

Being in the Grip of Bulimia: New Evidence on How Bulimia Relates to Addictive Behavior

Daniela Iorio and Michelle Sovinsky

December 12, 2013

Abstract

Using longitudinal data that tracks bulimic behavior among young girls (National Heart, Lung, and Blood Institute Growth and Health Study) from 1988 until 1997 in the United States, we examine (1) whether the persistence in bulimia nervosa (BN) can be attributed to slow learning about the deleterious health effects of BN or if it reflects tolerance formed from an addiction; and 2) whether bulimic behavior is consistent with addiction criteria as stated in the Diagnostic and Statistical Manual of Mental Disorders DSM-IV. To address the endogeneity of the past behavior, we use instrumental variables techniques, and show that the past four years of behavior positively and significantly impacts current behavior when controlling for individual heterogeneity. We also find that accounting for the “stock” of bulimic behavior are not negative, which casts doubt on the importance of learning. Making the case for treating BN as an addiction has important policy implications. First, it suggests that the timing of educational policy and treatment is crucial: preventive educational programs aimed at instructing girls about the deleterious health effects of BN, as well as treatment interventions, will be most effective if provided in the early stages. Second, it would put those exhibiting BN on more equal footing (from a treatment reimbursement perspective) with individuals with drug or alcohol addictions.

1 Introduction

Eating disorders (ED) are a growing health concern. Estimates from the National Eating Disorders Association indicate that as many as 20 million women in the US are battling with an ED (NEDA, 2012). It is estimated that up to 4% of females in the United States will engage in the bingeing/purging form of an ED, bulimia (BN) during their lifetime (NIMH, 2002). They typically start when they are teenagers. However, the onset age appears to be dropping, where the behavior is increasingly seen among children as young as 10 (Cavanaugh and Ray, 1999).

Bulimia is especially serious given that a primary characteristic is the increasingly compulsive nature of the behavior. Individuals suffering from BN report requiring more of the behavior to produce the same effect, parallel to the behavior associated with drug addictions. Also, it is well-documented that addicts exhibit higher BN prevalence rates relative to non-addicts (Bulik, et al. 1992). These findings suggest that there may be an addictive component to BN. On the

other hand, a common set of traits may predispose an individual to excessive behaviors. That is, some individuals may have strong (unobservable) tastes for bingeing and purging which are persistent over time or evolve slowly. As a result, the propensity to engage repeatedly in bulimic activities may arise solely from differences across individuals (individual heterogeneity) and/or could be driven by (true) state dependence, which is consistent with the potentially addictive nature of BN (i.e., a randomly chosen person becomes chemically/ biologically addicted to the process over time if they binge and purge now). Previous studies overlooked the relative importance of state dependence versus individual heterogeneity in explaining BN persistence. A notable exception is Ham et al. (2013), which is the first work to document that unobserved heterogeneity plays a role in the persistence of BN, but strikingly up to two-thirds of BN persistence is due to true state dependence (i.e., the causal effect of lagged BN). Having established robust evidence of state dependence in BN in Ham et al. (2013), the purpose of this work is now to use modern econometric techniques to examine the addictive nature of BN. Specifically, we examine whether persistent behavior can be attributed to tolerance or to slow learning about the deleterious health effects of BN.

In order to draw a quantitative link between addiction and state dependence, we use the National Heart, Lung, and Blood Institute Growth and Health Study (henceforth NGHS). A notable aspect of the data is that for each respondent it contains information about an Eating Disorders Inventory index developed by a panel of medical experts, which was designed to assess the psychological characteristics relevant to bulimia (Garner et al., 1983), and a number of indices that measure a respondent's potential for personality traits/disorders that are highly correlated with BN, such as tendencies toward perfectionism, feelings of ineffectiveness, and interpersonal distrust (Garner et al., 1983).

We exploit these longitudinal data on individuals' history of bulimic behavior to estimate a dynamic model where the past behavior may affect the current tendency towards BN. As in any dynamic model, we first need to address the endogeneity of the past behavior. To this end, we consider the lags of the personality traits/disorders as instruments for past BN, and use instrumental variables techniques to examine whether persistent behavior can be attributed to tolerance or to slow learning about the deleterious health effects of BN. Our results cast doubt on slow learning as a driving force in state dependence. We first show that the past four years of behavior positively and significantly impacts current behavior when controlling for individual heterogeneity. We also find that accounting for the "stock" of bulimic behavior are not negative, which casts doubt on the importance of learning. Finally, we link our findings to the Diagnostic and Statistical Manual of Mental Disorders DSM-IV (APA, 1994) addiction criteria, and combine our results with other evidence in the medical literature to make the case

that BN should be considered an addiction.

Treating BN as an addiction has important policy implications. First, if BN reflects an addictive component, policy timing is crucial and policy interventions will be more effective if provided in the early stages. Second, making the case for BN as an addiction would put those exhibiting BN on more equal footing (from a treatment reimbursement perspective) with individuals abusing drugs or alcohol. Recent estimates show that only 6% bulimics receive mental health care (Hoek and van Hoeken, 2003), while a majority of states cover treatment for alcohol and drug addiction (Robinson, et al. 2006). Daly (2008) found that typical coverage by insurance companies for EDs failed to provide adequate reimbursement for the most basic treatment as recommended by the American Psychiatric Association.

2 Data

We use longitudinal data collected in the NGHS for 2,379 girls starting in 1988, when the girls were aged 9-10. The same cohort was interviewed once a year until 1997. The follow-up rate was 89% after ten years.

The respondents were from schools in Richmond, California and Cincinnati, Ohio, and from families enrolled in a health maintenance organization (HMO) in the Washington, DC area. Schools were selected based on census tracts containing approximately equal fractions of African American and White children with the least disparity in income and education between the two groups. The majority of the cohort was randomly drawn from families with nine (or ten) year-old girls that participated in the HMO. A small percentage was recruited from a Girl Scout troop located in the same geographical area as the HMO population.

Bulimia Subscale Starting when the girls were aged 11-12, every other year the NGHS included questions about BN (refined in Striegel-Moore et al. (2000) to be easily understood by young respondents). These include 1) I eat when I am upset; 2) I stuff myself with food; 3) I have gone on eating binges where I felt that I could not stop; 4) I think about bingeing (overeating); 5) I eat moderately in front of others and stuff myself when they are gone; 6) I have the thought of trying to vomit in order to lose weight, and 7) I eat or drink in secrecy. The responses (“always”=1, “usually”=2, “often”=3, “sometimes”=4, “rarely”=5, and “never”=6) were used to construct an Eating Disorders Inventory Bulimia subscale (hereafter the ED-BN index). A response of 4-6 contributes zero points to the ED-BN index; a response of 3 contributes 1 point; 2 contributes 2 points; and 1 contributes 3 points. The ED-BN index is the sum of the contributing points. A value greater than 10 indicates clinical bulimia. Note

that the answers to the individual questions are not available in the data. See Garner et al. (1983) for details. The index is widely used to assess the psychological traits relevant to bulimia (Rush, et al. 2008). As Table 1 indicates, the mean ED-BN index is 1.2.

Personality Traits/Disorders The NGHS also contains questions used to measure personality traits/disorders, such as tendencies toward: perfectionism (the perfectionism index), feelings of ineffectiveness (the ineffectiveness index), and interpersonal distrust (the distrust index). We provide details on the underlying questions in Ham et al. (2013).

The correlations between the ED-BN Index with the perfectionism, ineffectiveness, and distrust indices are 0.23, 0.44, and 0.21, respectively. These are consistent with Pearlstein (2002) who reports several personality traits that increase risk for BN and with papers in the medical literature that document the high comorbidity of BN with personality characteristics (e.g., Bulik, et al. 2003, Lilenfeld, et al. 1998).

Demographic Variables Demographic traits include parental (or guardian) educational attainment (in brackets), household income (in brackets), age, and race (White or African American). The survey is an exogenously stratified sample, designed to be equally distributed across race, income, and parent’s education as the descriptive statistics in Table 1 confirm.

3 Empirical Strategy

We regress the ED-BN index, y_{it} , on a one-period lag (y_{it-1}) and demographic characteristics and personality traits (X_{it}). There may be an unobserved individual component that is persistent over time, which is denoted by δ_i . The v_{it} is a normally distributed shock that accounts for non-observable time changing factors. Specifically,

$$y_{it} = \beta_0 + \beta_1 y_{it-1} + \beta_2 X_{it} + \delta_i + v_{it}. \quad (1)$$

As in any dynamic model, we need to address the endogeneity of lagged behavior. In order to obtain a consistent estimate of β_1 we use an instrumental variables approach, namely the Two-Stage-Least-Squares (henceforth, 2SLS) approach. In words, we use the time changing portion of the personality traits X_{it-1} as excluded instrumental variables to obtain an estimate of β_1 that reflects only state dependence. To test if the personality traits are valid instruments, we consider two diagnostics: i) a test statistic for weak instruments; and ii) a Wald statistic to test the overidentifying restrictions that the instruments are valid. In calculating the former test, we take into account the fact that there will be heteroskedasticity in the first-stage regression equation for a censored dependent variable, and we use the conjecture by Hansen, Hausman,

and Newey (2008) that in the presence of heteroskedasticity, the Wald statistic for the null hypothesis that the excluded instruments are zero in the first stage, minus the number of instruments, should be greater than 32. In diagnostic ii), the critical value is $Chi2(l)$; where l is the degree of overidentification. Intuitively the test can be thought of as assuming that one of the instruments is valid, and then seeing whether the other instruments have a zero coefficient in the structural equation.

Both the diagnostics show that our instruments are not weak, and the overidentifying restrictions are not rejected. We refer the reader to Ham et al. (2013) for a thorough discussion of the methodology and tests.

We should note that our sample is limited by the fact that the personality indices are not available in wave 7. However, we can double our sample size if we assume that the personality indices values vary smoothly from waves 5 to 9, and use interpolated values in wave 7. We present the 2SLS estimates using the imputed data.

4 Results and Discussion

In this section, we examine the potential addictive nature of BN, and document a number of aspects of BN behavior that are consistent with it. According to the DSM-IV, a behavior (or use of a substance) is an addiction if it satisfies at least three of seven criteria in a given year: 1) experiencing a persistent desire for or an inability to reduce or control the behavior, 2) the behavior continuing despite known adverse consequences, 3) withdrawal, 4) tolerance (more is needed for the same effect), 5) engaging in the behavior for a longer period than was intended, 6) spending much time seeking or recovering from its effects, and 7) the behavior interfering with important activities.

It is straightforward to note that BN fulfils criterion 1 (inability to control its use) as one of the diagnostic criteria for BN involves loss of control over the eating process. Regarding criterion 2, Ham et al. (2013) document that young women persist in BN. More specifically, we first considered a model where the only explanatory variable is the (assumed to be exogenous) lagged dependent variable; its coefficient is estimated at 0.44 and, not surprisingly, it is very statistically significant. Regarding the effect of past ED-BN experience on current behavior, the coefficient can be interpreted as an elasticity since we would expect the mean of a variable and its lag to be equal, so we obtained a relatively large estimate of the elasticity of 0.44. After we added the controls X_{it} and we treated the lagged dependent variable as endogenous (employing the instrumental variable approach outlined above), we estimated an elasticity of 0.149, which

indicates that up to two-thirds of the variation in the persistence can be attributed to state dependence. Due to data limitations we are not able to determine if the respondents are aware of the negative consequences of their behavior, however a number of the adverse health effects will be readily apparent, such as an irritated esophagus, tooth decay, muscle weakness, gastric rupture, and anemia. In this sense the continued behavior is consistent with addiction criterion 2 (i.e., use continues despite known adverse consequences). There is separate scientific evidence of withdrawal (criterion 3) in laxative use, which is a purging behavior (Colton, et al. 1998). We now provide empirical evidence in favor of criterion 4 (tolerance), using the longitudinal data and econometric techniques described above.

Note that the presence of state dependence is necessary for BN to fulfill the tolerance criterion, but it is not sufficient. In fact, there may be competing explanations that generate state dependence, but that do not involve tolerance or increased use over time. For instance, it may be that individuals are initially uncertain of the deleterious side effects associated with bulimia, but they slowly learn through experimentation that BN is harmful. The slow learning explanation for state dependence has the implication that the longer individuals have experienced bulimic behavior the less likely they are to experience it in the future. We now explore the potential for the slow learning explanation, and test whether we can rule it out. We first consider an AR(2) process, that is both y_{it-1} and y_{it-2} matter in explaining y_{it} , and then construct an “intensity” stock variable that is the sum of the ED-BN index over all previous periods. We also consider a “threshold” stock in which past behavior contributes to the stock only if the girl engaged in more intense BN behavior in the past (defined as a value of the ED-BN greater than 6). The threshold stock reflects the idea that a person learns the harmful consequences of BN only when the intensity of the past behavior is relatively high. Note that while such stock measures could be problematic in samples with older individuals (as earlier behavior would be out of sample and thus unobserved), this is not an issue in our sample since the girls are quite young when first interviewed. The results in Table 2 provide strong evidence against the slow learning interpretation. All results are based on 2SLS estimation where we treat the lagged ED-BN index as endogenous and include demographics and personality indices.

For comparison purposes, in the first column we repeat the results from Ham et al. (2013), which include only the first lag of ED-BN index. Column (2) specifies an AR(2) process where one and two lags of the personality indices are used as instrumental variables. Column (3) includes one lag of the ED-BN index and the intensity stock, while column (4) replaces the intensity stock with the threshold stock. In columns (3) and (4) we use the lag and the sum over all previous waves of each personality index as instrumental variables.

Our results in column (2) show that the first and second lag coefficients (recall that each

lag is two years) are both statistically significant and equal to 0.12 and 0.11, respectively. These results cast doubt on slow learning as a driving force in state dependence, as the latter suggests that experiencing BN for four years would most likely reduce current behavior. Further evidence against the learning interpretation comes from columns (3) and (4). If learning was important we would expect the coefficients on the stock variables to be negative and statistically significant, but instead they are both positive and insignificant. Thus we conclude that slow learning does not explain state dependence in BN persistence. These findings corroborate our hypothesis that it is tolerance that explains state dependence.

In summary we argue that BN fulfills at least three of the DSM-IV criteria necessary to be classified as an addiction, which provides a basis for BN to be classified as an addiction. Corwin and Grigson (2009) note that other diagnostic criteria for bingeing related disorders approximate the DSM-IV criteria for addiction. These include binge-type consumption, (i.e., criterion 5); bingeing is followed by inappropriate compensatory behavior (i.e., criterion 2); bingeing occurs at least twice a week for 3 months (i.e., criterion 5). Their argument is not based on an empirical analysis, but rather on the relation between the DSM-IV addiction and BN criteria.

Our case can be made even stronger by noting that BN presents important similarities to drug and alcohol abuse. First, individuals suffering from ED report requiring more of the behavior to produce the same effect, parallel to the behavior associated with drug or alcohol addictions. Medical research has found that the auto-addiction-opioid theory posits that ED is an addiction to the body's production of opioids (see Vandereycken 2006 for a survey). Starving, bingeing, purging, and exercise increase the body's β -endorphin levels resulting in the same chemical effect as that delivered by exogenous opiates. Medical research provides further support of this hypothesis. For instance, Bencherif et al. (2005) finds that opioid receptor binding in the area of the brain involving the anticipation and reward of eating in bulimic women is lower than in healthy women, and this reaction has been found in other studies of addictive behavioral disorders, including drug addiction and gambling. Second, patients with BN seem to respond to treatment initially aimed at combatting drug and alcohol abuse. For example, Naltrexone, an anti-addiction opioid antagonist normally used in the treatment of alcohol dependence, has shown signs of success in normalizing eating patterns in those suffering from anorexia and bulimia (Marrazzi 1995).

These results suggest directions for policy aimed at combating BN. Our results strongly suggest that BN should be treated as an addiction. This is important in the sense that we argue those exhibiting BN should be treated in an analogous way (from a treatment reimbursement perspective) to those individuals abusing drugs or alcohol. In some states this is a current policy

issue, and in the majority of the states treatment for alcoholism and drug addiction is covered whereas treatment for ED is not covered in as many states (Center for Mental Health Services Report on State Parity Laws, 2008), and when available the coverage can be inadequate.

References

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (4th edition) (DSM-IV)*. Washington, D. C.: APA.
- Arellano, M. & Bond, S. (1991). Some Tests of Specification for Panel Data: Monte Carlo Evidence and an Application to Employment Equations. *Review of Economic Studies* 58, 277-97.
- Bencherif B., A.S. Guarda, C. Colantuoni, H. Ravert, R. Dannals, and J. Frost (2005) "Regional Opioid Receptor Binding in Insular Cortex is Decreased in Bulimia Nervosa and Correlates Inversely with Fasting Behavior" *The Journal of Nuclear Medicine*, 46: 1349-1351.
- Bulik, C., Devlin B., & Bacanu S. (2003). Significant Linkage on Chromosome 10p in Families with Bulimia Nervosa. *Am J Med Genet* 72, 200-207.
- Bulik, C. M., Sullivan, P. F., Epstein, L. H., McKee, M., Kaye, W. H., Dahl, R. E., et al. (1992). Drug use in women with anorexia and bulimia nervosa. *Int J Eat Disord*, 11(3), 213-225.
- Cavanaugh, C., & L. Ray (1999) "What we Know about Eating Disorders: Facts and Statistics" In Lemberg, Raymond and Cohn, Leigh (Eds) *Eating Disorders: A Reference Sourcebook*. Oryx. Press. Phoenix, AZ.
- Colton, P., Woodside D., & Kaplan A. (1998). Laxative Withdrawal in Eating Disorders: Treatment Protocol and 3 to 20-Month Follow-Up. *Int J Eat Disord* 25(3), 311-317.
- Corwin, R. & Grigson, P. (2009). Food Addiction: Fact or Fiction? *J Nutr* 139(3), 617-619.
- Daly, R. (2008). Few States Act on Parity Improvements This Year. *Psychiatr News* 43, 2-25.
- Department of Health & Human Services (2006). Agency for Healthcare Research and Quality, Management of Eating Disorders, Evidence Report/Technology Assessment, Number 135, 2006. AHRQ publication 06-E010. Senate Report accompanying the 2006 budget
- Garner, D., Marion P., Olmstead M., & Polivy J. (1983). Development and Validation of a Multidimensional Eating Disorder Inventory for Anorexia Nervosa and Bulimia. *Int J Eat Disord* 2, 15-34.

Ham, J., D. Iorio, and M. Sovinsky (2013) "Caught in the Bulimic Trap? Persistence and State Dependence of Bulimia Among Young Women," *Journal of Human Resources*. 48(3):736-767.

Hansen, C., J. Hausman and W. Newey (2008) "Estimation with Many Instrumental Variables" *Journal of Business and Economic Statistics* 26(4): 398-422.

Hoek, H.W., & van Hoeken, D. (2003). Review of the prevalence and incidence of eating disorders. *Int J Eat Disord* 383-396.

House of Representatives (2008). 6983, 110th Congress, Paul Wellstone and Pete Domenici Mental Health Parity and Addiction Equity Act of 2008

Lilenfeld, L., Kaye W., & Greeno C. (1998). A Controlled Family Study of Restricting Anorexia and Bulimia Nervosa: Comorbidity in Proband and Disorders in First-Degree Relatives. *Arch Gen Psychiatry* 55, 603-10.

Marrazzi, M. JP Bacon, J Kinzie, ED Luby (1995) "Naltrexone use in the treatment of anorexia nervosa and bulimia nervosa," *International Clinical Psychopharmacology*, 10, 163 -172.

National Eating Disorders Association (2012) URL: <http://www.nationaleatingdisorders.org/sites/default/files/ResourceHandouts/GeneralStatistics.pdf>. Accessed Oct 29, 2013.

National Heart, Lung, & Blood Institute (NHLBI) Growth and Health Study Research Group. (1992). Obesity and cardiovascular disease risk factors in black and white girls: The NHLBI Growth and Health Study. *Am J Public Health*, 82, 1613–1621.

NIMH (2002) The National Institute of Mental Health: Eating Disorders: Facts About Eating Disorders and the Search for Solutions. Pub No. 01-4901. Accessed Feb. 2002. <http://www.nimh.nih.gov/publicat/nedspdisorder.cfm>.)

Pearlstein, T. (2002). Eating disorders and comorbidity." *Arch Womens Ment Health* 4,67-78.

Reas, D., D.Williamson, C. Martin, and N. Zucker (2000) "Duration of Illness Predicts Outcome for Bulimia Nervosa: A Long-term Follow-up Study" *International Journal of Eating Disorders*, 27: 428-34.

Robinson, G., Connolly, J., Whitter, M., & Magaña, C. (2006). State Mandates for Treatment for Mental Illness and Substance Use Disorders (DHHS Pub. No. (SMA) 07-4228). Rockville, MD: Center for Mental Health Services, Substance Abuse and Mental Health Services Administration.

Rush, J., First M., & Blacker D. (2008). Handbook of Psychiatric Measures American Psychiatric Publishing: Arlington, VA, 2nd Edition.

Striegel-Moore, R., Schreiber G., Lo A., Crawford P., Obarzanek E., & Rodin J. (2000). Eating Disorder Symptoms in a Cohort of 11 to 16-Year-Old Black and White Girls: The NHLBI Growth and Health Study. *Int J Eat Disord* 27, 49-66.

Vandereycken, W. (2006) "The Addiction Model in Eating Disorders: Some Critical Remarks and a Selected Bibliography" *International Journal of Eating Disorders* 9(1): 95 - 101.