A RATIONAL THEORY OF ADDICTION: BULIMIA

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This paper investigates the hypothesis of rational addiction theory as it pertains to Bulimia Nervosa. Bulimia is currently classified as a mental health disorder, and while psychological studies often allude to the addictive nature of the disorder, it is imperative to reinforce theory with empirical data. A rational addiction model is derived through the maximization of the agent’s utility function, which incorporates measures of bulimia and bulimic risk factors that are supported by the existing literature. The theoretical model was tested on an empirical data set collected from a study conducted by the National Heart Lung and Blood Institute. The dependent variable was an index constructed for the purpose of measuring one’s severity of bulimic behaviors and tendencies. Emphasis was placed on the significance of the lagged variable on bulimia as a method of capturing persistence in behavior over time. The results found that lagged and lead variables of the bulimic index each accounted for about 12% - 20% of current bulimic behavior.

KEYWORDS: (Bulimia Nervosa, Rational Addiction Theory, National Heart Lung and Blood Institute)
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DEDICATION

I would like to thank my professor and advisor, Aju Fenn for supporting me through this process. I would also like to dedicate this paper to every girl who has ever struggled or continues to struggle with this eating disorder.
CHAPTER I
INTRODUCTION

The theory of rational addiction within the field of addiction economics has opened up a vast production possibility frontier for the capabilities of economic estimation. At first the integration of addiction theory and economic modeling may seem counterintuitive, but implications derived from these theories have the potential to raise serious policy reform questions about behavioral addictions and their validity for potential insurance coverage and preventative programs.

While addiction economics constitutes a wide range of topics, it is predominantly made up of studies that analyze an array of consumption addictions. The benefits of employing a discreet numeraire addictive good are that it can be numerically quantified and monetarily measured. The basic quantitative measures associated with consumption goods fortify the derivations and implications of demand functions. This paper is aimed towards the study of Bulimia Nervosa as an addiction, through the use of a rational demand model similar to Fenn (1998).1

It's not surprising that the dawn of a new age of the superficial, that has been sparked by the incredible speed with which technological advancements are accomplished, has shared an upwards trend with the incidence of bulimia in girls in

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young women.\textsuperscript{2} The Internet age has created an arena where judgment can occur anonymously behind closed doors. In a world where little seems private, the incidence of bulimia, which epitomizes the need for privacy, has grown at a fast rate. This paper will use the information imparted by the psychological research to develop a theory to explain broad and specific risk factors of bulimia nervosa that distinguish it has a behavioral addiction.

CHAPTER II
LITERATURE REVIEW

Although many contributions have been made to the study of Economic Addiction Modeling has predominantly focused on consumption addictive behaviors such as cigarette addiction. The literature falls short of the expansive set of behavioral disorders that exhibit addictive traits. As the study of behavioral addictions grows within the field of psychology, the perception of eating disorders as addictive is becoming widely accepted; thus it is important to analyze the addictive nature of eating disorders through econometric analysis.¹ This chapter will provide a review of the current literatures on both the economics of addiction and the psychological analysis of bulimia as an addiction.

Psychological Approach

This section will present the current psychological literature pertinent to the development of and persistence to the disorder. First, the dual quality of addictions will be discussed as a way to develop the sentiment this paper offers. The abusive family structure will be explored in detail, as well as the social pressures that guide girls to develop the disorder. The mind and body relationship from the perception the bulimic will be explored through the notion of permeable boundaries. This section will elucidate upon how the loss of boundaries within the abusive family construct leads to an

inability to comprehend limitations and balance, two ideals that are vehemently violated in the binge purge cycle.

Bulimia nervosa is seen as a psychosomatic disorder, affecting both the physical and mental configuration of a person; therefore, the causes are multidimensional in nature. Simona Giordano attributes the pathological behavior that bulimics exhibit to some neurological, physiological, endocrine, or biochemical disorder.\(^2\) Giordano defines pathological behavior as a consequence of an organic cause, and not as a choice made by the sufferer. This definition is analogous with her description of “hard” addiction as an irresistible urge to do something, which is rooted in psychological or neurophysiologic processes. For her, this implies that the person’s freedom is curtailed and the addiction is considered a dependency.

Marilyn Freimuth, Et. Al., propose that substance use disorder (SUD) rarely occurs in isolation and that dual diagnosis, the co-occurrence of an SUD and mental illness, is the norm.\(^3\) Research suggests that the most common pattern involves a mental health diagnosis that precedes the SUD by five to ten years. Epidemiological studies confirm that the use of one substance is correlated with using other substances, a trend that is often overlooked in the diagnosing process. SUDs also co-occur with mental disorders, which are also often unrecognized and complicate the accuracy of diagnosis and treatment.

Many trade-offs exist within eating disordered patients such as binge eating for shopping, vomiting for laxatives, and vomiting for exercising, bringing attention to the

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multidimensional nature of bulimic behavior. Addiction hopping often happens during an attempt to give up an addiction or go cold turkey. The addict often unconsciously transfers the impulsive and compulsive energies she had reserved for the former addiction towards an activity such as yoga that she perceives as positive. Over indulgence in yoga is not the problem, it is the imperceptible transfer of energy from one substance or behavior one end of the spectrum, such as alcohol, to another contradictory behavior at the other end of the spectrum, such as yoga, that invokes the inference that individuals may simply be addicted to being addicted. The idea that addiction to one behavior leaves a person susceptible to developing another addictive disorder is referred to as substance replacement, or addiction-hopping. The co-occurrence of multiple addictions and a propensity to transfer from one addiction to the next, suggests the existence of an addictive syndrome that can manifest in different forms. This analysis expounds upon an idea paramount to understanding bulimia as an addiction, that the addictive behavior i.e., smoking, drinking, bulimia, etc. is a symptom of a deeper mental disorder.

Bulimia can be perceived as a battle for self-control and autonomy; bulimics struggle with the desire to exert control and the urge to lose it.¹ In recent years researchers have found that this elusive fantasy often stems from childhood trauma and unstable family environments. The family environment is characterized as being

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disengaged, chaotic, and neglectful where conflict and hostility are apparent, but open expression is not encouraged.⁵

In their 1988 paper, Humphrey and Stern integrated Donald Winnicott’s construct of the *Maternal holding environment* and M. Klein’s formulation of *ego deficits at the level of part-object relations to the entire family system* to suggest that, “within families where a person has been diagnosed with bulimia, there are trans-generational developmental deficits that determine the level and quality of the internal psychological experience within the individuals, as well as the dynamic functioning of the larger family system. In this family construct there is a tension between unmet individual needs and equally or more powerful family needs that require individual self-sacrifice”.⁶ Igoin-Apfelbaum, in his (1985) study of family backgrounds of bulimic patients, found an interesting dichotomy between actual family dysfunction and the desire of the patients to achieve their family unit fantasy. From this incongruous perception, he theorized that the occurrence of bulimia might be related to a combination of a history or threats of violent separations.⁷

The dysfunctional family imposes threats of violence, often to coerce the individual to attain unreasonable goals of perfection. Simona Giordano reasons that although the dysfunctional family may impose an excess of high expectations and contradictory demands on the individual, the eating disorder is not developed out of the

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need to control these external pressures. She believes that the patients are rather made to suffer by their own adherence to a moral logic within which, disappointment is experienced as morally wrong. Giordana’s conception of the self, combined with the Apfelbaum’s assessment of the patient’s paradoxical rejection of and desire for family, provides a glimpse into the development of bulimic behaviors. The bulimic person often suppresses the hurtful feelings felt within the family unit, and unconsciously uses food as a means through which he or she can physically act out those emotional tensions. Many parallels can be drawn between food and family through the bulimic ritual of bingeing and purging. The bulimic cycle begins with restricting food, a behavior that is analogous to the individual’s desire to escape the harmful family. A binge occurs when the bulimic’s appetite becomes insatiable, as a result she consumes large quantities of food to fill the emotional void the family could not. Food becomes the fantasy of the perfect family unit that the individual yearned for as a child. When the bulimic realizes that overeating does not fill the void and that the fantasy is not real, shame and anger drive her to purge the guilt felt by desiring food, an allegory of the harmful family. Purging allows the individual to feel in control through the detachment from both food and family. In this model, the patient replaces her sense of self with the eating disorder, unable to identify the self from the disorder.

Lehoux and Howe, in their 2005 study, used cross-sectional data to compare women with bulimia to their sisters in order to empirically test the roles that perceived non-shared environmental factors and personality traits play in the development of bulimia. Using a survey-based system the researchers were able to identify different risk

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factors by comparing the bulimic girls to their sisters through regression analysis. A major contribution made by this study is the elucidation of the highly positive and significant variable “insecure attachment to fathers”. Insecure paternal attachment significantly increased the risk for bulimia by about ten times, after controlling for depression and anxiety. *Insecure paternal attachment* has been linked to an increased risk for developing psychiatric problems and may represent a perceived non-shared risk factor for women with bulimia. Insecure paternal attachments, reflect the feelings of desiring a close father-daughter relationship, combined with the fear of a sexualized relationship. The inability for the girl to separate sexual relationships with familial closeness reveals yet another apparatus through which misguided or lack of boundaries manifest. The fact that insecure paternal attachment is an underlying risk factor for various psychiatric problems contributes to the idea that many addictions are born out of similar mental deficits and trauma, and that the variance between different substances may reflect specific environmental factors and personality traits.

In Lehoux et al.’s 2005 study women with bulimia demonstrated higher scores on narcissism and reported experiencing more teasing than their sisters; both variables exhibited positive correlates with bulimia. Developmental teasing experiences, in conjunction with a high value on appearance, may have heightened the risk for body dissatisfaction and the propensity to diet. Lehoux argues that childhood teasing may be a retrospective correlate manifested through post-traumatic stress disorder. “Rorty and Yager (1996) argue that severe dieting and bingeing may represent attempts to manage

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painful emotions associated with intrusive memories of childhood trauma and be a way to regain a sense of control and identity cohesion”.

It is clear that the dysfunctional family creates a space conducive to the manifestation of eating disorders in an individual, but families also play a hereditary role in the development of bulimia. Over the past decade, there has been a surge of evidence supporting the influence of genetic factors on the etiology of eating disorders. Family and twin studies have shown that bulimia is a familial disease, and that a substantial portion of the observed familiality is due to genetic factors. The inheritability of bulimia nervosa has been estimated to be between 28% and 83%, with the remaining variance attributable to individual specific environmental factors. The serotonergic system has received considerable attention for its potential role in the development of eating disorders because it helps with the regulation of food intake and body weight. Several findings suggest dysregulation of serotonergic transmission and increased serotonin activity in patients with eating disorders. Several genes involved in the serotonergic system have been identified as possible “candidate genes” for eating disorders, but contradictory results have hindered the validity of these findings. Even though the exact genetic and biological elements that contribute the hereditary nature of eating disorders are uncertain, this research provides a new platform from which bulimia can be understood.


12 Ibid.
In order to examine the addictive nature of bulimia it is necessary to understand the underlying roots of other substance use disorders. Norman Miller and Mark Gold, in their 1990 paper, propose the adaptive model of addiction, which states that the addictive substance is used to replace a failure in adult integration, caused by childhood social and family problems. According to this model, the individual seeks an identity of the addict, which is preferable to that of admitting failure and dependence on the abusive family.13

Not only are the psychological issues that cause bulimia and substance abuse disorders similar, but both also share common etiologies. The shared etiology hypothesis views the relation of the two disorders as the result of common risk factors, including personality type, family history, and biological vulnerability, that predispose an individual to develop bulimia and/or a substance abuse disorder.14 For Rebecca J. Carbaugh, specific personality type is crucial to the shared etiology hypothesis. Personality traits common to both disorders are lack of control, craving, and denial. Individuals who are prone to either disorder display social anxiety, impulsivity in situations that have self-damaging resolutions, and have a deep desire for affection and acceptance.15 These personality traits cause people to be more inclined to develop bulimia or a substance abuse disorder because both are impulsive and self-damaging.

Bulik and Sullivan found that similar parental traits tend to contribute to the development of both disorders. Paternalistic characteristics include lacking enjoyment

15 Ibid.
as a father, having inappropriate seductive boundaries with daughters, and inhibiting
development of independence; while mothers contribute to the onset of both addictions
by primarily focusing on weight and appearance. Through these parental dynamics, the
child feels the need to please others by striving achieve external expectations,
disregarding her own needs and goals. The child becomes powerless to establishing
boundaries, allowing others to pervade the salient inner space that is the self. In turn, the
child loses respect for her worth and is unable to create boundaries for herself, leading
to the acquisition of the addiction. The addiction becomes a sacred and private medium
through which autonomy and control can be exercised. The individual indentifies with
the addiction as a manifestation of the inner self, without which he or she would
completely lose control.

The symmetry in the development and persistence of both bulimia and substance
addiction is evident through the analyses of the aforementioned studies, but in order to
test the addictive nature of bulimia through regression analysis, current econometric
models and empirical studies of addiction must be explored.

It is clear that the development of and persistence to bulimia is extremely
complex in its composition, which is confound by the interactions of highly contrasting
sentiments and actions, such as the desire for paternal attachment that is convoluted by
the fear of sexual intimacy. A lack of or misguided perception of boundaries causes
children to feel and act in extreme and often opposing ways. This intense push-pull
mentality is manifested through the binge-purge cycle, in which the bulimic violates the
physical boundaries of her body, manifesting the notion of permeable boundaries in a
physical and masochistic way. Although the specific risk factors and social dynamics
that lead one towards the development of bulimia may differ from the specific risk factors those of a smoker, but the impulsiveness and bipolarity that cause bulimia to persist, are often what cause substance addiction to persist.

**Economics of Addiction**

In their 1994 study, Becker, Grossman, and Murphy employed their model of addiction to empirically test whether cigarette smoking is addictive. The BGM model is derived from a utility maximization problem that depends on consumption in that period and in the previous period, subject to a lifetime budget constraint, because behavior is addictive if past consumption influences future consumption. They used cross sectional time series data to test the presence of addiction and to test whether a myopic or rational model is more accurate. The rational model considers consumers to be forward looking, such that future expected prices would effect current consumption, while the myopic model reflects non-forward thinking consumers, such that future price does not effect current consumption. The results show that future prices do influence current consumption, and are thus consistent with the rational model. The coefficient on past consumption is positive and significant, corresponding with the original hypothesis that cigarette smoking is addictive.

Fenn and Schroeter’s 1991 paper adds to the work of Becker et al., by testing the hypothesis that cigarette demand was myopic in nature prior to 1979, when the General Surgeon released information about the harmful effects of cigarette smoking, and rational from then on. Fenn developed a modified version of the BGM model, also

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17 Ibid.
using cross sectional time series data. Coefficients on past consumption are positive and significant on pre and post 1979 periods, supporting the hypothesis that cigarettes are an addictive substance. While their results exhibit evidence that cigarette demand experienced a structural break in 1979, the positive and significant coefficient on future consumption in periods prior to and after 1979 imply that cigarette demand corresponded with a rational pattern even before the Surgeon General’s warning was released.\(^{18}\) This paper contributes to and supports the earlier work by Becker et al., verifying the strength of the rational model of addiction and the proposal that cigarette smoking is addictive.

Other contributions have been made to empirical analysis of economic addiction, but mainly focus on addictive substances such as alcohol, cigarettes, sex, drugs, and gambling. There is however, one empirical economic analysis of bulimia as an addiction, inspired by the BGM rational addiction model. Goeree et al.’s, 2009 study used regression analysis to support their assumption that bulimia exhibits addictive behavior, consistent with the BGM rational addiction model. Through econometric estimation methods they found that lagged bulimic behavior is significant and influences future behavior, therefore bulimic behavior satisfies the economic conditions for addiction.\(^{19}\) A noteworthy conclusion this paper draws from the results is the fact that over half of the persistence in bulimia is due to the addictive nature of behavior, but that unobserved heterogeneity is still important. This is finding is important because it shows that the actual behaviors employed in bulimic episodes are addictive, controlling


\(^{19}\) Michelle S. Goeree, John C. Ham, and Daniela Iorio, *Caught in the Bulimic Trap: Do Eating Disorders Reflect Addictive Behavior?*, March 1, 2009.
for unobserved biological and genetic factors that may predispose a person to bulimia. While individual heterogeneity may contribute to the development of the addiction, the scientific literature lacks information about which genetic and biological traits predispose a person to developing an eating disorder. This would also be difficult to test through regression analysis because it would require time series micro data containing genetic and biological data on the individuals. This result, that actual state dependence accounts for over half of the persistence in bulimia, is the first economic contribution towards modeling bulimia as an addictive behavior.

The psychological literature presented in this chapter aids in delineating the risk factors that contribute to bulimia nervosa, and also sheds light upon the similarities between substance addiction and eating disorders. While this information is helpful starting point, it fails to provide a clear correlation between bulimia and addiction. The literature presented on the economics of addiction does not theoretically delve into the extensive set of behavioral addictions that exist, mostly because it is difficult to create an econometric model for a non-substance addiction. Therefore the next section will explore a method through which bulimic behavior is measured, and propose a theoretical economic model for bulimia using the framework of the BGM rational model.

Conclusion

The analysis of the psychological literature pertaining to bulimia reveals complex and convoluted nature of the disorder, but, from a simpler perspective, the impulsive, extreme, and volatile nature of bulimia is much more similar to the behavior of a substance abuse addict than a person struggling with anorexia. The next chapter
will develop a theory for the addiction to bulimia, and the specific constituents that make it more similar to substance abuse disorder than to anorexia.
CHAPTER III
THEORY

This section seeks to delineate the psychological disorders and specific risk factors that contribute to bulimia nervosa, such that a concise theory for both the onset of and addiction to this eating disorder can be constructed. This construction of bulimia is aimed to validate the etiological factors that lead to the addiction, which are laid out in the psychological literature. A rational consumer’s utility maximization problem will be developed later in this chapter in order to derive a function that represents the addict’s demand for bulimic behavior. The method for measuring bulimic behavior that will be used to quantify demand is referred to as the Eating Disorder Inventory (EDI), and will be described within this section. The construction and derivation of the utility maximization problem in this paper comes from Aju Fenn’s modification to the Becker-Grossman rational addiction model, which will be clearly described in this chapter. Finally the rational addiction model will be derived.

Risk Factors

Bulimia nervosa is caused by biological, social, familial, and personality linked risk factors, but the severity of each factor and the order in which they strike is hazy in the existing research. Many studies have found that a mental health diagnosis usually precedes an addiction by five to ten years, satisfying the aforementioned-shared etiology hypothesis, which claims that both eating disorders and substance use disorders
share common roots. In other words mental health disorders create a fertile space for any addiction to manifest, which adheres to the notion that addictions develop out of the need to fill an inner void. Mental illnesses, common to both types of addictions include anxiety, depression, and obsessive-compulsive disorder. Therefore, within this theoretical construction mental disorders make an individual vulnerable to an addiction, but specific environmental and biological risk factors channel the dependence towards bulimia.

**Abusive Family Construct**

The abusive family structure hinders children from understanding and developing personal boundaries, and is therefore considered a specific risk factor. Bulimia is often characterized by a struggle to create and maintain boundaries, and is elucidated within the binge purge cycle itself, i.e. having a convoluted sense of limits seen though starvation, overeating, and purging. The ability to set boundaries allows individuals to gain control and self-respect by mediating their interior space and identifying with themselves. Women with eating disorders have issues setting boundaries for themselves and others, allowing people to invade the interior space, which in turn deteriorates the sense of self and causes them to lose control. A child who grows up in an abusive household and experiences childhood trauma is not able to develop and understand boundaries, and therefore allows exterior forces to define her

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2 Ibid.

sense of self. The eating disorder becomes a metaphor for the individual’s identity and the binge purge cycle reflects her failed attempts to exercise autonomy.

In an abusive household where the child lacks a sense of boundaries, she becomes disillusioned by her needs and her parents’ demands. Often times, in this setting the child will strive to meet external pressures in order to gain approval from others. This extreme sense of perfectionism is a symptom of the dysfunctional family and is a correlate for bulimia.\(^4\) Within this family construct the child does not develop the defenses to protect herself from the biological factors that dispose her to the disorder. The hereditary component of this eating disorder is two-fold: 1. Genetics predispose an individual to the disorder, 2. Mothers who are bulimic or have disordered eating unintentionally impose pressure on their daughters to be thin.\(^5\) Either way, a family history with bulimia is a risk factor for the disorder.

Over the years psychoanalysis papers have speculated upon the notion that specific paternal and maternal attributes factor into the development of bulimia. “Freud conceptualized bulimia as hysterical symptoms resulting from unconscious sexual conflict involving oral incorporative mechanisms and Oedipal genital wishes.”\(^6\) He believed that vomiting was representative of an underlying oral sadistic sexual fantasy. Freud’s knack for over-sexualization must be taken into consideration upon analyzing his prescribed theory. A contemporary take on Freud’s theory views bulimic symptoms

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as a result of the conflict between the desire for closeness with the father and the misguided fear of incestuous intimacy. This interpretation is analogous with the results from Lehoux and Howe’s 2005 study, that insecure paternal relationships were a significant risk factor for bulimia.⁷ In the 1970’s Bruch, using Freud as her platform, saw eating disorders as expressions of the person's conflict between wanting independence and wishing to remain with the mother.⁸ She believed that gender sameness sustained the illusion that the girl and the mother were really one, and although she did not agree with Freud, this sense of mother-daughter sameness contributes to the fear of an Oedipal bond between father and daughter.

Social Pressures

Social pressures and societal expectations to be thin scrupulously contribute to the onset of bulimia such that they can penetrate the sturdiest of boundaries. The most notable form of social trauma is endured through teasing and ostracism. Girls who are teased for being overweight at a young age learn that being fat is bad and in turn place the desire to be thin on a pedestal. This form of exclusion causes girls to become anxious, depressed, and inspires within them a rigid drive to be thin, all of which are common symptoms of bulimic patients.⁹ Societal expectations to look a certain way reinforce the importance of physical appearances for these girls and foster a sense of narcissism within them. In conjunction with teasing, these pressures can compel a girl

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to place herself worth in her appearance and disregard her emotional and bodily constituents.

Rational Addiction Models

Becker and Murphy’s 1988 paper, *A Theory of Rational Addiction* introduced a theoretical model where forward looking utility-maximizing consumers could become addicted to a substance, and take future consequences of present day consumption of the substance into consideration. The key contribution this model bestowed upon the field of addiction economics was the switch from a myopic model, in which consumer choices are solely based on utility in the current and previous periods, to the rational foresighted consumer choice model. The employment of consumer foresightedness suggests that if the price of the addictive good is set to rise in the next period, consumers will cut back on present consumption.\(^{10}\)

The Becker-Murphy theoretical model explains elements of the addiction process, such as tolerance, binges, and cold turkey, through the existence of unstable steady-state consumption levels. The model was empirically tested by Becker et al. (1994), in which they examined whether changes in past and future cigarette prices would raise or lower current cigarette consumption.\(^{11}\) For the sake of concision this summary will only introduce the basic utility function of the model. The implications of the rational model will be clearly explored in the overview of Fenn’s modification to it. In the Becker-Murphy model a consumer’s utility depends on two goods, \(c\) and \(y\).

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\(^{11}\) Ibid.
They assume that \( u \) is strictly concave in all of its arguments. Therefore utility in the current period is a function of \( c(t) \), current consumption of the addictive good, \( y(t) \), current consumption of a composite good, and \( S(t) \), current period’s addictive stock. Past consumption of \( c \) affects current utility through the addictive stock, \( S \).

\[
\dot{S}(t) = c(t) - \delta S(t) - h[D(t)]
\]

\( \dot{S} \) is the rate of change over time in \( S \), \( \delta \) represents the instantaneous depreciation rate and measures the exogenous rate at which the physical and mental effects of past consumption of \( c \) diminish, and \( D(t) \) represents the consumer’s endogenous expenditures to depreciate the addictive stock.

Fenn made a modification to the Becker-Grossman model, specifically to the stock constraint, so at this point the discussion of the rational addiction model will shift to Fenn (1998). Equations (3.1) and (3.2) rewritten in discreet form:

\[
U_t = U(Y_t, C_t, A_t) \tag{3.3}
\]

\[
A_t = C_{t-1} + (1 - \delta)A_{t-1} \tag{3.4}
\]

As mentioned earlier, this states that the addictive stock in the current period is equal to the sum of last period’s consumption and the existing remnants of last period’s addictive stock. Fenn makes the observation that even if the last period’s addictive stock has fully depreciated such that \( \delta = 1 \), that the addictive stock in the current period will still equal the last period’s consumption level.\(^{12}\) In other words if \( C_{t-1} > 1 \), the current

The difference between equations (3.4) and (3.5) is that the latter represents the undepreciated portion of the last periods consumption rather than its full value. The rationale supporting this revision is that the quantity of the addictive substance, nicotine in Fenn’s case, consumed at any point in time begins to depreciate prior to consumption in the next period: “The prolonged use of nicotine over time causes the body to be accustomed to a certain level of the drug. If the body experiences a drop in the level of nicotine below the level that it is used to, then the person experiences withdrawal.”¹³ This adjustment provides a more general form of the stock constraint in which one is able to model demand for both addictive and non-addictive goods. A non-addictive good does not accumulate addictive stock and can be modeled when δ = 1.

The rational consumer’s utility function in period t is given by equation (3.6). Equation (3.7) represents the utility function substituting out for $A_t$, which is simplified because Fenn, keeping with Becker et al., assumes that only the previous period’s consumption enters into the addictive stock

\[
U_t = U(Y_t, C_t, A_t, e_t) \quad (3.6)
\]

\[
U_t = U(Y_t, C_t, (1 - \delta)C_{t-1}, e_t) \quad (3.7)
\]

Fenn makes the assumptions that marginal utility increases with consumption of both the addictive good and composite good, $U_{C_t} > 0$ and $U_{Y_t} > 0$, while it decreases with an increase in the addictive stock, $U_{A_t} < 0$. These assumptions allow for the model to

¹³ Ibid.
account for the occurrence of tolerance, withdrawal, and reinforcement. Fenn defines tolerance as the effect of obtaining a lower level of pleasurable response to the addictive good as the level of past consumption rises \( (U_{A_t} < 0) \). Withdrawal is the decline in utility endured from the reduction of the addictive good \( (U_C > 0) \). Reinforcement is the marginal utility from consumption of the addictive good as the consumer’s past experience with the substance rises, and is captured by assuming that \( U_{C_0>0} > 0 \).\(^{14}\)

The next steps in Fenn’s rational model follow the same path as the Becker-Murphy model. It is assumed that the consumer lives for \( T \) years, discounts future utility at the market rate of interest \( r \), and has a lifetime budget constraint. Based on these conditions the consumer’s utility maximization problem is shown in equation (3.8).

\[
\text{Max}_{C_t,Y_t} \sum_{t=1}^{T} \beta^{t-1} U(Y_t, C_t, (1 - \delta)C_{t-1}, e_t) \quad \text{s.t.} \quad \sum_{t=1}^{T} \beta^{t-1} (Y_t + P_t C_t) = W_t \tag{3.8}
\]

The first part of the equation is the consumer’s lifetime utility function where the agent’s discount factor is represented by \( \beta = \frac{1}{1 + r} \). The second part of the equation is the agent’s lifetime budget constraint \( W_t \) is the present value of lifetime wealth, \( P_t C_t \) is the current price of the addictive good multiplied by the amount of the addictive good consumed, and \( Y_t \) represents the amount spent on the composite good because the variable itself is numeraire. The Lagrangian and associated first order conditions with respect to \( Y_t \) and \( C_t \) are written as follows:

\[
L = \sum_{t=1}^{T} \beta^{t-1} U(Y_t, C_t, (1 - \delta)C_{t-1}, e_t) + \lambda \{ W_0 - \sum_{t=1}^{T} \beta^{t-1} (Y_t + P_t C_t) \} \tag{3.9}
\]

\[
\frac{\partial L}{\partial Y_t} = U_{Y_t}(Y_t, C_t, (1 - \delta)C_{t-1}, e_t) = \lambda \tag{3.10}
\]

\(^{14}\) Ibid.
Equations (3.10) and (3.11) are the first order conditions derived from the Lagrangian in equation (3.9). Equation (3.10) states that the marginal utility gained from consumption of the composite good is equal to the marginal utility of wealth, which makes intuitive sense because \( Y_t \) is equal to the money value of the composite good. Equation (3.11) states that the present value of the marginal utility of current consumption is equal to the current price of the addictive good multiplied by the marginal utility of wealth.\(^{15} \) This implies that the agent takes the impact of current consumption on the current period’s marginal utility into consideration, as well as its impact on the next period’s marginal utility through its contribution toward next period’s addictive stock.

**Rational Agent’s Utility Maximization Problem**

In both the Becker-Murphy and Fenn models of rational addiction, the addictive goods are cigarettes; a substance for which consumption can be quantified and price can be labeled. Because bulimia is a behavior it does not have an explicit price nor can it be quantified through levels of consumption, thus, a shift of perception is required to understand this model. The agent’s utility function in period \( t \) is given by equation (3.12).

\[
U_t = U(C_t, A_t, G_t, Y_t, e_t) \quad (3.12)
\]

Where:

\[
A_t = (1 - \delta)C_{t-1} \quad (3.13)
\]

\[
G_t = (X_t + H_t) \quad (3.14)
\]

\(^{15}\) Ibid.
In this utility function $C_t$ represents the EDI bulimic index, which, as stated earlier in this paper, is a measure of the severity of bulimia. In Fenn’s model $C_t$ represented consumption of cigarettes, but different levels of consumption can also be viewed as levels of severity of the addiction to cigarettes, in an effort to draw parallels. Here $A_t$ takes the same form as Fenn’s modification, the addictive stock is the un-depreciated portion of the last period's bulimic index. $G_t$ represents emotional and hereditary risk factors that contribute to bulimia, where $X_t$ is a vector of the EDI indexes, with the exception of bulimia, and $H_t$ is equal to the agent’s mother’s bulimic index. $Y_t$ represents a composite good or activity, and $e_t$ represents unobservable lifetime events.

By assumption marginal utility increases with in both the bulimic index and the composite good/activity, $U_C > 0$ and $U_Y > 0$, while it decreases with an increase in the addictive stock, $U_A < 0$. Since these assumptions are analogous with Fenn, the same theories of withdrawal, reinforcement, and tolerance apply to this model.

Assuming that the agent lives for $T$ years and discounts future utility at the market rate of interest $r$, the agent’s lifetime utility function and budget constraint are given by:

$$U(0) = \sum_{t=1}^{T} \beta^{t-1}U(C_t, A_t, G_t, Y_t, e_t)$$

(3.15)

Substituting for $A_t$ from equation (3.13) into (3.15) gives:

$$U(0) = \sum_{t=1}^{T} \beta^{t-1}U(C_t, (1-\delta)C_{t-1}, G_t, Y_t, e_t)$$

(3.16)

Subject to:

$$\sum_{t=1}^{T} \beta^{t-1}(Y_t + P_tC_t) = A^0$$

(3.17)
where the agent’s discount factor \( \beta = \frac{1}{1 + r} \).

Here \( P_t \) is still defined as the price of the addictive good, but corresponds to different implications because bulimia does not take an explicit monetary value. In this model \( P_t \) represents the negative events or circumstances that emotionally incline an individual to cope through bulimic behaviors. For example, an individual, who is disposed to this eating disorder for various reasons, experiences emotional trauma within her family unit that causes her family’s cohesion to decline and in turn triggers her propensity to indulge in the purge cycle of bulimia. In the next period her family’s cohesion increases so her binge, purge behavior declines in severity because the root of her emotional stress is better than it was in the previous period. Here family closeness represents a proxy for price within this model of bulimia. A strong assumption that must be made about price here, is that it is exogenous. It must be assumed that the individual has no role in the level of family cohesion that she experiences.

Lifetime wealth \( A^t \) represents the sum of the agent’s capacity and time. The Lagrangian for this problem is written as:

\[
L = \sum_{t=1}^{T} \beta^{t-1}U(C_t,(1-\delta)C_{t+1}(X_t + F_t),Y_t,e_t) + \lambda\{W_0 - \sum_{t=1}^{T} \beta^{t-1}(Y_t + P_tC_t)\} \quad (3.18)
\]

The first order conditions with respect to \( Y_t \) and \( C_t \) are:

\[
\frac{\partial L}{\partial Y_t} = U_y(C_t,(1-\delta)C_{t+1}(X_t + F_t),Y_t,e_t) = \lambda \quad (3.19)
\]

\[
\frac{\partial L}{\partial C_t} = U_1(C_t,(1-\delta)C_{t+1}(X_t + F_t),Y_t,e_t) + \beta U_2(C_{t+1},(1-\delta)C_t,(X_{t+1} + F_{t+1}),Y_{t+1},e_{t+1})*(1-\delta) = \lambda P_t \quad (3.20)
\]

Equation (3.19) states that the marginal utility gained from increasing the severity of bulimic behavior is equal to the marginal utility of wealth, and equation (3.20) states
that the present value of the marginal utility of current bulimic behavior is equal to the
current price of the addictive good multiplied by the marginal utility of wealth. The
first-order conditions in this problem are almost identical to Fenn’s, with the exception
of different exogenous variables, but their interpretations hold different implications
because money and wealth are not defined in numeraire terms, but rather the emotional
and psychological capacity of the body and mind.

The derivation of the demand equation is identical to Fenn’s (not shown in this
paper), where the utility function is quadratic in \( C_t, G_t, Y_t, \) and \( e_t \). The quadratic utility
function at time \( t \) is given by equation (3.21).

\[
U_t = U_1 C_t + U_2 A_t + U_3 G_t + U_4 Y_t + U_5 e_t + \frac{U_{11}}{2} C_t^2 + \frac{U_{22}}{2} A_t^2 + \frac{U_{GG}}{2} G_t^2
\]

\[
+ \frac{U_{yy}}{2} Y_t^2 + \frac{U_{ee}}{2} e_t^2 + U_{12} C_t A_t + U_{1g} C_t G_t + U_{2g} A_t G_t + U_{1y} C_t Y_t
\]

\[
+ U_{2y} A_t Y_t + U_{1e} C_t e_t + U_{2e} A_t e_t + U_{gy} G_t Y_t + U_{ge} G_t e_t + U_{ye} Y_t e_t
\]

(3.21)

Next substituting out for \( A_t \) using equation (3.13) and \( G_t \) using (3.14) yields the utility
function in equation (3.22).

\[
U_t = U_1 C_t + U_2 (1 - \delta) C_{t-1} + U_3 (X_t + H_t) + U_4 Y_t + U_5 e_t + \frac{U_{11}}{2} C_t^2 + \frac{U_{22}}{2} (1 - \delta)^2 C_{t-1}^2
\]

\[
+ \frac{U_{GG}}{2} [X_t^2 + H_t^2 + 2X_t H_t] + \frac{U_{yy}}{2} Y_t^2 + \frac{U_{ee}}{2} e_t^2 + U_{12} C_t (1 - \delta) C_{t-1} + U_{1g} C_t (X_t + H_t)
\]

\[
+ U_{2g} (1 - \delta) C_{t-1} [X_t + H_t] + U_{1y} C_t Y_t + U_{2y} Y_t (1 - \delta) C_{t-1} + U_{1e} C_t e_t + U_{2e} e_t (1 - \delta) C_{t-1}
\]

\[
+ U_{gy} (X_t + H_t) Y_t + U_{ge} (X_t + H_t) e_t + U_{ye} Y_t e_t
\]

(3.22)

Because this model is forward looking, the quadratic utility function \( U_{t+1} \), must also be
given.
\[ U_{t+1} = U_1 C_{t+1} + U_2 (1 - \delta) C_t + U_y (X_{t+1} + H_{t+1}) + U_{yx} Y_{t+1} + U_{ye} e_{t+1} + \frac{U_{11}}{2} C_{t+1}^2 + \frac{U_{22}}{2} (1 - \delta)^2 C_t^2 \]

\[ + \frac{U_{gg}}{2} [X_{t+1}^2 + H_{t+1}^2 + 2X_{t+1}H_{t+1}] + \frac{U_{gy}}{2} Y_{t+1}^2 + \frac{U_{ge}}{2} e_{t+1}^2 + U_{12} C_{t+1} (1 - \delta) C_t + U_{1g} C_{t+1} (X_{t+1} + H_{t+1}) \]

\[ + U_{2g} (1 - \delta) C_t [X_{t+1} + H_{t+1}] + U_{1y} C_{t+1} Y_{t+1} + U_{2y} Y_{t+1} (1 - \delta) C_t + U_{1e} C_{t+1} e_{t+1} + U_{2e} e_{t+1} (1 - \delta) C_t \]

\[ + U_{gy} (X_{t+1} + H_{t+1}) Y_{t+1} + U_{ge} (X_{t+1} + H_{t+1}) e_{t+1} + U_{ye} Y_{t+1} e_{t+1} \]

(3.23)

Using the quadratic utility function in equations in (3.22) and (3.23), the first order conditions in equations (3.19) and (3.20) can be written as:

\[ U_y + U_{yy} Y_t + U_{1y} C_t + U_{2y} (1 - \delta) C_{t-1} + U_{gy} (X_t + H_t) + U_{ye} e_t = \lambda \]  

(3.24)

\[ U_y + U_{yy} Y_{t+1} + U_{1y} C_{t+1} + U_{2y} (1 - \delta) C_t + U_{gy} (X_{t+1} + H_{t+1}) + U_{ye} e_{t+1} = \lambda \]

(3.25)

\[ [U_{11} + U_{11} C_t + U_{12} (1 - \delta) C_{t-1} + U_{1g} (X_t + H_t) + U_{1e} e_t + U_{1e} e_{t+1}] \]

\[ + \beta [U_{21} (1 - \delta)^2 + U_{22} (1 - \delta)^2 C_t + U_{12} C_{t+1} (1 - \delta)^2 + U_{2g} (1 - \delta) (X_{t+1} + H_{t+1})] \]

\[ + U_{2e} (1 - \delta) Y_{t+1} + U_{2e} (1 - \delta) e_{t+1}] * (1 - \delta) = \lambda P_t \]

(3.26)

Corresponding with Fenn, solving equations (3.24) and (3.25) for \( Y_t \) and \( Y_{t+1} \)

respectively, give equations (3.27) and (3.28).

\[ Y_t = \frac{\lambda - U_y - U_{1y} C_t - U_{2y} (1 - \delta) C_{t-1} - U_{gy} (X_t + H_t) - U_{ye}}{U_{yy}} \]

(3.27)

\[ Y_{t+1} = \frac{\lambda - U_y - U_{1y} C_{t+1} - U_{2y} (1 - \delta) C_t - U_{gy} (X_{t+1} + H_{t+1}) - U_{ye}}{U_{yy}} \]

(3.28)

Next, keeping with Fenn, substituting equations (3.27) and (3.28) into equation (3.25) for \( Y_t \) and \( Y_{t+1} \), subsequently solving for \( C_t \) gives the following demand equation.

\[ C_t = \alpha_0 + \alpha_1 C_{t-1} + \alpha_2 C_{t+1} + \alpha_3 P_t + \alpha_4 X_t + \alpha_5 X_{t+1} + \alpha_6 H_t + \alpha_7 H_{t+1} + \alpha_8 e_t + \alpha_9 e_{t+1} \]

(3.29)

The coefficients in equation (3.29) are defined as:
\[\alpha_+ = \frac{1}{\Omega} \left[ -U_1 - \frac{U_{1y}(\lambda - U_y)}{U_{yy}} - \beta U_2(1 - \delta)^2 - \frac{\beta U_{2y}(1 - \delta)^2(\lambda - U_y)}{U_{yy}} \right] \] (3.30)

\[\alpha_1 = \frac{1}{\Omega} \left[ -U_{12}(1 - \delta) + \frac{U_{1y}U_{2y}(1 - \delta)}{U_{yy}} \right] \] (3.31)

\[\alpha_2 = \frac{1}{\Omega} \left[ -\beta U_{12}(1 - \delta)^2 + \frac{\beta U_{2y}U_{1y}(1 - \delta)^2}{U_{yy}} \right] \] (3.32)

\[\alpha_3 = \frac{1}{\Omega} \lambda \] (3.33)

\[\alpha_4 = \frac{1}{\Omega} \left[ -U_{1g} + \frac{U_{1y}U_{gy}}{U_{yy}} \right] X_i \] (3.34)

\[\alpha_5 = \frac{1}{\Omega} \left[ -\beta U_{2g}(1 - \delta)^2 + \frac{U_{2y}U_{gy}(1 - \delta)}{U_{yy}} \right] X_{i+1} \] (3.35)

\[\alpha_6 = \frac{1}{\Omega} \left[ -U_{1g} + \frac{U_{1y}U_{gy}}{U_{yy}} \right] H_i \] (3.36)

\[\alpha_7 = \frac{1}{\Omega} \left[ -\beta U_{2g}(1 - \delta)^2 + \frac{U_{2y}U_{gy}(1 - \delta)}{U_{yy}} \right] H_{i+1} \] (3.37)

\[\alpha_8 = \frac{1}{\Omega} \left[ \frac{U_{1y}U_{y\epsilon}}{U_{yy}} - U_{1\epsilon} \right] \epsilon_i \] (3.38)

\[\alpha_9 = \frac{1}{\Omega} \left[ \frac{\beta U_{2y}U_{y\epsilon}(1 - \delta)^2}{U_{yy}} - \beta U_{2\epsilon}(1 - \delta)^2 \right] \epsilon_{i+1} \] (3.39)

where,

\[\Omega = \left[ U_{11} - \frac{U_{2y}^2}{U_{yy}} + \beta U_{22}(1 - \delta)^3 - \frac{\beta U_{2y}^2(1 - \delta)^3}{U_{yy}} \right] \] (3.40)

In order to examine the implications each coefficient has on its respective variable it is necessary to examine their signs. Incorporating an inference used by Fenn and introduced by Chaloupka (1991), it is assumed that the level of the bulimic index and
the level of addictive stock do not affect the marginal utility of the composite good.\textsuperscript{16}

This is shown by the expression given in equation (3.41).

\[ U_{1y} = U_{2y} = 0 \]  

(3.41)

This paper also assumes that the level of the bulimic index and the level of addictive stock positively influence the marginal utility gained from one’s psychological disposition. This assumption corresponds with the notion that eating disorders reflect a form of self-medication for individuals who fit the theoretical model.\textsuperscript{17} Therefore, an increase in the bulimic index increases the marginal utility of an individual’s mental state \((X_t)\), and perception of family influences and dynamics \((H_t)\). This relationship is shown in equation (3.42).

\[ U_{1g} = U_{2g} > 0 \]  

(3.42)

**Influence of Past Bulimic Behavior on Current Bulimic Behavior**

Using equations (3.42) and (3.40) to simplify (3.31) gives:

\[
\alpha_i = \frac{-U_{12}(1 - \delta)}{U_{11} + \beta U_{22}(1 - \delta)} \geq 0
\]

(3.43)

The coefficient, \(\alpha_i\), on past consumption is positive. This relationship represents the most important concept of addictive behavior, the ability of a unit of last period’s consumption to increase this period’s consumption.\textsuperscript{18}

\textsuperscript{16} Ibid.


Influence of Future Bulimic Behavior on Current Bulimic Behavior

Using equations (3.42) and (3.40) to simplify (3.32) gives:

\[ \alpha_2 = \frac{(-\beta U_{12} (1 - \delta))^2}{U_{11} + \beta U_{22} (1 - \delta)^3} > 0 \]  
(3.44)

“In keeping with Becker et al., the model developed in this paper predicts that current and future consumption will be adjacent compliments.”\(^{19}\) Becker and consequently, Fenn, both assert that future consumption will have a positive impact on current consumption, and is seen by examining the positive coefficient on future consumption in equation (3.44) in this paper.

The Influence of Price on Present Consumption

Using equations (3.42) and (3.40) to simplify equation (3.33) gives:

\[ \alpha_3 = \frac{\lambda}{U_{11} + \beta U_{22} (1 - \delta)^3} < 0 \]  
(3.45)

The negative coefficient on price implies that the model predicts current bulimic behavior to decrease when the current price of bulimia increases. Since price represents events that incline an individual to perform bulimic behaviors, it must be assumed that the agent has been educated about risk factors that cause bulimia for this statement to be true. In other words, the agent is only rational if she is aware of the negative life events, such as a disconnected familial life, that trigger bulimic behavior for this statement to be true.

The Influence of Heredity and Emotionality on Present Consumption

Using equations (3.24) and (3.40) to simplify equations (3.34) and (3.36) yields:

\[^{19}\] Ibid.
Equation (3.46) gives the expression for the coefficients on both the variable for heredity and the vector variable for emotions. Equation (3.46) expresses the fact that the current bulimic index increases with an increase in both variables, and that heredity and emotionality have the same magnitude effect on current bulimia.

Using equations (3.24) and (3.40) to simplify equations (3.35) and (3.37) gives:

\[
\alpha_4 = \alpha_6 = \frac{U_{1g}^{(+)}}{U_{11}^{(-\delta)(-\delta)} + \beta U_{21}^{(1-\delta)}} > 0
\]

Equation (3.47) gives the expression for the coefficients on the variables for heredity and emotionality in the future period. This coefficient is analogous to Becker’s depiction of current and future consumption as “adjacent compliments.”

This concludes the theory section of this paper. The next section will introduce the data and the empirical model that is constructed from the theoretical model derived in this section.
CHAPTER IV
METHODOLOGY AND DATA

The purpose of this section is to introduce the data set that will be used to empirically test the theoretical model that was developed in the third chapter of this paper. The details and fundamental purpose of each explanatory variable within the data will be described, along with each one’s expected relationship with the regressor. After reviewing the data, the empirical model will be constructed and the methodology for testing the model will be described. The regression results of the empirical model are given in the following chapter.

Data and Sources

The data set that is used for the empirical analysis of bulimia nervosa as a behavioral addiction in this paper comes from the National, Heart, Lung and Blood Institute (NHLBI) Growth and Health Study (hereafter abbreviated NGHS). The idea to utilize this data set comes from a similar paper by Goeree et al. (2009), who used the data from the NGHS to study the addictive nature of bulimia. The primary objective of the NGHS was to determine whether black-white differences in the development of obesity in females during pubescence could be explained by differences in dietary habits, patterns of social activity, socioeconomic status, or psychosocial factors. The


2 Michelle S. Goeree, John C. Ham, and Daniela Iorio, Caught in the Bulimic Trap: Do Eating Disorders Reflect Addictive Behavior?, March 1, 2009.
NGHS was a 10-year longitudinal study that followed about 1,100 black girls and 1,100 white girls. The participants were between the ages of 9 and 10 at the first examination and were interviewed and examined at five stages within the duration of the study. Although this study was not intended to analyze bulimia nervosa, the data set contains information pertinent to the subject. The aforementioned EDI indices were measured for each girl at years 3, 5, 7, and 9. These measurements, in conjunction with other explanatory variables within the data will be used within this empirical analysis.

As discussed earlier, a primary component of analyzing the addiction process is to measure the influence of past behavior on current behavior. Therefore this process requires that each participant has a complete set of data for each variable within each year. Due to enormity and duration of this study, the data is fractured in areas. A major component that contributes to this is the difficulty of actually retrieving data from each participant at each of the collection intervals. This requires intense participant accountability, which the institution itself could not control. Therefore, the empirical analysis will not include each girl in the estimation, but since the sample size is so large, this should not significantly misguide the results.

Methodology

The empirical model is displayed in equation (4.1).

\[
bulim_{it} = \alpha_0 + \alpha_1 * bulim_{it-1} + \alpha_2 * bulim_{it-1} + \alpha_3 * f f e \epsilon_t + \alpha_4 * scbul_{it} + \alpha_5 * (bodydis_{it} + dr4thin_{it}) + \alpha_6 * (inef \ c_t + perf \ c_t) + \epsilon_{it}
\]

\[ (4.1) \]

Table (4.1) provides a description of each variable in equation (4.1), along with the mean, standard deviation, and value range of each variable.

**Table 4.1**

Variable Definitions and Descriptive Statistics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Description</th>
<th>Mean</th>
<th>Value Range</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>$bulim_i$</td>
<td>EDI bulimic index for individual $i$ at time $t$.</td>
<td>1.77</td>
<td>(0, 21)</td>
<td>3.066</td>
</tr>
<tr>
<td>$bulim_{i-1}$</td>
<td>EDI bulimic index for individual $i$ at time $t-1$.</td>
<td>1.86</td>
<td>(0, 21)</td>
<td>3.12</td>
</tr>
<tr>
<td>$bulim_{i+1}$</td>
<td>EDI bulimic index for individual $i$ at time $t+1$.</td>
<td>1.67</td>
<td>(0, 21)</td>
<td>2.96</td>
</tr>
<tr>
<td>$scbul_i$</td>
<td>EDI bulimic index for the parent of individual $i$.</td>
<td>.10</td>
<td>(0, 21)</td>
<td>2.10</td>
</tr>
<tr>
<td>$mom2fat_i$</td>
<td>Yes-Yes Q: “Has your mother ever told you that you are too fat?”</td>
<td>1.83</td>
<td>(1,3)</td>
<td>.38</td>
</tr>
<tr>
<td>$dad2fat_i$</td>
<td>Yes-Yes Q: “Has your father ever told you that you are too fat?”</td>
<td>1.93</td>
<td>(1,3)</td>
<td>.40</td>
</tr>
<tr>
<td>$ffeel_i$</td>
<td>Measure of how individual $i$ perceives her family’s cohesion at time $t$.</td>
<td>2.84</td>
<td>(0, 4)</td>
<td>.98</td>
</tr>
<tr>
<td>$bodydis_{it}$</td>
<td>EDI measure of body dissatisfaction for individual $i$ at time $t$.</td>
<td>8.44</td>
<td>(0, 27)</td>
<td>7.67</td>
</tr>
<tr>
<td>$dr\text{4thin}_{it}$</td>
<td>EDI measure of drive for thinness for individual $i$ at time $t$.</td>
<td>5.20</td>
<td>(0, 21)</td>
<td>5.90</td>
</tr>
<tr>
<td>$inefct_{it}$</td>
<td>EDI subscale that measures feelings of inadequacy and loss of control of individual $i$ ’s life at time $t$.</td>
<td>2.99</td>
<td>(0, 27)</td>
<td>4.18</td>
</tr>
<tr>
<td>$perfct_{it}$</td>
<td>EDI subscale that measures extreme personal expectations for superior success.</td>
<td>6.48</td>
<td>(0, 18)</td>
<td>3.30</td>
</tr>
</tbody>
</table>

**Measurement of Bulimia**

In order to model bulimia as a function of the specific risk factors, it is necessary to introduce a system for measuring the severity of bulimic behavior. Garner et al. developed a measure called the Eating Disorder Inventory (EDI) in their 1983 study. “The EDI is a multi-scale measure designed for the assessment of psychological and behavioral traits common in anorexia nervosa and bulimia”.

The EDI is made up of eight sub-scales:

---

4 Ibid.
1. Drive for Thinness: excessive concern with dieting and entrenchment in an extreme pursuit of thinness.


3. Body Dissatisfaction: reflects the belief that specific body parts that change shape and size at puberty are too large.

4. Ineffectiveness: assesses feelings of general inadequacy, insecurity, worthlessness and the feeling of not being in control of one’s life.

5. Perfectionism: indicates extreme personal expectations for superior success.

6. Interpersonal Distrust: indicates a sense of alienation and a general reluctance to form close relationships.

7. Interoceptive Awareness: reflects one’s lack of confidence in recognizing and accurately identifying emotions and sensations of hunger or satiety.

8. Maturity Fears: measures one’s wish to retreat to the security of the preadolescent years because of the overwhelming demands of adulthood.\(^5\)

The EDI is a survey-based questionnaire in which subjects respond to 64 six point items by rating whether each item applies “always,” “usually,” “often,” “sometimes,” “rarely,” or “never.” The scoring is such that the most extreme eating disordered response is “always” earning a score of 3. “Usually” earns 2 points, “often” earns 1

\(^5\) Ibid.
point, and the other three choices earn 0 points. Each personality index is equal to the sum of the points administered by the subject’s responses to the items that correspond to each subscale. The items that correspond to each of the EDI indices are listed below, along with their respective point scales.\(^6\)

1. **Drive For Thinness; 0-21**
   a. I eat sweets and carbohydrates without feeling nervous. *
   b. I think about dieting.
   c. I feel extremely guilty after overeating.
   d. I am terrified of gaining weight.
   e. I exaggerate or magnify the importance of weight.
   f. I am preoccupied with the desire to be thinner.
   g. If I gain a pound, I worry that I will keep gaining.

2. **Interoceptive Awareness; 0-30**
   a. I get frightened when my feelings are too strong.
   b. I get confused about what emotion I am feeling.
   c. I can clearly identify what emotion I’m feeling. *
   d. I don’t know what’s going on inside of me.
   e. I get confused as to whether or not I am hungry.
   f. I worry that my feelings will get out of control.
   g. I feel bloated after eating a small meal.
   h. When I am upset I don’t know if I am sad, frightened, or angry.
   i. I have feelings I can’t quite identify.
   j. When I am upset, I worry that I’ll start eating.

\(^6\) Ibid.
3. Bulimia; 0-21
   a. I eat when I am upset.
   b. I stuff myself with food.
   c. I have gone on eating binges where I have felt that I could not stop.
   d. I think about bingeing.
   e. I eat moderately in front of others and stuff myself when they’re gone.
   f. I have the thought of trying to vomit to lose weight.
   g. I eat or drink in secrecy.

4. Body dissatisfaction; 0-27
   a. I think that my stomach is too big.
   b. I think my thighs are too large.
   c. I think that my stomach is just the right size. *
   d. I feel satisfied with the shape of my body. *
   e. I like the shape of my buttocks. *
   f. I think my hips are too big.
   g. I think that my thighs are just the right size. *
   h. I think that my buttocks are too large.
   i. I think that my hips are just the right size. *

5. Ineffectiveness; 0-30
   a. I feel ineffective as a person.
   b. I feel alone in the world.
   c. I feel generally in control of the things in my life. *
   d. I wish I were someone else.
e. I feel inadequate.

f. I feel secure about myself. *

g. I have a low opinion of myself.

h. I feel that I can achieve my standards. *
i. I feel that I am a worthwhile person. *
j. I feel empty inside (emotionally).

6. Maturity Fears; 0-24

a. I wish that I could return to the security of childhood.

b. I wish that I could be younger.

c. The happiest time in life is when you are a child.

d. I would rather be an adult than a child. *

e. The demands of adulthood are too great.

f. I feel happy that I am not a child anymore. *

g. I feel that people are happiest when they are children.

h. The best years of your life are when you become an adult. *

7. Perfectionism; 0-18

a. Only outstanding performance is good enough in my family.

b. As a child, I tried very hard to avoid disappointing my parents and teachers.

c. I hate being less than best at things.

d. My parents have expected excellence from me.

e. I feel that I must do things perfectly or not do them at all.

f. I have extremely high goals.
8. Interpersonal Distrust; 0-21

a. I am open about my feelings. *

b. I trust others. *

c. I can communicate with others easily. *

d. I have close relationships. *

e. I have trouble expressing my emotions to others.

f. I need to keep people at a certain distance (feel uncomfortable if someone tries to get too close).

g. I can talk about personal thoughts or feelings. *

The items marked with an asterisk denotes a negatively keyed item so that the subject earns 3 points for “never”, 2 points for “rarely”, and 1 point for “sometimes”. This measure of eating disordered behavior was tested and confirmed as significant in Garner et al. (1983).

In summary, the model of bulimia developed in this chapter attempts to explicate the addictive nature of the disorder in two ways. First the model proposes that a mental health disorder precedes and fosters an addictive personality within an individual, but that certain risk factors lead a person to choose one substance or behavior over the others. In other words, addictions can co-occur or trade off from one to another. Second, the emotional symptoms induced by each of the specific risk factors i.e., abusive families, genetics, societal pressures join to channel the individual’s addictive nature towards bulimia.
**Dependent Variable**

The dependent variable in the empirical model will be the EDI bulimic index, (BULIM). As stated in chapter 3, the EDI is a survey-based questionnaire in which subjects respond to 64 six point items by rating whether each item applies “always,” “usually,” “often,” “sometimes,” “rarely,” or “never.” The scoring is such that the most extreme eating disordered response is “always” earning a score of 3. “Usually” earns 2 points, “often” earns 1 point, and the other three choices earn 0 points. The bulimic index is made up of 7 items; such that a girl can score between 0 and 21, where a higher score indicates more problems related to the eating disorder. According to Garner et al. (1983) a score higher than 10 signifies that a girl is very likely to have a clinical case of bulimia.\(^7\)

**Independent Variables**

The main objective of this model is to test whether or not bulimia could be considered a behavioral addiction, and if so, which exogenous factors affect its severity. The psychological literature supports the notion that addictions develop when an individual acquires an increasing dependence to a substance or behavior due to his or her growing tolerance to the amount of addictive stock within the body.\(^8\) This idea emphasizes the significance that past consumption of the addictive good or behavior has on the development of the addiction, and is analogous with Becker et al.’s inference that past consumption reinforces current consumption when behavior is addictive. They

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\(^7\) Michelle S. Goeree, John C. Ham, and Daniela Iorio, *Caught in the Bulimic Trap: Do Eating Disorders Reflect Addictive Behavior?*, March 1, 2009.

assert that a good is addictive if and only if an increase in past consumption leads to an increase in current consumption, holding all other variables fixed.\textsuperscript{9}

Therefore, past bulimic behavior, \((LBULIM)\), is the fundamental independent variable in this analysis, and will be represented by the EDI bulimic index in the prior period. The purpose of holding all other variables constant is to test for state-dependence between past bulimic behavior and current bulimic behavior, controlling for individual heterogeneity, which would convolute the results.

The psychological literature acknowledges a hereditary component of bulimia, and although it is unknown as to whether the disorder’s transmittance is predominantly attributed to genetics or to learned factors, as with many other addictions, familial history with the disorder is a risk factor.\textsuperscript{10} The empirical model will employ \((SCBUL)\), the mother’s bulimic index, as the primary variable for measuring heredity. In order to explore the complex nature of heredity, a regressor containing both genetic and peripheral/learned familial factors will be constructed using three different explanatory variables. The NGHS data set contains a variable signifying one or both of the parents’ EDI bulimic indices for most of the participants, denoted by \((SCBUL)\), which was estimated at only period during the study. In an effort to remain consistent with the female-based study, only the mother’s bulimic index will be included. Two variables, \((MOM2FAT)\) and \((DAD2FAT)\) were observed over two periods during the study. These two variables indicate whether a girl has ever been called fat by her mother or father,


where yes denotes a value of 1, and no is given a value of 2. This combination heredity variable will thus be expressed as \[ \text{scbul}^2 / (\text{mom2fat}_i \times \text{dad2fat}_i) \], where the square of the mother’s EDI bulimic index is divided by product of (MOM2FAT) and (DAD2FAT). The mother’s bulimic index is squared due to it relative importance compared to the denominator. (MOM2FAT) and (DAD2FAT) are implemented as a means to reinforce the explanatory power of the hereditary variable. Since (SCBUL) takes values 0-21, where 21 denotes the most severe case of bulimia, and (MOM2FAT) and (DAD2FAT) take the values 1 and 2, where the value 1 corresponds to the emotionally abusive household and a value of 2 indicates that the girl has never been called fat by either her mother or father, this variable should approach \((\text{SCBUL}^2)\) in the most severe case, and 0 in the absence of hereditary influence on bulimia. In other words, as the severity of genetic factors increase, holding the denominator constant, and as peripheral familial influences increases, (decreasing in numerical value), holding the numerator constant, the hereditary variable, \[ \text{scbul}^2 / (\text{mom2fat}_i \times \text{dad2fat}_i) \], will increase. This newly constructed variable represents the variable \( F_i \) from the theoretical model in equation (2.29), and is expected to have a positive relationship with the incidence of bulimia in the participant.

In the case of bulimia, the psychological risk factor, \( X_i \) from equation (2.29), can be divided into two underlying components, body-image specific and general emotional psychological factors. While the general psychological factors such as anxiety and depression, have been identified as primary risk factors common amongst girls with eating disorders, these psychological factors are also highly correlated with
other addictions.\textsuperscript{11} The body-image psychological factors delineate the risk factors to a prescribed theory for the case of this eating disorder. The distinction between these two components, that comprise $X_r$, is central to the assertion that bulimic behavior is addictive. For example, in the event that cross section panel data is observed beginning at the onset of bulimia, it might be inferred that the general psychological factors would explain a greater amount of the variance in bulimic behavior, but that over time, the involvement in bulimic behaviors would shift from a generalized form of escapism to a physical and emotional dependence. In this case, the magnitude of the body-image specific psychological factors would increase and explain a greater amount of the bulimic behavior. This example corresponds to the dual addiction theory discussed in chapter 3, in which a general psychological disorder can precede an addiction by 10 years, and more formally, that general psychological disorders set the stage for any addiction to manifest, but that specific risk factors guide a person to choose one substance or behavior over another.\textsuperscript{12}

Using the EDI subscales, two variables will be constructed to more efficiently represent the dual quality of the psychological risk factor, $X_r$. The subscales DR4THIN and BODYDIS assess attitudes and behaviors related to body and shape that are fundamental aspects of the psychopathology of bulimia.\textsuperscript{13} Together these regressors will specify the body-specific psychological factors that corroborate bulimia. INEFCT; the


\textsuperscript{12} Ibid.

\textsuperscript{13} David M. Garner, Marion P. Olmstead, and Janet Polivy, "Development and Validation of a Multidimensional Eating Disorder Inventory for Anorexia Nervosa and Bulimia.," International Journal of Eating Disorders 2, no. 2 (1983): 15-34.
subscale measuring general feelings inadequacy, insecurity, worthlessness and the feeling of being out of control, has also been described by some as the underlying disturbance of the eating disorder.\textsuperscript{14} PRFCT indicates the perusal of extremely high expectations, and manifested in the bulimic cycle through the unrealistic expectation of food restriction. Together, PERFCT and INEFCT will be combined within a vector variable to represent the primary emotional-physiological factors common amongst girls with eating disorders and individuals with other addictions. At the surface ineffectiveness and perfectionism seem to contrast with each other in a very basic sense; ineffectiveness connotes internal shame and feelings of failure, while perfectionism suggests that an individual is aware of her high level of capabilities and desires to project them. Here, the former implies and inward, private personality, while the former is often indicative of success and external associations.\textsuperscript{15} This private external paradox is very similar to other addictions.

These two variables will be constructed to represent the dual nature of $X_s$, which is expected to have a positive relationship with the dependent variable.

The demand function used for the empirical analysis will not contain the future variables for psychological $F_{s+1}$ due to irreparable flaws within the data set. $F_{s+1}$ will not be specified by the empirical model because the variable used to represent $F_s$, scbul, was only measured during one year for most of the participants.

The notion that addiction is rational in nature implies that individuals are forward looking, such that current knowledge about the future consequences of the

\textsuperscript{14} Ibid.

addictive behavior will alter their current decision-making process. In the case of cigarette addiction, Fenn et al. infer that cigarette consumption became rational in nature after the disclosure of the Surgeon General’s Warning in 1979. Fenn asserts that cigarette demand decreased in the post-information period, which enlightened the public to the negative health consequences of nicotine addiction. This is similar to the earlier model developed by Becker et al. in which the current knowledge of an increase in the future price of cigarettes is expected to decrease current demand. In both models, information and price are negatively correlated with demand for the addictive good.

The theoretical model for bulimia developed in chapter three introduced the idea that the independent variable price represents an exogenous circumstance that compels bulimic individuals to alter the frequency of bulimic behavior. Empirically, price is intended to implicitly represent an uncontrollable factor that has the capacity to alter bulimic behavior through its emotional impact on individuals. An increase in a specific factor, used to denote price, causes an individual to consciously choose to cope by engaging in increased bulimic behavior as a response to the emotional strain. Apfelbaum cites the act of bingeing and purging as a reaction to negative family structures, and that girls who grow up in fractured homes are more likely to develop bulimia. In keeping with the psychological literature, family feels close will denote the proxy for the independent variable for price. This variable, FFEEL is an index, ranging between 1 and 4 that measures the level at which a participant perceives her family’s cohesion.

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The price variable in this model is unfortunately subjected to the drawback of using existing micro-data; that variable choices are limited to the existing variables in the dataset, leading to inaccuracy and misspecification of the variable. While FFEEL is not the ideal proxy for price in this model, the intuition supporting its exogenous and negative impact on bulimic behaviors, cited by Apfelbaum, validates its use within the model. Furthermore, as with price in Becker’s model, if the agent has current knowledge that FFEEL will exogenously decrease in the next period, she will be more likely to increase current bulimic behavior.

*Endogeneity in past and future behavior*

The existing rational addiction literature addresses a complication with autocorrelation in the variables that represent past and future addictive behavior: “past consumption $C_{t-1}$ and future consumption $C_{t+1}$ depend on several past and future prices which are absent from the regression equation. Thus, $C_{t-1}$ and $C_{t+1}$ are not truly exogenous.”\(^{18}\) Fenn uses a two-stage least squares estimation procedure in order to alleviate the problem. The endogenous variables $C_{t-1}$ and $C_{t+1}$ were separately regressed on each truly exogenous variable within his empirical model, along with the variables $P_{t-1}$ and $P_{t+1}$. In the second stage of estimation he ran an OLS regression of his empirical model, substituting the actual values of $C_{t-1}$ and $C_{t+1}$ with the predicted values generated by the first stage of the process.

Fenn acquired the solution to this endogeneity problem from Becker et al. (1994), who also analyzed cigarette addiction. In analyzing addiction to bulimia, several underlying elements, intrinsic to the disorder and the NGHS data set, exist that make

\[^{18}\text{Aju J. Fenn, The Impact of Addiction Information on Cigarette Consumption, thesis, Iowa State University, 1998.}\]
Becker and Fenn’s solution to the endogeneity problem unusable. First, the price of bulimia in this model is more abstract than the fixed monetary value given to cigarettes. The price of bulimia is represented by an exogenous factor that has an emotional hold on an individual, and bulimic behavior only depends on this “price” once the addiction becomes rational through the acquisition of information. Since there is no variable for information within the data set, the actual dependence of each individual’s bulimic behavior on price is unknown. Second, the proxy used for price in this model ffeel, family feels close, is not an optimal choice for price, but is the only workable variable due limitations of the data. When regressed on bulimic behavior, ffeel is not significant. Intuitively, this means that endogeneity in past and future bulimic behavior is not attributable to ffeel.

This does not imply that past and future bulimic behavior are not endogenous, but that the root of endogeneity is much more complicated and convoluted than the monetary value given to cigarettes. Price may vary between individuals depending on access to information, individual heterogeneity, and different experiences with the disorder. Due to these obstacles, the strict exogeneity assumption will be more relaxed in this empirical model.

**Estimation Procedures**

A random effects tobit model will be employed to estimate the demand for bulimic behavior: “A tobit model is relevant when the dependent variable of a linear regression is observed only over some interval of its support. Consider the annual household expenditure on a durable item…A cross-section survey would reveal a significant portion of households with zero expenditure and the rest with positive
levels...the sample will be a mixture of observations with zero and positive values."^{19}

In this situation an ordinary least-squares regression will not provide reliable parameter estimates because the censored sample is not representative of the population. Because the NGHS was not specifically targeted towards the study of eating disorders, a majority of the observations contain zeroes; thus, a tobit model most appropriately fits the data.

The NGHS dataset is defined as panel data, or longitudinal data, which is characterized by repeated measurements of the same individual over several time intervals. Table 4.2 provides a section of the NGHS dataset, exhibiting its multidimensional panel structure.

Table 4.2
Example of Panel Data

<table>
<thead>
<tr>
<th>RID</th>
<th>Year</th>
<th>Bulim</th>
<th>Scbul</th>
<th>Dr4thin</th>
</tr>
</thead>
<tbody>
<tr>
<td>131</td>
<td>3</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>131</td>
<td>5</td>
<td>5</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>131</td>
<td>7</td>
<td>4</td>
<td>2</td>
<td>12</td>
</tr>
<tr>
<td>131</td>
<td>9</td>
<td>1</td>
<td>2</td>
<td>13</td>
</tr>
<tr>
<td>181</td>
<td>3</td>
<td>8</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>181</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

^{19} Adrian Colin, Cameron and P. K. Trivedi, *Microeconometrics Using Stata* (College Station, TX: Stata Press, 2009).
The variable RID identifies each girl in the study. The three explanatory variables shown in the table, bulim, scbul, and dr4thin, were collected at each checkpoint interval for each girl. This is defined as panel data.

Dependent variables have the potential to vary over time and individuals. The Stata Manual defines within variation as variation over time or a given individual, and between variation as variation across individuals. This distinction determines which type of individual-effects model fits the panel dataset. “The total variation (around grand mean $\bar{x} = 1/NT \sum_i x_i$) can be decomposed into within variation over time for each individual (around individual mean $\bar{x}_i = 1/T \sum_t x_{it}$) and between variation across individuals (for $\bar{x}$ around $\bar{x}_i$).”\(^{20}\) The decomposition for the variance is:

\begin{align*}
    s_w^2 &= \frac{1}{NT-1} \sum_t (x_{it} - \bar{x}_t)^2 \\
    &= \frac{1}{NT-1} \sum_t (x_{it} - \bar{x}_i + \bar{x} - \bar{x}_i)^2 \\
    &= \frac{1}{NT-1} \sum_t (x_{it} - \bar{x}_i + \bar{x})^2 \\
    &= \frac{1}{NT-1} \sum_t (x_{it} - \bar{x}_i)^2 + \frac{1}{NT-1} \sum_t (\bar{x}_i - \bar{x})^2 + \frac{2}{NT-1} \sum_t (x_{it} - \bar{x}_i)(\bar{x}_i - \bar{x}) \\
    &= s_w^2 + s_g^2 + \frac{2}{NT-1} \sum_t (x_{it} - \bar{x}_i)(\bar{x}_i - \bar{x}) \\
\end{align*}

\(^{20}\) Adrian Colin. Cameron and P. K. Trivedi, *Microeconometrics Using Stata* (College Station, TX: Stata Press, 2009).
\[ s_0^2 = \frac{1}{NT-1} \sum_i (x_{it} - \bar{x})^2 \]  

(4.5)

where equation (4.3) displays within variance, (4.4) is the equation for between variance, and (4.5) is the equation for overall variance.\(^{21}\)

The fixed-effects model permits regressors to be correlated with the time-invariant component of the error term, while continuing to assume that the regressors are uncorrelated with the idiosyncratic error. The random-effects model assumes that the both the time-invariant and idiosyncratic errors are uncorrelated with the regressors. In the FE model the coefficient of a regressor with little within variation will be imprecisely estimated; therefore an RE model is used in the presence of greater between variation. The NGHS data set exhibits greater between variance, shown in table (4.6), therefore a random-model will be employed.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Variance Type</th>
<th>Mean</th>
<th>Std. Dev.</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>lbulim:</td>
<td>overall</td>
<td>1.855944</td>
<td>3.12149</td>
<td>0</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td>between</td>
<td>2.515818</td>
<td></td>
<td>0</td>
<td>17.5</td>
</tr>
<tr>
<td></td>
<td>within</td>
<td>1.952164</td>
<td>-7.47738</td>
<td></td>
<td>13.85594</td>
</tr>
<tr>
<td>fbulim:</td>
<td>overall</td>
<td>1.665114</td>
<td>2.957624</td>
<td>0</td>
<td>21</td>
</tr>
</tbody>
</table>

21 Adrian Colin. Cameron and P. K. Trivedi, *Microeconometrics Using Stata* (College Station, TX: Stata Press, 2009).
<table>
<thead>
<tr>
<th></th>
<th>between</th>
<th>within</th>
<th>scbul: overall</th>
<th>0</th>
<th>121</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2.397563</td>
<td>1.858375</td>
<td>1.870709</td>
<td>7.699763</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-5.00155</td>
<td>2.546414</td>
<td>-54.12929</td>
<td>57.87</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.943214</td>
<td>0.8268682</td>
<td>2.772589</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>.6262437</td>
<td>2.772589</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>.5580271</td>
<td>.0948215</td>
<td>3.791606</td>
</tr>
<tr>
<td>Bodythin</td>
<td>overall</td>
<td>13.56985</td>
<td>12.52753</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td></td>
<td>between</td>
<td>10.5158</td>
<td>0</td>
<td>46</td>
<td></td>
</tr>
<tr>
<td></td>
<td>within</td>
<td>6.87535</td>
<td>-12.93015</td>
<td>41.31985</td>
<td></td>
</tr>
<tr>
<td>Perfin</td>
<td>overall</td>
<td>9.427198</td>
<td>5.742437</td>
<td>39</td>
<td></td>
</tr>
<tr>
<td></td>
<td>within</td>
<td>4.633491</td>
<td>1</td>
<td>31</td>
<td></td>
</tr>
<tr>
<td></td>
<td>between</td>
<td>3.611946</td>
<td>-9.572802</td>
<td>26.4272</td>
<td></td>
</tr>
</tbody>
</table>

The command for a random effects tobit model specified to fit panel data is denoted by `xttobit`. The random effects tobit model specifies the latent variable $y^*_a$ to depend on the explanatory variables, an idiosyncratic error and an individual error. The tobit model assumes zero as a censored observation, thus the latent demand for bulimic behavior is not expressed until some known constant threshold, denoted by $L$ is passed.\(^{22}\) This means that the unobserved dependent variable, $y^*_{a}$, is only observed when $y^*_{a} > L$, when $L = 0$. The general representation of this model is expressed in equation (4.7).

\(^{22}\) Adrian Colin. Cameron and P. K. Trivedi, *Microeconometrics Using Stata* (College Station, TX: Stata Press, 2009).
\[ y^*_\alpha = x_\alpha \beta + \alpha_i + \varepsilon_\alpha \]  

(4.7)

where, \( \alpha_i \sim N(0, \sigma_\alpha^2) \) and \( \varepsilon_i \sim N(0, \sigma_\varepsilon^2) \) and the vector \( x_\alpha \) includes an intercept.

For left censoring at \( L = 0 \), the \( y_\alpha \) variable is observed where,

\[
y_\alpha = \begin{cases} 
y^*_\alpha & \text{if} \quad y^*_\alpha > 0 \\
0 & \text{if} \quad y^*_\alpha \leq 0
\end{cases}
\]  

(4.8)

The next chapter will use the variable described in this chapter, combined with the theory outlined in Chapter 2, to test the empirical model and in turn, draw implications and conclusions from the results.
CHAPTER V

RESULTS AND CONCLUSIONS

The final chapter of this study will discuss the results produced by the Random-Effects Tobit regression of the empirical model presented in the previous chapter. Each of the significant variables will be described and interpreted in the context of the parameters implicit to the theoretical model that was derived in chapter 3. In addition, the potential for misspecification, due to violations of the model assumptions, will be addressed and argued against through the analysis of the robust standard errors.\(^1\) The next section of this chapter will examine the marginal effect of each explanatory variable on the EDI bulimic index, and analyze the mechanisms through which these influences are transmitted. The shortcomings of this study will be discussed, and suggestions for future research proposed. The final section will outline and develop any conclusions and potential policy implications that may be drawn from the results.

**Results: Random-Effects Tobit Model**

Table (5.1) summarizes the results from the regression analysis of the empirical model expressed in chapter 4 using the NGHS dataset.\(^2\) Two different models were

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estimated using the random-effect tobit regression, and each model was regressed a second time using its respective robust standard deviations. The z-statistics are expressed in parenthesis below each coefficient. Models 1 and 2 estimate demand for bulimic behavior using varying combinations of the independent variables. The code for running each regression in STATA is presented in APPENDIX A.

### TABLE 5.1

Random Effects Tobit Regression Results

The Explanatory Risk Factors of Bulimia Nervosa

<table>
<thead>
<tr>
<th>Variable</th>
<th>Definition</th>
<th>Model 1</th>
<th>1.Robust</th>
<th>Model 2</th>
<th>2.Robust</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBULIM</td>
<td>EDI Bulimic index in the previous period</td>
<td>.3593</td>
<td>.3593</td>
<td>.3802</td>
<td>.3802</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(7.81)*</td>
<td>(6.67)*</td>
<td>(8.53)*</td>
<td>(6.95)*</td>
</tr>
<tr>
<td>FBULIM</td>
<td>EDI Bulimic index in the future period</td>
<td>.4858</td>
<td>.4858</td>
<td>.5071</td>
<td>.5071</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(8.16)*</td>
<td>(9.89)*</td>
<td>(8.60)*</td>
<td>(8.26)*</td>
</tr>
<tr>
<td>SCBUL</td>
<td>Mother’s EDI Bulimic index</td>
<td>.1319</td>
<td>.1319</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(2.02)*</td>
<td>(2.70)*</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>(SCBUL^2)</td>
<td>Combination of learned</td>
<td>-</td>
<td>-</td>
<td>.0194</td>
<td>.0194</td>
</tr>
<tr>
<td>(SCBUL^2)/m2</td>
<td>Combination of learned and genetic hereditary factors</td>
<td>-</td>
<td>-</td>
<td>.0194</td>
<td>.0194</td>
</tr>
<tr>
<td>FFEEL</td>
<td>Measure of the agent’s perception of her family’s coherence.</td>
<td>.0438</td>
<td>.0438</td>
<td>.0115</td>
<td>.0115</td>
</tr>
<tr>
<td>BODYTHIN</td>
<td>Vector of two EDI indices: (drive for thinness + body dissatisfaction)</td>
<td>.0696</td>
<td>.0696</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>FBODYTHIN</td>
<td>BODYTHIN in the future period</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>PERFIN</td>
<td>Vector of two EDI indices: (perfectionism + ineffectiveness)</td>
<td>.1372</td>
<td>.1372</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Interaction between BODYTHIN and PERFIN</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>
Table 5.1 displays the results from the regressions of two congruent models, that each seek to construct a viable general theory for the addiction to Bulimia. The columns titled 1.Robust and 2.Robust display the results from models 1 and 2 after substituting standard error calculations with their respective robust standard errors. The RE Tobit model relies heavily on the assumption of homoskedastic normally distributed errors for consistency. In the case of OLS regression, a test such as White’s would be used to check for the existence of heteroskedastic errors which, if found, would be corrected for through various methods: “More generally, it can be difficult to generalize highly parameterized cross-section non-linear model. Stata does not provide panel commands in this case.”

The calculation for obtaining heteroskedastic-robust standard errors differs from the calculation of homosekedastic standard errors. Although the former equation yields accurate values of the standard errors for both homoskedastic and

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3 Adrian Colin. Cameron and P. K. Trivedi, *Microeconometrics Using Stata* (College Station, TX: Stata Press, 2009).
heteroskedastic data, a predominant amount of statistical software uses the calculation for homoskedastic standard errors as the default. 4 In the second set of regressions results, the value of the robust standard errors will likely be equal to the standard errors displayed in the results from the corresponding models, while the occurrence of hederoskedasticity will yield different robust and non-robust standard errors. Due to the fact that there is no formal test for heteroskedasticity in STATA, along with the fact that each sequence of programming codes attempted manually inputted into STATA yielded heteroskedasticity.

Model 1 corresponds to the analysis of the core, basic risk factors that cause bulimia, and represents the original empirical regression from equation (4.1). All of the explanatory variables in model 1 are significant at the 5% level with the exception of (FFEEL), using both types of standard deviation measurements. Sigma_u is an individual-specific error with the distribution, sigma_u = (α ~ N(0,σ^2)). In model 1, sigma_u is 0.00 but it is only statistically significant using the robust standard errors, which could indicate either heteroskedasticity or some form of serial correlation within each girl over time, which is often attributed to the use of lagged and lead transformations of the dependent variable. However, the random effects model permits intraclass correlation of the error, therefore the random effects model permits serial correlation in the model error. 5 This is likely to be the case in model 1, so it is likely that a slight form of heteroskedasticity is present.


5 Adrian Colin. Cameron and P. K. Trivedi, Microeconometrics Using Stata (College Station, TX: Stata Press, 2009). 247.
Model 2 meaningfully complicates the interpretation of the regression by testing the multidimensional composition of the risk variables. Model 2 replaces (SCBUL) with $[SCBUL^2/(MOM2FAT \times DAD2FAT)]$, which captures both the learned and genetic hereditary factors that corroborate the disorder. Additionally, (PERFIN) and (BODYTHIN) are combined to form the interaction variable (PERFIN*BODYTHIN), which elucidates the jointly significant nature of broad psychological disorders and bulimic specific psychological disorders. Here, all of the variables are significant at the 5% significance level, with the exception of (FFEEL) and $[SCBUL^2/(MOM2FAT \times DAD2FAT)]$. The WaldChi2 test statistic is positive and significant in Model 2 when both the standard errors and the robust standard errors are used, implying that the model fits the data in both instances. Contrary to model 1, the value of sigma_u is insignificant when the robust standard errors are implemented. The significance of sigma_u in the regression of model 1, using the robust standard errors, means that the individual specific error will contribute to 0% of the variance in the overall error term. This suggests that either all of the individual heterogeneity that can corroborate bulimia is explained through the explanatory variables in the model, or that autocorrelation exists between the lagged and lead variable of bulimia.

The interpretation of the coefficients, produced by the tobit model, differ from those generated by the traditional OLS regression, in which estimated marginal effects are directly given by estimates of the slope coefficients.\textsuperscript{6} Alternatively, the parameters produced by the tobit regression are interpreted as partial derivatives of the latent

\textsuperscript{6} Adrian Colin. Cameron and P. K. Trivedi, \textit{Microeconometrics Using Stata} (College Station, TX: Stata Press, 2009). 80.
variable \( y^* \) with respect to \( x \). The marginal effect is defined as the effect on the conditional mean of the dependent variable to changes in the explanatory variables.\(^7\) There are several types of marginal effects in the censored model, but this study is will focus on marginal effect on the expected value of \( y \) for the non-censored observations, or the truncated mean. Table (5.1) reveals that the use of the robust standard errors correspond to more accurate estimations of the standard deviation, therefore table (5.2) summarizes the marginal effects of the truncated mean, \( E(y|x, y > L) \), from the robust measurements of models 1 and 2.

\(^7\) Adrian Colin, Cameron and P. K. Trivedi, *Microeconometrics Using Stata* (College Station, TX: Stata Press, 2009). 527
Table 5.2
Marginal Effects on Bulimia

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\frac{\partial y}{\partial x}$</td>
<td>$\frac{\partial y}{\partial x}$</td>
</tr>
<tr>
<td>LBULIM</td>
<td>0.1176</td>
<td>0.1252</td>
</tr>
<tr>
<td></td>
<td>(6.64)*</td>
<td>(6.88)*</td>
</tr>
<tr>
<td>FBULIM</td>
<td>0.159</td>
<td>0.167</td>
</tr>
<tr>
<td></td>
<td>(9.47)*</td>
<td>(8.33)*</td>
</tr>
<tr>
<td>SCBUL</td>
<td>0.0432</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>(2.73)*</td>
<td>-</td>
</tr>
<tr>
<td>SCBUL^2/m2</td>
<td>-</td>
<td>0.0063</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>(1.25)</td>
</tr>
<tr>
<td>FFEEL</td>
<td>0.0143</td>
<td>0.0038</td>
</tr>
<tr>
<td></td>
<td>(0.36)</td>
<td>(0.08)</td>
</tr>
<tr>
<td>BODYTHIN</td>
<td>0.0228</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>(6.46)*</td>
<td>-</td>
</tr>
<tr>
<td>PERFIN</td>
<td>0.0449</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>(4.34)*</td>
<td>-</td>
</tr>
<tr>
<td>PB</td>
<td>-</td>
<td>0.0018</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>(7.03)*</td>
</tr>
<tr>
<td>$\bar{y}$</td>
<td>3.14</td>
<td>3.1509</td>
</tr>
</tbody>
</table>

Significant at 5% level
Psychological Effects

The marginal effects, $\partial y/\partial x$, correspond to the coefficients in table (5.1). Table (5.2) reveals that, with the exception of LBULIM and FBULIM, PERFIN, the emotional component of the psychological factor, has the greatest marginal impact on the dependent variable, BULIM; in which a unit increase in PERFIN leads to a 4.49% increase in the bulimic index. The positive influence of the emotional component of the psychological risk factor is analogous to the positive parameter, $\alpha_4$, in equation (2.46).

PERFIN is the sum of the EDI indicies for ineffectiveness and perfectionism, two psychological syndromes prescribed to the vast array of addictions. The private external paradox alluded to in chapter three draws symmetry between these variables through the mechanisms through which they interact to aid in the construction of addictive personalities. This relationship is particularly salient to the empirical analysis of addiction. Individually, a perfectionist attitude may yield positive benefits, but when corroborated by the disparaging feelings characterized by ineffectiveness, the extreme pursuit of achievement comes at the cost of the individual. Here, ineffectiveness can be understood as an apparatus through which a perfectionist attitude manifests to develop the propensity towards addiction.

BODYTHIN in model 1 is also positive and significant, reflecting the sign of $\alpha_4$ in equation (4.6), where a unit increase in the EDI indicies, body dissatisfaction and drive for thinness yield a 2.28% increase in the bulimic index. These two EDI indicies reflect the eating-disorder specific psychological disorders, and since this sample population is heavily skewed to the right, so that the proportion of girls affected by the
disorder is very small, further implications of this variable would be difficult to test in this dataset.

**Hereditary Effects**

In model 1 SCBUL is significant at the 5% significance level, where a unit increase in a mother’s EDI index increases her daughter’s bulimic index by 4.32%, which is analogous with the positive coefficient on $H_i$ in equation (4.6), reinforcing the accuracy of the model. In model 2, $(scbul^2 / (mom2f at^* dad2f at))$, reflects an attempt to estimate the dual quality of the hereditary risk factor by combining the genetic component with its learned counterpart. This variable is insignificant, most likely due to its poor construction.

**Price**

The variable chosen to represent price in this model, FFEEL, is extremely insignificant with a p-value of .93. FFEEL was chosen as the proxy for price due to its exogenous and inverse relationship to bulimic behavior. This variable is intended to measure an independent variable that exogenously impacts the severity of bulimic behavior. Duemm et al. (2003), find that internalized social pressures lead to self-perceived body dissatisfaction through an exogenous pathway so that, controlling for individual heterogeneity, the increase of bulimic behavior due to social pressures is exogenous. Due to limited variables within the NGHS data set, price is poorly represented, but is still expected to be a significant determinant when accurately represented.

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Past and Future Bulimic Behavior

Past bulimic behavior is positive and significant in both models, where a unit increase in LBULIM leads to an 11.74% increase in current bulimic behavior in model 1, and a unit increase in LBULIM in model 2 leads to a 12.52% increase in current bulimic behavior. This difference is most likely due to the fact that model 2 contains more insignificant and potentially misspecified variables so that the additional variance in the dependent variable is attributed to LBULIM. The impact of lagged bulimic behavior on current bulimic behavior has the greatest influence in determining whether this eating disorder can be characterized by addiction. Persistence in bulimic behavior over time, accounting for individual heterogeneity, can be attributed to true state dependence.\textsuperscript{9} Addictive behaviors are unique in the sense that persistence is attributed to the phenomena of tolerance, in which agents must increase levels of behavior or consumption in order to gain consistent levels of utility over time, and where the marginal utility gained from an increased unit of behavior is positive but decreasing. The parameters on lagged bulimic behavior in the empirical model validate this notion. Because the estimated parameters measure the percentage that BULIM changes with respect to a unit increase in each explanatory variable, as the value of BULIM increases, the affect of a unit change in lagged bulimic behavior causes current behavior to increase at an increasing rate. The empirical results from this regression support the positive influence of past behavior on current behavior, and in turn, support the notion that bulimia is addictive in nature.

\textsuperscript{9} Michelle S. Goeree, John C. Ham, and Daniela Iorio, \textit{Caught in the Bulimic Trap: Do Eating Disorders Reflect Addictive Behavior?}, March 1, 2009. 18.
Future bulimic behavior is positive and significant in both models as well, where model 1 reveals a 15.9% increase in current behavior in lieu of a one unit increase in future behavior, and a 16.7% increase in present behavior due to a one unit increase in future bulimia in the second model. The positive and significant coefficients on FBULIM in both models are analogous to the positive parameter sign indicated by the theoretical model in chapter 3.

Overall, the parameter estimations produced by the regression of the empirical model support the hypothesis that bulimia is addictive in nature and reinforces the theory developed in chapter two, that most addictions originate out of a general psychological ailment and that specific risk factors drive the individual to developing a specific addiction. Furthermore, the multidimensional construction of the psychological and hereditary variables complicates the interpretation of the variables due to the intricate composition of human emotions.

Future Research

The primary shortcomings of this study could be attributed to the nature of the data set used. While the NGHS data set contains a multitude of useful variables, the purpose of the study was directed towards the identification of childhood risk factors in the development and persistence with obesity. It would be favorable for future researchers to collect their own data so that the sample population would reflect a more balanced portion of study targets. Also the identification of a “price” variable for bulimia would contribute to the current research in a very meaningful way. As Fenn (1998) analyzed, addiction does not become rational until the emergence of information pertinent to the future negative implications of the addiction are understood.
The classification of bulimia as an addiction rather than as a disorder could influence schools to teach girls and boys about long-term effects eating disorders inflict upon the lives of individuals affected by them. Goeree (2009) expounded upon the fact that the longer an individual struggles with bulimia, the harder it will be for her to overcome it.\textsuperscript{10} The acceptance and acknowledgment of Bulimia Nervosa as a behavioral addiction would benefit medical policy though the creation of preventative care programs. This paper finds that bulimia is addictive through the persistence of bulimic behavior over time, and is fortified by the mental health disorders and environmental pressures that inadvertently condone this addiction by disregarding magnitude and prevalence.

\textsuperscript{10} Michelle S. Goeree, John C. Ham, and Daniela Iorio, \textit{Caught in the Bulimic Trap: Do Eating Disorders Reflect Addictive Behavior?}, March 1, 2009.
APPENDIX A

STATA Code for Random-Effects Tobit Regression

The purpose of this appendix is to reveal the codes entered for the four models included in this paper.

1. REGRESSION
   xttobit bulim lbulim fbulim scbul ffeel bodythin perfin, ll(0)
   predict yhat
   summarize yhat
   mfx compute, predict(e(0,.))

2. REGRESSION (Boot)
   xttobit bulim lbulim fbulim scbul ffeel bodythin perfin, ll(0) vce(boot)
   predict yhatboot
   summarize yhatboot
   mfx compute, predict(e(0,.))

3. REGRESSION (INTERACTIONS)
   xttobit bulim lbulim fbulim par2 ffeel bodythin bp, ll(0)
   predict yhatint
   summarize yhatint
   mfx compute, predict(e(0,.))

4. REG INT (BOOT)
   xttobit bulim lbulim fbulim par2 ffeel bp, ll(0) vce(boot)
   predict yhatintboot
   summarize yhatintboot
   mfx compute, predict(e(0,.))
WORKS CITED


http://www2.econ.iastate.edu/classes/econ371/yerokhin/.


