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Evolutionary Explanations of Eating Disorders

Igor Kardum, Asmir Gračanin, Jasna Hudek-Knežević
University of Rijeka, Faculty of Arts and Sciences
Department of Psychology

Abstract

This article reviews several most important evolutionary mechanisms that underlie eating disorders. The first part clarifies evolutionary foundations of mental disorders and various mechanisms leading to their development. In the second part selective pressures and evolved adaptations causing contemporary epidemic of obesity as well as differences in dietary regimes and life-style between modern humans and their ancestors are described. Concerning eating disorders, a number of current evolutionary explanations of anorexia nervosa are presented together with their main weaknesses. Evolutionary explanations of eating disorders based on the reproductive suppression hypothesis and its variants derived from kin selection theory and the model of parental manipulation were elaborated. The sexual competition hypothesis of eating disorder, adapted to flee famine hypothesis as well as explanation based on the concept of social attention holding power and the need to belonging were also explained. The importance of evolutionary theory in modern conceptualization and research of eating disorders is emphasized.

Keywords: eating disorders, evolutionary theory, reproductive suppression, sexual competition

INTRODUCTION

There is a disagreement among scientists about the basic definition of mental disorder, and usually discussions concerning its definition encompass the role of social values as well as the centrality and the necessity of the presence of failures in evolved adaptive mechanisms (Lilienfield & Marino, 1995; Wakefield, 1999, 2005). Evolutionary reasoning can help to discriminate between conditions of an

✉ Igor Kardum, University of Rijeka, Faculty of Arts and Sciences, Department of Psychology, Slavka Krautzeka bb, 51000 Rijeka, Croatia. E-mail: *kardum@ffri.hr*

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organism that are the consequences of adaptive reactions designed by evolutionary processes and those that are the consequences of failures of these mechanisms, despite the fact that both kinds of conditions can be desired or undesired by an individual or society. Accordingly, mental disorders can be considered as conditions in which one or more psychological mechanisms do not compute according to the criteria that constitute their evolved function and in a way that is considered harmful (Cosmides & Tooby, 1999; Wakefield, 1999). This point of view is a base of the *harmful dysfunction* concept (Wakefield, 1999, 2005), with harmfulness being the only element included in the definitions of mental disorders given by the most scientists. Namely, concerning the evolved human traits, there is a question of whether a certain mechanism is operating in a way that produces the functional output it was designed to. It is well known that evolutionary processes result in three basic products: adaptations, which may be sometimes mulfunctional because of numerous reasons, by-products of adaptations, and evolutionary noise (Buss, Haselton, Shackelford, Bleske & Wakefield, 1998). Apart from malfunctioning of adaptive mechanisms, by-products and evolutionary noise may also in many cases be considered as pathology or described as abnormal, depending on the desired state of the individual. Adaptive mechanisms can produce painful and thus unwanted experiences even when triggered by the appropriate cues, and even when their intensity corresponds to their function. In addition, natural selection also results in the creation of other organisms that could be harmful and pathological. Many undesirable states of the organism may be created by the activity of different pathogens or other organisms that are in competition with an individual or species (e.g. predators or conspecifics). Evolutionary noise results from random mutations in genetic structure, which is the basic process of natural selection and occur relatively often. By its definition noise does not have any functional consequences but may result in the departure from the adaptive design created by evolutionary processes and thus may produce malfunction in adaptive mechanisms. Since in most cases genetically produced variations in each mechanism are held within the range of activation that is limited by evolutionary forces (e.g. level of angry behavioural tendencies), evolutionary noise most often produces variations which do not result in departures from adaptive design, and thus they can not be considered as functional failures. Anyway, mechanisms selected to yield certain adaptive traits may, through random genetic assortment, be inherited in intensities or combinations that result in organizations that do not perform functions for which the traits were selected or do not perform them in the range compatible with normal functions. Such extremes of normal variation can be evolutionary dysfunctions (Wakefield, 1999). One variant of this possibility can be found in cases in which some traits are inherited at high levels of intensity together with the low level of counterbalancing traits, and subsequently this combination leads to e.g. personality organization that precludes performing functions for which the traits were selected, despite the fact that each trait is expressed within the range defined by natural selection. In the context of reproductive success, failures of

adaptive mechanisms are usually maladaptive although they may be accepted by the social environment. For example, a genetically caused failure of adaptive mechanisms that are related to social domain may lead to a situation in which an individual unconditionally shares his/her own resources with others or lacks any form of aggression. Functional analysis will consider it a dysfunction although it can be praised by the modern society. In short, genetic mutations and evolutionary noise can often produce failures of evolved mechanisms that, depending on their harmfulness, may be considered as disorders.

Many unwanted conditions are direct results of natural selection (Nesse & Williams, 1999) and although they are not failures of adaptive mechanisms, they require attention and treatment. Adaptations themselves can sometimes appear to resemble psychopathology just because they represent compromises or trade-offs between competing functions or demands of the organism (Gilbert, 1998).

Each physical or psychological trait possessed by humans is in some way evolutionary trade-off. Regularly, advancement in one adaptive mechanism leads to the decrement in functionality of other mechanism. This may eventually lead to the heightened vulnerability to pathology. For example, if there is an evolutionary pressure posed to rabbits in the form of fast running predators, such as foxes, rabbits will develop features that enable faster running. Those Rabbits that run faster would probably be lighter, and consequently they would not be able to conserve enough body resources for the periods of food shortage. Also, their metabolism would be running faster, which could result in more pronounced tissue damage, finally leading to poorer physical health. A clear example of these trade-offs in humans is the evolution of upright posture. While it offers clear selective advantages because of freeing the hands for numerous useful activities, it also results in more pressure on the lower spinal discs, thus causing disability and subjective suffering (Nesse & Williams, 1999).

Because of the fact that genes, not individual organisms, are basic units of natural selection, it is reasonable to expect that genes would promote their interests in getting into the next generation even at the expense of the individual's health. A clear example is sex differences in longevity, with men living approximately seven years less than women (Sarafino, 2006). This is the consequence of intrasexual selection that leads to the remarkably higher levels of testosterone in males, which have deleterious effects on body tissues. The same process also makes males more prone to aggressive and risky behaviours, which additionally contribute to shorter longevity. Furthermore, men will seek additional sexual partners, despite knowing the risks of disease, jealous husbands' or their own wife's harmful reactions (Buss, 1994), and thus act in favour of spreading their genes and against their own well-being.

Any kind of pathology including psychopathology can represent a by-product or side-effect of an adaptive mechanism, rather than evolved mechanism itself. By-product can be necessary consequence of specific adaptive function or it can be

present because of the multiple effects of one gene. For example, Symons (1979) proposed that rape could be a by-product of different sexual adaptations in man, which include greater arousal of visual stimuli, greater autonomous sex drive and a greater desire for varied sexual partners. These adaptations could also interact with evolutionary noise and create serious psychopathology resulting in extremely deviant sexual behaviour. On the other side, genes that underlie some psychiatric conditions may be present because they give a reproductive advantage that outweighs the disadvantages produced by a specific condition. For example, there is a hypothesis that genes that underlie schizophrenia may provide some advantages for the people with that disorder, although these advantages may have nothing to do with psychological characteristics, but with some body functions such as immune system. Anyway, recent scientific evidence points to the existence of psycho(patho)logical trade-offs: individuals with manic-depressive tendencies indeed show increased creativity (Fodor, 1999), which indisputably has some selective advantages.

While many evolutionary trade-offs result in greater vulnerability to different dysfunctions, there are also numerous trade-offs that is direct cause of individual suffering and dysfunction in some evolved mechanisms. For example, in the recent past, decreases in amount of iron in blood were treated as a dysfunction, and in such cases physicians usually prescribed doses of iron. On the other hand, there is evidence that humans and other animals possess complex mechanisms designed to withhold iron when body is affected by bacteria that need this element for their survival (Weinberg, 1984). This mechanism shows that decreases in iron, often treated as anemia, are, in fact, an adaptive body reaction. The same principle holds for the elevated body temperature: while it has been thought of as a disorder, now it is well established that it is a defence against infection (Kluger, 1997). Other unwanted conditions with clear functions include pain, nausea, vomiting, fatigue, anxiety etc. All these mechanisms are defences protecting humans from dangers that were appearing regularly throughout the evolution, although it is easy to mistakenly interpret their manifestations as pathological because they produce painful subjective experience and have certain debilitating consequences. Although they may often have diminishing effects on the reproductive success of an individual, they also have beneficial effects on survival. For example, people lacking the capacity for pain usually die by their early 20s or 30s. Furthermore, vomiting is certainly an aversive and debilitating reaction, and people tend to interpret it as a symptom of illness, especially in the case when they do not immediately know what caused it. However, vomiting clears toxins and pathogens from the upper gastrointestinal tract, which is an adaptive mechanism that can, under some conditions, save one's life. Therefore, it is important to have in mind that these are the mechanisms that prevent pathology; they are not pathology themselves (Wakefield, 1999). They are produced by natural selection because they promote inclusive fitness, even though they may cause pain and dysfunctions of some other mechanisms. Cosmides and Tooby (1999) have noted that naturally

selected mechanisms are not designed to make us happy, as well as human standards of value do not have to correspond to the evolutionary standards of function. Consequently, an individual may suffer either from a harmful dysfunction as well as from a sore function.

Subjectively painful defences are often mistaken for dysfunctions, especially the ones in the emotional domain, such as the anxiety and the depression (Nesse & Williams, 1994). Anxiety, as well as other physiological and psychological defences, functions as the "smoke detector principle". If the cost of not reacting to potential danger is higher than the cost of false alarm despite of the subjective painful experience, the evolution will tailor defensive reactions to appear at the very first sign of possible threat. Because most anxiety responses are not expensive from the genes' perspective, and protect us against huge potential harms, an optimal anxiety system will express many unnecessary alarms. Therefore, anxiety has evolved as useful mechanism which protects us from various harms (Nesse, 2005).

Evolutionary explanations of severe depression consider it an excessive activation of naturally selected mechanism of low mood. Several evolutionary theories of depression view its milder version as a normal and adaptive reaction. *The conservation of resource theories* see depression as a mechanism that leads to the inhibition of appetitive functions which might be adaptive by enabling the individual to give up unattainable goals, to conserve the resources and to redirect them to more productive tasks (Klinger, 1975, 1993; Nesse, 2000). *Social competition theories* state that depressed mood is an answer to the descent in social hierarchy, and that specific behaviours accompanying this state correspond to the loss in social rank, serving an individual to escape possible attacks (Price, 1998), or to behave according to the perceived social resources of his/her own (Gilbert, 1997). Different variants of *the attachment theory* of depression propose that the depressive response serves as a distress call (Frijda, 1994), provokes a search for the lost relationship (Averill, 1968), or motivates the sufferer to avoid further deterioration of pre-existing bonds (Ingram, Miranda, & Segal, 1998).

Many behaviours can be considered pathological depending on their correspondence to the situation. For example, bereavement is almost indistinguishable from major depression, and the only difference between the two is that the former is an appropriate reaction designed by natural selection, while major depression results from the dysregulation of adaptive mechanism, and belongs to the harmful dysfunctions. Behaviours of individuals high in social rank may correspond to some aspects of manic disorder and they are still not considered pathological because they are produced by a phylogenetically developed apparatus of social rank modulation, which in this case produces a match between situation and behavioural output (Gardner & Wilson, 2004). The same principle holds for the correspondence of intensity and time of reaction and characteristics of situations. For example, an intense fearful response in the presence of evolutionary important

stimulus such as snake is considered normal, while the same intensity would be considered abnormal in the situation without any signs of danger.

Thus, lack of correspondence between reaction and situation may be an indicator of harmful dysfunction of a psychological mechanism. Certain adaptations such as anxiety or depression can be activated in inappropriate circumstances or with inappropriate intensity and length because of the failure of mechanisms that detect the situational cues and that regulate the intensity and length of response (e.g. because of the genetically caused malfunctioning in neurotransmitters' metabolism). These conditions are certainly unwanted, pathological and require treatment. However, there are a number of states that can not be considered as harmful dysfunctions because the mechanisms that produce them are working perfectly but they are still not tailored to the situation. First group comprises defence mechanisms that function as smoke detector. These states are not tailored to the specific situation but represent normal response that have the tendency to overreact. Second group includes the states that are not tailored to the situation, but occur because of the differences in ancestral and modern environment. In this context, Crawford (1998) proposes a simple typology which can help to categorize different kinds of mental disorders and comparable states. *True pathologies* are those states that could be considered harmful both throughout human evolutionary history as well as today (e.g. autism and schizophrenia). *Quasi-normal behaviours* are those that were maladaptive during our evolution while in today's environment they do not produce harm or even are desired by the individual or the society (e.g. adoption of unrelated children, close birth spacing). *Pseudopathologies* are states produced by the functioning of intact adaptive mechanisms but induce harm because of the differences in environment in which we evolved and the one we live in nowadays (e.g. excessive male jealousy, anorexia, wife abuse, nepotism etc). Pseudopathologies require special attention because of the enormous transformations of the human environment in the recent period of human history.

Environment of evolutionary adaptiveness (EEA) is a statistical composite of situational features or selective forces that created specific adaptation (Irons, 1998). Each adaptation has its own EEA and can operate in accord with its primary function only if actual conditions are in the range of situational features that correspond to those in EEA. Gilbert (1998) suggested that many states usually labelled as psychopathology may actually represent the consequences of the mechanisms that were adaptive during human evolution and are not well suited to modern conditions. Research concerned with the differences between ancestral and current environments, and the impact of these differences to our current anatomy, physiology and behaviour, is known as environmental mismatch theory (Bailey, 1995; Spinella, 2003).

For most human adaptations EEA is situated in the period of Pleistocene when our ancestors lived as hunter-gatherers. Since the environment has dramatically

changed in the last 12 000 years with the appearance of the agriculture, and also in the last 100 years because of the rapid technological advances, humans nowadays live in a very different environment compared to that in EEA (Crawford, 1998; Contaldo, Pasanisi, & Bellini, 2005). One example of the pathology resulting from the changes in the life style of modern humans is increased rate of breast cancer in modern societies probably related to common use of birth control, which was not possible in EEA, as well as earlier menarche, which is probably the consequence of superior diet. Namely, in the period between first menarche and first pregnancy, female breast cells are especially vulnerable to becoming cancerous and the prolongation of this period nowadays has deleterious consequences for many women (Eaton, Pike, & Short, 1994).

Evolutionary explanations of obesity

Although obesity is not yet included in any of the classification systems as an eating disorder, it is almost everywhere emphasized as a serious global epidemic in industrialized countries nowadays. Its prevalence is gradually increasing, especially in children and adolescents (Pinel, Assanand, & Lehman, 2000). Recent statistics state that approximately 312 millions of people in the world are obese (Wood, 2006). Adverse effects of obesity on health are well documented. It is related to many diseases such as cardiovascular and cerebrovascular disease, diabetes mellitus, several types of cancer, endocrine abnormalities, kidney dysfunction etc. (Pinel, Assanand, & Lehman, 2000).

As all other bio-behavioural phenomena, obesity is explained on different explanatory levels. Proximally, it is frequently explained by brain mechanisms that regulate eating (Hofbauer, 2002), and also by genetic factors. Namely, research results show substantial heritability for body weight, body mass index, obesity as well as genetic continuity of body weight from infancy to adulthood (Plomin, DeFries, McClearn, & McGuffin, 2008).

However, some authors note that the explanations of the prevalence of obesity based only on proximal mechanisms are not adequate, which suggest that some distal explanations based on evolutionary approach are also needed because the endogenous system regulating energy balance has developed during the course of evolution (Nesse, 2002).

From the evolutionary standpoint it is important to explain which selective pressures designed the mechanism that controls the quantity of food consumed. The most commonly accepted evolutionary explanation at present is that the increase in the number of obese people is, to some extent, the result of evolved adaptations which function was to resolve the problem of food shortage in our ancestral past and which is still working although food is nowadays abundant and people exercise little (Pinel, Assanand, & Lehman, 2000).

Namely, one of the main problems of the individuals during human and animal evolution was to consume enough food in order to survive and reproduce. However, in the environment of our ancestors appropriate food could be found only intermittently and unpredictably, and there were often intensive competition for food resources that led to its "high positive-incentive value". In those conditions, specific adaptive mechanism for hunger and eating has evolved, it's most important characteristics being to anticipate and prevent energy deficits. This mechanism promoted the level of consumption that maintain energy resources well above the level required to meet immediate needs and energy storage as a buffer against periods when food was scarce. The above-mentioned adaptive mechanism designed for the times of food shortages is still operating, and in modern industrialized countries in which food is cheap and easily available, it may be the cause of overeating and obesity (Pinel, Assanand, & Lehman, 2000). There are several potential biological mechanisms through which this adaptation works (e.g. Logue, 2004). Firstly, adipocytes, specialized cells that store fat, can only increase, but never decrease. Namely, number of adipocytes for lean as well as obese people is set during childhood and adolescence and the number of adipocytes for both categories vary little during adulthood (Spalding et al., 2008). Secondly, the restriction of caloric intake (loosing weight) lowers metabolism rate and increases fat storage for the possible future famine (Gatchel & Oordt, 2003). Thirdly, the more tasty foods are available the more desirable it becomes, and this could easily lead to overeating and resulting obesity (e.g. Logue, 2004). Also, pregnancy is one of the good examples of how this adaptation functions. Namely, additional fat stores are particularly adaptive in pregnant women, because they enable them to nurse their infants through a famine, while the infants of mothers who do not gain weight often die. Therefore, female body is well adapted to obtaining, and retaining the additional fat needed to maintain their pregnancy and subsequent lactation period (Raphael & Lacey, 1994).

Also, one of the important factors relevant for the prevalence of obesity in contemporary people living in industrialized countries is qualitative and quantitative differences in food consumption and life-style between modern people and their ancestors. For example, despite the significant energy requirements of paleolithic men that result from a diet containing three times more protein compared with the diet of the modern men, their food-intake was low in lipids and sodium (Wood, 2006). Also, compared to the ancestors, the diet of contemporary men is characterized by the scarcity of complex carbohydrates and fibre, as well as the excess of saturated fatty acid. Furthermore, the dietary regimes of our ancestors was based on animal proteins and supplemented by carbohydrates and fats from plants, while contemporary people combine a food high in energy and sedentary lifestyle insufficient of energy expenditure (di Costanzo, 2000; Eaton, Eaton & Konner, 1999). The accessibility of different food during our evolutionary past has had an impact on our genotype as well as phenotype. Since our metabolic and gastrointestinal processes are adapted to a diet typical to hunter-gatherer lifestyle,

the modern diet results in a challenge to the human genotype with the adverse effects on health and incidence not only of eating disorders but also of many diseases called "life-style diseases" (e.g. cardiovascular diseases, diabetes, cancer etc). Therefore, it is evident that the adaptation designed to resolve the problems of food scarcity in paleolithic men is today an obstacle to mental and physical health and efficient treatments of obesity. Consequently, a prerequisite for a successful use of the treatments of obesity is an improved understanding of distal mechanisms regulating energy balance.

Evolutionary explanations of eating disorders

Anorexia nervosa and bulimia nervosa are amongst most commonly studied and treated psychiatric problems. In discussing the causes of these disorders, generally an interaction of psychological, social and biological factors are brought up (Gatward, 2007). However, some authors suggest an approach that would be a synthesis of physiological and psychological explanations included within the wider biological framework of evolutionary theory (e.g. Palmer, 2000).

There is ample evidence of the increase in eating disorders in the last few decades (Steiger, Bruce, & Israël, 2003), although both anorexia and bulimia have been described much before the nineteenth century, with anorexia described as early as 17th century (Raphael & Lacey, 1994). Both disorders are more frequent in industrialized countries with almost all patients being women (95%) (Södersten, Bergh, & Zandian, 2006). It is estimated that at least 10% of school-aged girls in the developed world manifest partial anorexic or bulimic syndromes, together with dietary, psychological and medical problems (Steiger, Bruce, & Israël, 2003). Apart from being mostly female phenomena that have increased in prevalence relatively recently, both anorexia and bulimia share some more characteristics. Namely, they are both thought to have an almost identical geographical distribution and their basic psychopathology include disturbed body image and the striving for thinness. Abed (1998) notes that these shared features of anorexia and bulimia suggest the possibility of the same underlying etiology, which makes the attempt to explain these disorders through a single general mechanism plausible. The theory aspiring to give a comprehensive explanation of eating disorders should be able to give answers to the questions such as: Why eating disorders affect disproportionately more females than males? Why people strive for thinness? Why the increase in these disorders occurred recently and particularly in the industrialized countries?

The beginning of a systematic attention given to the explanation of eating disorders within evolutionary framework is the reproductive suppression hypothesis (Wasser & Barash, 1983). The authors propose that "females can optimize their lifetime reproductive success by suppressing reproduction when future conditions for the survival of their offspring are likely to be sufficiently better than present ones as to exceed the costs of the suppression itself'. In other

words, in the poor environmental conditions, in which pregnancy would be complicated and offspring would have a low chance of survival, it would have been adaptive for females to delay reproduction until a more advantageous conditions for childbearing would have occurred. Because low body fat results in the termination of ovulation and amenorrhea, extreme dietary behaviours can eventually lead to the loss of fertility and the suppression of reproduction (e.g., Frisch & Barbieri, 2002). Thus, preferences for thinner body ideals could be beneficial for women trying to control the timing of their reproduction. Surbey (1987) proposes one variant of the reproductive suppression hypothesis by claiming that anorexia nervosa may postpone puberty in girls predisposed to early maturity. Early and late maturers have different life histories in modern societies and it seems that developing anorexia nervosa alter a girl's developmental trajectory from that of an early maturer to that of a late maturer through the reduction of sexual fat. Namely, anorexic female body attract fewer males, and girl's libido is reduced, which leaves a girl more time for pursuing academic success and career that is highly valued in her family. When a girl has recovered from anorexia she is in a good position to obtain a mate with higher reproductive value (Surbey, 1987). Some research results support this hypothesis. For example, Juda, Campbell and Crawford (2004) found that women who perceive low levels of social support from their romantic partners and family reported increased dieting symptomatology and lower perception of parental readiness. Also, it has been shown that females of many species are able to postpone their fertility as a response to poor environmental conditions (Voland & Voland, 1989).

A critique regarding the reproductive suppression hypothesis is that it does not explain the function of distorted body image and hyperactivity of the anorexics, and does not give the answer to the questions such as why some other means that are less costly to stop menstruation were not evolved, why poor reproductive success is more likely to happen to wealthy and well-fed females and why sometimes men and postmenopausal women also may be anorectic (Guisinger, 2003).

Apart from the reproductive suppression hypothesis, Voland and Voland (1989) point out that kin selection theory (Hamilton, 1964) and the model of parental manipulation (Trivers, 1974) could also explain why suppressed reproduction leads to the increase of reproductive fitness in anorexic females.

From the kin selection theory standpoint, anorexia may be adaptive insofar as it allows the increase of intra-familial helping behaviour of an anorexic at the cost of her reproductive suppression. Research shows that members of anorexic families possess mutually overprotective attitudes, and that anorexic individuals especially, constantly worry about the well-being of their families (Minuchin et al., 1975). By reproductive suppression as well as by caring and self-sacrifice for one's own family, anorexic female helper allows an increase in her inclusive fitness by enabling successful replication of one's genes in collateral kin (Voland & Voland, 1989).

Parental manipulation model comprises three hypotheses. First states that parents exert a specific influence on the onset of or maintenance of anorexia. For example, there have been many reports of significant correlations between attitudes and behaviours of the dominant and overprotective mothers and the probability of anorectic reactions of their daughters (Steiger, Bruce, & Israël, 2003). Overprotection and domination is an effective strategy that prevents the finding of a partner and thus the reproduction of a daughter as well as daughter's detachment from their mothers. Second hypothesis within parental manipulation model posits that anorexia is positive for the parental fitness. Namely, anorexia and related suppression of the reproduction of daughters, especially from upper-middle and upper classes families, may function as a means for the concentration of the investment potential in the children with higher reproductive value, i.e. sons. Third hypothesis states that anorexia reduces fitness of patients. Suppressed reproduction is evidently biologically dysfunctional because it reduces the reproductive value of anorexics. To sum up, the hypotheses within parental manipulation model describe the subtle mechanisms by which parents prevent the development of a child's drives by changing them from self-centred and genetically based selfish behaviour (Voland & Voland, 1989).

Abed (1998) proposed the sexual competition hypothesis of eating disorders. It suggests that desirable female shape, often named "nubile", is an indicator of youth and reproductive potential and therefore acts as a sign of attraction to males and competition to other females. Female hour-glass appearance or low waist-to-hip ratio is a sign of her higher reproductive potential as well as an indication that she is not pregnant. Therefore, one behavioural strategy evolved in a female is to preserve nubile shape. This strategy operates through the drive for thinness and females who possessed this drive had a reproductive edge in the ancestral environment, especially in attraction and retention of long-term mates. Additionally, in the contemporary environmental conditions of western developed countries a progressive decline in fertility is documented, with older females having their first children, smaller number of children and greater inter-birth interval. Consequently, progressively older females are nowadays trying and are successful in retaining or recreating the nubile shape which results in the increase in female intra-sexual competition for high quality long-term mates. This competition sometimes, although not often, may lead to an over activation of the strategy designed to preserve relative thinness characteristic for a nubile shape, manifested as eating disorders, and especially anorexia.

Abed (1998) himself numbers several weaknesses of the sexual competition hypothesis. Namely, this hypothesis is based on the phenomenon of the declining fertility in western societies, which is in itself of unknown cause. Furthermore, the hypothesis does not predict proximal mechanisms underlying the development of eating disorders. Also, there is no conclusive evidence of the universality of "the concern about physical attractiveness", which is essential for this hypothesis, and

finally, by this hypothesis it is difficult to explain the existence of anorexia and bulimia in males.

Furthermore, in order to explain eating disorders Guisinger (2003), offers adapted to flee from famine hypothesis that integrates conventional theories and evolutionary perspective. He suggests that anorectic symptoms, including restricted food intake, hyperactivity, and denial of starvation, are manifestation of adaptive mechanisms to the conditions of famine that in our evolutionary past facilitated migration of our nomadic ancestors from the famine occurring in the depleted environment to the better one.

There is much evidence supporting this hypothesis. For example, many species refuse food in those circumstances when feeding competes with other activities such as migrating or breeding. Also, it has been noticed that, apart from humans, many animals increase activity in times of food shortage. This evidence has been supported by laboratory findings on rodents. Furthermore, several species at low body weight show anorexia like syndromes, and sometimes urges to move and refuse food are experienced as compulsions. Additionally, high heritability of anorexia has been found, suggesting that anorectic traits may have been evolved by natural selection. And finally, most neuromodulators and hormones regulating activity and appetite facilitate migration. However, this hypothesis can not explain why anorectics have a great resistance to eating when food is available and why hyperactivity is not always present in humans throughout the anorexia. Also, appetite-promoting neuropeptide Y and ghrelin have been found to be elevated in anorexic patients (Guisinger, 2003).

In order to explain eating disorders Gatward (2007) makes a synthesis of evolutionary ideas about response to threat. He notes that dietary restrictions and eating disorders could be explained by the concept of social attention holding power, and the need to belonging. The concept 'social attention holding power' is defined as an individual's ability to hold attention and gain investment from other members of the group (Gilbert, 1992), and this concept is closely related to the degree to which a person feels in control (Sloman, Gilbert, & Hasey, 2003; Marmot, 2004). Namely, those individuals who perceive themselves to occupy a high, dominant position, or status, within the group(s) to which they belong feel more in control comparing to those in a more submissive position (Marmot, 2004) and therefore, have higher social attention holding power. Furthermore, the need for belonging to a group evolved as one the fundamental human needs, as our ancestors that were not a part of a group would have been unlikely to survive for long. Because the survival of humans depended heavily on belonging to a group, people had to compete with others for resources and this competition could have lead to the threat of exclusion from the group. Competition for status in many animals involves threats or displays and in order to assess their value to the group an individual has to compare themselves with others, which includes comparison of physical appearance, health and reproductive potential in particular. Until relatively

recently obesity was a sign of good resources and status, since only the wealthy could afford to be obese (Stevens & Price, 2000). However, nowadays when food is easily available and relatively cheap, dietary restriction has become a sign of high status and self-control, and as it is evident by weight loss, restriction of food has become an attempt to demonstrate status and control. Being told that she is too fat could be a threat to self-perceived mate value of a girl. Thus, dietary restriction can further develop into anorexia, but only in those circumstances in which the weight loss sets off an ancient adaptive response to the threat of famine. Weight gain could be felt as threatening because it could mean re-entering the competition for status and belonging. The main weakness of this hypothesis concerns difficulties in its evaluation and ruling out alternative explanations.

General conclusion

Although the proposed evolutionary explanations and models may seem to be very different, they share one common element – response to threat. Namely, all of them – the reproductive suppression hypothesis and its variants (kin selection theory and the model of parental manipulation), the sexual competition hypothesis of eating disorder, the adapted to flee famine hypothesis as well as the explanation based on the concept of social attention holding power and the need to belonging could be explained as a form of a response to threat that leads anorexics (and especially girls) to develop symptoms of eating disorders.

The next important step for understanding eating disorders is an integration of their proximal and distal causes, i.e. the way internal and external triggers activate the evolved mechanisms that lead to eating disorders.

Also, all of the evolutionary hypotheses are very difficult to confirm or disconfirm but at least they provide new ways of looking to eating disorders, and maybe help in finding the more effective ways of treatment.

REFERENCES

Abed, R. (1998). The sexual competition hypothesis for eating disorders. *British Journal of Medical Psychology, 71,* 525–547.

Averill, J.R. (1968). Grief: Its nature and significance. *Psychological Bulletin, 70,* 721-748.

Bailey, K. (1995). Mismatch theory and paleopsychopathology. In C.B. Crawford (Chair), *Human behavior and evolution society conference*. Symposium conducted at the meeting of Human Behavior and Evolution Society, Los Angeles, California.

Buss, D. (1994). *The evolution of desire*. New York: Basic.

Buss, D.M., Haselton, M.G., Shackelford, T.K., Bleske, A.L., & Wakefield, J.C. (1998). Adaptations, exaptations, and spandrels. *American Psychologist, 43*, 533-548.

Contaldo, F., Pasanisi, & F., Bellini, O. (2005). Satiety and hunger: Some consideration from feast and famine aboriginal (stone age men) experiences. *Eating and Weight Disorders, 10*, e49-e51.

Cosmides, L., & Tooby, J. (1999). Toward an evolutionary taxonomy of treatable conditions. *Journal of Abnormal Psychology, 108*, 453-464.

Crawford, C. (1998). Environments and adaptations: Then and now. In C. Crawford, D.L. Krebs (Eds.), *Handbook of evolutionary psycholo*gy (pp. 275-302). London: Lawrence Erlbaum.

di Costanzo, J. (2000). Paleolithic nutrition: A model for the future? *Current Opinion in Nutrition and Metabolic Care, 3,* 87-92.

Eaton, S.B., Eaton, S.B.III, & Konner, M.J. (1999). Paleolithic nutrition revisited. In W.R. Trevathan, E.O. Smith, & J.J. McKenna (Eds.). *Evolutionary medicine* (pp. 313-332). New York: Oxford University Press.

Eaton, S., Pike, M., & Short, R. (1994). Women's reproductive cancers in evolutionary context. *Quarterly Review of Biology, 69*, 353-367.

Fodor, E.M. (1999). Subclinical inclination toward manic-depression and creative performance on the Remote Associates Test. *Personality and Individual Differences, 27*, 1273-1283.

Frijda, N.H. (1994). Emotions are functional, most of the time. In P. Ekman, R. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 97-177). New York: Oxford University Press.

Frisch, R.E., & Barbieri, R.L. (2002). *Female fertility and body fat connection*. Chicago: University of Chicago Press.

Gardner, R.J., & Wilson, D.R. (2004). Sociophysiology and evolutionary aspects of psychiatry. In J. Panksepp (Ed.), *Textbook of biological psychiatry* (pp. 597-625). Hoboken, NJ: Wiley.

Gatchel, R.J. & Oordt, M.S. (2003). *Clinical health psychology in the primary case setting*. Washington, D.C.: American Psychological Association.

Gatward, N. (2007). Anorexia nervosa: An evolutionary puzzle. *European Eating Disorders Review, 15,* 1-12.

Gilbert, P. (1998). Evolutionary psychopathology: Why isn't the mind designed better than it is? *British Journal of Medical Psychology, 71*, 353-373.

Gilbert, P. (1997). The evolution of social attractiveness and its role in shame, humiliation, guilt and therapy. *British Journal of Medical Psychology, 70,* 113–147.

Gilbert, P. (1992). *Depression: The evolution of powerlessness*. Hove, East Sussex: Lawrence Erlbaum.

Guisinger, S. (2003). Adapted to flee famine: Adding an evolutionary perspective on anorexia nervosa. *Psychological Review, 110,* 745–761.

Hamilton, W.D. (1964). The genetical evolution of social behaviour. I and II. *Journal of Theoretical Biology, 7*, 1-52.

Hofbauer, K.G. (2002). Molecular pathways to obesity. *International Journal of Obesity 26,* 18- 27.

Ingram, R.E., Miranda, J., & Segal, Z.V. (1998). *Cognitive vulnerability to depression*. New York: Guilford Press.

Irons, W. (1998). Adaptively relevant environments versus the environment of evolutionary adaptedness. *Evolutionary Anthropology, 6*, 194-204.

Juda, M.N., Campbell, L., & Crawford, C.B. (2004). Dieting symptomatology in women and perceptions of social support: An evolutionary approach. *Evolution and Human Behavior, 25,* 200-208.

Klinger, E. (1975). Consequences of commitment to and disengagement from incentives. *Psychological Review, 82,* 1–25.

Klinger, E. (1993). Loss of interest. In C.G. Costello (Ed.), *Symptoms of depression* (pp. 43-62). New York: Wiley.

Kluger, M. (1997). The adaptive value of fever. In P. Mackowiak (Ed.), *Fever: Basic measurement and management* (pp. 255-266). Philadelphia: Lippincott-Raven.

Lilienfield, S.O., & Marino, L. (1995). Mental disorder as a Roschian concept: A critique of Wakefield's "harmful dysfunction" analysis. *Journal of Abnormal Psychology, 104*, 411-420.

Logue, A.W. (2004). *The psychology of eating and drinking*. New York: Routledge.

Marmot, M. (2004). *Status syndrome*. London: Bloomsbury.

Minuchin, S., Baker, L., Rosman, B.L., Liebman, R., Milman, L., & Todd, T.C. (1975). A conceptual model of psychosomatic illness in children. *Archives of General Psychiatry, 32*, 1031-1038.

Nesse, R.M. (2005). Evolutionary psychology and mental health. In D.M. Buss (Ed.). *The handbook of evolutionary psychology* (pp. 903-927). Hoboken, NJ: Wiley.

Nesse, R.M. (2002). Evolutionary biology: A basic science for psychiatry. *World Psychiatry, 1,* 7-9.

Nesse, R. M. (2000). Is depression an adaptation? *Archives of General Psychiatry, 57,* 14–20.

Nesse, R.M., & Williams, G.C. (1999). On Darwinian medicine. *Life Science Research, 3*, 1-17.

Nesse, R., & Williams, G. (1994). *Why we get sick: The new science of Darwinian medicine*. New York: Random House.

Palmer, B. (2000). *Helping people with eating disorders: A clinical guide to assessment and treatment*. Chichester: John Wiley & Sons.

Pinel, J.P.J., Assanand, S., & Lehman, D.R. (2000). Hunger, eating, and ill health. *American Psychologist, 55,* 1105 – 1116.

Plomin, R., DeFries, J.C., McClearn, G.E., & McGuffin, P. (2008). *Behavioral genetics*. New York: Worth.

Price, J. (1998). The adaptive function of mood change. *British Journal of Medical Psychology, 71*, 465-477.

Raphael, F.J., & Lacey, J.H. (1994). The aetiology of eating disorders: A hypothesis of the interplay between social, cultural and biological factors. *European Eating Disorders Review, 2,* 143-154.

Sarafino, E.P. (2006). *Health psychology: Biopsychosocial interactions*. Chichester: John Wiley & Sons.

Sloman, L., Gilbert, P., & Hasey, G. (2003). Evolved mechanisms in depression: The role and interaction of attachment and social rank. *Journal of Affective Disorders, 74*, 107-121.

Södersten, P., Bergh, C., & Zandian, M. (2006). Understanding eating disorders. *Hormones and Behavior, 50,* 572-578.

Spalding, K.L., Arner, E., Westermark, P.O., Bernard, S., Buchholz, B.A., Bergmann, O., Blomquist, L., Hoffstedt, J., Naslund, E., Britton, T., Concha, H., Hassan, M., Ryden, M., Frisen, J., & Arner, P. (2008). Dynamics of fat cell turnover in humans. *Nature, 453*, 783-787.

Spinella, M. (2003). Evolutionary mismatch, neural reward circuits, and pathological gambling. *International Journal of Neuroscience, 113,* 503-512.

Steiger**,** H., Bruce, K., & Israel, M.(2003). Eating disorders. In G. Stricker, T.A. Widiger, & I.B. Weiner (Eds.) *Handbook of psychology, Vol. 8, Clinical psychology* (pp.173-194). New York: John Wiley & Sons Inc.

Stevens, A., & Price, J. (2000). *Evolutionary psychiatry: A new beginning*. London: Routledge.

Surbey, M.K. (1987). Anorexia nervosa, amenorrhea, and adaptation. *Ethology and Sociobiology, 8,* 47-61.

Symons, D. (1979). *The evolution of human sexuality*. New York: Oxford University Press.

Trivers, R.L. (1974). Parent-offspring conflict. *American Zoologist, 14,* 249-264.

Voland, E., & Voland, R. (1989). Evolutionary biology and psychiatry: The case of anorexia nervosa. *Ethology and Sociobiology, 10,* 223-240.

Wakefield, J.C. (1999). Evolutionary versus prototype analyses of the concept of disorder. *Journal of Abnormal Psychology, 108*, 374-399.

Wakefield, J.C. (2005). Biological function and dysfunction. In D.M. Buss (Ed.). *The handbook of evolutionary psychology* (pp. 878-902). Hoboken, NJ: Wiley.

Wasser, S.K., & Barash, D.P. (1983). Reproductive suppression among female mammals: Implications for biomedicine and sexual selection theory. *The Quarterly Review of Biology, 58,* 513-538.

Weinberg, E. (1984). Iron withholding: A defence against infection and neoplasia. *Physiological Reviews, 64*, 65-102.

Wood, L. (2006). Obesity, waist-hip ratio and hunter–gatherers. *An International Journal of Obstetrics and Gynaecology, 113,* 1110-1116.

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