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on

Risk and protection factors for anorexia nervosa

by

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Contents:

1. Introduction

There are some crucial points to make before we start to discuss risk and protection factors for anorexia nervosa (AN):

First of all, we need to remember the importance of multidimensional models of aetiology (Garner, Garfinkel et al. 1982), (Gillberg C 1998), (Treasure and Holland 1995), and thus the relevance of contributions from various scientific fields as there are biology, psychology, developmental psychopathology, and sociology for explanation of aetiology and maintenance of psychopathology in general and the severe illness AN, in particular.

Second, some factors can serve as risk and protective factors for disordered eating behaviour, depending on the processes of risk, the age of the individual at which the factor is working, etc. (Kraemer, Stice et al. 2001).

Third, there is an interdependence of risk factors (with mediating and moderating mechanisms poorly understood to date (Kraemer, Stice et al. 2001) from all fields and no scientific area can claim to have an exclusive interpretation of aetiological relevance for this severe disorder. Thus, a distinction into e.g., genetic, psychological, etc. areas of interest is arbitrary, as integration of these various approaches is the most interesting and difficult task in clarifying aetio-pathogenesis of AN.

The chapter tries to give an overview about some crucial methodological issues of risk factor research, some important results of recent studies of risk and also of the few protection factors found for eating disordered behaviour and wants to emphasise in particular the field of individual-specific risk for AN summarising recent findings from a behaviour-genetic point of view.

2. Methodological issues

2. 1 Risk factor definition

Studies concentrating on pre-morbid psychosocial, psychological and environmental problems in eating disorders have been performed since many decades. Unfortunately, there has been a lack of clarity in some crucial methodological issues as there is risk factor terminology. Recently, much progress has been made in terminology of risk factors through the work of Kraemer and Kazdin ((Kraemer, Kazdin et al. 1997), (Kazdin, Kraemer et al. 1997)). Unfortunately, these clarifications have not often been taken into account in recent studies.

A variable shall be called "a risk factors" if there are at least the following pre-requisites fulfilled (acc. to (Kraemer, Kazdin et al. 1997) and (Kazdin, Kraemer et al. 1997):

(1) The variable precedes the outcome of interest. This needs careful clarification of the time-line of onset of AN and investigating of the variables which have been experienced before onset.

(2) Most retrospectively investigated factors are correlates or, if they have preceeded the outcome of interest, retrospective correlates (variable risk factors).

(3) The variable shows a dose-outcome relationship. Experiencing a larger number of risk factors enhances the liability (probability) to suffer from AN within a defined population.

(4) Putative risk factors occur in various fields: extrinsic (such as adverse events) and intrinsic (such as genetic vulnerability). They may act as predisposing, triggering and maintaining factors.

(5) Risk factors of causal relevance can be manipulated, which would change the outcome. Most factors cannot be changed (e.g. race, gender, year of birth, genotype) and are therefore called fixed markers.

Only one review to date has tried to clarify the terminology of risk factors in the field of eating disorders (Jacobi C in preparation), (Jacobi C 2002). The progress made with this report will be fruitfully used in further risk factor studies and will set standards for methodological clarity.

2. 2 Risk factor study designs

2.2.1 Cross-sectional studies

Cross-sectional studies, aiming to identify variables as being correlated and associated with the disorder result in information about correlates, not risk factors. As sometimes statistical methods have been used which result in risk ratios (odds ratios) they should not be interpreted as predicting the outcome or giving any information about causality (Kraemer, Yesavage et al. 2000). Correlates relevant in currently ill AN patients may give a hint, which factors should be looked at also in retrospective or prospective studies.

2.2.2 Retrospective studies

Retrospective studies, however, can result in correlates and risk factors. If we take into account that retrospective accounts are biased by the present disorder (e.g. mood states bias the memory (Brewin, Andrews et al. 1993), (Maughan and Rutter 1997), (Richter J 2002), and memories are always a

subjective anew construction at any given time and not a given fact themselves), then retrospective accounts of experiences which happened before onset of AN are correctly called retrospective correlates. If we use behavioural descriptions of variables and use interview based methodology, examining the period before the occurrence of symptoms, we do our best to enhance reliability of retrospective accounts (Brewin, Andrews et al. 1993), (Maughan and Rutter 1997), and can find putative (variable or fixed) risk-factors. This has been done recently in some large population based studies (e.g. (Fairburn, Cooper et al. 1999)).

2.2.3 Prospective studies

The best way of risk factor research excluding the bias problems that retrospective studies have inherent would be prospective longitudinal studies with follow-up of large (birth-) cohorts. There is an eminent problem in AN research with this, cause the incidence of AN is very low, which would require very large samples of many thousands to find a sufficient number of AN cases at follow-up requiring formidable amounts of money and efforts. This makes prospective risk factor studies much more feasible for bulimia nervosa and disordered eating not reaching diagnostic thresholds which are much more common eating problems (e.g. a recent study (Johnson, Cohen et al. 2002)) has investigated 782 families from childhood to adulthood and found 52 eating disordered patients with only one having AN, 10 having BN and the rest having EDNOS).

2.2.4 High-risk studies

Specific mainly prospective studies are high-risk studies. Adolescent daughters of mothers (and fathers) with former eating disorders in general or AN, in particular, could be a high-risk population from a psychological and genetic point of view e.g. (Russell, Treasure et al. 1998), reviewed recently by (Patel, Wheatcroft et al. 2002). Several mechanisms could work setting these children at higher risk: genetic influences, wishing their children to be thinner, problems in general parenting, poor role modelling for eating attitudes and behaviours, and marital discord (Patel, Wheatcroft et al. 2002). Also specific populations with possibly high cultural risk loadings like athletes (Davis and Strachan 2001), (Smolak, Murnen et al. 2000) and ballet dancers ((Dotti, Fioravanti et al. 2002)) etc. are candidates, however, the latter cannot help us to generalize findings to all eating disorder types gathered in these specific environments.

3 Some important results on risk factors for anorexia nervosa

There are numerous relatively recent reviews of risk factors for eating disorders in the scientific literature from different perspectives (e.g. (Steiner H 1995), (Schmidt, Humfress et al. 1997), (Tyrka, Graber et al. 2000), (Keel, Leon et al. 2001), (Stice 2001), (Shisslak and Crago 2001), (Rastam and Gillberg 1991), (Schmidt, Tiller et al. 1993), (Smolak, Levine et al. 1996), (Steiner and Lock 1998)). In the following we are summarising some important results and recent reports.

3.1 Genetic risk (Twin studies, family studies, molecular genetic studies)

Much progress have been made in molecular genetics and behavioural genetics, being now also used in the field of eating disorders. As it is an important issue in molecular genetics, that findings have to be replicated in different samples from different cultures, no definite conclusion is possible at the moment. In the chapter by Halmi (this volume) the genetic issues in eating disorders are reviewed in detail. For our purpose we summarise only some recent ones, which give a hint where studies focussing on gene-environment correlation and interaction could be sensible.

Genetic research in eating disorders relies on family studies, twin studies, studies of chromosomal abnormalities, and molecular genetic studies (reviewed in (Strober 1991), (Strober 1991), (Woodside 1993), (Treasure and Holland 1995), (Hebebrand and Remschmidt 1995), (Klump, Kaye et al. 2001), (Gorwood, Bouvard et al. 1998), (Spelt and Meyer 1995), (Hinney, Remschmidt et al. 2000), (Collier, Sham et al. 1999), (Collier 2002)). It is important to emphasize, that in complex disorders etiologically relevant genes, once they are found, contribute to the development of the disorder, but play only one role within multidimensional models (Plomin and Rutter 1998). Genes "are neither necessary nor sufficient" ((Collier 2002)) for the development of AN.

Twin studies in AN

Twin studies indicate a greater concordance rate of AN in monozygotic (MZ) than in dizygotic (DZ) twins ((Fairburn, Cowen et al. 1999), (Bulik, Sullivan et al. 2000)). In AN concordance rate for MZ twins is high and - depending on the phenotypical spectrum - ranges from 30 to 65 % which is significantly greater than the concordance rate in DZ twins which is between 0 and 9 %.These figures

suggest that genetic factors are of importance in AN. However, environmental factors, specifically experienced by a person are responsible for between 30 and 65 % of the variance.
An etiological model of AN predicts that in an environment in which there are specific triggers such as stress and an emphasis in food restriction, those who are genetically vulnerable would be highly likely to develop AN.

Family studies in AN

If a proposed trait is to be considered genetic, clustering of an illness within families is necessary. A genetic component of AN was first considered when Theander (Theander 1970) noted an increased prevalence of AN in the sisters of anorexic patients. The prevalence rates in sisters range between 3 and 7 %. The rate of eating disorders in female first-degree relatives ranges from 4 to 10 %. Female relatives have an up to six-fold greater risk of developing an eating disorder than controls (e.g.(Strober, Morrell et al. 1985)). There have been several studies investigating the familial clustering of eating disorders with other psychiatric comorbidity (affective disorders, alcoholism, obsessive-compulsive disorders, obesity). Higher prevalences of affective disorders, and obsessive-compulsive disorders were found in first-degree relatives in BN and AN respectively, compared with controls. Co-twins of twins with AN were at significantly higher risk for life-time AN, BN, major depression, and current low body mass index (kg/m^2). However, given the rarity of eating disorders among relatives of patients with affective disorders (unipolar or bipolar disorder) it seems unlikely that affective disorders and eating disorders share a unitary familial liability, although the two conditions frequently coexist.
A family history of an eating disorder, mood disorder or perhaps alcohol and substance related disorder seems to enhance the risk for AN or BN in adolescents ((Hsu 1997), (Fairburn, Cooper et al. 1999), (Fairburn, Welch et al. 1997)).
There is evidence that both eating disorders run in families and that genetic as well as environmental factors are important. Family studies can not discriminate between inherited factors and environmentally transmitted factors. For this distinction, twin and twin/adoption studies are needed. Adoption studies are not existent in the AN field.

Molecular genetic studies in AN

As an example, a first case-control study carried out at the Institute of Psychiatry in London reported an association between the -1438 G/A polymorphism in 81 patients with AN, in which allele A and the A/A genotype was in excess compared with 226 controls. This polymorphism in the promoter region

indicated that the 5-HT_{2A} gene may be a susceptibility factor for AN. The 5-HT_{2A} receptor gene was analysed as a candidate for anorexia since: (1) it is involved in the regulation of appetite; (2) it is under the regulation of oestrogen, of which levels change during the onset time of anorexia near puberty; (3) it mediates the action of the antipsychotic drug clozapine, which induces weight gain. These findings have been replicated by three other research groups but could not be detected by another two and not by the largest molecular genetic study to date on a 5HT2A receptor gene polymorphism which also did not show a significant association of this marker with AN ((Gorwood, Ades et al. 2002)).
Concluding, it seems that there is some evidence that research is on the way to elucidate the role of the genes for the development of AN using molecular genetic strategies.

A recent linkage study (using affected sib-pairs) has found linkage of a specific marker on chromosome 1 with anorexia nervosa restrictive type ((Kaye, Lilenfeld et al. 2000), (Grice, Halmi et al. 2002), (Klump, Kaye et al. 2001)). Furthermore, a study incorporating the two behavioural covariates drive for thinness and obsessionality, three regions with suggestive linkage have been found on chromosomes 1, 2 and 13 (Devlin, Bacanu et al. 2002). However, these results have to be replicated in other samples.

3.2 Personality traits

It has been known for years that some personality traits are shared by patients with AN ((Vitousek and Manke 1994), (Sohlberg and Strober 1994)), however, it is difficult to distinguish traits which predispose to the illness and those which are a consequence of the disorder.
There are two well investigated personality traits among others, which are exceptionally important in AN: perfectionism and temperamental traits. Perfectionism ((Shafran and Mansell 2001)) is the strongest candidate for being related to AN yet being included into risk models ((Slade 1982), (Vitousek and Manke 1994)). It has been found before onset ((Fairburn, Cooper et al. 1999)), enhanced during illness ((Halmi, Sunday et al. 2000), [(Davis, Claridge et al. 2000), (Kaye, Greeno et al. 1998)) and present after recovery ((Srinivasagam, Kaye et al. 1995), (Bastiani, Rao et al. 1995)). However, no study to date has followed one sample prospectively from the pre-morbid to the post-illness period. Perfectionism has also been identified as risk factor for bulimic behaviour in women perceiving themselves as overweight ((Joiner, Heatherton et al. 1997)). Family members of AN patients had higher rates of obsessive-compulsive personality disorder (ODPD) and higher levels of

perfectionism. AN and OCPD may share familial risk factors ((Lilenfeld, Stein et al. 2000), (Price Foundation Collaborative Group 2001)).

Temperament is another very well investigated feature in AN. Beside numerous smaller studies, the largest one (Klump, Bulik et al. 2000) found higher harm avoidance and lower cooperativeness scores in An patients compared with healthy controls and lowest novelty seeking scores in restricting and purging AN. All differences found were independent form body weight.

A variety of personality traits are substantially influenced by genetic factors. Cloninger ((Cloninger 1986), (Cloninger 1998)) theorized about four genetically independent and highly heritable *temperamental personality traits*: novelty seeking, harm avoidance, persistence, and reward dependence and three *character dimensions*: self-directedness, cooperativeness, self-transcendence. In particular, subjects low on novelty seeking are seen as emotionally restrained, slow to change interests, methodological, and rigidly persistent. When harm avoidance and reward dependence are high the subject is found to be an obsessive worrier prone to anxious, dysphoric mood, unusually sensitive to the feelings and needs of others and prone to highly driven and repetitive reward-seeking behaviour even when frustrated and disappointed. All eating disordered patients were found to be high in harm avoidance; bulimic patients were also high in novelty seeking, whereas restrictive anorexics were high in reward dependence and persistence. Concerning the character dimensions, the finding of low self directedness in distinct bulimic patients could serve as a predictor of poor treatment outcome in this patient group.

These temperamental factors may interact with environmental conditions in AN. The result of genetic endowement and early learning experiences, individual positions on the three temperamental dimensions are deeply rooted and set powerful limits on affective experience and behaviour, strongly influencing the form of psychopathology.

Furthermore, specific neurotransmitter systems have been postulated and in part proved to be correlated with these temperamental traits. It seems that the temperamental dimensions are possible behavioural indicators of dopaminergic (novelty seeking), serotonergic (harm avoidance), and noradrenergic (reward dependence) functioning.

Thus, perfectionism and temperamental traits seem to be good candidates for studies with behavioural-genetic designs, looking at the role of gene-environment interaction in eating disorder aetiopathology.

3.3 Self-esteem

Children with low self-esteem seem to be at a high risk to develop an eating disorder in adolescence. The risk was reported to be eight times higher than in those with high self-esteem ((Button, Sonuga-Barke et al. 1996)), however, the participants were not assessed at baseline for eating disorder symptomatology. A low opinion of the self enhances the drive to be perfect and attain perfection in all areas of (academic as well as personal) life. Negative self-evaluation as found pre-morbidly via retrospective assessment by Fairburn (Fairburn, Cooper et al. 1999) seems to be a relatively potent risk factor for AN. High levels of shyness, loneliness and feelings of inferiority has been reported in adolescence preceding AN of the binge/purging type but not in AN of the restricting type (Troop and Bifulco 2002).

3.3 Environmental risk

3.3.1 Familial risk

An enormous literature has examined family factors, family interaction and family environments of AN patients ((Vandereycken W 1989)). Dieting by mothers and fathers predicted dieting concerns and behaviours in adolescent girls ((Smolak, Levine et al. 1999), (Stice, Agras et al. 1999)) as did encouragement by parents to loose weight (Smolak, Levine et al. 1999). Maternal restraint and disinhibition (assessed at birth) predicted higher body dissatisfaction and dieting behaviours even at the age of 8 (as found prospectively by Jacobi et al. (Jacobi, Agras et al. 2001)).

Some characteristics of families with an eating disordered child have been found (review by (Karwautz A 2002), (Vandereycken W 1989)), however, it remains unclear if specific interaction patterns exist which could be causally related to the onset of eating disorders, but it seems that this is not the case. Also some longitudinal studies have not proven specific interaction patterns as predictive for AN or BN ((Attie, Brooks Gunn et al. 1990), (Graber, Brooks Gunn et al. 1994)). Adversity in childhood does not predict AN as measured using the "Childhood Experiences of Care and Abuse interview" ((Webster and Palmer 2000)).

Summarizing the literature on family interaction, no marked differences have been found between families with an anorexic child and those without. AN seems to affect family functioning in a way similar to other chronic conditions such as diabetes. It has become clear that the family can contribute

much to overcoming the eating disorder. To blame the family for causing the illness is not appropriate based on evidence from research.

3.3.2 Life events

It has been recognized for centuries that psychosocial difficulties are important triggers to the illness and indeed these are now incorporated into some of the recent proposed definitions of eating disorders ((Russell 1995), (Szmukler and Patton 1995)). Environmental factors have even greater importance for BN than for AN. Life events and difficulties frequently preceed AN and BN. The difficulties commonly involve interpersonal difficulties such as teasing and bullying at school or negative life events concerning their parents. Pudicity events before onset appear to be of particular relevance for AN but they only occur in 20 % of the cases. A more general effect might be a life event which engender a conflict of loyalities, or an ethical dilemma e.g. need to suppress emotional reaction for a rational position.

However, in themselves, life events are mostly common experiences and cannot explain the specific development of an eating disorder. One suggestion to explain specificity in the psychological consequences of the event is that it is the meaning of an event that is significant; loss events are associated with depression; threat events with anxiety. Pudicity events associated primarily with sexual disgust appear to be common in anorexia nervosa, which raises the possibility that events in which the meaning is linked to the emotion of disgust and shame may trigger eating disorders. An alternative explanation may be that the response to the event is an important component in shaping the outcome. Patients were found to have a more *helpless coping response* ((Troop, Holbrey et al. 1994), (Troop, Holbrey et al. 1998)); patients with anorexia nervosa used avoidance strategies and those with bulimia nervosa in contrast ruminated on their problems. It is uncertain whether these varieties of coping style arise as consequences of the meaning of the event, as a result of developmental experiences or temperamental predisposition.

3.3.3 General cultural risk

Females are at a much higher risk than males (90-96 % females) (Gender is a potent non-specific fixed marker). Individuals in their mid teens are at highest risk for AN and in those in their late teens and early twens for BN. People from the white race are at higher risk (Gray et al. 1987, Fisher et al. 1994, Rand et al. 1990). Individuals with a heterosexual orientation seem to be more prone to the

illness. There seems to be no high correlation between social-class and the development of eating disorders. However, adolescents from a higher social class were found to desire a lower body weights, to diet more often, to binge more severely, and to exercise more for weight control (Drewnowski et al. 1994).

3.3.4 Peer group issues and social support

Children with higher scores on the Eating Attitudes Test believed that they would be better liked by their friends whether they would be thinner and engaged in dieting to be better liked by their peers (Maloney MJ 1989), (Oliver and Thelen 1996)). Teasing of girls by peers when gaining weight during adolescence causes negative feelings towards body shape and further development of eating disorders ((Thompson, Coovert et al. 1995)).

The perceived adequacy of the available social support is thought to influence vulnerability for mental disorders in general. Anorexia nervosa patients and BN patients have deficits in their social network. Lacking premorbid social adjustment seems to be a predictor of bad outcome in AN patients. BN patients have disturbances in both the size and perceived adequacy of social relationships. This highlights the importance of a good social network and good social adaption for protecting the development of eating problems.

3.3.5 Traumatic experiences:

For a detailed discussion of the relevance of experienced sexual abuse see this book chapter by Vanderlinden. This very important topic is extensively discussed in the recent articles (Schmidt, Humfress et al. 1997), (Fallon and Wonderlich 1997), (Wonderlich, Brewerton et al. 1997), (Wonderlich, Crosby et al. 2000), (Smolak and Murnen 2002), (Connors 2001), (Molinari 2001), (Connors and Morse 1993). Premorbid repeated sexual abuse and repeated severe physical abuse has been reported by bulimic subjects. Summarizing the literature, there is evidence that sexual abuse is a non-specific risk factor (retrospective correlate) for bulimia nervosa and in particular when there is psychiatric comorbidity, and is more strongly associated with bulimia than with restrictive anorexia nervosa (Fairburn, Cooper et al. 1999). There are few data on the moderation and mediation effects of other factors on the relation of sexual abuse and disordered eating.

3.3.6 Developmentally specific risk factors

Preschool years:

Optimality in the pre-, peri-, and neonatal period was found not to be reduced in later eating disordered subjects. Very preterm birth increased the risk 3 fold to develop AN ((Cnattingius, Hultman et al. 1999). In early childhood, digestive problems and picky eating ((Marchi and Cohen 1990) were found to predate anorectic behaviour and eating related fighting during mealtimes seemed to predict later bulimic behaviour. Furthermore, later anorexic adolescents have suffered more often from bellyache and severe gastrointestinal problems (47%) in childhood .Eating conflicts, struggles with food, and unpleasant meals in childhood (1-11 yrs of age) are shown to be risk factors for the later development of eating disorders, however, again the numbers of full diagnostic cases are very low (AN: n=4 vs BN: n=14 in late adolescence) (Kotler, Cohen et al. 2001).

School-age years:

It is important to know that nearly the half of children in elementary school desire to be thinner than they are and that more than a third tried some form of weight loss. This is alarming because negative feelings about the own body set the these girls at higher risk for developing eating problems. However, fortunately, only a small part of those children develop an eating disorder in a clinical sense.

Puberty:

Eating problems can be understood as response to developmental changes. Numerous studies reported on this issue ((Crisp 1980), (Attie, Brooks Gunn et al. 1990), (Attie and Brooks Gunn 1992), (Smolak and Levine 1996). To understand why AN and BN usually start in adolescence it is important to emphasize which role the *transitions of early adolescence and late adolescence* play for their development ((Smolak and Levine 1996)): **Transitions** involve reorganizations in personality, cognitive structures, relationships, and social roles. They require adaption, which can be positive or - under certain circumstances - problematic, depending on personality characteristics and the surrounding social system. Transitions follow an extended stress-coping (predispositon-trigger-adaption) model. As predisposition serve the previous development and the repertoire of behaviour, personality, cognitions, emotions, and the characteristics of social relationships. The transition is then triggered by internal (psychological or physical changes) and external events or changes (e.g. role expectations). To stand the developmental normative tasks adaption is required.

Predispositions of the development of eating disorders during the early adolescence transition are perfectionism, low-self-esteem, and repeated dieting. *Triggers* are changes in peer and family relationships, academic demands, and social role expectations, the biological correlates and the social

construction of puberty, early maturation and the recognition of the high value of attractiveness in society for success.

Predispositions of the late adolescence transition are the same as in the early transition, however, they are more consolidated: a longer history of dieting behaviour, the view of attractiveness and thinness as important for success, low self-esteem, self-consciousness and social anxiousness, and the need for social approval are important.

Triggers of eating disorder in that transition from adolescence to adulthood are sometimes pregnancies, finding a job, marrying, starting high school resulting in less social support.

A developmentally appropriate model emphasizes that a combination of different predispositions and various triggers increases the likelihood of developing an eating disorder ((Tyrka, Graber et al. 2000), (Keel, Leon et al. 2001), (Stice 2001), (Shisslak and Crago 2001)).

4 Risk factor models

To understand who is at risk to develop an eating disorder risk factor models have been created:

The development of AN is best explained by multidimensional or bio-psycho-social models (e.g. by (Garner, Garfinkel et al. 1982), (Johnson and Connors 1987), (Halmi and Garfinkel 1995),(Connors 1996), (Smolak, Levine et al. 1996), (Treasure, Collier et al. 1997), (Gillberg C 1998)).

Both disorders may be preceded by dieting (Hsu 1997). However, dieting is a common behaviour in adolescence and early adult age in westernised countries and not every woman develops an eating disorder. Female subjects who dieted were 18 times more likely to develop an eating disorder than those who did not. Controlling weight by exercise rather than dieting contributed less to the liability to develop an eating disorder (Patton, Selzer et al. 1999). However, again there was no full case of AN detected in the 3-year follow-up period.

In addition to dieting, putative risk factors are *biological vulnerability* (e.g. genetic, susceptibility to dysregulation of neurotransmitter systems which regulate eating behaviour), and *psychological vulnerability* (e.g. predisposing personality traits, difficulties in the management of affect, developmental experiences, family conflicts, intrapsychic conflicts).

An aetiological model of AN must also take into account the clinical and epidemiological pattern of this disorder, such as the age of onset, and the sex ratio. The relative stability in the incidence of AN over time and across cultures suggests that environmental explanations will need to be general or that

individual factors may be more pertinent. In contrast, the exponential rise in bulimia nervosa (BN) could be explained by some environmental aetiological factors. Dieting appears to be a prime candidate (Hsu 1997). However, this cannot explain individual susceptibility which is more likely to be explained by specific environmental factors and/or individual vulnerability.

Several risk factors have been identified for the development of psychiatric disturbance in general and for eating disorders (and more specifically for AN) in particular. This approach of measuring unspecific and specific putative risk factors has been used extensively by Fairburn et al. in their risk-factor studies on AN, BN, and BED ((Fairburn, Welch et al. 1997; Fairburn, Doll et al. 1998; Fairburn, Cooper et al. 1999)).

Risk factors for psychiatric disturbances are e.g. temperamental factors, problematic family environments characterized by a preponderance of negative interactions such as blaming and over-control, lack of parental support, lack of empathy and nurturance, and stressful life events such as sexual abuse or other traumatic events.

Beside the more general factors for psychopathology and difficulties in self-regulation (Connors 1996), more specific factors need to be present as prerequisites for the development of an eating disorder. These are risk factors for body dissatisfaction are e.g. higher body weight, experiences of having been teased about the own weight or size, parents who themselves dieted, cultural norms of weight and shape. A *combination of risk factors from both areas* could then lead to an eating disorder, which has been hypothesized (but not tested so far) by a convincing two-factor model developed by Connors (Connors 1996). Low self esteem, insecurity in relationships, and difficulties in managing affects, which are problematic for many adolescents, combined with body dissatisfaction could lead to marked dieting behaviour. Thinness as a solution to parental criticism and relationship insecurity is then therefore seen in women with low perceived self worth.

Another two-factor model indicates that low body esteem and maladaptive personality independently and interactively contribute to the likelihood of having an eating disorder (McLaren, Gauvin et al. 2001), however, the study was cross-sectional in nature resulting in correlates rather than risk factors.

5 Individual specific issues in risk factor research

5.1 Designs derived from behaviour genetics

As it is known from twin studies with modelling approaches of twin data, that there are additional genetic variance (A), shared environmental factors (common=C), and non-shared environmental factors (E) which are relevant for psychopathology (Plomin et al. 2001, (Reiss, Neiderhiser et al. 2000), (Hoffman 1991)). Individual-specific environment (E, non-shared environment = experiences which are unique to each sibling reared in the same family) in all investigated fields of psychopathology is the most relevant beside genetic variance. In eating disorders, there are only a few recent studies dealing with the role of non-shared environment. Recently, a detailed review of behavioural genetic strategies used in eating disordered populations up to 2000 has been published ((Klump, Wonderlich et al. 2002)).

Several twin studies have been published ((Wade, Martin et al. 2000), (Wade, Martin et al. 1999), (Klump, McGue et al. 2002), (Klump, McGue et al. 2000)) which are per se genetically informed. We know from these recent studies, that the non-shared part of the variance explaining case-ness in twins is large for AN (24-42 %), BN (17-46 %) and disordered eating behaviour (28-68 %) ((Klump, Wonderlich et al. 2002)). Several questions of etiological relevance have been risen from the view of behaviour genetics in order to ideally focus risk factor research: which risk factors are highly heritable, which are results from shred familial influences, which are largely a consequence of indiviual-specific experiences, and to what extent changes the vulnerability by age (Hewitt 1997).

5.2 Recent studies

Risk factor research with behavioural sensitive designs require the pre-requisites of general risk factor research, however, the inclusion of more than one person per family is needed. To our knowledge, there are only a few studies using this approach in risk factor studies in eating disorders.

We have retrospectively investigated 45 sister pairs discordant for AN (Karwautz, Rabe Hesketh et al. 2001) using the Oxford Risk Factor Interview developed by Fairburn (Fairburn, Welch et al. 1997) and the Sibling Inventory for Differential Experiences (SIDE) developed by Daniels and Plomin (Daniels and Plomin 1985). Nearly all risk factors for AN found by Fairburn (Fairburn, Cooper et al. 1999) using a case-control design with unrelated controls were also found by us in this study using a within-family design (Dick, Johnson et al. 2000), therefore, corroborating that these risk factors are non-shared in

nature. In particular, the risk factors in the personal and environmental vulnerability domains were predictive of being the eating disordered sister (also after using stepwise regression procedures).
The comparison of the discordant sister-pairs with the SIDE measure revealed no differences between the sisters in parental treatment and peer group characteristics, but much more jealousy in childhood towards the (later) healthy sister in the sister with AN.
In addition, we have investigated the psychiatric disturbances of these sister-pairs having occurred before onset of AN (Karwautz A 2002). The occurrence of major depressive episodes was significantly more common in the pre-onset period of AN patients. The rates of anxiety disorders and substance related disorders did not premorbidly differ between the sisters.
Temperament and character has also been investigated (Karwautz A 2002) in these sister-pairs and differed as expected from results of other studies. Patients were high in harm avoidance, persistence, and self-transcendence, and low in novelty seeking, self-directedness and cooperativeness. They did not differ in reward dependence. Furthermore, being low in self-directedness predicted strongly the comorbid presence of a personality disorder in these patients with AN (Karwautz A submitted).
A retrospective pilot study in sister-pairs discordant for eating disorders using the Child Behaviour Checklist (CBCL) regarding childhood before onset of the eating disorder has found significantly higher scores in the overall scale, the externalising, and the internalising behavioural problems scale in female AN/R, AN/BP, and BN patients compared with their healthy sisters. Aggressive behaviour, attention problems, social problems, anxious/depressive symptoms, somatic complaints, and social withdrawl were prominent in particular in the AN/R group (n=28) (Wagner G 2002).
Within-family studies offer the ideal opportunity to control for cultural bias and problems with ascertainment of appropriate controls (Dick, Johnson et al. 2000) and give the additional value tom study genetic risk factors in the same samples.
To study gene-environment interaction, one of the most promising areas are those, where replicated positive association results with genetic factors are found in populations with anorexia nervosa history. Then, in a next step, we may try to include measures of the premorbid (non-shared) environment and look at gene-environment correlation and interaction. Despite the low power of a discordant sister-pair study using 45 sister pairs and including various measures of non-shared environment and 4 different genetic regions (serotonin, and dopamin receptor genes, and COMT were included) we have proposed a design which should enable interesting results once the sample is larger and once, as

mentioned above, some genetic associations have been replicated in AN patients (Karwautz, Rabe Hesketh et al. 2001).

6 Protective factors

It is indeed important to elucidate factors contributing to resistance of youth to unhealthy dieting and eating disorders. However, despite some advances, which have been made between the two reviews by Rodin et al. (1990) diagnosing complete non-existence of research on this topic and Crago et al. (2001) giving a short summary of research since 1990 up to 2000, there are only a few papers dealing with protective factors for eating disorders so far. Furthermore, there are the same methodological problems within numerous studies: e.g. some studies have cross-sectional designs and give correlates instead of risk-factors, not specifying and attempting to assess factors within the premorbid period (e.g. most recently (McVey, Pepler et al. 2002)). How protective factors act has been summarized: they can decrease dysfunction directly, interact with the risk factors interrupting its effects, can disrupt the mediational chain through which the risk factors act or prevent the occurrence of risk factors itself (Crago, Shisslak et al. 2001).

A larger number of general and unspecific protective factors for mental problems and disorders have been identified. However, more specific protection factors should be identified, which would be informative for developing prevention strategies.

Participation in non-elite sports seems to serve as protective factor for eating problems ((Smolak, Murnen et al. 2000)). High self-esteem and high self-efficacy are protective (Button, Sonuga Barke et al. 1996). Problem-solving skills are important in protecting someone to get an eating problem ((Troop and Treasure 1997)).

7 Conclusions and further research

There are numerous methodological considerations to bear in mind in order to enhance the value of risk and protection factor study in the eating disorders. Risk factors have been much more intensely investigated than protective factors. Beside retrospective studies, prospective longitudinal studies would be needed also in this rare but very severe disorder, cause good prevention programs are based on well designed studies on risk and protection factors. This should be taken into consideration by funding institutions. The inclusion of genetic measures in order to elucidate further gene-environment interaction and correlation would be sensible.

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