de Lecea, 2017). All three hubs were also connected to the dorsal amygdala. This suggests that more than just the medial amygdala is involved in Reward Dependence. Connections of reward learning and motivation circuits to a part of the amygdala associated with learning or reacting to aversive stimuli may also support the notion that the phenomenon of frustrative non-reward is correctly attributed to both the BAS and FFFS (Corr, 2002; Carver, 2004, 2006; Chesworth & Corbit, 2017). Other connections, unsurprisingly, were to other areas within the caudate and putamen. The negative connections of the bilateral medial amygdala and left putamen were largely to regions in the frontal lobe, cingulate and paracingulate gyrus, and temporal lobe regions near the hippocampus. These regions are among those associated with the BIS and with the ECS. This suggests the negative connections contribute behaviorally to behavioral inhibition (Gray & MacNaughton, 2000).

Similar patterns of connectivity were observed for the bilateral nucleus accumbens. Unique to the nucleus accumbens however are positive connections to the thalamus, right orbital frontal cortex, and right insula. This may play a

role in the orienting of attention to novel responses. The right insula has been associated with increases in autonomic arousal and peripheral adrenaline levels as well as with both voluntary and involuntary orienting of attention towards salient stimuli (Critchley, Melmed, Featherstone, Mathias, & Dolan, 2002; Meyer, Strittmatter, Fischer, Georg, & Schmitz, 2004; Eckert et al., 2009). The nucleus accumbens also appears to have specialized processing for novel stimuli (Murty, Stanek, & Heusser, 2013; Zaehle et al., 2013). By contrast, the amygdala may have a stronger role in Reward Dependence by activating motor functions when a discriminative stimulus has been learned to predict the availability of reward given some operant motor program (Murray, Izquierdo, & Malkova, 2009; Wassum & Izquierdo, 2015; Volkow & Morales, 2015; Chesworth & Corbit, 2017). It is also possible that the aversive effects of frustrative non-reward driven by BAS-FFFS interactions mediated by the amygdala may be a motivating factor in Reward Dependence, maintaining behavior though negative reinforcement. That is, when the reward is obtained, not only do the rewarding effects reinforce the behavior chain, but the elimination of frustration also reinforces the fixation on a known reward. This is known to play a role in the maintenance of drug seeking behavior, which is arguably the most extreme example of Reward Dependence (Koob, 2013).

4 Discussion

The first aim of this study was to explore a broad array of existing personality assessments through factor analysis to test the hypothesis that there are personality traits corresponding to the systems of Reinforcement Sensitivity Theory. The second aim of this study was to use the graph theory metrics strength* and eigen-

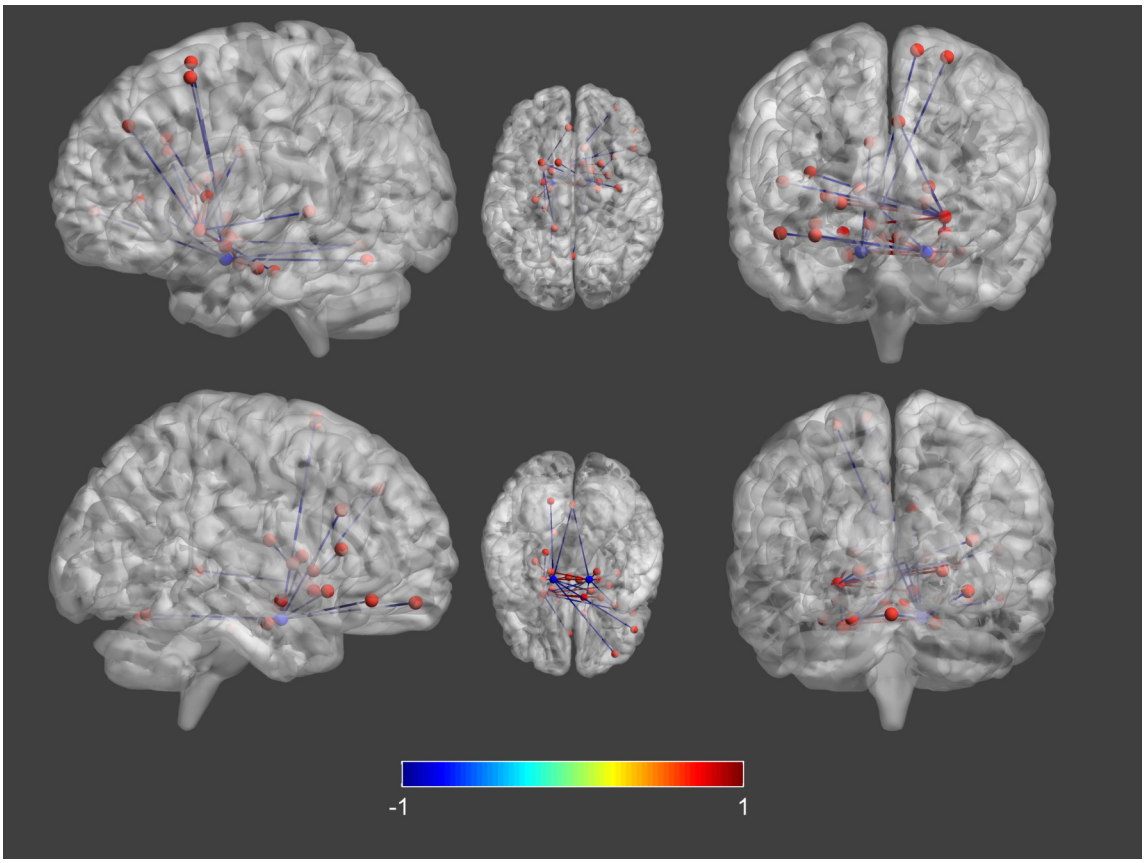

Figure 8: Strongest positive and negative connections of the left putamen and bilateral medial amygdala nodes, which predict the Reward Dependence trait in adults. The connections are partial correlations scaled to -1 to 1 range.

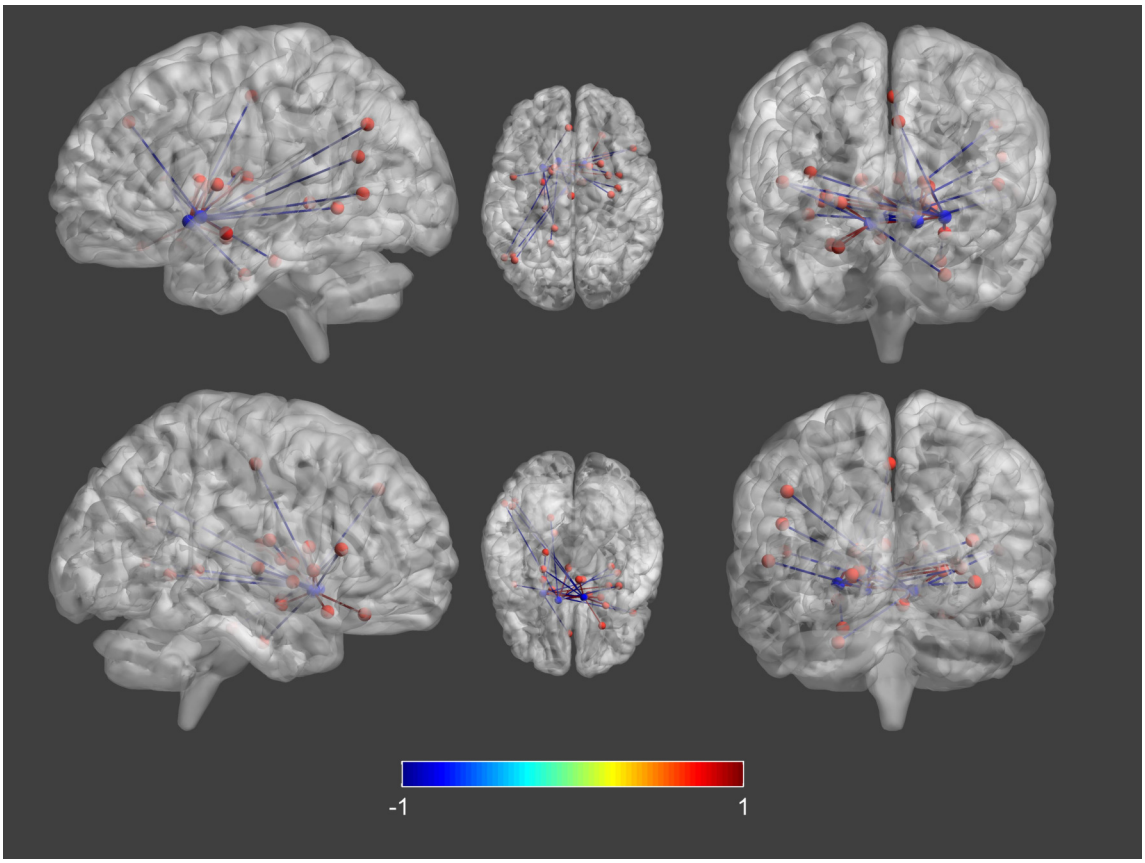

Figure 9: Strongest positive and negative connections of the left putamen and bilateral nucleus accumbens nodes, which predict the Novelty Seeking trait in adults. The connections are partial correlations scaled to -1 to 1 range.

vector centrality to characterize the resting state functional networks of the brain. It was hypothesized that brain regions corresponding to ones established as part of RST systems predicted the personality traits. The third aim of this study was to explore whether or not the neural correlates of personality were different in adolescents compared to adults. Reinforcement sensitivity theory has yet to extend to developmental questions. Network analysis utilizing graph theory allows for understanding the precise way certain circuits have been disrupted, and how that might influence the brain as it develops. This is an important frontier for the the field of neuroscience because if personality traits which place an adolescent at risk for mental health issues can be linked to developing brain circuits, there is potential for early intervention and therapies adapted to the developing brain.

The factor analysis revealed four factors underlying the battery of personality traits given to participants. One corresponded to the Behavioral Inhibition System (BIS) and Fight Flight Freeze System (FFFS), one to the Executive Control System (ECS), and two to different aspects of the Behavioral Approach System (BAS). The two BAS traits thematically seemed to correspond to Cloninger's Reward Dependence and Novelty Seeking (Cloninger, 1986). A factor analysis of a variety of personality assessments specifically designed to measure RST constructs also found that up to four factors are required to adequately account for the complexity of the BAS (Krupić, Corr, Ručević, Križanić, & Gračanin, 2016). Though not anticipated prior to the current study it is more likely than not that different capacities of the BAS would have different effects on behavior.

4.1 Behavioral Approach System (BAS)

The major distinction concerning the functions of reward learning circuitry is between wanting and liking. The first refers to a craving for some stimulus or outcome, and the later to the hedonic response to a stimulus or outcome. The function of wanting is to initiate movements organized into plans to obtain the desired outcome and is thought to depend on dopaminergic activity. Liking serves as a signal to terminate an ongoing response and is thought to depend on opioidergic and GABAergic function (Berridge, Robinson, & Aldridge, 2009; Peciña, 2008). On the other hand, the dopaminergic system also registers novelty and is released with diminishing magnitude as the familiarity of a stimulus increases (Schultz, Dayan, & Montague, 1997). Beyond this behavioral patterns can be detected in certain reward learning paradigms reflecting a preference for encountering novel stimuli or for certain novel stimuli that depend on different processes within the same circuitry (Krebs, Schott, & Düzel, 2009; Krebs, Heipertz, Schuetze, & Duzel, 2011; Houillon et al., 2013). It has also been suggested that behavior consists of both valuation and motivation stages. This means that an appraisal about the value of a stimulus or outcome is undertaken before a goal is formed (the 'wanting') to animate behavior, and both can be manipulated independently under certain experimental conditions (McNaughton & Corr, 2004; McNaughton et al., 2016). In summary, a variety of processes are attributed to the BAS as a whole including (in the order which they must occur in a behavior chain) valuation (determining whether a goal should be set), wanting (goal setting), motivation (vigor to animate behavior), and liking (the hedonic response indicating a goal has been achieved). In this view, the BAS may be better viewed as a collection of systems embedded within overlapping neural architecture or a 'supersystem'. BAS re-

lated personality traits then reflect stable patterns of function in different parts of the BAS supersystem. A future expansion of RST may need to add different conceptual systems to reflect this just as the original concept of the BIS was split into the FFFS and BIS when it became clear that fear and anxiety were overlapping but distinctive processes (Gray, 1982; Gray & MacNaughton, 2000) .

The regression results indicate that Reward Dependence is most distinctly related to the functional connectivity of the medial amygdala, while Novelty Seeking is most distinctly related to the functional connectivity of the nucleus accumbens. The putamen appears to be involved in both traits, although each trait was predicted by the left putamen node by different graph theory metrics. As mentioned previously, the amygdala's role in reward learning is the recognition of motivationally salient stimuli, including those learned through association (Murray et al., 2009; Wassum & Izquierdo, 2015; Volkow & Morales, 2015; Chesworth & Corbit, 2017). Reward Dependence as a trait is indicated by the current study to be largely tied to the amygdala. In particular the left amygdala may have greater brain-wide connectivity while the right amygdala may have a smaller degree of influence on the brain. This is consistent with results indicating the left amygdala has greater resting state functional connectivity with a greater level of BAS-related traits such as Extraversion (Pang et al., 2016). Extraversion has also been linked with greater gray matter volume in the left amygdala (Omura, Todd Constable, & Canli, 2005).

Novelty Seeking was more closely related to the nucleus accumbens. In particular, the less strongly connected the accumbens is to the rest of the brain, the greater score an individual has on Novelty Seeking. This may reflect low catecholamine levels, such that low noradrenergic or dopaminergic activity results in poor connectivity. Novelty Seeking behavior may then reflect a compensatory

mechanism to raise catecholamine through the seeking out of novel stimuli. On the other hand greater influence from the left accumbens on the brain results in a higher Novelty Seeking score. This positive relationship may be related to the strong functional connections the right accumbens has to the hypothalamus and insula found in the qualitative network analysis. Stimulation of the hypothalamus is known to induce exploratory behavior characteristic of Novelty Seeking, and the insula is known to play a role in autonomic arousal (Olds & Milner, 1954; Critchley et al., 2002). This could possibly reflect a greater degree of catecholamine input or greater sensitivity to such input, which is an apparent contradiction to the traditional explanation of novelty seeking. Such paradoxes have been observed in other domains, such as electro-dermal response (Stelmack, Plouffe, & Falkenberg, 1983). Novelty Seeking traits may also be moderated by sex, which was not accounted for in the present study (Cross, Cyrenne, & Brown, 2013). The particular valence and arousal level may also play a role, but it is not clear what would account for paradoxical observations in a resting state (Joseph, Liu, Jiang, Lynam, & Kelly, 2009). This apparent paradox may reflect the need for a greater number of factors related to the BAS to properly delineate the influences of different brain regions on different aspects of BAS-behaviors.

4.2 Executive Control System (ECS)

The Executive Control system is a complex system involving both approach and avoid tendencies. It is dependent on a variety of neurotransmitter systems, namely dopamine, serotonin, acetylcholine, and the ubiquitous GABA and glutamate. The ECS is responsible for the organization of behaviors into logical patterns in service of a goal, and hence is vital to the proper execution of wanting and moti-

vation stages of goal-directed behavior. Breakdown of the ECS results in the alternation between labile attention (mind wandering, environment scanning) and inflexible attention (hyperfocus or perseveration) characteristic of developmental disorders such as attention deficit hyperactivity disorder and autism spectrum disorders, respectively. At its most extreme, breakdown of the ECS results in completely disorganized patterns of behavior such as that seen from frontal lobe brain injury. Equally extreme are cases of ECS breakdown due to psychosis causing conditions such as schizophrenia, resulting in disorganized patterns of thinking, lack of spontaneous or voluntary behavior, and lack of insight concerning the veridicality of delusions and hallucinations.

If the BAS controls the sequence of valuation, wanting, motivation, and liking, the ECS is vital for the termination of this behavior chain. Deficits in this inhibitory process result in behavioral patterns such as addiction, where information concerning the risk or utility (specifically whether or not a behavior will result in the desired outcome) are not utilized in the ongoing valuation of an outcome. In addiction it is widely recognized that risk of death, injury, or illness are discounted despite something such as a drug no longer holding any intrinsic value (ie, loss of hedonic response due to tolerance to a substance). The ECS also plays an inhibitory role in the voluntary withholding of a response due to social expectation or needing to formulate a fully developed motor plan. Failure to appropriately inhibit a response either prior to its execution or after it has begun and failure to integrate goal-relevant information in decision making are fundamental features of impulsivity reflected in the Impulsivity trait recovered in the factor analysis. Addiction, antisocial personality disorder, ADHD, autism, and schizophrenia are notable clinical conditions that affect executive functions in different domains and severity (Hill, 2004; Egeland, 2007; Eisenbarth et al., 2008;

Crews & Boettiger, 2009).

The regression results for Impulsivity revealed a developmental difference in a major locus for the Executive Control Network. Maturation of impulse control appears to be mediated by a shift from the medial frontal lobe to the dorsal lateral frontal lobe. Furthermore, the influence of a node on the overall brain network, as measured by strength* and eigenvector centrality, predicts how impulsive an individual is likely to be. The greater the influence of the node for the ECS, the less impulsive someone will be. The medial frontal cortex playing the role of the 'executive' in adolescents is consistent with current knowledge about the developmental morphology of the frontal lobe. Gray matter maturation proceeds from the posterior to anterior direction. The dorsolateral frontal cortex is among the last to develop (Gogtay et al., 2004; Anderson, Jacobs, & Anderson, 2008). Evidence suggests that the medial part of the frontal lobe develops first as well, expanding from the medial to lateral parts. Medial frontal structures are involved in the initiation of movement in response to signals of reinforcement, as well as several other affective and motivation related processes (Fuster, 2002; O'Reilly, 2010). The particular medial area identified as an important node in the ECS for adolescents is a midline area overlapping with the paracingulate gyrus. This is consistent with the fact that the most anterior parts of the frontal lobe (the prefrontal cortex) have yet to fully develop, as well as the fact that the dorsal lateral part has yet to fully develop. The qualitative analysis of the connections suggest it shares many of the same functional connections as the dorsal lateral frontal cortex does in adults, although the connections tend to be within the same hemisphere. It is likely that the nature of white and gray matter development accounts for this hemispheric switch. Evidence suggests that the right-side white matter tracts and gray matter in the frontal lobes develops with greater speed than the left initially, then slowing

down while the development of the left side slowly catches up (Tanaka, Matsui, Uematsu, Noguchi, & Miyawaki, 2012).

4.3 Defensive Systems - BIS & FFFS

The Behavioral Inhibition System is another major system involved in behavioral monitoring and decision making. From an evolutionary perspective, all organisms require a system to guide behavior. Although other organisms have homologous brain regions to the ones in the human frontal-parietal (or Executive Control) system, the functional capacities yielding the abilities of human reason are simply not as complex and capable (Preuss, 1995; S. P. Wise, 2008). In other mammals in particular the BIS plays a large role in these processes. Lesions in the septal-hippocampal system in small mammals tend to produce deficits in respondent and operant learning tasks similar to those resulting from frontal lobe lesions in primates (Altman et al., 1973). The major task of the BIS is to monitor behavior for goal conflicts between the BAS and Fight-Flight-Freeze System (FFFS). The other major function is to monitor the proximity of threats (defensive distance) or other aversive situations. Anxiety about public speaking for instance may be present days prior to having to speak in front of a crowd, but grow stronger as the event draws closer. The BIS inhibits the FFFS, but as the perceived threat is more proximal it releases the FFFS from inhibition. Dysfunction of the BIS results in generalized anxiety disorder, allowing the FFFS to have undue influence on behavior and mood (Gray & MacNaughton, 2000). The FFFS by contrast is simply the defensive system in place to detect threats and react to them through the defensive behaviors of Fight, Flight, or Freeze. Panic disorder, PTSD, and specific phobias are examples of FFFS dysfunction, causing defensive reactions that can

be sudden, intrusive, and sometimes extreme (Gray & MacNaughton, 2000). The amygdala is most commonly associated with the FFFS, but projections to the different regions within the periaqueductal gray (PAG) trigger different defensive behaviors in a strategy selection process called the defense cascade (Kozlowska, Walker, McLean, & Carrive, 2015). The PAG in turn mediates many of its effects through connections the nucleus ambiguus (NA) and dorsal motor nucleus of the vagus nerve (DMNX) (Farkas, Jansen, & Loewy, 1997). The NA controls the phylogeneticallymore recent myelinated vagus nerve, present only in mammals, and releases the cardiovascular system from tonic regulation when the HPA axis releases a surge of corticosteroids in response to a stressor. The DMNX controls the phylogenetically older unmyelinated vagus nerve, which is normally only triggered in reaction to extreme danger or situations where fight or flight strategies are untenable. DMNX activation causes a sudden drop in blood pressure, suppresses breathing and bladder/bowel control, often causing a fainting response that has origins in the playing-dead and freeze responses (Porges, 2007, 2009).

Pathological activation of the FFFS is bound to produce approach-avoidance conflicts and shape avoidance behaviors. Higher order traits such as Neuroticism are designed to measure these longer term patterns of avoidance behaviors and frequency of experiencing negative affect. As a result, it was expected that only one trait would emerge related to the FFFS and BIS. This trait was named Threat Aversion. It was expected then that brain regions corresponding to both the FFFS and BIS would relate to this trait. As expected the amygdala and hippocampus were both related to Threat Aversion. The left dorsal amygdala predicted lower Threat Aversion and the right dorsal amygdala predicted higher Threat Aversion in adults. In adults it was found that the left and right hippocampus predicted greater Threat Aversion. Posterior regions of the hippocampus, rather than ante-

rior, were found to predict Threat Aversion. This implies the sensitivity to detecting goal-conflict or judging threats to be proximal relates to the posterior portion of the hippocampus, but the anterior portion is responsible for addressing a detected goal-conflict (Satpute et al., 2012).

The posterior hippocampus has also been more closely related to memory processes than the anterior hippocampus (Fanselow & Dong, 2010; Blum, Habeck, Steffener, Razlighi, & Stern, 2014). This is not surprising. If the posterior hippocampus is heavily involved with memory processes, then it has easy access to memories of threatening stimuli or circumstances. This would allow it to monitor the environment and compare stimuli against those in memory. Positive detection of a threat would result in an assessment of possible goal-conflict, signaling the anterior hippocampus to bias decision making in favor of the FFFS. This is evolutionarily sensible; a grazing zebra would be remiss to not assess the possibility of being eaten upon seeing a lion and decide in favor of fleeing. The qualitative network analysis of the posterior hippocampus showed connections largely to cortical areas in the temporal lobe, consistent with it being involved in accessing memories. These results support the idea that goal conflict does not need to be present to detect hippocampal involvement in trait-anxiety, allowing reinforcement sensitivity theory to be studied through resting state functional connectivity analyses.

4.4 Limitations and Future Directions

Although results are consistent with the predictions of reinforcement sensitivity theory, some caution is warranted in interpreting results. The sample size of the study is one major limitation warranting such caution. It has been suggested

to reliably detect personality related effects in the brain one needs a very large sample size on the order of around 200 participants (McNaughton et al., 2016). Also, fewer adolescents were enrolled in the study than adults. Due to this, the regression model lacked power to get sufficiently precise estimates in order to confidently state the direction of an effect for most estimates. This resulted in the selection of a small handful of brain regions to enter into the regression model. Ideally, every region of interest would be entered into every model to gain an understanding of how uniquely each brain region relates to a personality trait. Another limitation is that only 2 to 4 nodes were used as representatives of larger brain areas thought to be involved. This was done because it was probable that nodes within the same brain region would share similar properties and cause an undesirable amount of collinearity between predictors.

Future studies might also use personality assessments developed specifically with RST in mind. While the Carver and White BIS/BAS assessment is based on RST constructs, it is based on an early iteration of the theory which only posited a behavioral activation and behavioral inhibition system (Gray, 1970; Carver & White, 1994; Jackson, 2009; Reuter, Cooper, Smillie, Markett, & Montag, 2015). For the purposes of a study like this one, utilizing a battery of assessments specifically designed to measure the constructs of interest would necessitate the use of principal components analysis. The present study used exploratory factor analysis because it is most appropriate when looking for latent variables to explain a set of measured variables. However, a disadvantage of using factor scores from exploratory factor analysis is the issue of factor score indeterminacy. Due to the lack of perfectly unique factor solutions, determinate factor scores cannot be defined (J. S. Williams, 1978). Because of this, the method of computing factor scores could in theory affect the results. Principal components analysis (PCA) lacks this

problem (Grice, 2001). In a situation where all trait measures are directly conceptually related to RST systems PCA would be more appropriate to simply reduce the number of variables in any case.

A future study might also look at the effects of sex on personality and brain networks. Sex differences in personality have been found in many personality theories, including the Eysenck Personality Inventory, Cloninger's Temperament and Character Inventory, and Big Five (Miettunen, Veijola, Lauronen, Kantojärvi, & Joukamaa, 2007; Escorial & Navas, 2007; Weisberg, DeYoung, & Hirsh, 2011; Cross et al., 2013).

Nevertheless, results were consistent with predictions. The use of Bayesian methods allowed the use of a prior distribution that applies a small amount of shrinkage towards zero. This regularization helps avoid errors of magnitude. However, this does sacrifice a small amount of power in a small sample size study as smaller amounts of data grant a prior distribution more influence on the posterior, making it more difficult for an effect to be detected as significantly non-zero. This sacrifice is worth making in order to give positive findings greater credibility.

Although the current study was not able to adequately address developmental questions, it shows a future study with the improvements outlined here is feasible and could provide valuable information concerning the development of personality and its relationship to neurobiological and behavioral pathologies. The current study also supports the notion that reinforcement sensitivity theory is a useful 'unified theory' of personality for understanding the relationship of resting state functional brain networks to personality.

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