Academy for Eating Disorders Position Paper: Eating Disorders Are Serious Mental Illnesses

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Position

It is the position of the Academy for Eating Disorders (AED) that anorexia nervosa and bulimia nervosa, along with their variants, are biologically based, serious mental illnesses (BBMI) that warrant the same level and breadth of health care coverage as conditions currently categorized in this way (e.g., schizophrenia, bipolar disorder, depression, obsessive-compulsive disorder). As set forth below, we advocate this position unequivocally based on an emerging science that affirms with a reasonable degree of medical and scientific certainty that eating disorders are significantly heritable; influenced by alterations of brain function; significantly impair cognitive function, judgment, and emotional stability; and restrict the life activities of persons afflicted with these illnesses. Accordingly, the denial or restriction of equitable and sufficient treatment necessary to avert serious health consequences and risk of death is untenable and should be vigorously protested.

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Commentary

Overview

Eating disorders are still not considered serious forms of mental illness in some states and countries. In the United States, the failure to acknowledge the seriousness of eating disorders has resulted in a health care crisis for sufferers and their families. As of 2007, laws in some states (e.g., New Jersey, Illinois) exclude eating disorders from conditions considered to be "serious mental illnesses" (SMIs), "biologically based mental illnesses" (BBMIs), and in children, "serious emotional disturbances" (SEDs). The potential consequences of these exclusions are significant, as these categories can be used by insurance companies to determine which psychiatric illnesses are covered by their policies, and which are excluded, for reasons that are arbitrary or capricious. Unfortunately, there is no accepted definition of these categories, nor is there federal legislation that defines the terms.[1] For example, the United States Health and Human Services Substance Abuse and Mental Health Services Administration (SAMSHA) defines a SMI as a diagnosable mental disorder found in persons aged 18 years and older that is so long lasting and severe that it seriously interferes with a person's ability to take part in major life activities. For individuals younger than 18, SAMSHA reserves the term SED which they define as diagnosable mental disorder found in persons from birth to 18 years of age that is so severe and long lasting that it seriously interferes with functioning in family, school, community, or other major life activities (http://mentalhealth.samhsa.gov/features/hp2010/terminology.asp). Finally, in some states (e.g., New Jersey), a BBMI is defined as a condition that current medical science affirms is caused by a neurobiological disorder of the brain, significantly impairs cognitive function, judgment, and emotional stability, and limits the life activities of the person with the illness.

Because of this lack of consensus definition, individual states and insurance companies have been free to develop their own definitional criteria, resulting in the anomalous reality that eating disorders are excluded from coverage under these definitions in some areas of the country, but not in others.

Importantly, the debate about the seriousness of eating disorders is not limited to the United States. In the United Kingdom, Golderberg and Gournay (1999) argued that most eating disorders are mild cases of "somatised presentations of distress." In other parts of the world, eating disorders are sometimes ignored (e.g., bulimia nervosa (BN) in Romania[2]) and/or receive insufficient political and/or financial support for treatment services (e.g., Norway[3]).

It is thus essential to an informed public health care policy that governments have accurate information regarding the status of eating disorders as BBMIs, SMIs, or SEDs. In our role as the largest international organization of eating disorder scientists and clinical specialists, the AED has reviewed scientific data attesting to eating disorders as serious forms of mental illness. Although definitions for these categories all stress functional and biological impairment, the BBMI definition is most restrictive and we thus use this definition for evaluating the status of eating disorders:

> "A condition that current medical science affirms is caused by a neurobiological disorder of the brain, significantly impairs cognitive function, judgment, and emotional stability, and limits the life activities of the person with the illness."

We use the legal standard in the U.S. of within "a reasonable degree of medical or scientific certainty" (i.e., more likely than not) in evaluating this definition for eating disorders, in as much as we hold eating disorders to the same standard of certainty as other psychiatric disorders when determining whether they meet the definition of BBMI.

We evaluate this definition in relation to AN, BN, and, when possible, EDNOS. Empirical studies of EDNOS have lagged behind those of AN and BN due to their lack of inclusion in classification systems. However, studies of EDNOS have increased over the past decade in response to the recognition that these conditions are serious and debilitating in their own right. Thus, we review data on EDNOS when available and urge readers to monitor the empirical literature moving forward, as additional support for AN, BN, and EDNOS as serious forms of mental illness will likely accumulate.

Genetic and Neurobiological Data

The heritability of eating disorders is similar to that of other psychiatric conditions (e.g., schizophrenia, bipolar disorder, depression, OCD) that have been considered to be BBMIs, SMIs, or SEDs. Twin studies estimate that 50–83% of the variance in AN, BN, and binge eating disorder (BED; a form of EDNOS) are accounted for by genetic factors. These studies have included conditions meeting threshold and subthreshold criteria for AN, BN, EDNOS (specifically BED and subthreshold presentations of AN and BN).[4–10] Molecular genetic studies have begun to identify chromosomal regions and genes that may contribute to the genetic diathesis. Areas on chromosomes 1, 4, and 10 may harbor risk genes for AN and/or BN[11–13] and genes involved in the serotonin,[14,15] brain-derived neurotrophic factor (BDNF),[15–17] and opioid[14] systems may contribute to risk for AN. Although molecular genetic findings have been less consistent for BN,[15] and limited data exist for EDNOS, serotonin and BDNF genes are involved in food intake and also the anxious personality traits that are common in individuals with eating disorders (see "Cognitive and Emotional Functioning" below). Thus, the genetic diathesis for eating disorders may be linked to these systems.

When malnourished and emaciated, individuals with AN have alterations of brain structure (e.g. Refs. 18 and 19), metabolism,[20,21] and neurochemistry.[22]) Similar alterations are found in BN[23] where imaging studies show brain "atrophy"[24] and altered brain metabolism.[22,25] Moreover, in AN and BN, there are profound disturbances of brain serotonin,[26,27] neuropeptide systems,[22] and brain neurocircuitry[19,25,28,29] that frequently persist after recovery from the illness.[30,31] These alterations involve brain circuits known to modulate appetite, mood, cognitive function, impulse control, energy metabolism, and autonomic and hormonal systems.[32] It is important to note, however, that brain disturbances are not reflected in conventional laboratory blood test measures, as such tests do not directly assess brain function.

The role of biology in eating disorders is also supported by animal models. These models examine behavioral components of the disorders, as face validity for the cognitive aspects (e.g., fear of becoming fat, weight preoccupation) is difficult to achieve in nonhuman species. Nonetheless, data show disordered eating behavior in species as

diverse as rodents, sows, and primates.[33] For example, anorexic phenotypes (e.g., decreased food intake, significant weight loss, high activity levels) have been observed in rodents who: (1) are exposed to early stress; (2) have intermittent food restriction and access to running wheels; and/or (3) have autosomal recessive mutations or gene knockouts.[34,35] Some of these anorexic effects appear only after exposure to initial food restriction, a phenomenon that mimics early dieting in eating disorders. In addition, binge episodes (i.e., repeated consumption of palatable food in a short period of time) have been observed in rodents under conditions that increase risk for binge eating in humans (e.g., dietary restriction, stress).[36–40] A relevant new development is the identification of a subgroup of female rats who are "binge prone" and are naturally inclined to binge eat without experimental manipulations.[41] This rodent model has strong face validity as it models individual differences in binge eating in humans that likely reflect genetic and/or neurobiological differences in risk.

Overall, data are clear in showing that AN, BN, and EDNOS are heritable conditions in which the contribution of genetic factors is similar to that observed for other disorders considered to be biologically based (e.g., schizophrenia, bipolar disorder, OCD, recurrent major depression).[42–49] While it is true that the identification of confirmed risk alleles for these disorders awaits additional research, current knowledge is not unlike that of other BBMI disorders (e.g., OCD[43]). Neurobiological abnormalities are clearly present in AN and BN during the active illness, and some aspects of impaired brain anatomy and/or neurochemistry may persist after recovery. At this time, long-term prospective studies of neurobiological risk factors are lacking, although the presence of neurobiological alterations in unaffected family members of individuals with BN[50] suggest that these biological dysfunctions may contribute to illness onset. Finally, the presence of disordered eating phenotypes in nonprimate animals suggests a biological basis for many aspects of the disorders.

Cognitive and Emotional Functioning

Eating disorders are also associated with the deficits in cognitive and emotional functioning emphasized in BBMI definitions. Individuals with AN and BN exhibit difficulties with executive functioning (e.g., difficulties with set shifting)[51] and a weakness in contextual integration (i.e., getting the gist or the bigger picture).[52–54] Individuals with BN also exhibit a disinhibited pattern of responding, particularly in the context of negative emotions (more characteristic of BN disorders[55,56]), while individuals with AN have impaired decision making ability[57,58] and social cognition.[59] These deficits are pronounced during the acute phase of the illness and significantly interfere with judgment and interpersonal relationships. In particular, they may impact the progress of psychological therapy in AN, making engagement and joint work towards change more difficult.[60,61] Some deficits (e.g., set shifting) are also present after recovery from AN and in family members who do not have eating disorders.[62]

Impairments in emotional functioning are evident in significant comorbid psychopathology. The most common comorbid psychiatric conditions in AN include major depression and anxiety disorders (including, but not limited to, OCD, social anxiety disorder, and generalized anxiety disorder).[63–67] Anxiety disorders often predate the onset of AN,[64,68] and depression and anxiety persist after recovery.[69–71] Commonly comorbid conditions in BN include anxiety disorders,[63,66,67,72,73] major depression,[63,66,67,72,74,75] dysthymia,[75] substance use disorders,[67,74,76–78] and personality disorders.[66,67,78] Approximately 80% of individuals with AN and BN are diagnosed with another psychiatric disorder at some time in their life.[78] Comorbidity profiles of EDNOS have been shown to be comparable to or exceed those of BN.[79,80]

As with genetic/neurobiological alterations, substantial deficits in cognitive and emotional functioning are present in individuals with eating disorders. These deficits are similar to those observed in mood disorders[81] and anxiety disorders[82] and are themselves associated with their own set of genetic and biological risk factors.[46,83]

Limited Life Activities

According to the BBMI, SMI, and SED definitions, a serious mental illness limits the "life activities" of individuals suffering from the condition. Extensive data document the myriad ways in which eating disorders fit this criterion. Individuals with AN and BN rate their quality of life as low.[84] Social adjustment tends to be impaired,[85] as social communication skills are poor[59] and social networks tend to be small.[86] Vocational and educational functioning in individuals with AN and BN is below that expected, with absences from work and school (e.g., only 5.5 months per year in school over a 2-year period).[87–89] Social adjustment tends to remain poor even after recovery from BN,[85,90] highlighting the large "cost" of eating disorders to individuals who have suffered from the disorder. This

"cost" extends beyond the individual to the family and society at large as well. Women with AN have higher rates of pregnancy complications than women without eating disorders,[91] and their children may have later emotional and nutritional problems.[92] Carers of individuals with AN and BN have high levels of psychological distress.[93,94] Finally, eating disorders result in significant economic burden and health service use. A recent study on hospital admissions for adult psychiatric illness in England found that eating disorders had the highest proportion of admissions of all psychiatric disorders, with a length of stay over 90 days (26.8%) and the longest median length of stay (36 days).[95] More child and adolescent psychiatric beds are occupied by young people with eating disorders than any other diagnostic group (about 20% of inpatients).[96] In the U.S., individuals with eating disorders have higher health care utilization than individuals with other forms of mental illness, including depression.[97] High health care use tends to be similar across countries (e.g., in UK—see Ref. 87) and types of eating disorders (e.g., AN, BN, and EDNOS).

Medical complications represent significant forms of disability present in individuals with eating disorders. Indeed, eating disorders have one of the highest rates of medical complications of any psychiatric disorder.[98] Medical complications include hair loss, growth retardation, osteoporosis, loss of tooth enamel, gastrointestinal bleeding, bowel paralysis, dehydration, electrolyte abnormalities, hypokalemia, hyponatremia, and cardiac arrest.[23,98] The degree and type of medical consequences are related to the type of eating disorder behaviors (e.g., starvation, self-induced vomiting, binge eating, use of Ipecac syrup, etc.) and their severity,[98] with individuals with AN tending to experience the largest number of these complications.

Medical consequences can, and do, lead to death in some cases. Standardized mortality rates in AN are the highest of any psychiatric disorder[99–106] and are 12 times higher than the annual death rate from all causes in females 15–24 years of age.[106,107] Mortality rates for BN and EDNOS are harder to determine, being partially complicated by the relatively high degree of "cross-over" diagnoses from EDNOS and BN to AN and vice versa.[108] However, current estimates suggest that mortality rates in BN may not be elevated or only slightly elevated.[99,103,109] Notably, however, mortality in EDNOS may be as high as that observed for AN.[102] Increased risk of death in eating disorders is frequently due to medical complications described above[98,109] or suicide.[100,110]

Overall, eating disorders are associated with some of the highest levels of medical and social disability of any psychiatric disorder. These conditions carry significant costs to the individual, their family members, and to society at large. Indeed, the "life activities" of eating disorder sufferers are significantly impaired, sometimes to the point of early death.

Discussion

Our review indicates that, within a reasonable degree of medical and scientific certainty, AN and BN fit the routinely accepted definitional categories of BBMI, SMI, and SED based on evidence from studies of heritability, their association with significant neurobiological abnormalities, cognitive and emotional deficits, and social and medical disabilities. Moreover, in the light of accumulating data attesting to the severity and significant personal costs of EDNOS, it is expected that future studies will confirm their status as BBMIs, SMIs, and SEDs as well.

Like other BBMI conditions, the etiology of eating disorders is multifactorial and includes a combination of genetic, biological, and temperamental vulnerabilities that interact with environmental circumstances to increase risk. Nonetheless, the lack of recognition of the seriousness of eating disorders has implications for the status of eating disorders globally. In the U.S., eating disorders should be designated as BBMIs, SMIs, and SEDs and receive health care coverage and research funding that is equal to that of medical disorders as well as psychiatric conditions categorized as serious forms of mental illness. In other regions of the world, eating disorders should be recognized as serious forms of mental illness that deserve national recognition and funding. Changes in these designations and practices will ensure equal access to treatment and resources for all forms of serious mental illnesses.

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