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Through the looking glass

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Chapter 1

Introduction

This thesis aims to explore several aspects of the epidemiology of eating disorders in primary care and the community, with an emphasis on the effects of changed diagnostic criteria, a changing sociocultural environment over time, and the impact of self-perceived and peer-perceived social status on the occurrence of eating disorders. The following paragraphs provide a background to the topics and research questions addressed in this thesis.

EATING DISORDERS

Eating disorders are severe mental health problems with detrimental consequences for physical and psychosocial health.^{1,2} In a meta-analysis of excess mortality in the 1990s,³ the eating disorder anorexia nervosa (AN) was associated with the highest rate of mortality among all mental disorders; according to a recent meta-analysis, one out of twenty AN patients will have died after a decade.⁴

The core pathology of the most well-known eating disorders AN and bulimia nervosa (BN) is an overvaluation of weight and shape.¹ Most people derive their sense of self-worth and self-esteem from different sources, such as their competence at work or at school, interpersonal relationships, athletic achievements, etcetera. In eating disorder patients, however, self-esteem is entirely dependent on their weight and body shape.

Another central feature of both AN and BN is dietary restriction.¹ In individuals with AN, this results in underweight, or – in children and adolescents – a failure to meet expected increases in weight and growth according to age.² People with BN, on the other hand, who are by definition of normal weight or overweight, at length fail in their attempt to restrict their food intake, resulting in binge eating followed by compensatory behaviors to prevent weight gain. The essence of a binge-eating episode is the subjective sense of loss of control over eating;⁵ the sense that one cannot stop eating until all available food is consumed, or one has to stop because of severe abdominal discomfort. During a binge-eating episode, often large amounts of palatable foods, rich in sugar and/or fat, are rapidly consumed. Though binge eating can initially provide relief from negative feelings and food craving,⁶ an episode of binge eating is invariably followed by feelings of regret, guilt and shame. Strategies to counteract the anticipated consequences of binge eating – primarily weight gain – encompass purging, including self-induced vomiting and the use of laxatives and diuretics, and non-purging compensatory behaviors, such as excessive and compulsive exercise, or prolonged fasting. Since fasting increases the risk

for binge eating, a vicious cycle is thus created. Binge eating and purging can also occur as a symptom of AN, referred to as AN of the binge-purge subtype. In the other AN subtype, only restriction of intake is prominent. AN patients often have a markedly disturbed body image: they experience their body as fat, even when body weight is dangerously low.²

Eating disorders are usually classified according to the diagnostic criteria of the Diagnostic and Statistical Manual of Mental Disorders (DSM). A reliable guideline to classify mental disorders offers a universal and transparent language to clinicians to inform treatment decisions in an individual patient; besides, it is an essential tool to examine the prevalence of disorders to guide health care policy, to select patient groups for scientific study purposes, and to document morbidity and mortality rates.² The fifth edition of the DSM, DSM-5, was released in May 2013,² almost two decades after publication of the previous edition, the DSM-IV, in 1994.⁷ In those two decades, a wealth of research in the fields of cognitive neuroscience, neuroimaging, genetics and epidemiology has expanded our knowledge of mental disorders. Thus, an important limitation of the DSM-IV was uncovered: it is too rigid a categorical system. Clinical and scientific observations have shown that the boundaries between certain disorder categories are not clear cut at all, and that some symptoms are not bound to one disorder, but can occur with varying severity in a whole range of disorders.² This limitation of the DSM-IV also pertains to the section on eating disorders (e.g., Fairburn & Cooper⁸ and Attia et al.⁹).

The DSM-IV specifies only two eating disorders, AN and BN.⁷ In DSM-5, the significantly revised eating disorder section specifies three eating disorders: AN, BN, and binge-eating disorder (BED).² In BED, binge-eating episodes are – in contrast to BN – not followed by compensatory behavior. Therefore, BED is often seen in obese individuals. The decision to establish BED as a mental disorder was not without controversy. A major complaint – not limited to BED – about classifying certain behaviors as mental disorders is that behaviors that may seem quite common (such as binge eating) are unnecessarily medicalized.¹⁰ Compared to obesity per se, however, binge eating is associated with higher levels of psychopathology and weight- and shape concerns, and lower quality of life.¹¹ Obesity on the other hand, though also considered for the status of a mental disorder,^{12,13} was eventually not included in the DSM-5 (as it had not been in the DSM-IV either) because evidence that obesity is primarily caused by a mental dysfunction was unconvincing.¹³ It is nevertheless intriguing to imagine the immense – and probably unaffordable – mental health care policy implications of such a status, considering the fact that 35% of the adult US population is obese,¹⁴ and would thus be diagnosed

Table 1.1 Severity rating of eating disorders in DSM-5

	AN	BN	BED
Mild	BMI \geq 17	1–3 episodes of inappropriate compensatory behaviors per week	1–3 BE episodes per week
Moderate	BMI 16–16.99	4–7 episodes per week	4–7 BE episodes per week
Severe	BMI 15–15.99	8–13 episodes per week	8–13 BE episodes per week
Extreme	BMI <15	14 or more episodes per week	14 or more BE episodes per week

AN: anorexia nervosa; BN: bulimia nervosa; BED: binge-eating disorder;
 BMI: body mass index (kg/m^2); BE: binge eating

with a ‘new’ mental disorder. Hence, it is an important question how changes in diagnostic criteria and categories affect the number of people with a mental disorder diagnosis; in this case, with an eating disorder diagnosis.

A new feature in the DSM-5 is the introduction of a severity rating for disorders, ranging from mild to extreme, in order to help clinicians delineate treatment and track a patient’s progress. Key severity parameters for eating disorders have been defined (see Table 1.1). Clinicians can increase severity ratings based on the presence and severity of other symptoms, and the degree of functional disability.² The validity of the severity ratings for eating disorders is unknown.

EPIDEMIOLOGY

Epidemiological studies provide information about the occurrence of disorders. Morbidity and mortality rates derived from epidemiological studies inform health care policy and the planning of treatment services. Incidence studies may shed light on risk factors. As mentioned before, a reliable classification is a *sine qua non* for this purpose. The other way round, epidemiological data can also shape the development of a classification. The decision to establish BED as a specific eating disorder, for example, was partly informed by epidemiological data, proving its construct validity by showing a distinct profile of clinical characteristics and correlates of BED compared with the other eating disorders.¹¹

Longitudinal epidemiological studies investigate trends in the frequency of disorders over time. To reliably examine time trends in morbidity rates, it is essential that the classification used does not change over time. Differences in definitions of disorder make comparisons between studies difficult – if not impossible –, and prove to be a major challenge for long-standing (e.g., spanning decades) epidemiological studies.¹⁵ Time trends in morbidity rates provide clues to the etiology

of a disorder, especially to sociocultural factors, as it is assumed that the biological make-up of a species does not change within a few decades. Thus, epidemiological studies examining secular trends may uncover risk factors that affect the society at large. In the case of eating disorders, this knowledge is salient since sociocultural factors are thought to play a major role.¹⁶

The etiology of eating disorders is relatively poorly understood.^{17,18} Although many risk factors have been described,¹⁹ the exact mechanism through which an individual develops an eating disorder is unknown.¹ Moreover, though present in virtually all eating disorder patients, the risk factor body dissatisfaction – widespread among women in the Western world – is hardly sufficient to develop an eating disorder.²⁰

In accordance with most other mental disorders,² risk factors for eating disorders are categorized along social, psychological and biological lines (e.g., Jacobi. et al.¹⁹); in other words: from influences from the society at large and the social environment to the psychological and biological characteristics of the individual itself. Examples of societal risk factors include living in a Western culture, which places great value on the thin-body ideal, especially for women.¹⁶ This thin-body ideal is promoted and reinforced through the media¹⁶ and through ‘micro societies’ such as family and peers.^{21,22} Many girls and women feel that their bodies do not adhere to this standard and develop body dissatisfaction.²³ Psychological risk factors include low self-esteem, perfectionism (especially in AN), anxiety and depression, and difficulties with identity and autonomy development; salient processes in adolescence.^{16,19,20} Biological factors include a genetic predisposition (e.g., female sex) and neuroendocrine disturbances, for example in the central control of appetite.¹⁷

A factor associated with a certain disorder may only be called a risk factor if it is established that the factor in question precedes the outcome. Prospective, longitudinal studies are the gold standard to examine precedence.¹⁹ This type of study is relatively scarce in the field of eating disorders, because longitudinal studies, especially for such low-prevalent disorders, are expensive and time-consuming to conduct. Moreover, to distinguish consequences or concomitants of a disorder from true risk factors, a longitudinal study would ideally have to start before onset of the disorder. Since eating disorders usually develop in adolescence,¹⁵ early adolescence would be an advantageous starting point for longitudinal research into putative risk factors. While examining time trends may provide knowledge about risk factors on a macro (societal) level, studying a cohort of early adolescents may offer insight into sociocultural risk factors on a micro level (e.g., at school or at home). The micro environment of – future – eating disorder patients has been

studied before (e.g., Polivy & Herman²⁰), but mostly through self-report, which may be biased.²⁴ Therefore, a call for multiple informant data (e.g., peers) in eating disorder research has been made.²⁴

OUTLINE AND SCOPE OF THIS THESIS

Research questions addressed in this thesis include: Has the incidence of eating disorders changed over time? How do changes in the diagnostic criteria from DSM-IV to DSM-5 affect the number of people with an eating disorder diagnosis? Furthermore, an attempt is made to advance our understanding of how eating pathology develops by examining the role of self-perceived and peer-perceived social status in early adolescence as a potential risk factor.

Chapter 2 provides a comprehensive review of the literature on the epidemiology of eating disorders, focusing on the basic epidemiological parameters incidence, prevalence and mortality rate.

Chapter 3 examines changes in the incidence of anorexia nervosa and bulimia nervosa in the Netherlands during the 1980s, 1990s and 2000s, using data from a nationwide network of general practitioners, serving a representative sample of the total Dutch population.

Chapter 4 discusses the literature on epidemiology, course, and outcome of eating disorders in accordance with the DSM-5. The first part describes the consequences of the revised diagnostic criteria for the incidence and prevalence of anorexia nervosa, bulimia nervosa, binge-eating disorder, and the residual diagnosis of not otherwise specified eating disorders. The second part reviews course and outcome studies regarding the three specific eating disorders in DSM-5 – anorexia nervosa, bulimia nervosa and binge-eating disorder.

Chapter 5 reports on the prevalence and severity of DSM-5 eating disorders in a community cohort of Dutch adolescents. The validity of severity ratings is examined by linking severity level to detection and treatment rates by (mental) health care.

Chapter 6 investigates whether self-perceived and peer-perceived social status in early adolescence is associated with eating pathology in young adulthood. Data from the same community cohort of Dutch adolescents as described in Chapter 5 are used.

Chapter 7 provides a general discussion of the main findings of this thesis.

REFERENCES

1. Fairburn CG, Harrison PJ. Eating disorders. *Lancet* 2003;361:407-416.
2. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. (5th ed). Washington, DC: American Psychiatric Publishing; 2013.
3. Harris EC, Barraclough B. Excess mortality of mental disorder. *Br J Psychiatry* 1998;173:11-53.
4. Arcelus J, Mitchell AJ, Wales J, Nielsen S. Mortality rates in patients with anorexia nervosa and other eating disorders. A meta-analysis of 36 studies. *Arch Gen Psychiatry* 2011;68:724-731.
5. Wolfe BE, Baker CW, Smith AT, Kelly-Weeder S. Validity and utility of the current definition of binge eating. *Int J Eat Disord* 2009;42:674-686.
6. Berg KC, Crosby RD, Cao L, Peterson CB, Engel SG, Mitchell JE, et al. Facets of negative affect prior to and following binge-only, purge-only, and binge/purge events in women with bulimia nervosa. *J Abnorm Psychol* 2013;122:111-118.
7. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. (4th ed., text rev. ed). Washington, DC: American Psychiatric Association; 2000.
8. Fairburn CG, Cooper Z. Eating disorders, DSM-5 and clinical reality. *Br J Psychiatry* 2011;198:8-10.
9. Attia E, Becker AE, Bryant-Waugh R, Hoek HW, Kreipe RE, Marcus MD, et al. Feeding and eating disorders in DSM-5. *Am J Psychiatry* 2013;170:1237-1239.
10. Frances A. Saving normal: an insider's revolt against out-of-control psychiatric diagnosis, DSM-5, Big Pharma, and the medicalization of ordinary life. New York, NY: William Morrow; 2013.
11. Wonderlich SA, Gordon KH, Mitchell JE, Crosby RD, Engel SG. The validity and clinical utility of binge eating disorder. *Int J Eat Disord* 2009;42:687-705.
12. Volkow ND, O'Brien CP. Issues for DSM-V: should obesity be included as a brain disorder? *Am J Psychiatry* 2007;164:708-710.
13. Marcus MD, Wildes JE. Obesity: is it a mental disorder? *Int J Eat Disord* 2009;42:739-753.
14. Flegal KM, Carroll MD, Kit BK, Ogden CL. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. *JAMA* 2012;307:491-497.
15. Hoek HW, van Hoeken D. Review of the prevalence and incidence of eating disorders. *Int J Eat Disord* 2003;34:383-396.
16. Gordon RA. Eating disorders. Anatomy of a social epidemic. (2nd ed). Oxford: Blackwell Publishers; 2000.
17. Treasure J, Claudino AM, Zucker N. Eating disorders. *Lancet* 2010;375:583-593.
18. Walsh BT. The enigmatic persistence of anorexia nervosa. *Am J Psychiatry* 2013;170:477-484.
19. Jacobi C, Hayward C, de Zwaan M, Kraemer HC, Agras WS. Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. *Psychol Bull* 2004;130:19-65.
20. Polivy J, Herman CP. Causes of eating disorders. *Annu Rev Psychol* 2002;53:187-213.
21. Webb HJ, Zimmer-Gembeck MJ. The Role of Friends and Peers in Adolescent Body Dissatisfaction: A Review and Critique of 15 Years of Research. *J Res Adolesc* 2014;24:564-590.
22. Voelker DK, Reel JJ, Greenleaf C. Weight status and body image perceptions in adolescents: current perspectives. *Adolesc Health Med Ther* 2015;6:149-158.

23. Stice E, Shaw HE. Role of body dissatisfaction in the onset and maintenance of eating pathology: a synthesis of research findings. *J Psychosom Res* 2002;53:985-993.
24. Stice E, South K, Shaw H. Future directions in etiologic, prevention, and treatment research for eating disorders. *J Clin Child Adolesc Psychol* 2012;41:845-855.