

# Pathogenesis of Anorexia Nervosa

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## Abstract

This paper provides a recent review of studies investigating the eating disorder anorexia nervosa, with a view to summarising current knowledge on the pathogenesis of this condition. Genetic factors, the role of dieting, family functioning, and societal influences are each discussed in turn, and the application of current data to the treatment setting is also outlined.

**Key words:** *Anorexia nervosa, Eating disorders*

## Introduction

Anorexia nervosa (AN) is an intriguing disorder. Its incidence is increasing in the West, and it is among the most lethal of all psychiatric disorders.<sup>1-4</sup> It seems to selectively affect young, intelligent, attractive women of higher social standing.<sup>5</sup> Within a given society, its prevalence varies among different ethnic groups and increases as a society becomes more westernised.<sup>5</sup> It is a familial disorder.<sup>6</sup> A comprehensive theory of pathogenesis would need to encompass biological, developmental, psychodynamic, familial, and cultural perspectives. The search for such a theory is a daunting task. This article summarises current knowledge concerning the pathogenesis of AN. Bulimia nervosa (BN), a seemingly related condition sharing many of the clinical features of AN, will be mentioned only with regard to findings of relevance to AN.

## Genetics and Biology

There is strong evidence supporting a genetic basis to AN. Three studies have specifically addressed this aspect by studying twin pairs in a clinical setting.<sup>7-9</sup> Holland et al published data on 2 different sets of twins.<sup>7,8</sup> In the first study of 34 twin pairs and 1 set of triplets, pairwise concordance rates of AN were much higher among monozygotic (MZ) than dizygotic (DZ) twins (55% vs 7%).<sup>7</sup> In the second study of a further 15 female twin pairs, a significant difference between MZ and DZ twins was again found (71% vs 10%).<sup>8</sup> A later German study of 25 twin pairs

also reported a similar difference in concordance rates between MZ and DZ twins (57% vs 9%) with respect to AN.<sup>9</sup>

Data from 2 of 3 large scale population-based twin studies also support a genetic basis for AN. In the first study, Walters and Kendler administered adapted sections of the Structured Clinical Interview for *Diagnostic and Statistical Manual of Mental Disorders-III-R* (SCID-III-R) to 2163 Caucasian female twins on the Virginia twin register.<sup>10</sup> They found that the co-twins of twins with AN were more likely to have a current low body mass index, and also had a significantly higher lifetime risk for AN, BN, and major depression. Wade et al, in an Australian twin register study of 3869 female twins, found that environmental rather than genetic influences best explained the weight, shape, and eating concerns seen among twins with BN.<sup>11,12</sup> However, major methodological limitations, including poor case ascertainment undermine the validity of these findings. Data on AN was not reported.

In a Danish study, Kortegeard et al sent a questionnaire to 34,142 twins on the Danish twin register.<sup>13</sup> They found a higher pairwise concordance rate for broadly defined AN among MZ twins (15%) than among DZ twins (7%). Again, the method of case ascertainment — case status was determined by 2 questions in a 59-item questionnaire — and the relatively low response rate (82%) overall, weakened the findings.

Although these population twin studies have major methodological flaws, the findings do lend support for the role of genetic factors in the pathogenesis of AN.<sup>14</sup> What could be genetically transmitted to make an individual vulnerable to the development of AN? There are a range of possibilities to consider in this regard. The disorder itself could be transmitted similar to other conditions such as neurofibromatosis. However, it is unlikely that AN is the result of a single, dominant gene inheritance pattern, and such direct inheritance would still leave unexplained the biological mechanisms producing extreme dieting behaviour, and the characteristic mental attitude of weight phobia. Searches for defects in 1 or more of the hormones regulating weight and eating behaviour, such as leptin, neuropeptide Y, and peptide YY, have so far been negative.<sup>15</sup> There is a persistent interest among some researchers in the possibility of central serotonin disturbances in AN.<sup>16-17</sup>

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This hypothesis is based on the finding that elevated concentrations of 5-hydroxy indoleacetic acid (5-HIAA) was identified in cerebrospinal fluid (CSF) from patients who had recovered from AN. Elevated serotonin activity has been found to be consistent with the observation of increased obsessiveness and harm-avoidance traits in weight-recovered patients with AN.<sup>18-19</sup> There are some preliminary data to support the idea that obsessive perfectionism and harm avoidance may be heritable traits in families with eating disorders (ED), but the search for an association between eating disorders and serotonin receptor genes have yielded conflicting results.<sup>20-22</sup> Finally, it is possible that the genetic influence in AN may be an indirect one. For instance, it is well-known that body fat distribution and the amount of body fat differ between men and women.<sup>23</sup> Perhaps a propensity towards increased weight is genetically transmitted, resulting in rigorous dieting to control body weight, which in turn triggers the disorder.

In summary, studies of twins suggest that there is a genetic basis to AN. Studies to find a biological marker have been largely inconclusive, with the exception possibly of elevated CSF 5-HIAA levels in recovered patients with AN. The search for the genetic basis of AN continues.<sup>21</sup> Family studies data suggesting that families with ED may have a preponderance of mood and anxiety disorders is reviewed elsewhere.

## Cultural Influences and the Role of Dieting

The Minnesota Study convincingly demonstrated that prolonged semi-starvation can lead to the development of abnormal eating behaviors.<sup>24</sup> The most common antecedent to AN is dieting. An initial desire to lose a few pounds is soon overtaken by an obsessive desire to lose weight. Being able to lose weight and be thin is seen and felt to be ‘good’, such that gradually the dieting and weight loss come to be perceived or experienced as being in control, or being the only solution to life’s problems. When pressed to explain their self-starvation, anorexics often say “when the chips are down, the only way forward is to lose weight” or “losing weight is the only thing I am good at”.

At least 6 longitudinal studies have been conducted to explore the role of dieting in the pathogenesis of AN. Five of these have been the subject of a previous review, and are summarised only briefly here.<sup>25</sup> King utilised a 2-stage design to determine the prevalence of eating disturbances among 534 women and 186 men (aged 16 to 35 years) in several London general practices.<sup>26</sup> At 18 months follow-up, twice as many dieters had developed bulimic symptoms compared with those who had stopped dieting. Patton et al similarly used a 2-stage design to study the prevalence of eating disturbances in 15-year-old London schoolgirls (n = 1011).<sup>27</sup> At 1-year follow-up, 13 of the original 61 dieters had developed an eating disorder, 23 remained dieters, while 25 had become non-dieters.

Rathner and Messner completed a questionnaire survey to determine the prevalence of eating disturbances among

517 girls aged between 11 and 19 years in northern Italy.<sup>28</sup> At interview 1 year later, 8 of the 50 girls identified as being ‘at-risk’ at the initial survey had become bona fide patients. Methodological issues such as a failure to interview the subjects at baseline and poor case definition undermined the findings of this study. Poor case definition and lack of details also marred the study undertaken by Marchi and Cohen.<sup>29</sup> This 10-year follow-up study of 333 males and 326 females in upstate New York, found that eating disorders in late adolescence and early adulthood were preceded by picky eating in childhood and dieting behaviour in early adolescence.<sup>29</sup>

Finally, in a further 10-year follow-up study, Schleimer interviewed 97 of 130 young women who had earlier reported eating disturbances, including 12 with AN.<sup>30</sup> Over the 10-year period, 4 patients who did not have AN at baseline had developed AN, yielding a lifetime prevalence of 3.4%. This figure is substantially higher than the commonly accepted prevalence for AN of 0.5%.<sup>31</sup>

The sixth longitudinal study, conducted by Leon et al, followed a sample of more than 850 girls and 800 boys (grades 7 to 10) for 3 to 4 years to identify risk factors for eating disturbances.<sup>32-34</sup> The initial report from this study stated that the strongest predictor of year 3 risk status was years 1 and 2 risk scores for eating disorders.<sup>34</sup> Scores were compiled from various measures including dieting behaviour. Later reports noted negative affect to be a significant predictor, while the most recent report found negative affect/attitude at baseline to be a significant predictor of the final year risk score.<sup>32-33</sup> The use of risk score for eating disorder as an end point is problematic, and very few of those with a high risk score at year 3 actually had clinical AN. Also, because dieting behaviour was common among the subjects, its assessment was difficult and its role as an antecedent was not clearly established, other than in the initial report. Nevertheless, this study highlights the importance of other risk factors in the pathogenesis of AN, in addition to the presence of dieting behaviour.

Clearly not all dieters become individuals with an eating disorder. In fact, the majority of dieters will never develop an eating disorder. Nevertheless, 5 of the 6 longitudinal studies strongly suggest that dieting behaviour places an individual at higher risk for the development of an eating disorder. It could therefore be expected that eating disorders would increase in a culture where dieting behaviour has become more prevalent.<sup>5</sup>

What are the risk factors that distinguish dieters who develop an eating disorder from those who do not? The issue of genetic predisposition has already been discussed. The studies by Patton et al<sup>27</sup> and Leon et al<sup>32-34</sup> suggest that negative affect may also play a role — individuals who have a low self esteem or poor body image, or are prone to depression may be vulnerable to AN if they embark on dieting. What cultural influences might encourage an individual to go on a diet? The societal emphasis on a thin body figure has been well-discussed,<sup>35-37</sup> and is a continuing

issue in modern society. The prevalence of AN is known to increase in populations in which the emphasis on thin body figure is intensified, for example ballet dancers.<sup>36</sup> There is evidence that the pursuit of thinness is more intense and more prevalent among women of higher social standing,<sup>38</sup> and that younger girls are increasingly becoming pre-occupied with this quest for thinness.<sup>39-40</sup>

Other cultural factors may also be relevant. Brumberg provides a thoughtful discussion of possible factors:<sup>41</sup>

- the breakdown of meal patterns and meal times so that increasingly people are eating and drinking ‘on-the-go’
- the increase of ‘grazing’ behaviour rather than regular meals
- the availability of fast foods
- decreased importance of the social aspects of eating
- the wider range of foods readily available
- lack of awareness of appropriate food to eat at each mealtime.

All of these factors could contribute to a breakdown of regular eating patterns and lead to the chaotic eating so commonly found in patients with eating disorders. However, there is little hard data to support such contentions.

Crisp has argued that AN may be a defence against fears concerning sexual maturation.<sup>42</sup> It would be reasonable to assume that such fears may have intensified among young women given the increasing breakdown of sexual mores, the rise of the AIDS epidemic, and the resurgence of certain sexually transmitted diseases. Others have argued that AN is a misguided quest for individual identity.<sup>43</sup> If so, then the increasingly complex role of women in post-modern society may have encouraged the development of AN among women perplexed by such role diffusion.<sup>44</sup> These intriguing ideas are discussed in greater detail later in the review.

## Family Functioning

The idea that specific family dysfunction may lead to AN has been advanced by many authors.<sup>45-46</sup> Family therapy has been found to be effective treatment for recent onset AN in young patients but this cannot be used to infer that family factors may be pathogenetic.<sup>47</sup> Early research on family functioning in AN showed some promise.<sup>48-50</sup> Recent research, however, has produced no major new findings, and more often than not findings are conflicting or unconvincing. Research into family functioning is often tedious, and findings may be influenced by the patient’s age and the composition of the family, with each family member having a different view.<sup>51-53</sup> Whether any detected dysfunction is the cause or effect of AN is also difficult to disentangle. Recent findings in this area will be presented briefly.

Dare et al<sup>54</sup> and Le Grange et al<sup>55</sup> found low levels of expressed emotions (EE) in families of both anorexic and bulimic patients. The authors interpreted this as evidence of conflict avoidance. Also, perhaps surprisingly, there was no evidence of emotional overinvolvement, postulated by Minuchin as a characteristic of psychosomatic families. Patients and parents perceived their family interaction differently. Low EE at baseline predicted better outcome at 6 months. If confirmed, this finding may suggest that conflict

avoidance is actually beneficial to the recovery of AN, a conclusion unlikely to find favour among family therapists.

Waller et al<sup>52,53,56-58</sup> and Calam et al<sup>59</sup> found that family interaction ratings completed by patients with eating disorders predicted diagnosis, that is, it differentiated patients from control subjects. Patients with ED perceived their families as lower in adaptability (more rigid) and lower in cohesion (more disengaged) than controls. Findings by this group of investigators, suggest that there is a specific family dysfunction associated with ED. However, it appears difficult to specify this dysfunction beyond describing characteristics of rigidity and disengagement.

North et al<sup>51</sup> and Gower and North<sup>60</sup> found that family interaction as assessed by patients with AN was normal, although interviews conducted by a clinical researcher found general but not specific dysfunction in these families compared with families of psychiatric patients and community control groups. Both findings are in contrast to those of Waller et al<sup>52,53,56-58</sup> and Calam et al.<sup>59</sup> Better family functioning predicted better outcome at 1 year, but clinical improvement in AN was not matched by improvement in family functioning, at least as perceived by the patients. More inexplicably, family dysfunction correlated inversely with severity of AN. The authors interpreted this as evidence that family dysfunction was not a result of AN. Dare and Key have suggested that AN may serve to decrease family dysfunction.<sup>61</sup> If such is the case, then their own finding that low EE, presumably a measure of family dysfunction, predicted better outcome would be hard to explain.

In summary, the findings with regard to family functioning and AN are conflicting. Whether family dysfunction is a factor in the pathogenesis of AN remains speculative.

## Vulnerable Individuals

Any theory explaining the pathogenesis of AN must address why the disorder is so much more prevalent among women than men. The majority of published studies have found a female to male preponderance of about 10:1. As already mentioned, body fat differs between the 2 sexes, but this biological fact alone appears insufficient to account for differences in incidence figures. Many clinicians and researchers have suggested that the process of psychological developmental places some individuals at risk for the development of an eating disorder.

Early writers such as Bruch<sup>43</sup> and Crisp<sup>42</sup> conceptualised AN as a disorder of identity formation, for example. However, they did not clarify why young women are more vulnerable with regards the development of AN than young men in this process. Perhaps the first author to approach the pathogenesis of eating disorders from a feminist perspective was Boskind-Lodahl.<sup>62</sup> Since then, many articles and volumes have articulated this perspective. The views of a number of authors on this topic offering useful insights have been presented.<sup>41,44,63-66</sup>

### ***Culturally-determined Factors***

In western culture, women are encouraged to define themselves in terms of their relationships with others, that is, they define their identity in relational terms. Consequently, their self worth is determined by their ability to initiate, engage, and maintain close relationships. For most young women, a romantic relationship with a man is of primary importance.

Attaining the culturally defined beauty ideal is beneficial in this regard because it can bring success in such relationships. Putting a premium on relationships also renders women vulnerable to others' opinions, and women therefore tend to define their self worth in terms of how they appear to others. This relational orientation, which is thought to be culturally determined, presumably increases a young woman's desire for thinness, and thus may be a risk factor for the pathogenesis of AN.

Other culturally-determined factors may also underlie women's preponderance for eating disorders. For instance, it has been argued that women may be under greater pressure to conform to societal norms (in this case the demand for a slim figure) because of their inferior social status relative to men. Females are also more likely to be victims of childhood sexual abuse which is a risk factor for the development of a psychiatric disorder and perhaps also specifically for an eating disorder.

The increasingly complex role of women in modern society leading to role diffusion may also be a contributing factor. The ideal of a thin body figure may be biologically unattainable by many women because of the female propensity for greater body fat, unless they resort to extreme dieting. Of note in this regard, premorbid obesity has been identified as a risk factor for the development of bulimia nervosa.<sup>14</sup>

In sum, the power inequity between the sexes, the greater exposure of women to certain risk factors, the unrealistic female beauty ideal, and female relational identity formation are proposed as the explanation for the gender difference seen in the prevalence of eating disorders. These ideas are intriguing. The evidence that women are under greater pressure than men to achieve a slim body figure is overwhelming. Studies to investigate these tenets further, such as the link between relational identity formation and the development of an eating disorder, are still needed.

### **Towards a Coherent Theory of Pathogenesis**

At this point in our understanding of AN, a coherent theory of its pathogenesis is elusive. However, available data suggest that a young woman with a family history of eating disorder, depression, or obsessive compulsive disorder, living within a culture that emphasises thinness, may be more likely to develop AN if she embarks on a rigorous diet to lose weight, particularly:

- if rigorous dieting arises from low self esteem
- the young woman defines her identity primarily according to how others perceive her

- she was exposed to other risk factors for AN such as childhood sexual abuse or childhood obesity.

### ***Clinical Implications***

How can a theory of pathogenesis help a clinician in treating a patient with AN? Firstly, it can assist in providing psychoeducation to the patient and the family. Psychoeducation is particularly important in engaging both the patient and family during the initial phases of treatment. Faced with a sullen, silent, and resistant patient, the clinician may utilise a strategy such as the following to engage the patient: "Very often people don't know why they want to go on a diet. However we do know ... ." The clinician can then go on to describe the findings of the Minnesota study<sup>23</sup> or any of the longitudinal studies such as those of Patton et al.<sup>27</sup> The family may particularly appreciate such information, since they almost always find the patient's behaviour incomprehensible.

Secondly, as the patient is often reluctant to relinquish her thinness, helping her to weigh the pros and cons of using thinness to achieve a sense of identity or control may open the way for further treatment. A clinician may initially agree with the patient's stance saying, for example: "Not eating and being thin is not always bad. Thinness is often perceived as being special." The clinician can then describe the Garner and Garfinkel<sup>35</sup> or the Wiseman et al<sup>37</sup> studies from this perspective. The risks of dieting may then be raised using findings from the Minnesota study to indicate that dieting and weight loss in fact bring very little actual gain.

Thirdly, a knowledge of pathogenesis can assist the treatment of AN by guiding the clinician to focus on factors such as negative emotionality, relational identity formation, or possible sexual abuse.

### **Concluding Comments**

Readers who are familiar with my previous reviews on the pathogenesis of AN will realise that progress in our understanding of this disorder has been painfully slow.<sup>23, 67</sup> Fresh ideas and meticulous research from a new generation of researchers is urgently needed.

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