

Academy for Eating Disorders Position Paper: The Role of the Family in Eating Disorders

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Position

It is the position of the Academy for Eating Disorders (AED) that whereas family factors can play a role in the genesis and maintenance of eating disorders, current knowledge refutes the idea that they are either the exclusive or even the primary mechanisms that underlie risk. Thus, the AED stands firmly against any etiologic model of eating disorders in which family influences are seen as the primary cause of anorexia nervosa or bulimia nervosa, and condemns generalizing statements that imply families are to blame for their child's illness. The AED recommends that families be included in the treatment of younger patients, unless doing so is clearly ill advised on clinical grounds. The position articulated in this article is in line with the World Wide Charter for Action on Eating Disorders.

Commentary

Overview

Anorexia nervosa (AN) and bulimia nervosa (BN) are serious mental and behavioral disorders with significant psychiatric and medical morbidity.¹ Although, it is now well appreciated that the etiology of these conditions is complex and that their treatment possess unique challenges, certain fam-

ily-based theories of causation, now recognized as overly simplistic and erroneous, are still in circulation. These posit, for example, that particular styles of interaction among family members are not only unique to eating disorders but also they play a specific role in the etiology or the maintenance of illness behavior. Reminiscent of such discredited pejoratives as the "schizophrenogenic" and "autis-togenic" mother, the idea that certain parental attitudes or family patterns could be "anorexogenic" has endured even though empirical support for this notion is weak at best.²

It is thus disturbing to note recent examples of misinformation disseminated to the public. For instance, one high-profile model recently blamed parents and families for the occurrence of eating disorders following media attention given to the death of several runway models from complications of AN. In response to assertions regarding causal roles for families in general, the aim of this article is to briefly review what is known about family influences in AN and BN. As a comprehensive review of this area is well beyond the scope of Academy for Eating Disorders (AED) Position Papers, the material cited is necessarily selective to support the position, but we believe it fairly summarizes current knowledge.

Historical Footnotes

The importance of family support and the possible detrimental role of parental inaction in the face of a child's life-threatening malnutrition were first introduced in accounts of AN appearing in the late 19th century.³ Indeed, Gull's seminal description of the illness asserted that it was justified, if not essential, to limit parental-child contact during treatment to prevent enabling of the illness by parental complicity in behaviors that had the effect of thwarting refeeding. It was not until the 1960s that the role of the family was reframed by Minuchin et al.⁴ in what was hailed by many as a fundamentally new conceptual model of AN. This model of the "psychosomatic family" had profound influ-

Accepted 21 July 2009

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Published online in Wiley InterScience
(www.interscience.wiley.com). DOI: 10.1002/eat.20751

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ence, placing emphasis on pathological interactive familial processes in the pathogenesis of AN, and advocating a particular form of family therapy as an effective means of arresting the condition in younger patients by altering family structure. However, early efforts to substantiate the model^{2,5} failed to identify any particular familial pattern linked to AN.

A paradigm shift originated with work at the Maudsley Hospital in London in the late 1970s.⁶ This paradigm shift directed attention away from models that presumed a central etiologic and maintaining role for family dynamics to see the family as a potential resource in therapy, easing parents' burden of guilt, and promoting an attitude of inclusion that is reflected today in the AED "World Charter for Action on Eating Disorders", and in current treatment guidelines applied to the pediatric age patient. Thus, current family therapy models for AN and BN now focus more on facilitation of emotional communication and emotional literacy, and assisting family members in developing skills to better negotiate differences of opinion and attitude, recognizing that some rigidity of behavior and emotionality are at times associated with eating disorders.

Family Factors in the Etiology of Eating Disorders

Despite these paradigm shifts, past and current risk factor research has still focused on the role of the family in causing or contributing to the eating disorder. Developmentally informed research^{7,8} on family risk factors in eating disorders remains sparse, and to date, data do not support the assertion that families are causal. Although cross-sectional studies and research seeking to identify characteristics that precede onset of illness are vitally important in generating hypotheses regarding causal factors, they are not without limitations. Similarly, whereas longitudinal, prospective studies that seek to identify predictors of future illness⁷ are crucial to the development of more rigorous models of risk, they have lacked adequate statistical power to identify risk factors associated with relatively rare or uncommon disorders such as AN and BN.

Risk Factor Research: Cross-sectional Studies. Numerous studies have sought to identify parenting- and family-functioning characteristics that may occur in advance of the onset of eating disorders. Among noteworthy findings are that inappropriate parental pressures uniquely distinguished eating disorder patients from psychiatric and normal controls⁹; that early-life "overprotective/high-concern" parenting behaviors were more common in mothers of

patients with AN compared to controls¹⁰; and that parental indifference, family discord, lack of parental care, and greater adversity distinguished BN, mixed AN/BN, and depressed groups from normal controls, whereas participants with AN displayed a premorbid developmental profile similar to normal controls. A series of community-based studies, using retrospective data, showed significantly more change in family structure (e.g., a parent leaving or a step-parent entering the family) in the year before onset of illness in a group of women with BN relative to the equivalent time frame for normal controls,¹¹ and that high parental expectations, low parental contact, and more family criticism about shape and weight occurred before onset of illness more often in persons with BN compared to mixed psychiatric and normal control groups.¹² In another case-control community-based study by the same group of investigators that compared developmental histories in groups of participants, AN, BN, normal controls, and psychiatric controls, individuals with AN were shown to have experienced significantly more parental problems compared to healthy controls, including separation, arguments, criticism, high expectations, over-involvement, under-involvement, low affection, and critical comments from family about shape, weight, or eating; however, participants with AN did not differ either from psychiatric controls, or participants with BN, on these variables.

In light of the methodological limitations of retrospective/cross-sectional research, these findings suggest that family/parenting factors that precede the onset of AN or BN increase risk for psychopathology in general; a further reasonable speculation is that such general risks then interact developmentally with inherent, and possibly more specific, sources of biological rooted vulnerabilities that ultimately give shape to particular phenotypes of disordered eating (see Klump et al.¹).

Longitudinal Risk Factor Research. Some prospective studies that have investigated the effects of parenting- and family- functioning variables in predicting later onsets of eating disorders or eating-related psychopathology have failed to find such factors to be of significance,¹²⁻¹⁴ but others have.^{10,15,16} The strength of these findings, both positive and negative, is however called into question by absence of psychiatric controls to determine specificity of the prospective associations that have been found, lack of adequate power to test for the prediction of full-blown cases of eating disorder, and reliance on risk factor assessments of questionable reliability and validity.

The Role of Genetics. Family, twin, and molecular genetic studies have all been conducted in eating disorders, with increasing evidence that heritable influences underlie susceptibility to both AN and BN.¹⁷ While the transmission of disease susceptibility genes represents a type of “family” influence, it is an influence of a different type. Moreover, no specific disease conferring genes or genomic regions of interest have been clearly replicated; the idea that genes alone account for the development of eating disorders seems implausible¹⁸; the nature of this transmissible effect remains unknown; and it is surely the case that whatever is being transmitted genetically is intertwined in complex ways with non-genetic social and family effects, elements of which are exceedingly difficult to parse. In this regard, as with other complex psychiatric conditions, it is likely that a multiplicity of risk factors—genetic, developmental, psychological, and cultural—will be shown to influence susceptibility to eating disorders, informing not only the development of empirically supported models of etiology, but also of prevention as well.^{17,19–23} For example, if adverse family factors can activate genetic susceptibility, perhaps protective family behaviors can be mobilized for the purposes of prevention. Whether or not it may be possible to buffer the effects of at least some susceptibility factors through use of family-based strategies that promote stabilizing behaviors (e.g., modeling of healthy, non-restrictive eating on the part of parents, regular family meals) and in doing so prevent the development of full-blown illness in high risk individuals, remains an important question for future study.

The Role of the Parents in the Resolution of Eating Disorders

Treatment studies, especially those utilizing parents in therapy, can help in our understanding of the role of the family in eating disorders. However, controlled treatment trials for both AN and BN remain small in number. Even so, a consistent finding in work completed recently is that family involvement appears to be useful in reducing both psychological and medical morbidity, especially for younger patients with a short duration eating disorder,^{24–26} and that this form of treatment is acceptable to parents and patients alike.^{27,28} Moreover, utilizing parents in treatment could at least in part explain lower attrition rates in adolescent treatment studies, roughly 15%, compared to treatment studies of adults where drop out rates average 50%.²⁹ Clearly, more definitive, randomized controlled studies of family therapy are needed along

with studies comparing family therapy to other modalities of treatment³⁰ and research assessing the value of skills based learning for carers of adults with eating disorders.³¹

The applicability of family therapy for BN has been the subject of only a few studies.^{32,27,33,34} For the subset of adults with BN who were treated with family therapy in the original Russell et al. study,³² the effects of family therapy were small. Subsequently, after a small case series demonstrated the feasibility of family therapy for adolescent BN,³³ two randomized clinical trials demonstrated that family treatment may be useful for some adolescents with BN.^{27,34} Schmidt et al. found that family therapy compared favorably to a self-help form of cognitive behavioral therapy for adolescents with BN, but that more adolescents refused family treatment.³⁴ Le Grange et al.²⁷ found that manualized family-based treatment³⁵ was superior to a non-specific individual therapy, but the proportion of participants showing robust improvement was small. Taken together, these results suggest that family-based treatment methods may hold promise for some adolescents with BN, but definitive studies are sorely needed.

Family members caring for persons with an eating disorder often experience general distress, anxiety, and diminished quality of life.^{36–38} It is therefore hypothesized that reducing caregiver strains might be important to improving outcome.³⁷ While attempts have been made to better understand both positive and stressful components of caregiving, there is still limited knowledge of how to best understand and ameliorate caregiver distress, especially when patients remain seriously ill.³⁹ Literature for parents is enormously important in providing education about the illnesses in the hope of reducing levels of distress, negative caregiving experiences, as well as guilt and shame.^{31,40,41}

Discussion

There appears to be no consistent structure or pattern of functioning in families with a member who suffers from an eating disorder; rather, eating disorders evolve a multiplicity of family contexts. As the role of family life in a child’s emotional and psychological well-being is uncontested, so too is the potential value of including families in the treatment of children and adolescents, and in identifying resources within the family that can hasten

recovery. Just the same, although no evidence exists supporting the concept of “anorexogenic” parents or families, in certain cases involvement of the family may be clinically contraindicated (e.g., severe parental psychopathology). Thus, the assessment of families requires close attention to the parents’ competencies, motivation, and history of adverse or traumatizing events. But even when such adverse circumstances are present, the development of a play to help and support sufferers and how to ease family burdens should take precedence over accusation and blame. Thus, it is our position that families should be involved routinely in the treatment of most young people with an eating disorder. Exactly how such involvement should be structured, and how it will be most helpful will vary from family to family.

The authors thank Michael Strober, PhD, for his invaluable contribution in the final version as well as earlier drafts of this paper. The authors would also like to thank the AED Board of Directors for their careful review of this paper.

References

1. Klump KL, Bulik CM, Kaye WH, Treasure J, Tyson E. Academy for eating disorders position paper: Eating disorders are serious mental illnesses. *Int J Eat Disord* 2009;42:97–103.
2. Dare C, Le Grange D, Eisler I, Rutherford J. Redefining the psychosomatic family: The pre-treatment family process in 26 eating disorder families. *Int J Eat Disord* 1994;16:211–226.
3. Silverman J. Charcot’s comments on the therapeutic role of isolation in the treatment of anorexia nervosa. *Int J Eat Disord* 1997;21:295–298.
4. Minuchin S, Rosman B, Baker L. *Psychosomatic Families: Anorexia Nervosa in Context*. Cambridge: Harvard University Press, 1978.
5. Humphrey LL. Observed family interactions among subtypes of eating disorders using structured analysis of social behavior. *J Consult Clin Psychol* 1989;57:206–214.
6. Dare C, Eisler I. Family therapy for anorexia nervosa. In Garner DM, Garfinkel PE, Editors. *Handbook of Treatment for Eating Disorders*. New York: The Guilford Press, 1997, pp. 307–324.
7. Jacobi C, Hayward C, De Zwaan M, Krawmer HC, Agras S. 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.
8. Kazdin AE, Kraemer HC, Kessler RC, Kupfer DJ, Offord DR. Contributions of risk-factor research to developmental psychopathology. *Clin Psychol Rev* 1997;17:375–406.
9. Horesh N, Apter A, Ishai J, Danziger Y, Miculincer M, Stein D, et al. Abnormal psychosocial situations and eating disorders in adolescence. *J Am Acad Child Adolesc Psychiatry* 1996;35:921–927.
10. Shoebridge P, Gowers SG. Parental high concern and adolescent-onset anorexia nervosa: A case-control study to investigate direction of causality. *Br J Psychiatry* 2000;176:132–137.
11. Welch SL, Doll HA, Fairburn CG. Life events and the onset of bulimia nervosa: A controlled study. *Psychol Med* 1997;27:515–522.
12. Graber JA, Brooks-Gunn J, Paikoff RL, Warren MP. Prediction of eating problems: An 8-year study of adolescent girls. *Dev Psychol* 1994;30:823–834.
13. McKnight Investigators. Risk factors for the onset of eating disorders in adolescent girls: Results of the McKnight longitudinal risk factor study. *Am J Psychiatry* 2003;160:248–254.
14. Nicholls D, Viner R. Childhood risk factors for lifetime anorexia nervosa by age 30 years in a national birth cohort. *J Am Acad Child Adolesc Psychiatry* 2009;48:791–799.
15. Beato-Fernandez L, Rodriguez-Cano T, Belmonte-Lario A, Martinez-Delgado C. Risk factors for eating disorders in adolescents: A Spanish community-based longitudinal study. *Eur Child Adolesc Psychiatry* 2004;13:287–294.
16. Johnson JG, Cohen P, Kasen S, Brook JS. Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. *Am J Psychiatry* 2002;159:394–400.
17. Striegel-Moore RH, Bulik CM. Risk factors for eating disorders. *Am Psychol* 2007;62:181–198.
18. Bulik CM, Reba L, Siega-Riz AM, Reichborn-Kjennerud T. Anorexia nervosa: Definition, epidemiology, and cycle of risk. *Int J Eat Disord* 2005;37:S2–S9.
19. Stein A, Woolley H, Cooper S, Winterbottom J, Fairburn CG, Cortina-Borja M. Eating habits and attitudes among 10-year-old children of mothers with eating disorders: Longitudinal study. *Br J Psychiatry* 2006;189:324–329.
20. Mazzeo SE, Zucker NL, Gerke CK, Mitchell KS, Bulik CM. Parenting concerns of women with histories of eating disorders. *Int J Eat Disord* 2005;37:S77–S79.
21. Loeb KL, Le Grange D, Lock J. *Family-based treatment for the prevention of anorexia nervosa*. Mount Sinai School of Medicine, New York, 2005.
22. Neumark-Sztainer D, Eisenberg ME, Fulkerson JA, Story M, Larson NI. Family meals and disordered eating in adolescents: Longitudinal findings from Project EAT. *Arch Pediatr Adolesc Med* 2008;162:17–22.
23. Martinez-Gonzalez MA, Gual P, Lahortiga F, Alonso Y, De Irala-Estevez J, Cervera S. Parental factors, mass media influences, and the onset of eating disorders in a prospective population-based cohort. *Pediatrics* 2003;111:315–320.
24. Eisler I, Dare C, Russell GFM, Szmukler GI, Le Grange D, Dodge E. Family and individual therapy in anorexia nervosa: A five-year follow-up. *Arch Gen Psychiatry* 1997;54:1025–1030.
25. Eisler I, Simic M, Russell G, Dare C. A randomized controlled treatment trial of two forms of family therapy in adolescent anorexia nervosa: A five-year follow-up. *J Child Psychol Psychiatry* 2007;48:552–560.
26. Lock J, Couturier J, Agras WS. Comparison of long term outcomes in adolescents with anorexia nervosa treated with family therapy. *Am J Child Adolesc Psychiatry* 2006;45:666–672.
27. Le Grange D, Crosby R, Rathouz P, Leventhal B. A randomized controlled comparison of family-based treatment and supportive psychotherapy for adolescent bulimia nervosa. *Arch Gen Psychiatry* 2007;64:1049–1056.
28. Lock J, Agras WS, Bryson S, Kraemer H. A comparison of short- and long-term family therapy for adolescent anorexia nervosa. *J Am Acad Child Adolesc Psychiatry* 2005;44:632–639.
29. Halmi CA, Agras WS, Crow SJ, Mitchell J, Wilson GT, Bryson S. Predictors of treatment acceptance and completion in anorexia nervosa: Implications for future study designs. *Arch Gen Psychiatry* 2005;62:776–781.
30. Le Grange D, Eisler I. Family interventions in adolescent anorexia nervosa. *Child Adolesc Psychiatr Clin N Am* 2009;18:159–173.
31. Treasure J, Smith G, Crane A. *Skills-based Learning for Caring for a Loved One with an Eating Disorder: The New Maudsley Method*. London: Routledge, 2007.

32. Russell GFM, Szmukler GI, Dare C, Eisler I. An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Arch Gen Psychiatry* 1987;44:1047–1056.
33. Dodge E, Hodes M, Eisler I, Dare C. Family therapy for bulimia nervosa in adolescents: An exploratory study. *J Fam Ther* 1995;17:59–77.
34. Schmidt U, Lee S, Beecham J, Perkins S, Treasure JL, Yi I. A randomized controlled trial of family therapy and cognitive behavior therapy guided self-care for adolescents with bulimia nervosa and related conditions. *Am J Psychiatry* 2007;164:591–598.
35. Le Grange D, Lock J. *Treatment Manual for Bulimia Nervosa: A family-based Approach*. New York: The Guilford Press, 2007.
36. Treasure J, Whitaker W, Whitney J, Schmidt U. Working with families of adults with anorexia nervosa. *J Fam Ther* 2005;27:158–170.
37. Kyriacou O, Treasure J, Schmidt U. Understanding how parents cope with living with someone with anorexia nervosa: Modelling the factors that are associated with carer distress. *Int J Eat Disord* 2008;41:233–242.
38. Cottee-Lane D, Pistrang N, Bryant-Waugh R. Childhood onset anorexia nervosa: The experience of parents. *Eur Eat Disord Rev* 2004;12:169–177.
39. Szmukler GI. Caregivers for people with mental health problems. *Eat Disord Rev* 2007;18:7.
40. Lock J, Le Grange D. *Help your teenager beat an eating disorder*. New York: Guilford Press, 2005.
41. Bryant-Waugh R, Lask B. *Eating Disorders: A Parents Guide*, Revised ed. New York: Brunner-Routledge, 2004,