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## Facial Emotion Recognition Impairments in Subclinical Depression

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Facial Emotion Recognition Impairments in Subclinical Depression

Senior Project submitted to

The Division of Science, Mathematics and Computing of Bard College

by Charles Leighton

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**Table of Contents**

Abstract.....1

Introduction.....2

    Emotion Recognition Impairments.....5

    Treatment.....14

    The Proposed Study.....22

Methods.....23

Predicted Results.....32

Discussion.....34

References.....50

Appendices

    Appendix A.....57

    Appendix B.....59

    Appendix C.....61

### **Abstract**

Depression brings with it a wide variety range of symptoms. One of the least studied symptoms in depression is an impairment in the ability to recognize the emotions on the faces of others. Previous literature has shown both that many people without diagnosed depression still display some depressive symptoms as well as that the impairments in emotion recognition are an extremely common symptom. These impairments are frequently associated with an increase in the severity of other symptoms, which makes their presence in subclinical populations especially important to uncover. In this proposed study, 400 students who don't meet the diagnostic criteria for depression would be tested on their ability to detect emotion (happiness and sadness) in rapidly presented, masked images of faces. Their detection accuracy would then be compared to their scores on the Beck Depression Inventory II. Subjects will score in the lowest range of scores of the BDI-II; from 0-13 (which is the "minimal depression" range of scores on the BDI-II) since higher scores would prevent them from participating in the study.

A negative monotonic relationship is predicted between subjects' ability to detect emotions (measured using  $d'$ , a measure of discriminability), and their self-reported scores of depressive symptoms on the BDI-II. This predicted pattern of results is consistent with the idea that there is a causal relationship between emotion recognition impairments and the development of more severe depressive symptoms. Although this study is designed to show that the impairments exist in a subclinical population rather than prove causality, it may help direct future studies towards researching this connection in order to more accurately identify people at risk for clinical depression.

Many people, especially college students, suffer from depression. Out of the population in general, college-aged adults have the highest percentage of depression diagnoses out of any age group (Richards, 2011). According to a 2012 study by the National Alliance on Mental Illness, 27% of college students reported having a primary diagnosis of depression, while some have comorbid mental illnesses as well (Grudattaro & Crudo, 2012). This number is significantly higher than the reported number of people with depression in the general population, which still comes out to 19.5% (Richards, 2011). In addition to the already high rate of depression, nearly half of that 19.5% with depression will suffer from chronic depression for their entire lives (Bourdon, Rae, Locke, Narrow, & Regier, 1992).

The large number of reported cases of depression might not be the full scope of the problem presented by depression. This is likely an underestimate. The 19.5% figure is based on those who are formally diagnosed with depression. Without a diagnosis, receiving treatment is exceptionally difficult. Insurance companies need an affirmative diagnosis before being able to provide coverage for treatment. Due to the difficulties of receiving treatment, many people who do receive a depression diagnosis do not receive treatment afterwards. For example, a 2008 study found that 85% of college students with moderate to severe depressive symptoms were not receiving any treatment (Garlow et al., 2008). Additionally, Angst & Merikangas (1997) suggest that even more people experience depression than are diagnosed, finding in one study that 45% of diabetes patients were found to have undiagnosed depression.

Part of why major depression is so hard to treat is that it has extremely heterogeneous symptoms. People with the same diagnosis can have vastly different experiences of the disorder. To capture this wide variety of symptoms, clinicians use a list of symptoms and requirements found in the Diagnostic and Statistical Manual of Mental Disorders. The DSM is a long-

established source of information and diagnostic guides for psychologists, and has gone through numerous additions and five separate versions. It is internationally well-revered, and has become the standard for informing clinical diagnoses.

The fifth and latest version, the DSM-V, has four main requirements for someone to be diagnosed with depression (American Psychiatric Association, 2013). They must experience a depressed mood or loss of interest or pleasure in daily activities for at least two weeks (this must represent a change in mood from their baseline functioning; i.e., if someone experienced a manic phase and then returned to baseline, this subsequent loss of pleasure wouldn't be counted as a depressive episode). This change must also lead to social, occupational, or educational impairments. On top of that, the patient must meet at least five of nine symptoms: 1: depressed mood or irritability; 2: anhedonia (a pronounced loss of interest in activities that were once enjoyed); 3: significant weight loss or weight gain; 4: insomnia or hypersomnia (sleeping too little or too much); 5: psychomotor agitation or retardation; 6: fatigue or loss of energy (separate from hypersomnia); 7: feelings of guilt or worthlessness; 8: an inability to concentrate; and 9: suicidal tendencies. Lastly, these symptoms cannot be better explained by substance abuse, other medical illness or psychiatric disorders, or bereavement/grief. If someone does not meet these requirements they then do not meet the criteria for diagnosis, and cannot be diagnosed with depression.

Due to the strictly categorical nature of depression diagnoses (you either have it or you don't) currently described by the DSM-V, even subclinical individuals can display depressive symptoms, many of which can be socially debilitating and frequently worsen with time (Bistricky, Ingram, & Atchley, 2011; Angst & Merikangas, 1997). A problem with this style of diagnostic requirements -- where many of those who aren't able to be diagnosed nonetheless

display some of the symptoms -- is that it is very difficult to decide how few symptoms are too few for diagnosis. This issue is exacerbated by how depressive symptoms often increase in severity if left untreated. Because of this, even subclinical individuals are at risk of becoming clinically depressed. Depression diagnoses have to be early enough to catch patients before their illness develops untreated for too long yet also have to not be thrown around too lightly and risk misdiagnosing people.

This is a problem because of the frequent cases of chronic, lifetime depression. The chronic nature of their depression is important to notice, as chronic illness is often caused by a late diagnoses (Garland & Solomons, 2002). When people are diagnosed late enough, their symptoms have developed to such a point that fully treating them becomes extremely difficult due to the habits and thought patterns built before diagnosis. The importance of early diagnosis and treatment for people with depression is in large part due to the development of symptom severity in depression mentioned earlier. Patients with depressive symptoms often develop more and more severe symptoms unless they receive effective treatment. This presents a grim situation for undiagnosed individuals, as the developing symptoms make functioning socially, professionally, and occupationally increasingly difficult.

Why don't symptoms remain static at the same level of severity? This is due to the nature of the symptoms themselves. Anhedonia, sleeping and eating pattern disruptions, sadness, and the myriad of possible symptom combinations pile on top of each other and negatively affect interpersonal relationships and daily life. This leads to a negative cycle, where symptom progress decreases the quality of life, which in turn leads to the development of more severe symptoms. For instance, take someone who is only slightly depressed. They might, for instance, lose sleep, gain weight, and are often irritable. Due to these personal, internal changes, their self

esteem can decrease as can their professional performance. On top of this, further symptoms of depression are a loss of hope, guilt, or feelings of worthlessness (American Psychiatric Association, 2013). These new stressors can exacerbate the existing symptoms, making them more severe or leading the individual to develop additional symptoms. This new progression in turn affects their daily life, which in turn leads to a worsening of symptoms. It is a vicious cycle. It is easy to see how these symptoms can lead to both someone's personal life and their current mental status to quickly devolve.

In addition to nine symptoms listed by the DSM-V, the feedback between social relationships, and symptom development, another factor can lead to depression worsening, one unlisted by the DSM-V. This unlisted symptom - an impairment in the recognition of the facial emotions of others - seems to contribute to social dysfunction and negative thinking, making early detection critical (Bistricky, Ingram, & Atchley, 2011). Even if fewer than five of the nine depression symptoms listed by the DSM-V are present, the presence of emotion recognition impairments frequently leads to the development of increasingly severe symptoms, if not also ones not experienced previously.

### **Emotion Recognition Impairments**

In the emotion recognition impairments that frequently occur in depression, people are not only less able to recognize the emotions of other people, but actually misidentify the emotions to others. Douglas and Porter studied 68 patients with severe depression compared to 50 healthy controls on their ability to recognition emotions. Not only did the depressed group misinterpret neutral faces as sad faces significantly more often than the healthy control group, but they also misinterpreted neutral faces as happy faces significantly less than healthy controls.

The 2010 study by Punkanen et al. also measured the abilities of depressed patients to recognize emotions compared to healthy controls. However, their study measured their ability to recognize emotions not in faces, but in music. Seventy-nine depressed and alexithymic patients (alexithymia is a common symptom of depression in which one is unable to identify and describe their own or others' emotions and is also not listed in the DSM-V as a possible symptom to be considered during a diagnostic interview) and 30 healthy controls were played 30 musical excerpts. Each excerpt was previously rated by healthy controls on how well they represented each of five basic emotions: anger, fear, happiness, sadness, tenderness. In the study, subjects rated each song on a Likert scale for each of the five basic emotions. Subjects also took the Montgomery-Asberg Depression Scale (MADRS) to determine the clinical severity of their depression and the extent of their alexithymia. Increasing levels of clinical severity depression and alexithymia were correlated with increasingly lower scores on their ability to recognize the emotion in the musical excerpts played to them.

The negative effects of this impairment in emotion recognition can be seen when added to the previously described cycle of someone with an initially low level of depression whose symptoms negatively affect their day-to-day relationships and career, which in turn worsens their symptoms. Facial emotion recognition impairments would lead people to believe others are acting more negatively towards them, speeding up the rate at which their lives are negatively affected by their depressive symptoms and relationships. Instead of only feeling guilty for a poor performance at work and tired from a lack of sleep, people with these impairments might think that people are being negative to them, preventing them from using social support as effectively as they once could have. Ironically, the time when people need support the most is also when they are the least likely to look for it. This worsens their already existing symptoms and speeds

up the process at which their depression develops as their other symptoms worsen and their emotion recognition impairments get more pronounced. This leads to further symptom development and the falling apart of their interpersonal relationships as the once mistaken negative reactions of others develop into actual negative reactions in response to the depressed individual's behavior.

In addition to not measuring predictive symptoms like emotion recognition impairments, current diagnostic practices for depression have split depression into several subtypes of depression based on groupings of symptoms. This practice has been critiqued as being too specific and exclusionary through trying to understand and categorize the plethora of symptoms that can be developed during depression into different subcategories of depression (Bourdon, Rae, Locke, Narrow, & Regier, 1992). The concern is that by focusing on the minutiae of the disease like the individual symptoms, clinicians are preventing their diagnoses from being as accurate as possible. Bourdon et al. also discuss the problem of the DSM's increasing specificity and exclusivity of diagnoses. Later editions of the DSM have made being diagnosed increasingly difficult as they add more requirements that must be met in order to be diagnosed (Carmassi, Corsi, Gesi, & Bertelloni, 2017). As our understanding of depression has changed, so too have the requirements for being diagnosed with it changed as well.

The increasing list of requirements for being diagnosed serves an important purpose. The wrong diagnosis can not only prevent recovery, but set it back through ineffective treatment. Yet while the continuous revisions to diagnostic practices that better reflect our understanding of depression are extremely beneficial, the increasingly exclusive nature of diagnosis has led many people that deserve a diagnosis for depression to not meet the diagnostic criteria that the DSM-V requires. This would not be a problem were it not for the fact that many of them still display

depressive symptoms — just not enough or the right ones to be diagnosed according to the DSM-V. For this paper, these specific subclinical groups can be called “fringe groups,” as they are on the edge of being diagnosed. Were the requirements less exclusive, they likely would receive a diagnosis. But, by being a categorical diagnosis — patients either have or do not have depression — people who display some but not all of the required symptoms cannot be diagnosed. If people display some symptoms for depression but do not fit the full requirements for diagnosis, they still will not receive a diagnosis. Despite going to a clinician to hopefully receive help, many people just don’t meet the full requirements for diagnosis regardless of their existing symptoms. It is because of this diagnostic division that clinicians face the problem of determining how much of which symptoms are enough for diagnosis. Making the requirements too relaxed will allow for misdiagnoses, while making them too stringent will keep some people from receiving much needed treatment. Currently, that division is informed by the DSM-V, although the placement of that division is under debate in regards to the issues of the frequent existence of subclinical symptoms, late diagnosis, relapse, and chronic depression.

This is problematic for several reasons. To begin with, not possessing all of the symptoms required for diagnosis does not mean that the symptoms are not debilitating for the patient. By withholding diagnosis, people are often left with untreated symptoms. One of the problems that this leaves fringe groups with is how to get treated without a diagnosis. Due to pressure from insurance companies, medical practitioners and clinicians frequently are unable to provide affordable service to patients if their medical problems do not meet requirements (such as a primary diagnosis) set forth by the insurance companies in order to help pay for treatment (Weissman & Rosselli, 2016). Without meeting these requirements, many people cannot afford treatment.

This is not to say that there should not be a cutting off point for diagnoses. At a certain point, the symptoms are so minor that they cannot cause social impairment or significantly impact someone's life and behavior. However, the diagnostic requirements for depression exclude more than just the minorly afflicted. Many of the symptoms of depression worsen with time, and even patients who are barely on the fringe for diagnosis can often spiral into diagnosable depression as their symptoms escalate.

The specificity of the diagnostic requirements isn't the problem with diagnosis. The problem instead is that the specific symptoms that people experience can and often change as their illness progresses. Depression has many symptoms, more even than the nine listed by the DSM-V, as can be seen by their omission of the emotion recognition impairments. These symptoms can mix and change overtime on a very individual bases, and yet these heterogeneous symptoms have instead been grouped into ten different subtypes of depression, which clinicians then assign people to based on their symptoms (See Appendix D). This presents a problem considering how often people's symptoms change over time. People's symptoms can vary so much over the course of their illness that they actually meet the requirements for several different subtypes of depression depending on their current symptoms.

The problem of heterogeneous symptoms was addressed by Kanter, Busch, Weeks, and Landes (2008) in their study on the traditional behavioral models of depression. They explicitly state that if depression is going to be grouped into subtypes based on different groups of symptoms, then there are as many subtypes of depression as there are people with depression. It seems to me that this is especially important in regards to arguing against the multiple subtypes of depression. Depressive symptoms differ largely from individual to individual, and while using cookie-cutter methodology to label patients as one subtype or another does make the

clinician's job of recognizing symptom patterns easier, it does not necessarily more accurate. Kanter et al.'s argument is further supported by the fluid, volatile nature of depressive symptoms. Depression tends to be volatile, and people with depression frequently switch symptoms and swing from one subtype to another.

The idea that depressive symptoms shouldn't be used to categorize depression into subtypes does not mean that symptoms shouldn't be measured at all. Symptoms are how depression presents itself, and without — for instance — performing the equivalent of a neural litmus test for depression, they are how depression will be diagnosed. The symptoms being displayed, however, should be used to identify whether or not the patient has depression and not to assign the patient to a subtype of depression with its own specific treatment plans when the patient's symptoms will likely change so much that they meet the requirements of an entirely different subtype.

This inclusive view of depression did not start with Kanter, Busch, Weeks, and Landes, however. Over a decade before, Angst & Merikangas (1977) recognized that the depression spectrum must be far broader than was currently believed. They found that many patients with depression experienced multiple subtypes of depression over the course of their illness. These changes were relatively quick, with patients often displaying multiple subtypes and little stability in symptomatology over a 15- year period. Additionally, many people who didn't meet the specific criteria for depression were nevertheless given treatment by their clinicians who deemed it appropriate due to their poor mental state. This suggests that even clinicians acknowledge that the DSM's requirements often miss the mark. Diagnostic criteria and insurance policies have continued to get stricter since 1997, and receiving treatment without a diagnosis now is extremely difficult. Although it is completely necessary and important to have a diagnostic

threshold, having an increasingly specific and exclusionary one may be preventing some people from receiving the treatment they need, as does the implied idea that depression is categorical and unable to fluctuate continuously.

While this more inclusive view towards diagnosing depression could be seen as leaving openings for bias in the forms of human error or subjective self-report on behalf of the patient, the evidence suggests that the opposite would occur. If anything, diagnosing people from a more holistic approach seems like it would improve diagnostic accuracy. More and more research has shown that the problem with diagnosing depression is not over diagnosing, but rather underdiagnosing (Bourdon, Rae, Locke, Narrow, & Regier, 1992; Angst & Merikangas, 1997; Bistricky, Ingram, & Atchley, 2011). A more inclusive style of diagnosis would act as a safety net for people currently in the fringe group of diagnosis described earlier, allowing some to receive treatment that currently are unable to. The inclusion criteria don't have to be drastically changed, either, as depression severity exponentially decreases in frequency. There are far more people who are subclinical (according to current requirements) than there are with severe depression. Increasing the accuracy of diagnoses through lowering the diagnostic requirements therefore would not require the diagnostic requirements to be changed drastically, only enough to let in those currently on the fringe. Rather than focusing on the many possible subtypes of depression and insisting that patients be strictly categorized according to their current symptoms, this change in how we see mental illness could lead to more inclusive diagnostic practices once clinicians stop focusing on the minutiae of the symptoms.

A problem with diagnosis beyond being too exclusive with diagnostic requirements is that many of the people that do receive a diagnosis for depression either relapse after their initial remission or suffer from chronic depression and never successfully treat their depression. The

need for changes in diagnostic practices is further highlighted by the number of patients with depression that deal with chronic symptoms for much if not all of their lives and never experience full remission. This pattern of diagnosis/lack of recovery was seen in a 1986 study by Faravelli, Ambonetti, Pallanti, & Pazzagli. In the study, 101 patients with depression were followed for at least a year after their initial recovery from their first major depressive episode. Fifty-one of the 101 patients relapsed during the course of that year. The relapsed patients additionally displayed predictive signs for relapsing despite an initial recovery. They displayed not only significantly higher levels of residual symptoms than the 50 non-relapsed patients but also poorer social skills and higher levels of pathology, despite similar treatments. All of this supports the idea that incomplete recovery is likely to lead to relapse within just a year after treatment.

This pattern of residual symptoms after treatment developing into relapses of depression was also found by Lewis, Paulus, and Schettler in 2000. They found that residual sub threshold depressive symptoms remaining following the completion of even the first major depressive episode frequently signifies the continuation of the depression, often leading to a more debilitating and chronic prognosis. Additionally, they recognized that even if the ongoing symptoms are currently subclinical, they still show that the illness is currently active and that continued treatment should be advised.

One of the symptoms that often remains after remission is the impairment in emotion recognition. This is cause for special concern considering how this impairment often causes other symptoms to develop over time. One study suggesting this compared 54 women with major depressive disorder (the technical term for depression) in remission for at least two months who had no comorbidity and were on antidepressants to healthy controls on their ability to

recognize emotions (Biyik, Keskin, Oguz, Akdeniz & Gonul, 2015). Remission is the point at which symptoms are either absent or much reduced. The remission group scored significantly higher than healthy controls on their ability to recognize sad faces, supporting the idea that people who have previously had depression are more aware of negative stimuli than those who haven't had depression. This unusually high sensitivity could be a the residual version of the impairments in emotion recognition.

While not everyone with residual symptoms relapses, this pattern of results has consistently been found in patients with remitted depression. In a two year cross-sectional study, 59 subjects with both active and remitted MDD and healthy were compared using a facial emotion recognition task (Shiroma, Thuras, Johns, & Lim, 2016). Depressed patients had a much lower sensitivity to happiness, especially at moderate intensity of stimuli. Surprisingly, this pattern was the same for those patients in remission with a complete absence of or minimal symptoms. Even remitted patients with a complete lack of symptoms showed the same negative biases as the active MDD group, albeit to a slightly lower degree. Even after the remission of other symptoms, these biases can significantly change how people recognize the emotions of others.

The combined effects of residual symptoms following remission can be seen clearly in a 2013 study by Loi, Vaidya, & Paradiso on the ability of patients with depression to recognize emotion in body language and their social abilities. Their study included 51 patients with active major depressive disorder (MDD), 68 patients who had been in remission for at least two months (MDD-R), and 69 healthy controls (HC). The subjects were tested on their ability to read emotion from body language as well as on their social adjustment. As expected, the subjects with active MDD scored were significantly less accurate at recognizing body language than both

of the other groups. Surprisingly, however, even the patients with MDD-R scored lower than HC emotion recognition. Both of the depressive groups scored lower than the HC on social adjustment, although the active MDD group scored even lower than the MDD-R group.

The observation that subjects with MDD in remission performed significantly worse than healthy controls shows that even after remission, people who have experienced depression still possess some of their symptomatology after the disappearance of most of the major symptoms. This may influence both the high rate of people that relapse as well as the low rate of people who experience permanent remission. This is exactly why late diagnoses is such a problem in the medical community, especially the mental health community. Once some symptoms have been introduced and developed, it becomes increasingly hard to treat the illness to the point where the patient is rid of all of their maladaptive behaviors.

To summarize, people with the observed emotion recognition impairments are hypersensitive to negative emotions and hyposensitive to positive emotions. Additionally, they often misattribute emotional value to a neutral face, and while there are conflicting results regarding the direction of the neutral to emotional misattribution, the majority of studies find that the neutral faces are typically identified as negative. This leads people with these impairments (even the subclinical ones) to often view others' emotions as more negative than they really are, causing them to react more strongly to the perceived negative emotions of others and less strongly to sincerely positive ones.

## **Treatment**

Treatment itself is very complicated and multifaceted. People do not recover from depression for a variety of reasons. Depression is often both chemical and situational in nature, and

combatting one without the other, or only being able to treat one without effectively treating the other, is a recipe for chronic depression. This is why so many treatment plans use both medication and therapy like CBT (Cognitive Behavioral Therapy) to both change how chemicals like serotonin are released in the brain why also trying to change how the patient thinks and sees themselves and their relationship to the world.

Even the effective combination and use of various, complementary treatment techniques do not guarantee remission. One of the main causes for this is a late diagnosis. When people are diagnosed with depression too late (or any mental illness for that matter), their symptoms have progressed to such a point that they are hard to fully treat. Either the symptoms alone or their daily habits and thought patterns have advanced to such a degree that the removal of such ingrained phenomena is incredibly difficult.

The existence of these symptoms even after treatment and the remission of other symptoms makes a full recovery and prevention of relapse difficult, but recent research suggests that the impairments in emotion recognition themselves can be treated with the right methodology (Wolf, Pujara, & Baskaya, 2016). Subjects with lesions in their ventromedial prefrontal cortex (vmPFC) were found to be impaired at recognizing moderate intensity expressions of anger compared to healthy controls. These impairments, however, can be lessened by teaching the lesioned subjects to fix their gaze at important emotional sections of the face such as the eyes and mouth. These findings suggest that 1) the vmPFC is important for emotion recognition by directing visual attention to emotional parts of the face and 2) that teaching subjects with similar symptoms where to fixate may help reduce impairments. If gaze manipulation can decrease emotion recognition impairments present in patients without damaged

vmPFC's, then patients with remitted or current depression would have a valuable tool to prevent relapse and help with the prevention of symptom escalation.

The prefrontal cortex not only seems to be involved in the emotion recognition impairments present in Wolf et al.'s subjects, but also seems to be related to the development of depression in general. In a study by George, Ketter, and Post (1994), dysfunction of the prefrontal lobe was found in both primary and secondary depression. The study found that the prefrontal lobe in these patients was frequently hypometabolic, with less activity than was observed in healthy controls. They propose that this observed prefrontal lobe dysfunction could play a large role in producing many of depression's symptoms through poorly managing the emotional center of the brain — the limbic system.

How could these impairments apply to subclinical populations that don't meet the categorical definition of the DSM-V? If these impairments exist in these fringe groups, there's a large chance that they will lead to a worsening of symptoms despite the individual originally not meeting diagnostic criteria. These impairments, if present in fringe groups, could lead to the development of either larger impairments of emotion recognition as well as additional and more severe depressive symptoms, leading them to eventually meet the requirements for a diagnosis of depression. This possible turn of events is problematic, as if people with subclinical symptoms but impairments in emotion recognition seek out a clinician for a diagnosis and don't receive it, they may still develop depression after being cleared. One of the holes in current diagnostic practices is that these impairments are often neither asked about nor are they considered when looking at symptoms. By not measuring or at least enquiring about a patient's ability to recognize emotions, clinicians risk missing a large warning flag that the patient's symptoms might worsen in time.

There are good reasons for why emotion recognition impairments have not been included in diagnostic criteria. To start with, they aren't easily measurable in the fashion that diagnostic interviews typically use. They are difficult to self-report — in fact, they are typically measured using a facial emotion recognition task on the computer or another version such as the test on song emotion recognition — so a checklist of symptoms such as the DSM-V and self-report questionnaires like the BDI-II aren't able to easily capture them. In addition to the difficulty of measuring them without equipment, the way in which these impairments are measured has not been standardized yet. The literature surrounding these emotion recognition impairments reports conflicting results based on the different techniques used to measure them. These impairments are typically measured using an emotion recognition task, but the task itself has several variables that can affect the results such as the faces used, the duration of the image shown, and the positioning of the face. In these emotion recognition tasks, subjects are usually shown quick images of faces and then asked to identify the emotion or answer a forced choice question where they have to pick between two options in an attempt to identify the expression on the face (these options are typically emotional and neutral).

One of the variables with the largest impact on results is display time. Different studies have used different display times for the faces, ranging from subliminal images shown for only seven milliseconds to an unlimited display time where the subject is allowed to examine the face for as long as they need before answering. Shorter display times seem to be better at detecting emotion recognition impairments. This can be seen when comparing the predictive factors found in studies that used either very short or very long display times. In one study, 26 depressed patients and 26 healthy controls had the event related potentials (ERPs) of their occipital P1, occipito-temporal N170, and Parietal P3 measured while being subliminally shown slides happy,

sad, and neutral faces (Zhang, He, Chen & Wei, 2016). ERPs are fluctuations in the brain's electrical activity in response to stimuli and are measured using an electroencephalogram (EEG) on the scalp. Depressed patients responded significantly slower to happy faces than did HC as well as responding faster to sad faces than HC. Both faces responded faster to sad faces than neutral faces, but the patients displayed this more than the HC. Despite the differences in ERP response time, neither groups were able to identify the faces they were shown above chance, ensuring that the reactions to the faces were subliminal.

Zhang et al.'s participants were shown a subliminal image of either a happy, angry, or neutral face for 7ms, followed by a mask and a probe (a question). The subjects had to decide whether the face shown to them was emotional (sad or happy) or neutral. In this study, the results had a significant relationship with depression, with the depressed group performing significantly less accurately than the nondepressed group. Conversely, in another 2016 study by Fieker, Moritz, Kother and Jelinek, 45 women with clinical depression and 30 women without any psychiatric diagnoses were tested on their ability to recognize the emotions of others in photographs as well as their confidence in their responses. Participants were shown 63 photographs that were balanced across gender for as long as they needed to respond. Both groups performed with the same levels of accuracy, but accuracy was associated with response confidence *instead* of depression. While the two are related, response confidence depends less on depression and more on other personality features. Accuracy on the task varies between individuals even without displaying any impairments. Some people are naturally faster at detecting emotions and recognizing patterns than others, and the unlimited time frame allowed subjects to think carefully over their decisions, possibly allowing some of the faster depressed subjects to think over their answers and influence the results.

The accidental change of the variable that influences task accuracy is a very important distinction. By allowing the subjects to view the faces for as long as needed, the task becomes too easy to effectively detect the impairments. It leads to a large ceiling effect where even those with the emotion recognition impairments can score well. This leads to a problem with the internal validity of the study. Through the choice of an unlimited display time, Fieker et al.(2016) changed the variable predictive of task accuracy from depression to answer confidence. While testing for response confidence is a very good idea, allowing the participants to take as long as they want with each photograph is very unrealistic. People often don't hold expressions for long periods of time and the ability to make accurate snap decisions of what emotion another person is feeling might still be affected by depression.

The external validity of the study is also impacted by the change in display time. While external validity isn't always a priority for researcher, the emotion recognition impairments are frequently too subtle to be seen using methodology that doesn't present enough of a challenge to subjects to receive a diverse spread of scores. To be clear, these impairments don't alter every interaction that patients have with other people; sometimes they do know what emotion the other person is displaying. The difficulty that the emotion recognition impairments present is that oftentimes, the emotions displayed by others aren't as easy to discern, even for the unafflicted. Faster display times better mimic these subtler, faster interactions (such as micro expressions or brief changes in emotion), and through them the external validity of the study is also increased.

By diagnosing depression early through the inclusion of emotion recognition impairments in diagnostic practices, symptoms can begin to be treated before they reach the point of no return (or at least, the point of difficult treatment). Doctors do not wait for a condition to develop fully before treating it, and clinicians should begin to follow their example to better diagnose and

more effectively treat their own patients. By diagnosing depression before the full onset of symptoms, treatment becomes easier as the person is more optimistic/mentally healthy and has stronger interpersonal relationships. If used in initial diagnostic interviews, if someone who is subclinical according to the DSM-V still possesses these symptoms, a follow-up interview could be recommended. These symptoms so often lead to the development of other symptoms and so negatively affect interpersonal relationships that someone who displays these impairments is at serious risk for their other symptoms to develop from nonclinical to clinical. By recognizing the potential for symptoms to worsen, people with a subclinical diagnosis who are still at risk can hopefully receive the help they need.

Presymptomatic warning signs and diagnosis are not exclusively useful for mental illness. Medical fields other than psychology have been moving towards increasingly early warning signs and preventative measures rather than waiting for the onset of symptoms before beginning treatment. In cancer, heart disease, AIDS, and even dental treatment, warning signs and preventative measures are largely encouraged and looked for. It is largely because of this early detection that medical treatment has become so much more effective in recent years. The entire principles behind vaccines and flossing are to be preventative measures. Despite this change in treatment in the medical world, mental illness treatment still depends on the outbreak of symptoms to occur first, with very few preventative measures being proposed.

In addition to being used as an additional symptom to consider in diagnosis, these impairments could also be used as a method of early detection. As was mentioned earlier, these impairments frequently lead to the development and worsening of other symptoms. One of the main reasons for the high rates of relapse and low rates of remission in depression is a tendency towards late diagnosis and treatment, to the point where the individual with depression has

developed such severe depressive symptoms that it is very difficult to treat completely. If emotion recognition impairments are found during an initial diagnostic interview while other symptoms remain minimal, the psychologist treating the individual with depression would have been given at least a warning that other depressive symptoms may follow. If necessary, the clinician could recommend a follow-up visit to ensure that symptoms remain subclinical.

The emotion recognition impairments present in depression have not only been found to lead to the development of other depressive symptoms, but have also been found to predict therapeutic outcome (Dannowski, Kerstin, Donges, 2006). In this study, the initial levels of emotion recognition impairments in twenty depressed individuals were observed to predict the success of their treatment over the course of seven weeks. This is very promising in regards to the useage of these impairments as early warning signs for clinicians, but one problem is left before any changes can be made to how depression is diagnosed, the changes have to be backed up by more than relationships found in previous literature. These impairments have to be shown to exist in people who haven't yet met the current criteria for depression according to the DSM-V. If there is no one who scores subclinically has these impairments, then their use as an early warning sign is nonexistent. They have to be shown to exist in currently subclinical populations. The benefit of these emotion recognition impairments extends to early detection and helping detect at risk individuals, but if these symptoms only develop once the major depression has already set in along with other symptoms, they won't be very helpful in regards to preventing those other symptoms. In order to show that these emotion recognition impairments do exist, I'm proposing a study that will show what the likely course of events were the experiment to be run.

### **The Proposed Study**

The experiment itself is simple. While previous studies have looked for these impairments in subjects with depressed or those in remission in comparison to nondepressed control groups, the proposed study will look for signs of these impairments in a subclinical populations. The subjects will undergo a clinical interview with a trained clinician to ensure none are diagnosable, and they will fill out the BDI-II and take an emotion recognition task on a lab computer. The emotion recognition task will consist of 160 trials. Each trial will be a forced choice response following the subliminal display of a face, which will be either emotional (angry or happy) or neutral. Subjects will have to identify whether or not the face they were shown was emotion or neutral. Subliminal imaging will be used due to the strong results found by previous subliminal imaging studies on emotion recognition impairments (Zhang et al., 2016).

Due to the number of subclinically depressed individuals who still display other depressive symptoms, there is a predicted significant relationship between depression symptom severity scores as measured by the BDI-II and the extent of the emotion recognition impairments as measured by an emotion recognition task. This task will be based on the methods of the Zhang et al. (2016) study due to the effectiveness of their methods at showing a relationship between the same two variables in a clinical population. In this study, however, the methods will be presented to people with specifically nonclinical conditions. In the task, subjects will be asked whether the faces shown to them were happy, sad, or neutral. The relationship between symptom severity and task accuracy is predicted to be a negative and monotonic one, whereas BDI-II scores increase, accuracy on the emotion recognition task decreases.

## METHODS

### Participants

Five hundred students will be recruited from colleges in the Dutchess County area through advertisements on bulletin boards and directed to an online screening process in the first step of a screening process to ensure that the subjects have no previous diagnosis of mental illness or history of medication or treatment for mental illness. Of those original five hundred students, approximately four hundred will meet the requirements for participation in this study. When the subjects pass the initial online screening, they will be invited to take part in an in-person study as well as given a date for participation in the study and a follow-up screening by a trained clinician to ensure that they fit additional requirements for their continued participation in the study.

Since I want to study a population with subclinical depression, I need to study individuals that do not meet the DSM-V's diagnostic criteria for depression. However, the diagnosis approach of the DSM-V is categorical; depression is either diagnosed or it isn't. This makes comparing depression severity in subclinical individuals to the continuous variable of task accuracy difficult. In order to do so, the BDI-II will be used in order to compare depression severity to task accuracy. The BDI-II will allow subjects to provide a continuous measure for their levels of depression. Although the BDI-II is not a diagnostic tool and was designed to track symptom severity rather than to diagnose, the BDI has been observed to have a great deal of predictive ability regarding depression (Veerman, Dowrick, & Ayuso-Mateos, 2009). In this study, the population mean on the BDI-II for dozens of towns in England, Finland, and Norway was found to predict the average rates of depression for each locality.

This group of interest — people who don't quite meet the symptom requirements for a diagnosis of depression — should score from 0-13 on the BDI-II. This range of scores is listed as “minimal depression” (Beck, Steer, & Brown, 1996) and is strongly related to subclinical depression diagnoses (M. Campbell, D. Maynard, J. Roberti & M. Emmanuel (2012). This range of scores seems like the most likely range that people with undiagnosed depression would score. Higher scores than that represent more severe symptoms, and if they score above the minimal depression range of scores on the BDI-II it's likely that they're diagnosable according to the DSM-V's requirements.

The number of subjects was determined after reviewing the distribution of BDI scores from 15, 233 American college students in a 2015 study on college rates of depression (Whisman & Richardson, 2015). In this study, 75% of the subjects scored a 13 or lower, placing them in the Minimal Depression range of scores on the BDI-II. According to the National Center for Education Statistics, 20.5 million students enrolled for classes in the fall of 2016. Seventy-five percent of America's 25,500,000 college population is 19,125,000. With a population this size, 400 subjects are needed to achieve a 95% confidence level that the relationship between subject accuracy on the emotion recognition task and the BDI-II scores are not by chance along with a 5% confidence interval. While this is a proposal study, I am sensitive to the time and costs required for this study to actually be run, and choosing to run 200 more students would be a huge jump in both of those resources. The budget for the experiment can be found in the appendix (See Appendix C). To get 400 useable subjects, at least 534 college students will have to be screened ( $400 / .75 = 533.33$ ). To accommodate additional obstacles like participant dropout during the experiment and human error, slightly more students may have to be screened.

While the Whisman et al. (2015) study does cite slightly higher rates of depression than have previously been found, the number of subjects required for a 5% confidence interval with a 95% confidence level remains 400 until a population of 222,639, at which point the required number of subjects slowly decreases. For fewer subjects to be required in the current study, America would have to have 1.1% the number of college students who score in the 10-13 range of the BDI than predicted. This would be about a tenth of the number of students who *are* depressed in America at any given time (there are roughly 6.7% of the population in America with depression at any given time, which amounts to 21,775,000 people). Due to the exponentially decreasing pattern of scores on the BDI-II (and based on that, on the exponentially decreasing pattern of depression intensity), it would be impossible for there to be *fewer* subclinically depressed individuals than positively diagnosed individuals, so the relatively high participant number of 400 is still necessary. It is because of this exponential pattern of scores on the BDI-II that so many participants are needed. With most of the population scoring in the low single digits, enough participants are required to make sure that some of them score on the higher end of the minimal depression range (i.e., 8-13).

### **Measures**

The Beck Depression Inventory-II will be used in both the online pre-screening procedure and the follow-up screening in the beginning of the experiment. It consists of 21 questions with a range of possible scores from 0-63 (See Appendix B). The questions cover a variety of symptoms including depressed mood, guilt, lack of sex drive, weight gain, and a lack of energy and are scored from 0-3 (0 signifying complete disagreement and 3 complete agreement). It has been shown to be able to discriminate between depressed and non-depressed subjects and have

an internal consistency of .9 as well as a test-retest reliability between .73 and .96 (Wang & Gorenstein, 2012). While the BDI-II is not technically a diagnostic tool, the strong relationship between its scores and depression diagnoses as well as its high internal consistency and test-retest reliability make it a useful tool for both assessing depression levels in subjects and providing a continuous variable that can be compared to accuracy on the emotion recognition task (Veerman, Dowrick, & Ayuso-Mateos, 2009; Campbell, Maynard, & Roberti, 2012). Despite being one of the most frequently used measures for assessing the severity of symptoms in depression, the BDI-II does not include emotion recognition impairments among its 21 items. The best way to show that emotion recognition impairments are an effective means of early detection is to compare it against the BDI-II. If such symptoms can be observed in a sample that has been cleared by such a highly regarded and commonly used tool for symptom assessment and depression severity, it suggests that non-self report measures like the DSM-V might have to be adapted to include the additional symptom.

Subjects will perform an emotion recognition task based on a study on the unconscious emotional processing of patients with major depression (Zhang, He, & Chen, 2016). The task uses subliminal images of happy and sad emotional faces followed by a “mask” created by a scrambled neutral face (See Appendix A for time display). The decision to borrow their use of subliminal images rather than a different procedure and image display time is based on the difference in results between their study and another study by Fieker, Moritz, & Kother (2016). While the study by Zhang et al. supported their hypothesis that there would be a difference in detection accuracy related to the depression severity of their subjects, the study by Fieker et al. was unable to support the same claim when allowing the faces to be displayed as long as the subjects required.

A possible objection to the use of the mask following the subliminal image is that it might distract the participant. Due to its scrambled nature, the mask doesn't provide any information about the face shown previously. What the mask does so, however, is correct for something called iconic memory. Iconic memory is an aspect of memory that allows people to reference the last image they've seen after the image has been shown. For example, imagine the afterimage that looking at the sun leaves on your retinas. Iconic memory is very similar. When shown the subliminal image (the emotional or neutral face), subjects automatically store an iconic memory of the image despite that it was only shown very briefly — for 7ms. While the brain doesn't consciously recognize what it's done, these iconic memories allow people to access information from the picture for a short period of time, unless attention is paid to specific parts of the image, which then allows it to be translated to working memory. Iconic memory only happens when very close attention is paid to an image. Much like staring at the sun, the focus has to be relatively intent for an afterimage to make a mark on your mind (Mack, Erol, & Clarke, 2015). For this study, subjects are expected and instructed to pay close attention to the screen. Due to both this and based off of the findings by Mack et al. (2015), subjects are expected to store iconic memories of the faces shown to them on screen.

This is where the scrambled mask of the neutral face becomes useful. By showing a scrambled mask after the subliminal image, the iconic memory of the original face from the subliminal image is “wiped away,” replaced by the iconic memory of the scrambled mask. This mask, however, possesses no useful information that the participant can access during the probe following the images and so does not affect results. Not only does the visual mask erase the iconic memory, but the quick presentation of the stimuli followed by the visual mask also means that subjects aren't able to use their higher-level conscious processing to think about the stimuli

because they can't refer back to either the iconic memory nor promote it to working memory due to the replacement of it by the mask.

The Fieker study (2016) found that accuracy on the emotion recognition task did not predict depression severity, but I believe this is because of the lack of challenge and realism in their methods. People's faces are constantly moving, and displaying an emotionally stagnant image of a model smiling or frowning for as long as the subject requires, or even for a few seconds, doesn't replicate quick body language and social cues the way that the Zhang et al. (2016) study's use of subliminal images does. Another study found similar results despite different methods (LeMoult, Joormann, Sherdell, Wright, Gotlib, 2009). Lemoult et al. found no differences between depressed subjects with recurrent depressive episodes and never depressed controls on emotion recognition accuracy. Subjects were shown faces that changed from neutral to the extreme of one of seven emotions over a period of time. While there were no significant differences in accuracy between the two groups, depressed subjects took significantly longer, and therefore required more intense facial expressions, than healthy controls. This supports the idea that some methods are too easy for depressed subjects and measure another variable instead, such as emotion intensity required, rather than emotion detection accuracy. By making the stimulus too easy to remember or decipher, the emotion recognition impairments biases aren't activated.

The emotion recognition task will be presented using E-Prime and will consist of 80 faces total (20 happy, 20 sad, 40 neutral), as well as 40 additional neutral faces to create scrambled masks. Unlike the original study by Zhang et al. (2016) that used a blocked design for the trials, this task will intermix trials randomly to prevent any possible artifacts from task mastery. There will be 160 total trials, with each of the unscrambled faces being displayed twice in total and

each scrambled face being shown four times in total. For each probe, there will be a 500ms fixation cross and a 500ms blank interval, followed by the emotional (sad or happy) face for 17ms, after which the scrambled mask will be shown for 150ms (See Appendix A; Figure 2 for an illustration of the time course). After being shown the mask, subjects will have 2000ms to answer if the face shown was emotional or neutral using either of two buttons on the keyboard, followed by a 500ms blank interval before the next trial. Seventeen milliseconds will be used for the length of the subliminal image display to mimic the Zhang et al. (2016) study's methods as closely as possible. They used this timing and were able to find a significant difference between depressed subjects and healthy controls on their ability to detect both happy and sad faces, unlike the Fieker (2016) study that did not have a limited display time. Additionally, the Dannlowski et al. (2006) study also found that subjects reported no knowledge of being shown images at this exact duration.

### **Procedure**

Subjects will begin participation in the study by signing an online consent form before taking the BDI-II and filling out an online self-report questionnaire about their psychiatric history to rule out anyone with a clinical diagnosis. This first step in the screening process will narrow down possible participants into a group comprised entirely of undiagnosed individuals with BDI-II scores in the minimal depression range of 0-13. The subjects that meet the requirements for participation will be invited to participate in the study and given a timesheet in which they can choose a time and date that they can meet the experimenters at for participation. In the follow-up meeting, subjects will be met by an experimenter and reminded of the purpose and content of this study.

Upon giving written informed consent the subjects will be led to the clinician, who will have them fill out the BDI-II and run each subject through a standard structured diagnostic interview using the categorical DSM-IV definition to ensure that none of the subjects meet the criteria for depression. Their scores will be labeled with a unique and confidential participant number following their participation to ensure privacy. Subjects' data will only be included if they are not diagnoseable according to the DSM-IV as well as if they score a 13 or below on the BDI-II, which is the "minimal depression" range of scores, on their second time filling out the BDI-II. This second testing it to make sure that subjects' symptoms have not increased in severity nor number since they passed the screening procedure. One concern brought up by the use of the BDI-II immediately before having the subjects take the emotion recognition task is that it might put the concept of depression in their minds, thereby altering their results on the task. While a valid concern, previous research suggests that it is much harder to affect results on emotion recognition tasks than through only having subjects take a brief questionnaire beforehand (Wolf, Pujara, & Baskaya, 2016). In their research, extensive training was required to alter participants' scores on an emotion recognition task. In comparison, having subjects fill out a brief questionnaire before the task will likely not have an effect on task accuracy.

After the interview, subjects will be led to quiet room with a computer for the emotion recognition task on E-Prime and made sure that they understand how to perform the task. The experimenter will then leave the room so that the subject can focus on the task. Upon completion, subjects will be debriefed (See Appendix C) and will be allowed to ask any questions they have about the research. Once all the subjects have been run there will be a drawing for four \$200 Amazon gift cards which can be mailed or e-mailed to the subjects if they choose to leave that information with the experimenters.

### **Analytical plans**

This study compares scores on two continuous variables (scores on the BDI-II from 0-13 and accuracy on the emotion recognition task). Because the type of relationship between scores on the BDI-II and accuracy on the emotion recognition task is unknown, a Spearman's correlation will be run to test for a monotonic relationship between the two. Spearman's correlation is ideal for this because while it tests for a monotonic relationship (as variable  $x$  increases, variable  $y$  increases OR decreases, but not both), the relationship tested does not need the data it is using to be normally distributed. Values from Spearman's correlation range from  $-1 < r_s < 1$ . A value of -1 would mean a perfectly negative monotonic relationship (as  $x$  increases,  $y$  always decreases) whereas a value of 1 would mean that there was a perfectly positive monotonic relationship (as  $x$  increases,  $y$  always increases). A value of 0 would mean that there is no monotonic relationship. It is important to note that a value of 0 does not mean that there is no relationship, only that there is no reliable monotonic relationship detected. In Pearson's correlation (a similar statistical procedure), the more that the data differ from a linear relationship the lower the value will be. However, in Spearman's correlation, even if the data have an exponential relationship (let's say that as BDI-II scores increase from 0-13, accuracy on the emotion recognition task exponentially decreases) it will still have a high value if the relationship is monotonic.

## **PREDICTED RESULTS**

### *Demographic Characteristics*

The predicted age of participants is 18-24, based on federal research that found that the majority of college students are in this age range (National Center for Educational Statistics, 2016). This

age was found in a population survey from 1970-2014 by the U.S. Department of Commerce. The predicted gender ratio of participants in this study is predicted to be 57% female. This information was retrieved from federal data that shows that 57% of the enrolled college population has been female for the past decade (National Center for Educational Statistics, 2014). The predicted racial distribution is 71.4% Caucasian, 9.5% Asian, 4.8% African American and 14.3% other; and 4.8% identifying as Hispanic or Latino (National Center for Educational Statistics, 2016).

Age, gender, and racial background are not predicted to have any significant predictive value on task accuracy when entered into a multiple regression as predictors after controlling for BDI-II scores. These four predictors are expected to have some predictive value for BDI-II scores themselves, as participant gender, sex, college year, and race have been found to affect depression scores (Harris, 2004). While these variables have been found to affect rates of depression with women and minorities displaying significantly higher rates of depression than males and Caucasians, they are not predicted to increase the effect of the emotion recognition impairments being studied independent of depression scores.

*Power analysis.* 400 participants yields 99% power assuming  $\alpha = .05$ , and  $r = -.3$ . (Punkanen, Eerola, & Erkkila, 2010).

*Data preparation.* The relationship between scores on the emotion recognition task and scores on the BDI-II will be analyzed using a Spearman's Correlation. The typical Pearson's Correlation requires a normal distribution of scores, an assumption that the population's BDI-II scores don't fit. Most of the population scores at the bottom of the range of BDI-II scores, displaying a large

floor effect. Using a Spearman's Correlation will allow for any existing relationship to be found between the two variables despite the lack of normativity in BDI-II scores.

### ***Predictions***

Subjects are predicted to display emotion recognition impairments despite not meeting the BDI-II's requirements for depression. These impairments will be measured using the 160-trial facial emotion recognition task. Participant accuracy on the emotion recognition task will be measured using  $d$  prime.  $D$  prime (or  $d'$ ) is an aspect of signal detection theory, which is the way in which one discerns between significant patterns and random noise (significant results and results at a chance level, respectively). Signal detection theories can include the use of hits, misses, false alarms, and correct rejections. In regards to the emotion recognition task, hits would be when the subject correctly identify that the face shown was emotional. Misses would be when subjects answer that the face shown was not emotional when it actually was. False alarms would be when subjects falsely respond that a neutral face is emotional (which is predicted to occur significantly more frequently in the sad emotion trials for people who display these impairments). Correct rejections occur when the subject correctly identifies a neutral face as neutral instead of emotional.

Because both the hyposensitivity to happiness and hypersensitivity to sadness have been displayed by people with depression (LeMoult, Joormann, Sherdell, Wright, & Gotlib, 2009; Douglas & Porter, 2010; Bistricky, Ingram, & Atchley, 2011; Zhang, He, Chen, Wei, 2016),  $d$  prime will be calculated using the false alarms and hits from both the happy and sad emotion recognition trials. In this task, a hit would occur if the subject correctly answered that the face shown to them was an emotional one. A false alarm would occur if the subject identified an

emotion but the face shown to them was actually neutral. Higher values show that the subject was more accurate while very low values would show that the subject barely scored above chance level.

As the subjects' BDI-II scores increase, so too are their  $d$  prime values expected to decrease. There is expected to be a weak but significant relationship between the two variables. This is based on both the findings of Angst & Merikangas (1997) who found that a significant portion of people in treatment (with symptoms to be treated) don't meet the diagnostic requirements for depression and the important role of these impairments in the worsening of symptom severity. Because of this, there will be enough people in the "fringe" group of depression out of the 400 subjects to have a significant relationship despite the majority of subjects who are expected to display very few symptoms. Using Spearman's correlation, a significant negative monotonic relationship is predicted ( $p = <.001$ ,  $r_s = -.3$ ; see Appendix A; Figure 3).

## DISCUSSION

In this the predicted results of this proposal study, subjects' accuracy (scored using  $d$  prime) on an emotion recognition task had a significant, negative monotonic relationship with their scores on the BDI-II. This pattern of results is consistent with existing literature; depression has long been associated with impairments in facial emotion recognition. Specifically, subjects in this study are predicted to display the same type of emotion recognition impairments that have been found in depressed and formerly-depressed subjects in previous studies (Zhang, He, & Chen, 2016; Punkanen, Eerola & Erkkila, 2010; Loi, Vaidya & Paradiso, 2013; Biyik, Keskin, & Oguz, 2015; Berg, Ballard, & Luckenbaugh, 2016; ) This unique

symptom of depression that this study proposes to measure causes a hypersensitivity to negative emotions and a hyposensitivity to positive ones.

This study differs from existing literature on these facial emotion recognition impairments in regards to the population being studied. Previous works have compared depressed subjects to control groups, remitted-depressed subjects to control groups, depressed subjects to remitted-depressed subjects, and have experimented with gender differences by studying only women or only men in some cases. This current proposed study would be unique in that it would be the first to measure the existence of these symptoms in never-diagnosed subjects that don't meet the requirements of the DSM-V for diagnosis. This is a distinction compared to the previous literature on these impairments that have examined depressed patients in remission. While both groups of subjects are subclinical according to the DSM-V, the current study's subjects would have never been diagnosable, as opposed to previous work that has studied residual symptoms in previously diagnosed patients.

This difference is important when considering the role of these facial emotion recognition impairments in relapses of depression. Previous studies have examined remitted-depressed patients for residual symptoms partially because of how residual symptoms often lead to a relapse in depression. One of the symptoms that has been found in remitted-depressed patients is the emotion recognition impairments this current study is designed to measure. These emotion recognition impairments play a large role in the development of both other and more severe symptoms, and their presence in a patient with remitted-depression makes a full recovery more difficult. Their existence in a never-diagnosed and currently undiagnosable (according to the DSM-V) group of subjects would mean that if these symptoms were present, even people

without a history of mental illness can be at a serious risk of developing more severe depression even after an original lack of other, more commonly diagnosable symptoms.

Facial emotion recognition impairments are a debilitating set of symptoms that make it more difficult for people to accurately recognize the emotions displayed on others' faces. Because of this, people with these impairments frequently experience downwards spirals in their social lives as their inappropriate reactions to the perceived negative reactions and emotions of others prevent them from interacting properly on a personal, educational, and professional level. Additionally, facial emotion recognition impairments frequently lead to the development of other, more severe symptoms. When combined with the downwards spiral in depressed individuals, the long term effects that these impairments can have on patients are crippling. The proposed results would suggest that many currently undiagnosable people may not only display a symptom that is thought to play a large part in depression's debilitating social effects (Bistricky, Ingram, & Atchley, 2011) but also that by not diagnosing them, clinicians are increasing their likelihood of developing more severe symptoms and possibly making full treatment more difficult due to a late diagnosis.

This problem is due in part to the focus of the DSM-V and other symptom trackers such as the BDI-II on first the development of symptoms followed by the categorization of the symptoms found in depression that are experienced by the patient. By waiting until symptom develop, the clinician is increasing the likelihood of a late diagnosis and the development of severe enough symptoms to make a full recovery more difficult. This focus on waiting until their symptoms have developed is also harmful because of the high number of symptoms required to be diagnosed. Without five of the nine depressive symptoms listed by the DSM-V, the patient's other existing "subclinical" symptoms can develop in severity without acquiring the

necessary fifth symptom for diagnosis. Without considering the severity of symptoms as well as the number of them, clinicians risk waiting too long to diagnose a patient. This only increases the likelihood that the patient's symptoms will develop to a permanently harmful level of severity.

The high number of depression subtypes that currently exist in the DSM-V is another aspect of diagnosis that shows the hyper attention given to symptoms. There are a full twelve subtypes of depression (See Appendix A; Figure 1). These cover the surface of a volatile disorder, one in which people's symptoms often change and one that they struggle with for years, often even after receiving treatment. Patients with long term depression frequently develop new symptoms and lose previous ones throughout the course of their illness; often moving between these ten depression subtypes often. The subtypes themselves don't even account for the full heterogeneous nature of symptoms. The individual experience of depression on a patient-by-patient basis is hugely varied, and there are far more than 10 different ways of experiencing depression. Current diagnostic procedures don't account for the frequent development of new or more severe symptoms, nor have other purely symptomatic treatments proved especially effective.

### **Limitations**

One of the problems with using these facial emotion recognition impairments in diagnosing depression is that these impairments are also found in both patients with schizophrenia and patients with anxiety. If these impairments are present in anxiety, depression, *and* schizophrenia, their predictive value for depression is reduced because they can signify the onset or continued development of two other, very different mental illnesses. Additionally, all three of these mental

illnesses are often comorbid with each other. Anxiety and depression have long been known to be frequently tied together both symptomatically and diagnostically, and depression has been shown to be extremely prevalent in patients with schizophrenia as well (Herniman, Allot, & Killackey, 2017). However, rather than act as evidence against the depression-specific nature of these impairments, the presence of these impairments in each of these three disorders can be used to support the role of depression's responsibility for these similar symptoms in anxiety and schizophrenia.

In the case of comorbid anxiety and depression, facial emotion recognition impairments are present in both. How can researchers be sure that what they see in depressive patients isn't an artifact of comorbid anxiety? Data from a study measuring both anxiety and depression and their effects on emotion recognition impairments suggests that the opposite relationship is true (Berg, Ballard, & Luckenbaugh, 2016). Rather than the case being that comorbid anxiety provides the emotion recognition impairments present in depression, their results suggest that it is depression that provides the impairments in facial emotion recognition. The 2016 Berg et al. study attempted to examine the effects of both in a comorbid population. Subjects with anxious-depression (AD) (n=14) and non-anxious depression (NAD) (n=14) completed the Emotional Expression Multimorph Task. In this task, emotional faces were slowly morphed along an intensity spectrum ranging from unemotional to very emotional. The NAD group was significantly more sensitive to sad expressions when compared to the AD group and required lower levels of intensity, which supports the hypersensitivity to negative emotions pattern found in the emotion recognition impairments. Anxiety was negatively correlated with sensitivity, such that higher anxiety scores required greater intensity of negative emotion to be detectable. Interestingly, hyper vigilance to angry and fearful faces was not displayed in the NAD group,

despite frequently being associated with anxiety. Contrary to the original idea that the emotion recognition impairments present in depression are the same found in anxiety, the patients with both anxiety and depression displayed different patterns of recognition than the patients with only depression.

Patients with comorbid anxiety and depression have different patterns of emotion recognition impairments than patients with only depression (Berg et al., 2016). While this suggests that the impairments found in the two disorders are at least somewhat different in nature, it also allows the impairments found in depression to still be used in assisting with diagnosing depression. With anxiety leading to measurably different emotion recognition impairments, the risk of misdiagnosis informed by the depression-specific impairments is bypassed. If the emotion recognition impairments display a pattern that better matches those found in anxiety, then the impairments shouldn't be used to inform a depression diagnosis.

A possible problem with this is the extraordinarily high rate of comorbidity between anxiety and depression. The rate of comorbidity has been found to be as high as 60% between the two disorders, as opposed to the chance of comorbidity of 2% (Cameron, 2007) based on the possible number of diagnoses. With this large amount of crossover, might the anxiety affect the results of the impairments? For the very nature of the difference in impairments between the two disorders, I would argue that this direction of causality seems unlikely. If the impairments found following the actual running of this experiment don't replicate those found in depression, it could indicate an instance of comorbidity with another disorder that is changing the direction of the facial emotion recognition impairments. This non-depression specific pattern of impairments could lead the clinician to consider testing the patient for additional diagnoses as well. By not

testing for these impairments, clinicians are missing a very useful opportunity to determine if the patient displays symptoms that better represent one disorder or the other.

Since anxiety displays different patterns of emotion recognition impairments than depression, these impairments can be used to possibly also distinguish between the two disorders as well as identify them. Emotion recognition impairments have also been found in schizophrenia, a mental illness already known for its incredibly debilitating social effects. Considering how crippling its negative symptoms are already (social withdrawal, apathy, reductions in speech, and impaired attention), schizophrenia seems like it might possess its own impairments in recognition independent of comorbid depression. Does schizophrenia also display a unique pattern of impairments in emotion recognition similar to how anxiety displays its own unique pattern?

Despite its debilitating effects, research suggests that schizophrenia's role in emotion recognition impairments is minimal. One 2006 study measured patients with schizophrenia compared to healthy controls on their ability to detect differences between emotions (Kee, Horan, Kynn, Mintz, Green, 2006). In this experiment, 47 schizophrenic patients and 31 healthy controls were shown 88 images of one of two models that showed four pairings of emotions (happy-sad, fearful-happy, angry-fearful, angry-sad) comprised of 11 possible ratios: 100% emotion A - 100% emotion B going by 10% increments. Contrary to expectations, the schizophrenic group did not display a negativity bias but did have shallower, less strict divisions between emotions on the displayed images compared to the control group. While less consistent in their ability to determine which emotions are which, the schizophrenic group still did not display the same pattern of results caused by these impairments.

Conversely, depression can be seen to increase the sensitivity to negative emotions in schizophrenic patients. In addition to facial emotion recognition impairments, both disorders are also known for impairments in prosody (vocal/tonal) emotion recognition. In a study by Herniman & Allot (Herniman, Allot, & Killackey, 2016), first-episode schizophrenic patients (FES) — patients experiencing their first schizophrenic episode — were compared to FES schizophrenic patients with comorbid major depression (MDD). This study aimed to determine whether subjects with FES and comorbid major depression have overall less accurate emotion recognition than those without MDD, or even a different pattern of emotion recognition impairments. Using 82 young adults (comorbid MDD:  $n=24$ ), it was found that those with comorbid MDD had more accurate recognition of sadness than those with only FES, following the pattern of hypersensitivity to negative emotions found in these emotion recognition impairments. The severity of the comorbid MDD was also significantly related to more accurate sadness recognition. These results only applied to facial emotion recognition, not prosody emotion recognition, but since prosody emotion recognition can be so varied (Punkanen, Eerola & Erkkila, 2010), the absence of a relationship may be due to a difference in methodology.

Could the emotion recognition impairments that are part of depression be the same emotion recognition impairments that have been observed in schizophrenia? The results from the study depressive schizophrenia support this idea. Schizophrenia has been shown to display the same emotion recognition impairments present in MDD while not showing the same pattern in patients with exclusively schizophrenia. The depression levels present in schizophrenic patients in the Herniman et al. study predicted their facial emotion recognition accuracy rather than schizophrenia alone.

Similar to anxiety, schizophrenia also has an unusually high rate of comorbidity with depression. According to current estimates, these rates of comorbidity have been found to be as high as 50%, with 50% of schizophrenia patients having a comorbid diagnosis of depression. To be clear, the reverse is *not* true. Only 1.2% of the population in the United States is diagnosed with schizophrenia (Nemade & Dombeck, 2009). With the current rate of depression in the United States being 6.7% and the rate of depression comorbidity with schizophrenia being 50%, only 10% of the depressed population have comorbid schizophrenia, compared to the half of schizophrenic patients that also have depression.

This large difference in the rates of comorbidity (50% of schizophrenic patients have depression while only 10% of depressed patients have schizophrenia) shows that the impairments in emotion recognition found in schizophrenia are possibly caused by depression rather than being caused by the schizophrenia. Were the latter to be the case, then the depression-only patients would likely not display the same patterns of impairments that schizophrenia patients do. However, as they do display the same patterns of impairments, the cause of the facial emotion recognition impairments can be guessed at due to the high number of depression cases in schizophrenia and the low number of schizophrenia cases in depression. To be clear, this is only in reference to the use of these facial emotion recognition impairments as a diagnostic tool for depression and schizophrenia. Both illnesses have many other symptoms that the other one does not. The emphasis being placed on these emotion recognition impairments is for when a patient displays the impairments but not enough other symptoms to be diagnosed with either depression or schizophrenia. A distinction in the pattern of emotion recognition impairments of the two disorders needs to be found in order for the two to be discriminable based on the impairments alone for subclinical “fringe” cases.

An interesting connection between schizophrenia and depression is the involvement of the basal ganglia in their symptomatology. In both mental illness, the basal ganglia has been found to have some relation to their respective symptoms (Douglas & Porter, 2010; Bernard, Russell & Newberry, 2017). In the study by Douglas and Porter (2010), 68 patients with severe depression and 50 healthy controls were compared on their abilities to correctly recognize emotions using a Facial Expression Recognition Task. The depression group displayed the usual hypersensitivity to negative emotions compared to healthy controls that is normally found in depressed patients. The depression group was significantly more likely to interpret neutral faces as sad and less likely to identify them as happy than the control group. In addition to the usual findings, the depression group also displayed a deficit in recognizing disgust compared to the control group. Douglas and Porter theorized that this might suggest that the basal ganglia is involved in these impairments due to its role in processing disgust.

The implication of the basal ganglia can be seen in schizophrenia's symptoms as well. In a meta analysis by Bernard, Russell, and Newberry (2017), 42 neuroimaging studies were looked at that measured basal ganglia activation in schizophrenia. Their findings showed that patients with schizophrenia frequently display a significant decrease in basal ganglia activity compared to healthy controls. The possible involvement of the basal ganglia in both patients with schizophrenia and patients with depression makes the distinction between the emotion recognition impairments found in the two disorders difficult. This does not necessarily mean that the basal ganglia leads to schizophrenia's other symptoms, however. Rather, the finding that the basal ganglia is often hypoactive in schizophrenia can possibly be explained by the frequent comorbidity of depression in patients with schizophrenia. With 50% of schizophrenic patients also possessing a diagnosis for depression, the basal ganglia involvement found by Bernard et al.

(2017) could be an artifact from the comorbid depression. Future studies could examine the activation of the basal ganglia in patients with schizophrenia after controlling for comorbid depression in order to determine whether or not the basal ganglia's hypoactivation is unique to depression or shared by both illnesses.

Another limitation of this study was deliberately chosen through necessity. Only college students will be used, which reduces its applicability to the real world. This study depends on its subjects being subclinical. However, it also depended on them displaying the emotion recognition impairments that have been identified in depression. While this is predicted to be the case for some of the subjects, many of the 400 subjects in this study will likely not display these impairments. To ensure that enough subjects do display the impairments to be noticeable, the sample will have to be taken from a population that has high rates of depression. This will increase how many people in the sample will be in the "fringe" group of depression (subjects that possess subclinical symptoms yet still are still significantly more impaired than the general undiagnosed public).

To accomplish this, this study uses college students as its subjects. College students display the highest rates of depression in the country, with 27% of college students reporting a primary diagnosis of depression according to a 2012 study by the National Alliance on Mental Illness (Grudattaro & Crudo, 2012). While this increases how many subjects are in the region of interest (subjects in the "fringe" group of depression), it also decreases external validity and applicability to the rest of the population. As this study would be the first to look for these unique facial emotion recognition impairments in a never-diagnosed group, finding the impairments in the fringe group is more important than making the study perfectly representational of the public. Should this study's proposed results be replicated, future research

could explore the presence of these impairments in other populations, examining the effects of age and background on the impairments as well as depression levels.

The purpose of this study was to establish to presence of these facial emotion recognition impairments in a subclinically depressed population. This does not address causation, only correlation, and does not prove that these subclinical individuals will develop depression, only that they display the impairments frequently associated with symptom development. Future studies informed by this one could address this possible causal relationship. Subjects could be identified with subclinical depression who possess these emotion recognition impairments and compared to a control group with the same level of subclinical depression but without the emotion recognition impairments. Their depressive symptoms could be monitored over time in a longitudinal study. If the impairment group develop depression while the control group don't, the role of the impairments in symptom development would be supported.

### **Practical Implications**

Were the predicted results of this study to be found following the actual running of this experiment, then both our understanding of how we diagnose patients and actual diagnostic practices could be improved. The emotion recognition impairments focused on in this study are frequently responsible for the development of both other symptoms as well as the severity of the symptoms (Bistricky, Ingram, & Atchley, 2011). They negatively affect people's perceptions of others emotions, leading them to find negatively emotional faces more negative than they really are, misinterpret neutral faces for negative ones, and find positively emotional faces less positive than they actually are. They are socially debilitating as well as risk making the depression worse the longer the illness goes untreated. If people don't display other symptoms but do display

these emotion recognition impairments, they still might be at risk for developing depression and other symptoms. That makes these symptoms useful for both early warnings of possible depression and symptom management.

These emotion recognition impairments are extremely common in depression. Despite their prevalence and frequent responsibility for symptom development, they aren't taken into consideration during diagnosis. Using their prevalence and unique pattern of bias in emotion recognition, these impairments could be used to identify people at risk of developing depression — similar to how high blood pressure is often recognized by doctors as a warning sign for a possible heart condition.

Further research could study the connection between alexithymia and the emotion recognition impairments found in depression. As they both affect the visual processing of others' emotions, the two symptoms could be connected. If a strong connection between the two is found, then questionnaires measuring alexithymia could be given during diagnostic interviews as a simpler method of testing for these impairments. This would make the inclusion of the emotion recognition impairments into diagnostic practices far simpler than having patients take an emotion recognition task. While this correlation between the two symptoms would be useful, it seems unlikely to be the case. This has to do with the nature of the two symptoms. In depression, the facial emotion recognition impairments are negatively biased. People with this symptom are hypersensitive to the negative emotions on other people's' faces and hyposensitive to the positive ones. In a very real way, they are extra sensitive to at least some of the emotions of others. This is a sharp contrast to the effects of alexithymia, which is characterized by an inability to recognize or describe the emotions of oneself or others, i.e., a lack of sensitivity to emotion.

## **Conclusion**

In summary, these predicted results suggest that facial emotion recognition impairments, an extremely common symptom in depression responsible for the continued development of other symptoms, would exist in college students with subclinical symptoms (according to the requirements of both the DSM-V and the BDI-II). While these symptoms alone aren't cause for concern, the predicted results display a pattern of impairments that closely mimics that of fully diagnosed depression. As the subjects' BDI-II scores rise (and presumably the severity of their depressive symptoms), so too are their scores on the emotion recognition task predicted to decrease. The relationship between these two factors is not causal, but it does show that there is value in these impairments for predicting depression. When considered alongside the way these impairments have been shown to influence and lead to the development of other symptoms, it is not a stretch to think that they might play a larger role in depression than they have currently been given credit for.

In a sample of 400 college students with subclinical depressive scores according to the DSM-V and BDI-II, their scores on the BDI-II are predicted to have a significant and negative relationship with their accuracy on an emotion recognition task. Their ability to recognize the emotions of others are predicted to decrease as their depressive symptoms increased — even at what is now considered a subclinical level by both the DSM-V and the BDI-II.

These findings would be exceptionally important for diagnostic practices. By displaying a pattern of impairments found in already-depressed patients where the impairments increase as does the severity of depression despite not meeting diagnostic criteria for depression, the subjects of this study show that there might be individual differences in the form of these emotion

recognition impairments undiagnosed patients that make them susceptible to developing more severe symptoms and eventually depression. However, by not meeting the requirements for a depression diagnosis according to the DSM-V, they would currently be unable to receive treatment that requires a diagnosis before anything can be started. This would not be a problem were it not for the role that these facial emotion recognition impairments play in the development of other symptoms and symptom severity. Can clinicians afford to strictly follow diagnostic criteria if it risks that the patient might develop worse symptoms, the existence of which will make successful treatment more difficult as their time spent untreated increases? I would argue that the answer should clearly be “no.” If following the DSM-V’s criteria for diagnosis to the letter leads to a large part of the population is not receiving treatment that could benefit them, it seems like the criteria should be changed to better help those in need. Put differently, if clinicians actually need to break the rules to help some of their patients because the rules would otherwise exclude them from receiving a diagnosis, the rules should be changed to include the patients are unable to receive the needed treatment.

Instead of forcing clinicians to have to bend the rules of the DSM-V to treat some of their patients that otherwise would have been excluded from a diagnosis, current diagnostic practices should be changed to use the impairments in facial emotion recognition as a means of early detection or an additional, measurable symptom to be included in the symptom list of the DSM-V. The importance of this change can be seen when considering two statistics about depression diagnosis. First, many people who don’t meet the full diagnostic criteria for depression still display some depressive symptoms. For instance, they could only display four of the nine depression symptoms listed by the DSM-V rather than the required five. Second, there is a high rate of relapse and chronic illness for depression patients that is likely caused in part by a late

diagnosis. By diagnosing after the onset of symptoms, the patient's recovery is made much harder for having to lose bad habits and thinking patterns. Considering the frequent role of these impairments in symptom development and the common problem of late diagnoses, these results should inspire a change in the requirements and techniques used to diagnose depression; either through encouraging clinicians to make diagnosis requirements more inclusive or by persuading them to measure the emotion recognition impairments themselves as an additional symptom of depression.

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## Appendix A

Figure 1: DSM-V Depression Subtypes

- 1: Depressive Episode
- 2: Recurrent Depressive Episode
- 3: Dysthymia
- 4: Melancholic depression
- 5: Vascular depression
- 6: Bipolar 1 Depression
- 7: Bipolar 2 Depression
- 8: Mixed Depression and Anxiety
- 9: Depressive Psychotic Episode
- 10: Atypical Depression
- 11: Seasonal Depressive Episode
- 12: Brief Recurrent Depressive Episode

Figure 2: Emotion Recognition Task Time Course

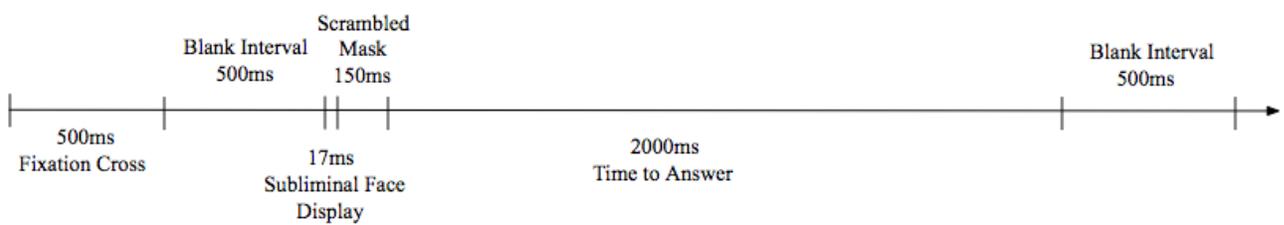
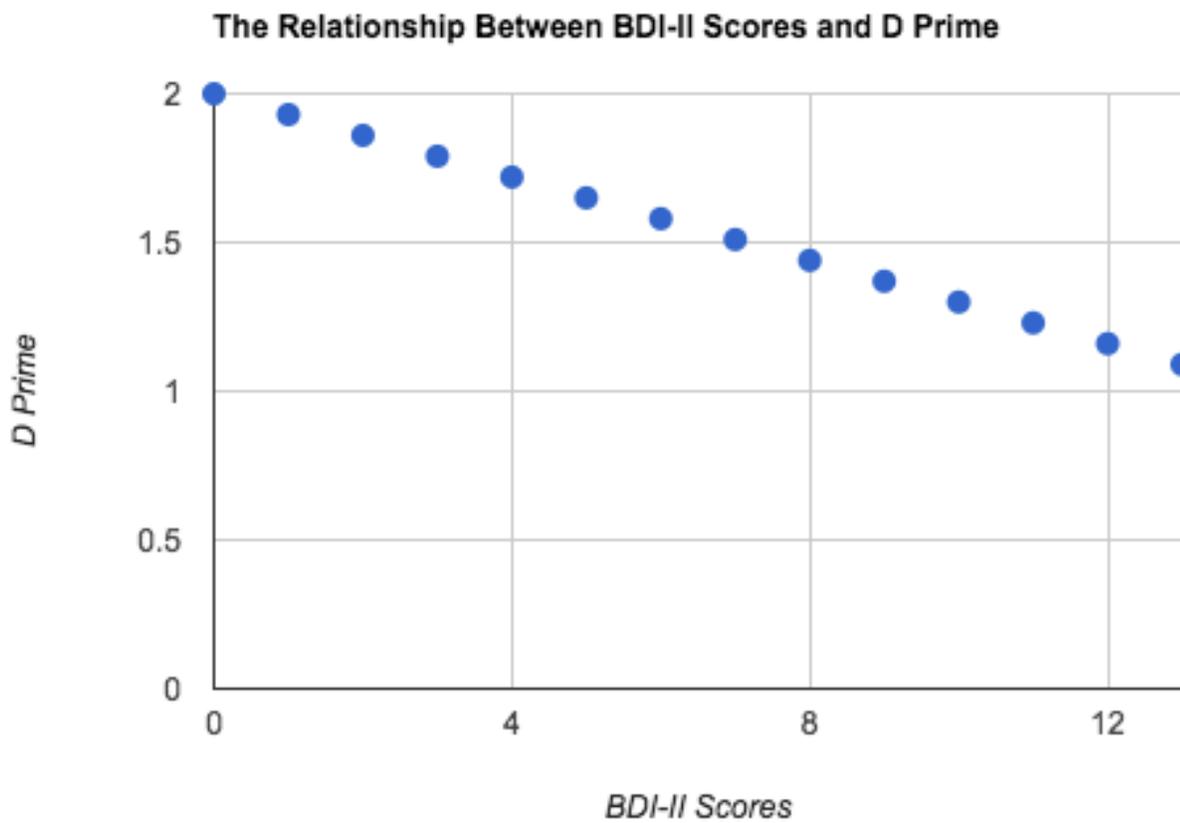


Figure 3: The predicted negative monotonic relationship of BDI-II scores and task accuracy ( $r_s = -.3$ ).



## Appendix B

BDI-II Items:

### 1. Sadness

- 0 I do not feel sad.
- 1 I feel sad much of the time.
- 2 I am sad all the time.
- 3 I am so sad or unhappy that I can't stand it.

### 2. Pessimism

- 0 I am not discouraged about my future.
- 1 I feel more discouraged about my future than I used to be.
- 2 I do not expect things to work out for me.
- 3 I feel my future is hopeless and will only get worse.

### 3. Past Failure

- 0 I do not feel like a failure.
- 1 I have failed more than I should have.
- 2 As I look back, I see a lot of failures.
- 3 I feel I am a total failure as a person.

### 4. Loss of Pleasure

- 0 I get as much pleasure as I ever did from the things I enjoy.
- 1 I don't enjoy things as much as I used to.
- 2 I get very little pleasure from the things I used to enjoy.
- 3 I can't get any pleasure from the things I used to enjoy.

### 5. Guilty Feelings

- 0 I don't feel particularly guilty.
- 1 I feel guilty over many things I have done or should have done.
- 2 I feel quite guilty most of the time.
- 3 I feel guilty all of the time.

### 6. Punishment Feelings

- 0 I don't feel I am being punished.
- 1 I feel I may be punished.
- 2 I expect to be punished.
- 3 I feel I am being punished.

### 7. Self-Dislike

- 0 I feel the same about myself as ever.
- 1 I have lost confidence in myself.
- 2 I am disappointed in myself.
- 3 I dislike myself.

### 8. Self-Criticalness

- 0 I don't criticize or blame myself more than usual.
- 1 I am more critical of myself than I used to be.
- 2 I criticize myself for all of my faults.
- 3 I blame myself for everything bad that happens.

### 9. Suicidal Thoughts or Wishes

- 0 I don't have any thoughts of killing myself.
- 1 I have thoughts of killing myself, but I would not carry them out.
- 2 I would like to kill myself.
- 3 I would kill myself if I had the chance.

### 10. Crying

- 0 I don't cry anymore than I used to.
- 1 I cry more than I used to.
- 2 I cry over every little thing.
- 3 I feel like crying, but I can't.

**11. Agitation**

- 0 I am no more restless or wound up than usual.
- 1 I feel more restless or wound up than usual.
- 2 I am so restless or agitated that it's hard to stay still.
- 3 I am so restless or agitated that I have to keep moving or doing something.

**12. Loss of Interest**

- 0 I have not lost interest in other people or activities.
- 1 I am less interested in other people or things than before.
- 2 I have lost most of my interest in other people or things.
- 3 It's hard to get interested in anything.

**13. Indecisiveness**

- 0 I make decisions about as well as ever.
- 1 I find it more difficult to make decisions than usual.
- 2 I have much greater difficulty in making decisions than I used to.

**14. Worthlessness**

- 0 I do not feel I am worthless.
- 1 I don't consider myself as worthwhile and useful as I used to.
- 2 I feel more worthless as compared to other people.
- 3 I feel utterly worthless.

**15. Loss of Energy**

- 0 I have as much energy as ever.
- 1 I have less energy than I used to have.
- 2 I don't have enough energy to do very much.
- 3 I don't have enough energy to do anything.

**16. Changes in Sleeping Pattern**

- 0 I have not experienced any change in my sleeping pattern.
- 1a I sleep somewhat more than usual.
- 1b I sleep somewhat less than usual.
- 2a I sleep a lot more than usual.
- 2b I sleep a lot less than usual.
- 3a I sleep most of the day.
- 3b I wake up 1-2 hours early and can't get back to sleep.

**17. Irritability**

- 0 I am no more irritable than usual.
- 1 I am more irritable than usual.
- 2 I am much more irritable than usual.
- 3 I am irritable all the time.

**18. Changes in Appetite**

- 0 I have not experienced any change in my appetite.
- 1a My appetite is somewhat less than usual.
- 1b My appetite is somewhat greater than usual.
- 2a My appetite is much less than before.
- 2b My appetite is much greater than usual.
- 3a I have no appetite at all.
- 3b I crave food all the time.

**19. Concentration Difficulty**

- 0 I can concentrate as well as ever.
- 1 I can't concentrate as well as usual.
- 2 It's hard to keep my mind on anything for very long.

**20. Tiredness or Fatigue**

- 0 I am no more tired or fatigued than usual.
- 1 I get more tired or fatigued more easily than usual.
- 2 I am too tired or fatigued to do a lot of the things I used to do.
- 3 I am too tired or fatigued to do most of the things I used to do.

**21. Loss of Interest in Sex**

- 0 I have not noticed any recent change in my interest in sex.
- 1 I am less interested in sex than I used to be.
- 2 I am much less interested in sex now.
- 3 I have lost interest in sex completely.

## Appendix C

### Consent Form

#### Emotion Recognition Accuracy in Subclinical Depression

In this study, you are being asked to participate in a study investigating the relationship between accuracy at emotion recognition and subclinical scores on the Beck Depression Inventory II scale (BDI-II). You are being asked to take part because you expressed interest and passed our online screening form. Please read the rest of this form carefully and feel free to ask any questions you have about the study before you agree to participate.

This study is about the relationship between emotion recognition accuracy and scores on the BDI-II to determine if current diagnostic practices can be improved by including the emotion recognition impairments in possible symptoms for depression. To participate in this study you must have no prior diagnosis of mental illness nor any treatment for mental illness.

In this study, you will be interviewed by a trained clinician to ensure that you do not meet the requirements for diagnosis. Following this, you will be asked to fill out the BDI-II. After filling out the BDI-II, you will be asked to take the emotion recognition task. These should take just over an hour to complete.

#### Risks and Benefits:

The material involved in this study is of a sensitive nature. By agreeing to participate, you may receive a diagnosis for major depressive disorder, which would exclude you from participation. Additionally, the questions on the BDI-II are somewhat sensitive and pertain to your mood and daily habits as well as some changes in sleeping and diet.

By participating in this study, you may benefit by receiving the very diagnosis that excludes you from participation. Depression can be extremely serious, and early detection is one of the best methods of treatment. Additionally, if you are eligible to continue in the study, you may benefit from the knowledge that you are helping possibly millions of depressed individuals receive the diagnosis and treatment they need.

Your responses to the BDI, the results of your interview with the clinician, and the your score on the emotion recognition task will be kept completely confidential and identified only with an anonymous participant number. These responses will all be kept private and secure; only the experimenters will have access to them, and they will only be labeled with your participant number. In the case of publication or any sort of public event, you are guaranteed anonymity and that none of your answers will be able to be identified as yours.

Participation in this study is completely voluntary. You may choose to leave the study at any time and you will still be entered in the raffle for the \$50 Amazon Gift Card. If you do choose to withdraw from the study, it will not affect the anonymity and privacy of your responses.

Compensation:

By participating in this study, you may enter into a raffle to win one of four \$50 Amazon Gift Cards. If you win, you will be notified by either e-mail or mail if you choose to leave that information with us.

If you have any questions about the study, feel free to ask the experimenter anything you'd like about the study, current research, or possible directions of research after this study. If you have questions after the study, you can reach the experimenters at [sproj17@bard.edu](mailto:sproj17@bard.edu). If you have any

questions or concerns about your rights as a participant you can learn more by visiting the International Review Board's website at <http://www.bard.edu/irb/> or contact its members at [rb@bard.edu](mailto:rb@bard.edu).

You will be given a copy of this form to keep for your records.

Statement of Consent: I have read the above information, and have received answers to any questions I asked. I consent to take part in the study.

Your Signature \_\_\_\_\_ Date \_\_\_\_\_

Your Name (printed) \_\_\_\_\_

Signature of person obtaining consent \_\_\_\_\_

Date \_\_\_\_\_

Printed name of person obtaining consent \_\_\_\_\_

Date \_\_\_\_\_

### **Debriefing Statement**

The study you just participated in examined the relationship between subclinical scores on the Beck Depression Inventory II and accuracy on an emotion recognition task. Currently, there is a debate over whether or not the diagnostic practices for depression are too categorical

and exclusive. Impairment at emotion recognition is a common symptom of depression, but is infrequently measured by diagnostic tools. This study was an attempt to determine if these symptoms are present in what would currently be called a subclinical population. If they are, both the psychological and medical fields may have to reconsider how they diagnose depression. Thank you for your participation in this study. If you have any questions about the study or future research or participation, contact the experimenters of this study at sproj17@bard.edu.

### **Costs**

The cost of the study will be a roughly \$14,223. The four \$200 Amazon Gift Cards will add up to \$800, and a 2016 study hired trained clinicians for \$30 an hour. Each clinical interview should take about an hour (but this may vary from participant to participant), which comes to about \$1200 for clinician fees. The print manual of the BDI-II costs \$83 without shipping. The emotion recognition task can be created on E-Prime, the license of which costs \$995 for a single user license. E-Prime can be run on any computer that has a Pentium i3 Processor with 2GHz or higher, 2GB of RAM, a video card, and a USB port. The Acer Aspire E15 E5-575-33BM 15.6-Inch laptop fulfills all of these requirements and can be bought for \$345 on Amazon. Lastly, the photographs of faces can be accessed for free from the Karolinska Directed Emotional Faces (KDEF) database as long as this study isn't published.