“Every year at least 30 million people of all ages and genders suffer from an eating disorder in the U.S. and every 62 minutes at least one person dies as a result from an eating disorder” (“Eating Disorder Statistics”). As eating disorders have the highest mortality rate of any mental illness and the number of individuals impacted each year continues to increase changes need to be made for the lives and survival of these individuals. There are many misconceptions around eating disorders such as “they are a choice” and that people will “be fine if they just eat”. These misconceptions suggest that many lack knowledge and full understanding on this issue. As these statements are not true research has been conducted to investigate the causes, symptomology, and recovery tactics of eating disorders to increase understanding of the disorder such as my research aims to do. Some of the most prevalent research on eating disorders currently is on the strong relationship between eating disorder and anxiety disorders. These studies have found rates of anxiety disorder 2-3 times greater in with individuals with eating disorders than in the general community (Godart, Flament, Lecrubier, & Jeammet, 2000). These findings have led researchers to question when and how anxiety disorders accompany these eating disorders. Current research regarding the temporal relationship of an anxiety disorder to an eating disorder has found that an anxiety disorder commonly precedes an eating disorder. Childhood and lifetime diagnosis of at least one anxiety disorder has been found in 13-75% of women with bulimia nervosa and 20-55% with anorexia nervosa (Godart et. al, 2000). These anxiety disorders include most commonly generalized anxiety disorder, obsessive compulsive disorder, and social phobia (Raney, 2008). These statistics demonstrate the strength of the
relationship of the two disorders and the prevalence of anxiety disorders in the eating disorder population. The implications of these findings are that methods of early detection need to be completed with individuals with anxiety disorders in order to prevent and detect eating disorders.

I am interested in the temporal relationship between anxiety and eating disorders as well the implications of possible early detection to save lives as I am one of the 30 million people suffering from an eating disorder in the US, but I wish I wasn’t part of this statistic. As an individual who has had childhood anxiety, then a later eating disorder, and then combated these two disorders at the same time I have realized the power of these disorders. I will never forget my father to carrying my limp, trembling, eighty-five-pound body into the emergency room of the hospital not far from death. Not far from becoming another individual who dies every 62 minutes due to an eating disorder. Maybe I wouldn’t have gotten to that point though if anyone considered that years before this I had been suffering with anxiety and had many alarming personality traits for a child so young. Maybe if people acknowledged the fact that childhood anxiety can lead to an eating disorder, or if I was checked for disordered eating symptomology due to my extreme anxiety in other aspects of life I wouldn’t have ended up in the hospital at that point or ended up in college still fighting both an eating and anxiety disorder. As I am living proof that an anxiety disorder can lead to eating disorder I am here to argue that an anxiety disorder can precede an eating disorder and that early detection methods need to be put in place based on this temporal relationship in order to save the lives and health of many like myself.

While many researchers have conducted studies to further the phenomenon that childhood anxiety precedes eating disorders the issue that remains is how these disorders are connected and how do anxiety disorders precede an eating disorders. Researchers such as Silberg, Deep, Godart, Raney have all conducted studies to further the phenomenon that
childhood anxiety precedes eating disorders from a biological perspective. The explanations among these researchers for this phenomenon vary as some argue for the dysregulation of a serotonin pathway, others for genetic causes, or others argue that they are due to altered striatal dopamine function. My view is that no matter the explanation all need to be considered in order to detect these aspects in individuals with anxiety disorders for possible detection of eating disorder.

The first two sources that I began with were “Anxiety disorders in anorexia nervosa and bulimia nervosa: Co-morbidity and chronology of appearance” by Godart from the Journal of European Psychiatry and “Premorbid Onset of Psychopathology in Long-Term Recovered Anorexia Nervosa” by Deep from The International Journal of Eating Disorders. I chose these two articles first as they both focus on the relationship between anxiety disorder and eating disorders and similarly focus on the biological vulnerability of two disorders due to dysregulation of a serotonin pathway. Serotonin is a neurotransmitter that regulates things such as mood and appetite therefore dysregulation of this neurotransmitter can cause abnormalities. Godart’s research publication is based on a study conducted to assess the childhood history of anxiety disorder in patients with eating disorders. In his research he found that “83% of AN (anorexia nervosa) subjects and 71% of (bulimia nervosa) subjects had a lifetime diagnosis of an anxiety disorder, most frequently social phobia” (Godart et. al, 2000). This data he found furthered the concept that anxiety disorders precede eating disorder, but also led him to find common abnormalities in the serotonin system. His research led him to the conclusion that the serotoninergic dysfunction, or abnormal amount of serotonin in the brain, could be the biological connection between anxiety disorders and eating disorders (Godart et. al, 2000). Similarly, in a study by Deep conducted to determine if any illnesses predated the onset of anorexia and the
time course of onset of psychiatric illness, his findings also led him to the argument that the biological vulnerability of anxiety to eating disorders takes the form of disturbed serotonin activity. He suggests that “disturbed serotonin activity may contribute to anxiety and OCD” and that “increased serotonin activity may contribute to inflexible behaviors” (Deep, 1995), all of which he found to be linked and to possibly lead to eating disorders. This suggests the connection between serotonin dysregulation and symptoms found in individuals with anxiety disorders and their relation to eating disorders. His findings that “78% of individuals with childhood anxiety developed childhood anxiety about 5 years before their eating disorders” furthered their conclusion that anxiety disorders precede eating disorder and that these childhood anxiety disorders are due to serotonin dysregulation. These findings imply that serotonin dysregulation can act as an early precursor to both these disorders. Both researchers conclude their publications with similar ideas that the serotonin dysregulation is the first behavioral expression of a biologic vulnerability in subjects who develop eating disorders (Godart et. al, 2000, Deep, 1995). These research publications brought up a new biological insight to the relationship of anxiety and eating disorders by introducing the common factor of the dysregulation of the serotonin system. Both authors acknowledge that the dysregulation of the serotonin system is most likely one of a number of factors that explain the relationship between anxiety and eating disorders, but they acknowledge the fact that their research has limitations as they weren’t able to include all possible perspectives to the relationship of these disorders due to the limitations of their research. Both authors chose to focus their research on the biological factor of serotonin dysregulation therefore other possible explanations were omitted. I respect both author’s acknowledging the fact that their research does not show the full picture as I also agree that there are additional contributing factors. These two sources introduced a possible
explanation for the relationship between anxiety and eating disorders and provide research to further the phenomenon of anxiety disorders prior to eating disorders. Importantly their findings suggest a possible brain location to observe for detection of eating disorders in individuals with anxiety disorders. As these sources gave one biological explanation for the relationship for anxiety and eating disorders I wanted to then find other explanations researchers are providing.

Although some researchers argue that biological vulnerability of anxiety and eating disorders is due to serotonin dysregulation, others believe that genetics are responsible for the biological vulnerability that causes the phenomena of anxiety disorders prior to eating disorders. In the Journal of Children of Psychology and Psychiatry I discovered a study done on the developmental association between eating disorders symptoms and symptoms of depression and anxiety in juvenile twin girls. This study was conducted to discover the role of biological genetic factors in the development of symptoms of eating disorders. Silberg argued for the idea that childhood anxiety is a genetic pathway towards the development of an eating disorder. This argument was based on their findings of a common genetic factor that influences patterns of symptoms strongly associated with eating disorders, depression, overanxious disorder, and separation anxiety disorder during development. They also found a genetic effect that influences the susceptibility to over anxiety, separation anxiety, depression, and eating disorders in development. Overall, they argued that childhood anxiety is an important genetically mediated pathway toward the development of an eating disorder (Silberg & Bulik, 2005). This research provides another explanation for the relationship between anxiety and eating disorders from a different biological perspective. The explanation of a genetic factor that this research provides adds diversity to the biological perspective by providing additional explanation and another biological aspect to be looked at for early detection. I disagree though on the strength of genetics
as a causal factor due to my own experience as I have no relatives with eating disorders, but I acknowledge the fact that this could be a possibility for others. Additionally, I think their research needs to be proven further with more specific evidence of a specific genetic factor such as a gene or chromosome to make their findings more specific and legitimate. Like most research publications this research overlooked other explanations for the relationship due to limitations when conducting research. This source though is valuable as it does provide another explanation for the relationship from an alternative angle, but I feel that more specific research on this topic needs to be conducted. Due to the lack of specificity of genetics in this article I then went to articles to find further evidence of these genetic factors.

In my process of finding more information on genetic factors as explanations for anxiety and eating disorders I came across a study in *The International Journal of Eating Disorders* which argues for the genetic explanation of anxiety preceding eating disorders through the commonalities of personality traits of people with the two disorders. In the study to explore the impact of overanxious disorder on the expression eating disorder symptoms and personality traits it was found that women with previous overanxious disorder reported “greater body dissatisfaction, harm avoidance, trait anxiety, parental criticism, higher parental criticism, higher personal standards, greater concern over mistakes, greater doubts about actions, lower extraversion, and higher neuroticism compared to women without a history of overanxious disorder” (Raney, 2008). These common personality traits suggest another means to detect early signs of anxiety disorders. These personality traits were found to be the same personality traits found in women with eating disorders, therefore suggesting that the genetic component of personality traits of anxiety disorders act as a precursor for an eating disorder. Raney also states that these genetic personality traits were magnified during one’s eating disorder when they had a
history of overanxious disorder. This suggests that personality traits that accompany anxiety can also reappear during one’s eating disorder as their anxiety is likely to be increased. This implies that anxiety can also reoccur during the eating disorder which contradicts his findings but this idea also serves as another angle to look at the relationship between eating and anxiety disorders. This research publication then cited another study done by researcher Diaz-Marsa to find how temperament and personality traits influenced the development and course of eating disorders. This study found that individuals with eating disorders appeared to have the genetic personality traits of neuroticism, neurotic-emotionally unstable, and low self-directedness which is suspected to lead to the development of eating disorders. More specifically, they found that individuals with bulimia nervosa also had personality traits of harm avoidance, dependent personality disorders, and impulsivity while individuals with anorexia nervosa were found to have avoidant, obsessive-compulsive, and persistence personality disorders (Diaz-Marsa, 2000). These findings acted to back up the idea of common personality traits presented by Raney and also suggests that these traits can serve as warning signs to an anxiety disorder. Collectively these research publications furthered the idea of genetic personality traits as explanation for the relationship between these two disorders. I agree with this explanation as many of the personality traits they discussed I have myself and have found discussion of by other researchers. Even though these studies are limited to personality traits and explanation they work to strengthen the idea of genetic factors causing a biological vulnerability for eating disorders. These findings also act to identify personality factors that can be assessed in individuals with anxiety disorders for early detection of disordered eating symptomology.

As Raney’s publication in The International Journal of Eating Disorders provides evidence for the genetic connection between anxiety and eating disorders his findings also
suggest altered striatal dopamine as another possible explanation for the coupled disorders.

Similar to serotonin, dopamine is a neurotransmitter that regulates mood and motivation and when released in abnormal amounts in the brain causes issues. Altered striatal dopamine therefore means that there is an abnormal release of dopamine in the brain’s striatum, also known as the reward circuit. Raney’s research goes on to discuss the how altered striatal dopamine is related to uncertainty and how intolerance for this uncertainty then causes anxiety in individuals. He related his data with preexisting data which found that individuals with eating disorders often have heightened sensitivity to uncertainty causing extreme distress reducing mechanisms such as restricting or purging. This idea from Raney’s study connects to the article “How Uncertainty Fuels Anxiety” by Julie Beck as her article discusses how high levels of intolerance of uncertainty can lead to anxiety disorders, eating disorders, and depression. In her article she discusses how “IU (intolerance of uncertainty) seems to be a causal risk factor-meaning it’s not just linked to GAD (generalized anxiety disorder), but that higher IU has actually been shown through experimental manipulation to lead to more worry” (Beck, 2015). Beck makes the point of the severity of this uncertainty as it can become a never-ending cycle. She suggests that this cycle then can set into motion the pathway leading to an eating disorder in individuals who have a heightened sensitivity to uncertainty and react with reducing mechanisms such as restricting or purging. From personal experiences I strongly agree with Beck’s argument about uncertainty and anxiety as I have personally experienced great anxiety from uncertainty especially when making decisions with ambiguous outcomes. Raney’s findings on altered striatal dopamine provides insight for an explanation for uncertainty and heightened anxiety explained by Beck which allows these author’s arguments to support each other. The issue with these authors’ research though is that they highlight anxiety disorders while underscoring their connection to eating
disorders, but they still provide valuable information as they contribute to the broad concepts of anxiety and eating disorders. As this information is omitted by these authors their work has led to explanations for purging and restricting mechanisms for found individuals with eating disorders.

As a whole Silberg, Deep, and Godart, and Raney’s findings all supported the phenomenon of childhood anxiety disorders preceding eating disorders due to biological vulnerabilities. Even though their explanations for the biological vulnerabilities differ among the researchers they all contribute to the biological perspective on anxiety preceding eating disorders. Collectively, all these researchers have similar implications for all their studies. The data collected by these researchers suggest the idea that actions need to made to enact preventative methods to reduce and detect eating disorder symptoms before they manifest themselves fully. Judy Silberg suggests “the importance of screening young girls presenting problems with anxiety or depression for signs of developing eating disorders…screening anxious or depressed girls for eating problems may improve both detection and secondary and tertiary prevention efforts in eating disorders” after finding evidence for biological vulnerability due to genetics (Silberg & Bulik, 2005). Raney suggests that his data implies “early detection of OAD as a potential means of averting the emergence of both later anxiety and eating disorders symptoms” (Raney, 2008). On top of early detection, researchers such as Godart believe that their findings imply that in recovery of the eating disorder the anxiety disorder also must be recognized for successful treatment (Godart et. al, 2000). Even researchers, such as Spindler, who argue that anxiety disorders don’t so much appear before an eating disorders but simultaneous to the disorder believes that “a patient evaluation that incorporates a comprehensive assessment of comorbidity and an examination of interactions between comorbidity and eating disorder symptoms” needs to be completed early for prevention and if
not during recovery (Spindler, 2007). If we combine the findings of these researchers an
extensive evaluation procedure can be formulated to create screenings that can be administered
by pediatricians and school counselors. Not only do these findings provide insight for early
detection of eating disorders through anxiety, but also for more targeted recovery strategies. Both
improved detection and recovery could then act to save the health and lives of many so that we
no longer have “30 million people of all ages and genders suffer from an eating disorder in the
U.S.” (“Eating Disorder Statistics”).
Works Cited


Silberg, J., Bulik C. (2005). The developmental association between eating disorders symptoms