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C-REACTIVE PROTEIN, DEPRESSION, AND SENSATION SEEKING

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ABSTRACT

Depression is becoming a leading cause of disability in the world today (Mathers & Loncar, 2006). In recent literature, relationships have been found between depression and sensation seeking (Carton, Morand, Bungenera, & Jouvent, 1995; Farmer et al., 2001) and between depression and the biological inflammatory marker C-Reactive Protein (CRP) (Ford & Erlinger, 2004; Henningsson et al., 2008; Liukkonen et al., 2006; Pikhart et al., 2009). In order to answer the question "Do people that seek a lot of new sensations have low levels of CRP which could make them resilient to depression?" this study aimed to recreate the negative correlational relationship between depression and sensation seeking, recreate the positive correlational relationship between CRP and depression, and find the connection between all three of these components. A packet containing saliva collection samples and the SSS-V and CES-D questionnaires were distributed and completed by a population of college students (n=158). Results found that the Thrill and Adventure Seeking (TAS) subscale of SSS-V was negatively correlated (-.189) with the CES-D in women. The Boredom Susceptibility (BS) subscale was negatively correlated (-.325) with CRP in women. Trends were found between BS and the CES-D and between CRP and the CES-D. It is possible that certain subscales of the SSS-V measure a protective trait against the onset of depression by way of CRP levels.

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Chapter 1

Introduction

The health of an individual is an extremely important aspect of their life. The biological, behavioral, and psychological levels of health exist within an intricate network, and the relationship between these three levels is often overlooked because of its complexity. For example, there is a distinct positive association between C-Reactive Protein (CRP), which is an inflammatory response marker, and Depression, where individuals with current depressive symptoms produce more CRP. Epidemiological studies have also shown that Depression is negatively associated with Sensation-Seeking. The goal of my study is to find the connection between all three components; CRP, depression, and sensation-seeking. The main question addressed in my study examines this relationship and asks "Do people that seek a lot of new sensations have low levels of CRP which could make them resilient to depression?"

Chapter 2

Background

CRP and Depression

C-Reactive Protein (CRP) is a marker of inflammatory response. CRP does not have the ability to pinpoint where in the body inflammation occurs but it is a general measure of systemic inflammation (Parks, 2008). Recently, high levels of CRP have been found to have a positive relationship with depression (Ford & Erlinger, 2004; Henningsson et al., 2008; Liukkonen et al., 2006; Pikhart et al., 2009). It has been projected that by the year 2030 depression will be the second major cause of disability worldwide and the main source of disability in high-income countries (Mathers & Loncar, 2006). It is a chronic disease that goes into remission but then reoccurs. The average number of episode reoccurrence is eight in a lifetime (Andrews, 2008). This can be debilitating considering that a quarter of a million respondents over 60 countries concluded that major depression is more disabling to their health than angina, arthritis, asthma, or diabetes (Moussavi et al., 2007).

A connection between CRP and depression could potentially relieve or anticipate this debility. This could be achieved first by defining the direction of this association. Unfortunately, the direction of the relationship between CRP and depression is still unclear and therefore it is commonly speculated that it is bidirectional. In other words, depression in an individual may cause elevated CRP levels, or an inflammatory state may be causing depression (Ford & Erlinger, 2004). In studies done on the anti-inflammatory properties of antidepressants (desipramine and fluoxetine), it is found that they reduce systemic inflammation which may be one mechanism by which they decrease depression (Roumestan et al., 2007). Depending on the direction of this relationship, we can use this information to prevent, treat, or predict certain diseases or psychological disorders. For example, we may be able to predict future risk of depression, find that anti-inflammatory treatment is helpful in treating depression, or it could predict oncoming comorbid diseases marked by inflammation when diagnosed with depression. A relationship

between CRP and depression can add to an increasing amount of information about how psychological disorders are linked to our biology, our behavior, and specifically to our immunological functions.

There would be a more distinguished correlation if we look at young adult populations for this relationship. This is because young adults have lower rates of medical comorbidity which can confound any association found between depression and CRP levels (Ford & Erlinger, 2004). It is also important to include studies that examine populations in non-western countries. Depression is affected by socioeconomic status (SES) and its correlating risk factors. Populations considered low SES may not have reasonable access to healthcare and therefore may not have a way to diagnose depression or they would not seek treatment. These are included in reasons as to why depression is still on the rise (Andrews, 2008). To eliminate this SES bias, data should be collected from both eastern and western countries (Pikhart et al., 2009). Lastly, we must include studies that recruit large populations in order to distinguish the relationship between CRP levels and depression.

Below, I review in more detail some of the studies that have investigated the relationship between CRP and depression. Hynek Pikhart et al. (2009) conducted a study to examine the most basic aspect of the association between CRP and depression. They decided to investigate the statistical robustness of the association. In order to evaluate depression, Pikhart et al. used a depressive symptom scale called the Center for Depressive Symptoms for Epidemiologic Studies Depression; it is more commonly referred to as the CES-D (Pikhart et al., 2009). This scale of depression was found to provide reasonable levels of sensitivity and specificity (Shean & Baldwin, 2008). CRP levels in Pikhart et al.'s study were collected through fasting venous blood sample and analyzed using a CRP assay. They set out to establish if the relationship between CRP and depression was genuine. Pikhart et al. conducted their study in the Czech Republic since it is considered a non-western European country; this allowed them to validate this relationship across different populations and consequently examine a diverse social gradient (Pikhart et al., 2009). Pikhart et al. also examined the possibility that depression and CRP are the result of a chronic disease. In addition to chronic disease, their study controlled for education, material deprivation, and BMI. After this, further factors controlled for smoking, alcohol consumption, physical activity, serum

lipid levels, and systolic blood pressure. After all of this they still found a statistically significant association between CRP levels and depression. Participants considered healthy also had this association. In fact, they found an approximately linear association between CRP concentration and the CES-D scale. It is important to note here for future reference that Pikhart et al. found that both females and males had a strong association between depressive symptoms and CRP and therefore they pooled their data (Pikhart et al., 2009). Other studies that looked at this relationship found that females had either no correlation, or a weaker association between CRP and depression (Ford & Erlinger, 2004; Howren, Lamkin, & Suls, 2009). This may also be due to the fact that Pikhart et al. used participants who were 45-69 years of age and CRP levels tend to increase with age. To review, Pikhart et al. found an association between CRP and depression in a large population, in a non-westernized country, after they adjusted for socioeconomic and behavioral covariates, and in subjects free of existing disease. From their results, Pikhart et al. concluded that the positive association between CRP and depression was genuine and robust (Pikhart et al., 2009).

Although Pikhart et al. (2009) found that there was a definite association between CRP and depression; they were not able to determine the causal direction of this relationship. This is because their study did not assess its subjects over time to define if elevated CRP levels precede or follow the onset of depressive symptoms. Other limitations in Pikhart et al.'s study are that they did not include recent infection information, which is important because it is associated with both CRP levels (inflammation) and psychological distress. They also mentioned that the CES-D may not be extremely accurate because it does not measure clinical depression and it may identify some other personality characteristics rather than just depression (Pikhart et al., 2009). Pikhart et al. validated the positive association between CRP and depression but they did not look at the difference in gender or the causal direction of the relationship; therefore it is important to further examine other studies that test the details of this positive association between CRP and depression.

A study that did focus on sex difference was one done by Ford and Erlinger (2004). The two researchers set out to find the association between major depression and elevated CRP levels in a nationally representative cohort. They conducted their study in the United States on young adults (18-39)

so that they could eliminate any comorbidity. In their study they decided to use latex-enhanced nephelometry, which quantifies CRP by measuring the amount of light that passes through the sample at an angle. Ford and Erlinger used the Diagnostic Interview Schedule to determine major depression in participants; this measurement is an interview that diagnoses a patient based on DSM-III criteria (Ford & Erlinger, 2004). This test is much more accurate at finding clinical cases of depression than the CES-D, which is designed more for depressive symptoms in a general population. Ford and Erlinger decided to stratify data based on sex because of the different depression rates in women and the previous effects of estrogen on CRP levels. This stratification of sex in the data played an important role in their results. When combined, Ford and Erlinger found a significant association between lifetime history of major depression and current elevated CRP levels. When they separated males from females they found that the association between depression and elevated CRP levels existed for men but not for women. This is different from the results of Pikhart et al.'s study. Ford and Erlinger still did not find an association in women after changing the threshold for elevated CRP level and after excluding any women pregnant or using estrogen or progesterone medications (birth control). This control for hormonal effects did not largely change the results (Ford & Erlinger, 2004). It is possible that a sex difference exists for this relationship because there is an unknown separate mechanism either causing systemic inflammation or marking systemic inflammation in women that is not CRP.

Ford and Erlinger found a rather interesting sex difference; however, they still cannot answer the question of the causality of the relationship. They mentioned the idea that the association is bidirectional; the inflammatory state may be causing depression or the depression may cause elevated CRP levels or the association may have a third factor that is unmeasured and affecting both variables. The reason it is difficult for Ford and Erlinger to find the direction of causality in this relationship is because their study was cross-sectional rather than longitudinal. Another weakness of their study is that they used a lot of self-report to determine health conditions and lifestyle. They also found that their data might imply that inflammatory states may return to normal whenever depression dissipates (Ford & Erlinger, 2004). This is

an important finding because it indicates that the association between CRP level and depression is state-dependent rather than trait-dependent.

A very large study done on a birth cohort in northern Finland had similar findings as Ford and Erlinger. Liukkonen et al. (2006) cited Ford and Erlinger in their article and hoped to find the same results. However, Liukkonen et al. used highly sensitive immunoassay methods to measure CRP concentrations in the blood. They determined depression with 13 questions on a Hopkins Symptom Checklist and required the participants to self-report any earlier lifetime doctor-diagnosed depression. Just as Pikhart et al. controlled for confounding variables of depression, Liukkonen et al. controlled for alcohol intake, body mass index, smoking, systolic blood pressure, physical inactivity, and social class to eliminate any confounding factors (Liukkonen et al., 2006). Liukkonen et al. found that in a genetically homogenous population, there is an association between current single depressive episode and elevated CRP levels in young male subjects (31 yrs.), but there is no association for young female subjects. These results were in concordance with Ford and Erlinger's study. Liukkonen et al. try to explain this phenomenon by citing another study (Penninx et al., 2003) that looked at this association in elderly subjects. Both Pikhart's study and Penninx's study, which included subjects 45-69 years old and 70-79 years old respectively; found that there is a positive association between CRP levels and depression in both genders. Liukkonen et al. point out that this may be due to the fact that men and women have a similar hormonal environment at an older age in comparison to the steep difference between hormonal environments in younger populations (Liukkonen et al., 2006). This is a valid hypothesis, although it is noteworthy to look at the previously mentioned idea about comorbidity. Younger populations may have a cleaner association between CRP levels and depression because they were less likely to have comorbid diseases (Ford & Erlinger, 2004). Therefore the CRP-depression association in men and women of older ages may exist because of this comorbidity rather than the fact that their hormonal environment is similar.

Once again in Liukkonen et al.'s study, the causality of the relationship between CRP levels and depression could not be investigated because the study was cross-sectional. This study also recognizes that depression may be under-represented because the self-report and questions about depression do not

diagnose depression as accurately as a structured clinical interview would. Analogous with Ford and Erlinger's prediction of causality in this relationship, Liukkonen et al. mentions that the association may be bidirectional. They mention that it may be that systemic low-grade inflammation and elevated CRP cause depression, or it may be that depression is promoting an inflammatory response and in turn activates the immune response (Liukkonen et al., 2006).

A study that focuses more on the direction of the CRP-depression causality was conducted by Jesse C. Stewart et al. (2009). Their study set out to define the directionality of the depression-inflammation relationship between CRP levels and depression to determine if depression is a cause or a consequence of increased inflammation. In order to address trends over time, Stewart et al. recruited 263 participants who were already involved in the Pittsburgh Healthy Heart Project (PHHP). They measured depressive symptoms and inflammatory markers at baseline and at 6 years from baseline. Depressive symptoms were calculated with the Beck Depression Inventory (BDI-II), which examines the diagnosis of current depression, past year depression, and lifetime depression than it does depressive symptoms (as the CES-D does) (Shean & Baldwin, 2008). CRP and inflammatory markers were quantified through fasting blood levels. The causality of the relationship was observed with three distinct path analytic models. Stewart et al. looked at the change in IL-6, change in CRP, and change in BDI-II (Stewart, Rand, Muldoon, & Kamarck, 2009). The primary result of this study was that depression may lead to elevated inflammation rather than being a result of elevated inflammation. However, it is important to document that Stewart et al. also found a weak bidirectional relationship between depressive symptoms and CRP levels. This is the more important finding when investigating CRP levels because their study combined CRP and IL-6 as markers of inflammation. Therefore the direction of the relationship may be convoluted in respect to CRP levels. In addition, this bidirectional relationship was not statistically significant which suggests that there are other mechanisms playing a role in this relationship. Although Stewart et al. presented new data that sheds some light on the inflammation-depression relationship; it still concludes that the relationship is bidirectional and complex (Stewart, Rand, Muldoon, & Kamarck, 2009).

This study was important to begin research that explores the direction of the relationship but it is not without limitations. Stewart et al. mention in their article that there are other articles such as Kiecolt-Glaser et al. (2003) that contradict the findings in Stewart et al.'s study. Furthermore, the sample size may have not been large enough to see a significant trend within a population, especially when this population is considered healthy. I also found it unfortunate that Stewart et al.'s study did not segregate their data by sex. Given the out of ordinary results that were found in previous CRP-Depression studies, analyzing the sex difference in a longitudinal study may result in interesting findings. This would be an appealing measure to include in future longitudinal studies looking at CRP levels and depression. Stewart et al.'s article also mentions that future studies may want to include community and patient samples in order to make conclusions about healthy versus non-healthy individuals. Lastly, Stewart et al. examined a third time point but it was disregarded due to a clerical error (Stewart, Rand, Muldoon, & Kamarck, 2009). This time point would have been critical considering previous articles' claims that the CRP-depression interaction may be state-dependent. If this is the case, then a six year time difference may have been long enough for an individual to become depressed and recover. Depression can go into remission and on average only has two symptoms present during remission (Conradi, Ormel, & de Jonge, 2010). This could also mean that CRP levels rose in accordance with depression and diminished with its remission as well. With these drawbacks we can indicate areas that need further inspection in future studies.

From a review of all of these studies we can conclude that there is a positive relationship between depression and elevated CRP levels. An important trend that we notice from the analysis of several studies is that separating the data by sex offers some interesting insight into gender differences. When measurements were segregated by sex we found that men had a significant positive correlation between CRP and depression, whereas women had a non-significant weak positive correlation in older women (ages 45-79) and no correlation in younger women (ages 18-39) (Ford & Erlinger, 2004; Liukkonen et al., 2009; Penninx et al., 2003; Pikhart et al., 2009). A fascinating look into this sex difference was offered by a recent meta-analysis. In this meta-analysis, CRP levels and depression provided the same results as the

above studies, however, studies that looked at the inflammation marker IL-6 found that women had a significant positive relationship between depression and IL-6 whereas this relationship in men was not significant (Howren, Lamkin, & Suls, 2009). This provides a look into the biological divergences between men and women. CRP may be a possible indicator of inflammation in men and IL-6 may be its corresponding indicator in women. Both men and women may have a positive relationship between biological inflammation and depression, but the marker and the way it is expressed may be through different pathways. This would be an important issue to look at in future studies.

Further knowledge to gain from these studies is that this relationship and effect may not be clearly observed unless studied in larger populations; it would be beneficial to use considerable numbers of participants in future analyses. We may also want to collect prospective data from both clinical and community samples. This way we can determine that a relationship between inflammation and depression exists in both healthy and unhealthy populations. Examining this inflammation-depression association and the causality of this association may prove beneficial to future treatments of depression or may provide a valid predictor of depression. Understanding the inflammatory processes in the brain may be the key to combining psychiatry and medicine (Miller & Manji, 2006). Defining the relationship between CRP and depression could potentially lead to extinguishing the widespread malady known as depression.

Sensation Seeking and Depression

Sensation seeking is a trait within an individual that is most often defined as a 'need for varied, novel, and complex sensations and experiences and willingness to take physical and social risks for the sake of such experiences' (Zuckerman, 1969). The term sensation seeking was first coined by Zuckerman in 1964. He and his colleagues developed a Sensation-Seeking Scale that quantified sensation seeking from a series of specific questions about behavior and personality dimensions. In order to accomplish this Zuckerman et al. broke down sensation seeking into four different dimensions. The first of these dimensions is Thrill and Adventure Seeking (TAS), this aspect measures sensation seeking in relation to components of speed and danger (Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972). The

majority of questions measuring this dimension include extreme outdoor sports or activities. Next there is Experience Seeking (ES), this component focuses more on internal sensation seeking. This includes leading an unconventional lifestyle and defiance to conform or listen to authority (Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972). The third dimension is Disinhibition (Dis), this dimension concentrates on self-indulgent attitudes when it comes to social life, sex life, and gambling (Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972). The last and fourth dimension is Boredom Susceptibility (BS) which assesses aversion to repetitive and routine people or experiences (Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972). In combination, all of these components of the Zuckerman's Sensation Seeking Scale (SSS) measure a type of extraversion that goes further than just social extraversion. It is a more uninhibited, wild, and non-conformation extraversion (Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972).

Throughout this literature there has been a correlation found between sensation seeking and depression (Carton, Morand, Bungenera, & Jouvent, 1995; Farmer et al., 2001). The relationship most often found between these two factors is a negative one; the higher sensation seeking someone is, the less likely they are to be depressed. However, even if the direction of association is suggested to go a certain way, the causal relationship between depression and sensation seeking still remains unclear. Depression may lower an individual's interest to pleasure seek and therefore result in a lower sensation seeking score (Carton, Morand, Bungenera, & Jouvent, 1995). On the other hand, particular personality traits defined as sensation seeking may predispose someone to be resilient to depression (Akiskal et al., 1983). There is promising potential in the treatment and prediction of depression if we can find the vulnerabilities and the protective factors that lead to the onset of depression.

A study done by Carton and colleagues in 1995 looked at 183 depressed patients who were diagnosed according to DSM-III-R criteria for major depression. Within the depressed patients, 85 were re-assessed after treatment for their depression. Carton et al. also matched the 183 depressed patients with a general population control that matched the age and sex of the depressed patients. One of the first major results from Carton et al.'s study was that depressed subjects scored lower than controls on all of the

sensation seeking subscales. This result is consistent with other studies and establishes the relationship between sensation seeking and depression. The other considerable result was that there was no significant difference between baseline and after-treatment sensation seeking scores. This is noteworthy because it suggests that sensation seeking may be more of a personality trait than a state consequence. This further suggests that low sensation seeking may be able to predict depression rather than low sensation seeking being a result of depression. However, Carton et al.'s study did not go without limitations. For example, it is unclear what affect a depressive episode has on an individual's sensation seeking. Sensation seeking scores were not taken before admittance for depression and therefore Carton et al. cannot conclude if sensation seeking scores were equal or decreased in contrast to premorbid levels. It would have also been beneficial if Carton et al. had followed up on these patients to find out the variability and change in their sensation seeking through different emotional experiences (Carton, Morand, Bungenera, & Jouvent, 1995).

Another study looking at sensation seeking and depression was conducted by Farmer and her colleagues at Cardiff University. The researchers decided to look at the difference in siblings in order to find any evidence of sensation seeking being a personality trait. Their hypothesis differed from my own; they predicted that higher scores of the sensation seeking questionnaire would mean more unpleasant life events which in turn made a person more likely to become depressed. To test this hypothesis, Farmer et al. recruited one hundred and five initial patients with depression who were ages 18-65 years which they called D-probands. The initial patients also had to have a sibling willing to be studied as well called D-sib. They then matched these initial patients with a control population who also had a sibling willing to volunteer; they were titled C-probands and C-sib respectively. Farmer et al. used the Zuckerman sensation seeking questionnaire and the Beck Depression Inventory to measure their two variables.

Farmer and colleagues found that sensation seeking scores were significantly negatively correlated with the Beck Depression Inventory scores. This trend appeared in the D-proband group. In addition to this finding, it was also found that sensation seeking scores of C and D proband groups were significantly correlated with their appropriate sibling group. It was even found that the C-sibs had a

higher sensation seeking score than the D-sibs; this remains consistent with the correlation between probands and siblings. Overall, Farmer et al. found that their results did not match their hypothesis. They found that rather than higher sensation seeking increasing depression, it actually had a reduced risk of depression. This outcome corresponds with my own hypothesis. It is important to mention here that Farmer et al. also found that twenty four of the subjects who were currently well but had a past history of depression did not differ from the subjects who had never reported being depressed. This result may suggest that sensation seeking is more state dependent rather than trait dependent. However, contradicting this finding is the correlation discovered between subjects and their siblings. This implies that sensation seeking may be a somewhat stable and familial personality trait. Furthermore, this personality trait may be a protective factor against depression. As a whole, it may be that there is a complex bi-directional relationship between sensation seeking and depression that can be a personality trait that is affected by mood (Farmer et al., 2001).

From the background presented, it is evident that there is clear association between Depression and CRP and Depression and Sensation-Seeking. I would postulate that Depression can be affected by a psychological response due to current circumstances or an event. This can then lead to an alteration in behavior, which can then influence biology, and a cycle of influences begins between these three components. Consequently, those with high depressive symptoms will be associated with higher CRP levels and low sensation-seeking. It is likely that in college students depression may not have been diagnosed or developed yet. It is also likely that a younger and healthier population would have lower inflammation markers. Therefore the relationship may not be strong within this age group. I have conducted a study of my own to further test this relationship among a college population. I predict that CRP will be inversely related to sensation-seeking. In combination, both high CRP levels and low sensation-seeking may predict a greater chance for depressive symptoms. If depression can be predicted by CRP levels and sensation seeking levels, then it can eventually be prevented. A small study such as mine can lead to a much larger cause. Ultimately, I hope to find that higher sensation seeking provides a protective factor against the onset of depression indicated by lower CRP levels.

Chapter 3

Methods

Overview

In order to test this hypothesis I had to measure C-reactive protein, sensation seeking and depression. I accomplished this by providing two distinct questionnaires to various Penn State classes with the incentive of extra credit or a chance to win a Penn State bookstore gift card. My questionnaires were handed out in a large packet of questionnaires and saliva collection tubes as a part of a larger study. Participants delivered completed packets to our Biobehavioral Health office. Once a sufficient number of packets were dropped off I began scoring the questionnaires, running saliva assays, and entering the results.

Subjects

Subjects included in this study were Penn State undergraduates both male and female ages 18-32, averaging 21 years of age. Overall there were 158 participants who fully completed the questionnaires. 130 of them were female and 28 of them were male.

Questionnaires

I used two questionnaires for this study. The first questionnaire was Zuckerman's Sensation Seeking Scale Form V (SSS-V), a shorter sensation seeking form that analyzes the four factors involved in sensation seeking (Zuckerman, Eysenck, & Eysenck, 1979). These factors included Thrill and Adventure Seeking (TAS), Experience Seeking (ES), Disinhibition (Dis), and Boredom Susceptibility (BS); all of which are not independent measurements but make up the general sensation seeking scale (Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972). Within the SSS-V there were 40 questions, each subscale of the SSS-V (TAS, ES, Dis, BS) had 10 questions of these total 40. The format of the question depended on the dimension of sensation seeking that was being measured. TAS questions

focused on aspects about extreme sports with questions like “I would like to try parachute jumping” (Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972). ES measured internal attitude defined by questions similar to “I like to have new and exciting experiences and sensations even if they are a little frightening, unconventional, or illegal.” Dis described social extraversion with questions such as “Keeping the drinks full is the key to a good party,” and BS measured the dislike of repetition with inquiries like “I can’t stand watching a movie that I’ve seen before” (Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972). SSS-questionnaires were scored by counting up the predetermined sensation seeking defined answers and reporting an individual score for TAS, ES, Dis, and BS; and then recording an overall sensation seeking score.

The second questionnaire was the Center for Epidemiological Studies Depression Scale (CES-D). The CES-D is used to describe and measure depressive symptoms in a community population. It does not make a clinical diagnosis of depression in the individual taking the questionnaire (Roberts et al., 1983). The CES-D included 20 questions with answers consistent with a Likert Scale. The majority of questions measured depressive symptoms with questions such as “I felt lonely” or “I could not get going” with a scale of how relative this statement was to the participant. There were four questions that were reverse scored because they were positive statements such as “I enjoyed life.” In order to score these questionnaires I added up the number of points for each response; more points indicating more depressive symptoms with the exception of reverse scored questions.

CRP Saliva Assay

I ran the Human CRP ELISA Kit in order to measure C-Reactive Protein levels for each participant. ELISA stands for enzyme-linked immunosorbent assay and it measures bound and immobilized CRP found within the samples through a technique called quantitative sandwich immunoassay. This is accomplished by pre-coating the microtiter plate wells with a monoclonal CRP specific antibody. Once unbound components are washed out, another layer of CRP-specific antibody

conjugated with the enzyme horseradish peroxidase is added to each well. The two react within a short incubation time and the reaction is stopped by adding sulphuric acid solution. The wells that contain CRP will note a change in color and this is quantified using spectrophotometrics (Human CRP ELISA Kit, 2004).

Saliva used for this immunoassay was collected using passive drool methods. Participants collected their saliva at three time points; once at wake before getting out of bed, another 30 minutes after waking up before brushing their teeth, drinking or eating, and another at bed time. Once a time point had been collected the participant was advised to store their sample in the freezer. Women participants were directed to collect their time points either during menstruation or two days after the end of menstruation. It is within this time frame that women's hormone levels are the lowest and vary the least. Saliva samples were excluded from the study if participants were using beta-blockers, anxiolytics, herbal supplements, or steroids.

Procedures

Students were approached at the beginning of classes and were given an explanation as to what the study was about in general terms and then were asked if they wanted to participate. Participants were handed manila envelopes that contained questionnaires and a saliva collection package. Oral instruction and written instruction were given to complete the surveys at any time for the males and during menstruation for the females. The subjects were then told to return completed packets to the office of my research advisor. Once I received a sufficient number of packets I proceeded to score the questionnaires as described previously and I stored the saliva samples in our -80C freezer until the time of assay.

Statistical Plan

Data collected from the SSS-V and the CES-D were analyzed by SPSS statistics software. The relationship between SSS-V and CES-D questionnaires was tested with a correlational test using each of

the subscales and the total score of the SSS-V and the CES-D. Subjects were then split by sex and similar correlational tests were performed for each sex separately. The relationship between the SSS-V questionnaire scores and the CRP values was also measured by a bivariate correlation test. This was conducted with females only because only female CRP data was measured because too few males participated in the study to arrive at a satisfactory sample. A p-value <0.05 was defined as significant. In addition to Pearson bivariate correlations, a multiple regression was performed in order to control for any confounding variables.

I charted a histogram for each variable within my data set in order to see if frequency distribution was normal. The only distribution that was not normal was CRP. To meet the assumptions of the statistical program I took the log-transformed data of CRP to facilitate normal distribution. In addition to running bivariate correlations, I ran a multiple regression so as to control for correlations between variables with the aim of finding any trends that may otherwise be confounded.

Chapter 4

Results

Previous literature has established a relationship between both sensation seeking and depression (Carton, Morand, Bungenera, & Jouvent, 1995; Farmer et al., 2001) and CRP levels and depression (Ford & Erlinger, 2004; Henningsson et al., 2008; Liukkonen et al., 2006; Pikhart et al., 2009). My study replicated the negative relationship between sensation seeking and depression with only one of the four subscales of the Zuckerman sensation seeking scale. Looking at Table 1, the Thrill and Adventure Seeking (TAS) subscale showed a significant negative correlation with CES-D (-.189) for females and a trend (-.159) for males. I did not find significant correlations between the other sensation seeking subscales or total and depression. Pearson correlation between CRP and CES-D was also not significant. However, a positive trend was found between CRP and CES-D in the multiple regression (Table 3).

I hypothesized that CRP would be inversely related to sensation-seeking and that the combination of both high CRP levels and low sensation-seeking may predict a greater chance for depressive symptoms. There was no significant correlation between CRP and total sensation-seeking in either direction. However, one of the sensation-seeking subscales (BS) did have a significant negative correlation with CRP (-.325) as seen in Table 2. Other trends were found when running a multiple regression with CES-D as the independent variable. The subscale of BS had a negative trend with CES-D and age had a positive association with CESD.

Table 1: Depression and TAS subscale correlation for a.) females and b.) males.

Correlations[a]

		TAS	CESD
TAS	Pearson Correlation	1	-.189*
	Sig. (1-tailed)		.020
	N	124	119
CESD	Pearson Correlation	-.189*	1
	Sig. (1-tailed)	.020	
	N	119	123

*. Correlation is significant at the 0.05 level (1-tailed).

a. Sex = Female

Correlations[b]

		TAS	CESD
TAS	Pearson Correlation	1	-.159
	Sig. (2-tailed)		.438
	N	28	26
CESD	Pearson Correlation	-.159	1
	Sig. (2-tailed)	.438	
	N	26	26

b. Sex = Male

Table 2: BS subscale and CRP correlation

Correlations[a]

		BS	lnCRP
BS	Pearson Correlation	1	-.325*
	Sig. (1-tailed)		.037
	N	123	31
lnCRP	Pearson Correlation	-.325*	1
	Sig. (1-tailed)	.037	
	N	31	33

*. Correlation is significant at the 0.05 level (1-tailed).

a. Sex = Female

Table 3: Multiple regression with Age, TAS, BS, and CRP

Coefficients[a,b]

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	Std. Error	Beta	t	Sig.
(Constant)	-34.608	18.736		-1.847	.077
Age	3.138	.905	.540	3.466	.002
TAS	-1.126	.396	-.425	-2.845	.009
BS	-1.437	.821	-.284	-1.750	.092
lnCRP	-1.405	.732	-.296	-1.919	.066

a. Sex = Female

b. Dependent Variable: CESD

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.685[a]	.469	.384	6.581

a. Predictors: (Constant), lnCRP, TAS, Age, BS

b. Sex = Female

Chapter 5

Discussion

Overall, I was able to significantly replicate the negative relationship between sensation-seeking and depression with the TAS subscale and only in women. I also found that women that scored themselves as being highly susceptible to boredom had lower salivary CRP levels than women that were less susceptible to boredom. In addition to these significant findings, I found a negative trend between boredom susceptibility and depression and positive trends between CRP salivary levels and depression and age and depression.

Out of the four subscales that made up the SSS-V I found that a relationship only existed between TAS and the CES-D, and it only existed in women. To recollect, TAS measured the want for extreme sports or activities (Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972). This would include outdoor sports like parachute jumping, waterskiing, and skydiving. This can be explained by a few consequential ideas. It may just be that individuals who have depressive symptoms or are feeling depressed are less likely to seek high sensations through extreme activity. In this case low sensation seeking would be a result of being in a depressed state. However, as found in the Cardiff Depression Study there is evidence of a familial trait of sensation seeking (Farmer et al., 2001). This idea is also supported by another study which found that even after treating depression, those who were depressed still scored significantly lower on the sensation-seeking scale than controls (Carton et al., 1995). Therefore another possible explanation that would support my hypothesis is that high TAS serves as a protective factor against depression. Individuals who seek out these intense hobbies may prove less susceptible to depression because they have a high sensation seeking trait. When it comes to the relationship between total sensation seeking and depression, my data do not follow previous literature. Results on the subscales are mixed but the majority of literature agrees that higher sensation seeking means lower depression (Farmer et al., 2001; Zuckerman, Eysenck, & Eysenck, 1979; Carton, Morand, Bungenera, & Jouvent, 1995; Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972).

When my data were separated into female and male categories the TAS and CES-D correlation existed among the women but not among the men. This is most likely due to the small sample size of men (28) and not enough power to see a significant correlation. A similar pattern was seen for men among TAS and CES-D (-.159); but there was not enough variance to create a significant finding. In fact, most literature found that overall sensation seeking and two of the four subscales (TAS and Dis) have a higher correlation for males than females (Farmer et al., 2009; Carton, Morand, Bungenera, & Jouvent, 1995). The BS and ES subscales have mixed literature when it comes to sex differences (Zuckerman, Eysenck, & Eysenck, 1979; Carton, Morand, Bungenera, & Jouvent, 1995). This variation between subscales found in literature may be due to the fact that Boredom Susceptibility and Experience Seeking represent more of a lifestyle and inner attitude of sensation seeking whereas Thrill and Adventure Seeking and Disinhibition signify more physical manifestations of sensation seeking. This physical adrenaline and excitement may be more attractive to males than females. In my study, CRP was analyzed only in females because of the small sample size of males. Therefore all the following conclusions and analyses are based on the female data set only.

An interesting finding was a significant negative relationship seen between BS and CRP levels. This would mean that women scoring higher on Boredom Susceptibility have lower levels of CRP and vice versa. Recollect that BS measures the dislike of repetition with inquiries like "I can't stand watching a movie that I've seen before" (Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972). Currently there is no previous literature that examines the relationship between sensation-seeking and CRP, let alone BS and CRP, therefore this is an entirely new idea and relationship that has been found. There are a few possibilities that I formulated. First, we must recognize that CRP has been connected to coronary artery disease (CAD) and that higher levels of CRP can indicate cardiac risk (Howren, Lamkin, & Suls, 2009). We can also speculate that individuals who are more easily bored and are sick of repetition are more likely to try new things and consequently may become more active. Thus, individuals who are more active (higher BS score) are less likely to form CAD and therefore may have lower CRP levels. Although this may be a prospective affiliation, I must also note that my study only examined the CRP levels of

women and the CRP/CAD correlation is predominantly seen in men (Howren, Lamkin, & Suls, 2009). Another option is that the BS sensation seeking subscale is the missing link between CRP and sensation-seeking. From previous literature we know that sensation-seeking and depression are negatively correlated and we know that CRP and depression are positively correlated; therefore, by my hypothesis, CRP and sensation-seeking should be negatively correlated. If we look at BS as a measurement of sensation-seeking, we can conclude this assumption to be true. It may be that BS serves as a protective mechanism against depression by way of lowering CRP levels. To make this relationship even more interesting, I reference the multiple regression results.

I decided to run a multiple regression in order to control for any possible confounding variables. In particular, I found that age had a positive correlation with depression within my sample of 18-32 year olds. There is a possibility that younger populations have not yet developed depression or may not be diagnosed yet and I wanted to control for this factor. After controlling for age, I found that BS was also negatively associated with the CES-D (depression). This trend completes the circle between the three components assessed in my study. To put it all together, in women, higher dislike of repetition (BS) is in concordance with lower CRP levels and they are less likely to be depressed. This would be consistent with the positive correlation found between depression and CRP levels. The correlational trend may have been significant because age was acting on both the depression scores and the BS scores; the trend may have only been visible after controlling for this factor.

Zuckerman ran a study in 1972 to find correlations between his sensation seeking scale and distinguished personality traits by using SSS and 16 Personality Factor Questionnaire (appendix A). Early results from these tests may help explain any current findings. For example, in females BS showed high positive correlations with dominance, surgency, bohemian, and radicalism and high negative correlations with super-ego (appendix A). This pattern goes along with the pattern of general SSS and the 16 PF scales. Conclusions from a study by Gorman (1970) with the PF scales found similar patterns and proposes that sensation-seeking suggests a more dominant, impulsive, non-conforming type of

extraversion (Zuckerman et al., 1972). This may give us insight into why women with high BS scores are less depressed and have lower CRP levels. It is not that women are just seeking new and novel experiences such as a new hobby; but they are going to extremes in order to resist boredom. This may insinuate that sensation-seeking is more of a personality trait rather than a simple act of seeking out new experiences. Therefore BS as a personality trait may be providing a protective factor against depression by way of lowering CRP levels.

Why then, did TAS not correlate with CRP as well? TAS was the only other subscale to have a correlation with depression. Interestingly, TAS and BS do not correlate with one another like the other subscales of the SSS-V scale-see Appendix B (Zuckerman et al., 1972). This may be a result of TAS and BS measuring and predicting different health outcomes or different underlying traits. They may be using different mechanisms of action and they may be affected by different confounding variables. This may also explain why we saw a significant correlation with depression for one (TAS) and not the other (BS).

In my results I was also able to replicate the relationship between CRP and depression by way of a trend in multiple regression. This finding may not have been significant in my study for a few reasons. First, my sample size was not extremely large (n=158) and therefore did not have high variance. Second, my data were only from females; most of the literature on this relationship has found a significant relationship for only males (Ford & Erlinger, 2004; Howren, Lamkin, & Suls, 2009). Third, in the multiple regression, age was found to be positively correlated with both depression and CRP levels and therefore may have served as a confounding factor.

My study cannot make these conclusions without recognizing limitations in my data collection and my population. I had a rather homogenous sample and thus my findings did not represent the general population. My sample also consisted of mostly college-aged individuals; this population has been found to have higher sensation seeking scores and have higher undiagnosed depression rates than older ages. Age is found to be significantly negatively correlated with sensation seeking (Farmer et al., 2001) and depression has been found to be the leading psychiatric disorder on college campuses (Beck & Young, 1978). Age is an important factor to consider in this triangular relationship.

Further limitations include a bias when it comes to individuals who returned the packets. Higher sensation seeking individuals have been found to not follow directions and are less likely to adhere to medications (Akerblad et al., 2008) and therefore may not have completed the packets correctly or have even completed them at all. This would rule out potentially high sensation seekers. The same case could go for depressed individuals. It has been found that more than a quarter of students report trouble functioning due to depressive symptoms (American College Health Association, 2005); therefore a portion of Penn State students may have not completed the packet because it was considered too much of an effort.

Discovering a potential protective factor against depression and understanding a biological mechanism for this protection is very useful in health prediction and prevention. Since depression is so prevalent in the world today and will become the second major source of disability worldwide by 2030 (Mathers & Loncar, 2006); there is a possibility that we might one day be able to asses one's risk of being diagnosed with depression. Precautions such as establishing a support system, identifying behavioral traits that can decrease risk, and exploring preventative medication can become an option that is improbable today.

Future studies may want to use CRP by blood sample rather than by saliva sample; although collecting blood samples is more invasive and requires more effort, it is more accurate at measuring CRP levels. Future studies may also find it beneficial to include a larger sample of both men and women of all ages. These studies should explore the direction of the relationship between sensation seeking and depression and CRP and depression. Is it depression that precedes low sensation seeking? Or is high thrill and adventure seeking preventing individuals from becoming depressed? Is CRP only a marker of depression? Or is it a risk factor? Longitudinal studies and data may be able to determine if sensation-seeking is a protective personality trait and decipher if CRP contributes to the onset of depression. Answering these questions can prove essential to the future treatment and possibly prevention of depression.

Appendix A

Correlations among SSS and the 16 Personality Factors

16 PF	SSS Subscale									
	General		TAS		ES		Dis		BS	
	M	F	M	F	M	F	M	F	M	F
Cyclothymia	11	-11	13	07	18	13	18	07	12	04
General Intelligence	03	-19	00	-17	11	-06	05	02	-11	02
Ego Strength	-02	-09	-05	10	-20	-13	-10	-35**	-06	-09
Dominance	52**	44**	38**	25	44**	43**	39**	37**	50**	48**
Surgency	42**	32*	42**	14	42**	36**	60**	36**	34**	40**
Super-Ego	-38**	-39**	-23*	-17	-55**	-49**	_41**	-50**	-32**	-35**
Adventurous	48**	37**	43**	21	34**	36**	25*	20	40**	25
Sensitive	07	-07	-03	-05	16	10	-14	01	09	-16
ParanoidTendencies	19	29*	13	15	25*	38**	31**	35**	20	21
Bohemian	29*	41**	13	09	31**	29*	-01	02	34**	43**
Sophisticated	-21	-03	-14	07	-30**	-16	-16	-17	-12	08
Guilt	-09	08	-03	-11	07	04	05	30*	-19	08
Radicalism	39**	39**	31**	15	44**	23	18	06	39**	34*
Self -Sufficient	-18	-10	-26*	-33*	-07	-18	-21	-09	-06	10
Controlled	-17	-29*	-20	-12	-34**	-24	-36**	-34*	-25*	-17
Tension	08	11	14	-01	13	17	02	02	-02	07

Note.—Decimals omitted. Males *N* — 71; females *N* = 51. M = male; F = female.

Appendix B
CORRELATIONS AMONG SSS FACTORED SCALES

SSS subscale	Males			Females		
	Study IA (*N* = 60)	Study IB (*N* = 36)	Study V (*N* = 151)	Study IA (*N* = 82)	Study IB (*N* = 69)	Study V (*N* = 248)
TAS vs. ES	43**	48**	39**	35*	40**	37**
Dis	21	16	35**	-01	30**	21**
BS	17	20	25**	17	22	28**
ES vs. Dis	54**	56**	54**	33**	69**	51**
BS	41**	41**	51**	60**	67**	62**
Dis vs. BS	48**	34*	44**	30**	62**	34**

Note.—Decimals omitted.
* $p < .05$.
**$ < .01.

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