CASE REPORT

Reducing symptoms in women with chronic anorexia nervosa. A pilot study on the effects of bright light therapy

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Abstract

OBJECTIVE: To examine the effect of bright light therapy on the sleep-wake rhythm, the menstrual cycle, mood, and key eating pathology symptoms in chronic anorexia nervosa.
METHODS: Five chronic anorectic women (mean duration of illness: 15.3 years) received 5 daily sessions of 30 minutes bright light therapy (10,000 LUX). Participants completed a diagnostic interview and questionnaires at pre-test, post-test and at a three month follow-up.
RESULTS: At follow up there was a slight improvement on core eating pathology, a fair decrease of depressive symptoms and an clinically important improvement on global distress.
CONCLUSIONS: Bright light therapy has on short term a positive effect on the physiological and psychological well being of chronic anorectic women. However, at follow-up the effects were partly lost. It is recommended to enhance the exposure period and repeat the treatment after 3 months.

INTRODUCTION

Anorexia nervosa is an eating disorder that is characterized by a severe disturbance in eating behaviour and a refusal to maintain minimal normal body weight. According to the DSM-IV the individual is intensely afraid of gaining weight and exhibits a significant disturbance in body perception (American Psychiatric Association 1994). Depression and anxiety disorders are common comorbid disorders (Halmi *et al.* 1991; Herzog *et al.* 1992; Bulik *et al.* 2000; Ivarsson *et al.* 2000; Herpertz-Dahlmann *et al.* 2001; Fairburn & Harrison 2003). The aetiology and pathogenesis of anorexia nervosa are still unknown (Rotenberg 2000), although there is some evidence that it is partly genetically based (Grice *et al.* 2002).

The DSM-IV classification of eating disorders is controversial (Eddy *et al.* 2009). Anorexia nervosa and bulimia nervosa are recognized as eating disorders. There is a residual category eating disorder NOS (EDNOS) which includes the provisional binge eating disorder. The majority of people seeking treatment for eating disorders receives the

diagnosis EDNOS (Button *et al.* 2005), which shows that the current criteria of eating disorders do not differentiate very well and therefore have little diagnostic and therapeutic relevance. It is suggested that all eating disorders are mainly cognitive disorders which share a core pathology, namely an overevaluation of body shape and weight and controlling them (Fairburn, 2008, Cooper & Shafran, 2008). Emphasizing the clinical importance of this core pathology, the current DSM-IV classification of eating disorders seems to overestimate the differences and to ignore the similarities of all eating disorders (Daaansen & de Jong, 2009) hindering the development and application of new evidence or practice based treatment.

Cognitive behavioural therapy is effective in the treatment of bulimia nervosa or binge eating disorder (Lewandowski *et al.* 1997; Agras *et al.* 2000; Thompson-Brenner *et al.* 2003), but there is little research addressing treatment outcome of anorexia nervosa. There is in fact no evidence-based treatment at all (Fairburn & Harrison 2003; National Institute Clinical Excellence 2004; Wilson *et al.* 2007; Bulik *et al.* 2007). A review of 119 studies on anorexia reveals that after treatment, roughly 47% of all anorectic patients recovers fully, 33% recovers partly and 20% becomes chronic. The overall mortality rate amounts to 5%. As far as regaining weight and normalization of menses were taken into account the results were slightly better, but on the other side the rate of normalisation of eating behaviour was slightly worse (Steinhausen 2002). In a Finnish community study participants with anorexia nervosa achieve significant clinical recovery after 5 years (Keski-Rahkonen *et al.* 2007). However, psychological, social and emotional criteria are mostly neglected and should be taken more seriously when striving for and measuring the rate of recovery and preventing relapse (Noordenbos & Seubring 2006). Clinical treatment hardly can improve the outcome. On average this setting leads to only 30% improvement (Richard 2005). Predictors of treatment failures in clinical and day treatment are duration of illness, amenorrhoea and low BMI (Howard *et al.* 1999). And even after treatment many patients still suffer from another psychiatric disorder with or without an eating disorder such as anxiety disorders (25,%), mood disorders (25%), substance abuse (14%) or personality disorders (borderline: 17,5%, histrionic: 16,5%, obsessive-compulsive: 31%) (Steinhausen 2002; Halvorsen *et al.* 2004). In conclusion methodological shortcomings in most studies aggravate a comparison of the outcome data: samples are small, recovery is not well defined, drop-out data are seldomly mentioned, severity of the illness is unknown or subtreshold anorectics are included, the follow-up period is short or major psychological problems as low self-esteem or cognitive distortions, both predictors for relapse, are neglected. Also, not much is known about the migration of diagnosis within the eating disorder spectrum over time (Milos *et al.* 2005; Fairburn 2008), initial treatment response or relapse (Pike 1998). As a consequence, due to the lack of good research data and recovery criteria it is difficult to define a chronic course of anorexia nervosa. Actually, at this moment we know little about the aetiology and pathogenesis of anorexia nervosa at all. Fairburn's (2008) suggestion to focus on the core pathology might be helpful to improve the outcome, but nevertheless new treatments seems to be mandatory.

BRIGHT LIGHT THERAPY

Bright light therapy is a rather common and effective therapy for seasonal affective disorders (Neumeister et al.1997; Brinkhuisen *et al.* 2003). There are indications that the disorder is caused by disturbances of the circadian rhythms. The exact aetiology is still unclear, but it is hypothesised that the symptoms are the result of the metabolism of melatonin: an increased level of melatonin in the dark winter months and, as a consequence, a low level of serotonin (Breiling & Argisle 1996) A low serotonin levels affects mood as well as other body processes such as sleeping, eating, hormone and temperature regulation. Similar features are found in anorexia nervosa (Brewerton & Jimerson 1996). Therefore we expect that the appliance of bright light therapy to anorectic patients might result in similar somatic and psychological improvement as in seasonal affective disorder.

In the field of eating disorders, hardly any studies on the effects of bright light therapy have been done. As far as we know there are no studies on anorexia nervosa and only a few small on bulimia nervosa. The few outcomes are not consistent. It is reported that in eating disorders the circadian rhythm of food intake is abnormal. Bright light therapy synchronizes the circadian rhythm of hunger, it has an anti-depressant effect on patients with eating disorders and reduces symptoms of binge eating and purging (Lam *et al.* 1994; Yamamotova *et al.* 2008). In a case study a 17 year old adolescent with eating disorder behaviour and winter exacerbation the eating improved only slightly, as the Eating Attitude Test revealed. However, the depression measured by the BDI decreased from severe to mild (Ash *et al.* 1998). In two other studies no long-term effect was found in the decrease of depression after ending treatment (Braun *et al.* 1999; Blouin *et al.* 1996) The effect on binging was different: while in one study there was no effect on frequency, size or content of the binges, in another study there was a positive effect on binging and purging in bulimics after three weeks of treatment with bright light therapy, but after ending the treatment there was a return to pre-test-baseline.

OBJECTIVE

We are aware that the biological mechanisms underlying anorexia nervosa are still not be clear and therefore the appliance of bright light therapy is rather specula-

tive. But the phenotypic overlap with depression which reacts well on bright light therapy and the poor outcome on practiced based cognitive behavioral therapy and the important biological and psycholigical impairment of anorexia nervosa justifies this new experimental intervention. It was postulated that the same biological intervention might be effective. The objective of this pilot study was to examine the effect of bright light therapy on the sleep-wake rhythm, the menstrual cycle, mood, and key eating pathology symptoms.

METHODS

Six female patients were recruited for this study, which was approved by the Research Department of the Psychomedical Centre PsyQ in The Hague, The Netherlands. Written consent was obtained from all participants. Before the start of the study, all patients took part in a rehabilitation program for patients with a chronic eating disorder. We considered patients as being chronic, if they had suffered at least five years or more of anorexia nervosa. This cut-off score was chosen, because of the already mentioned spontaneous high somatic recovery rate after five years (Keski-Rahkonen *et al.* 2007). Additionally, patients had to have been treated unsuccessfully at least twice with cognitive behavioural therapy in a day care or clinical setting for a period of nine months or more prior to the current rehabilitation program. Treatment outcome was defined as unsuccessful if no substantial weight gain was attained during treatment or if there had been a fundamental relapse within six months. The average number of earlier treatments was 3.2. At the moment of intake for the rehabilitation program the patients fulfilled the diagnosis of anorexia nervosa by DSM-IV.

Assessment

To assess current and lifetime psychopathology immediately prior to the start of the study, all patients were interviewed by a well trained independent psychiatrist with more than 10 years of experience in the treatment of eating disorders with the Structured Clinical Interview for DSM-IV (SCID) (First *et al.* 2002). In five cases the diagnosis anorexia nervosa was confirmed. One patient was diagnosed with bulimia nervosa. For that reason she was later excluded from the study. Nevertheless, she received light therapy during one week. Besides the diagnosis of an eating disorder two patients had also been diagnosed with Generalised Anxiety Disorder, two had a Major Depressive Disorder and one patient a Dysthymic Disorder (DSM-IV). According to the literature (Haffmans *et al.* 2006) it was expected that one of the first effects of bright light therapy would be a change in the menstrual cycle, therefore special attention was paid to it. None of the patients had a normal menses (one hysterectomy, two had amenorrhoea, one a very irregular period). One woman used oral anticonception. Her menstrual cycle was probably the result of this medication, because her BMI was only 12.7 kg/m^2. The average age of the six women was 34.2 years (range: 24–53 years, SD= 7.9). On average, the duration of illness was 15.3 years (range: 8–22 years). The average BMI for all 6 patients was 17.1 kg/m^2 (range: 12.7–20.8, SD=2.631, corrected for the five anorexic patients: 16.8 kg/m^2 and average weight of all patients was 47.7 kg (SD=7.9).

Three questionnaires were used for evaluating the effect of treatment. Overall psychopathology was measured by the Dutch Version of the SCL-90 (Derogatis 1977). The SCL-90 is an multidimensional self-report questionnaire often used for screening and evaluation of psychological problems and symptoms of psychopathology. It is also useful for measuring patient progress and treatment outcomes. The global index of distress (GSI) is the average value of all 90 items. The subscales 'anxiety' and 'sleeping problems' were used to measure changes in these topics due to treatment. Mood and affect were measured by the Dutch Beck Depression Inventory (Beck *et al.* 1996). Core eating disorder behaviour was assessed by the Dutch version of the EDI-II (Garner 1991). The questionnaire provides clinical information regarding the psychological and behavioural dimensions of an eating disorder. Also the EDI-II can be used for treatment evaluation. The EDI-II comprises 11 subscales. In this study, only four measuring subscales were used: 'drive for thinness, bulimia, body dissatisfaction and interoceptive awareness'. They were considered to represent the core symptoms of anorexia nervosa.

A sleep/wake diary was used to evaluate the quality of sleep. The patient monitors the moment of falling asleep, the frequency of waking up during the night, the time period she is sleeping and any sleep medication.

Procedure

This pilot study is a so-called multiple single case study. The chosen design is an ABA design. The baseline phase (A) is followed by a treatment phase (B) that again is followed by a baseline phase. In this design, patients are considered to be their own controls.

All questionnaires were filled out before beginning with the treatment. The patients started one week before treatment with the sleep/wake diary. During 5 daily sessions of thirty minutes each, starting at 8.30 in the morning, the patients were exposed to bright white light (10,000 LUX) from a bright light therapy device with constant background lighting. The patients had to sit at a table in front of the device at a distance of one foot. They were allowed to read, or drink a cup of coffee as long their eyes were exposed to the light.

One week after treatment the patients again had to fill out all questionnaires and monitor their sleep. Also, they had another appointment with the psychiatrist for a check-up on their menstrual cycle and psychiatric condition. In a follow-up after three months all procedures were repeated.

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RESULTS

To determine whether bright light therapy has an effect on anxiety, depression, eating behaviour, menstrual cycle and sleep a 'repeated measures analysis of variance' was done. On the subscale SCL-90 subscale 'depression' [F(1.16, 4.65)=2.02, p=0.19], 'anxiety' [F(1.2, 4.8=3.27, p=0.13], 'sleeping problems' [F(1.92, 7.67)=0.34, p=0.71] and total score [F(1.16, 4.66)=4.88, p=0.22] no significant change was found. Also on the BDI scores there were no significant differences between pre-test, post-test and follow-up [F(1.32, 5.27)=2.03, p=0.22]. Finally there was no significant change found on the EDI-II subscales 'drive for thinness' [F(1.40, 5.60)= 0.39, p=0.31], 'bulimia' [F(1.21, 4.84)=4.00, p=0.60], 'body dissatisfaction' [F(1.92, 7.68)=1.47, p=0.29] and 'interoceptive awareness' [F(1.79, 7.16)=4.23, p=0.06]. All other subscales were neglected.

Considering the limited size of the study group the lack of significant outcome was expected. Therefore, we decided to analyze on a qualitative level.

Global distress

Global distress of the individual is measured by the total score of all 90 items on the SCl-90. The Dutch version consists of 8 subscales, slightly different from the original version. For the interpretation of the raw scores we used the Dutch norm groups of outpatients (very high, high, above moderate, moderate, below moderate, low and very low). On group level there is an average decrease of global distress from high to below average and an overall improvement of about 43% between pre-test and follow-up. Patient 1 improved from very high to above moderate, patient 2 from very high to low, patient 3 from above moderate to low and patient 5 from very high to high. The global index of distress of patient 4 was stable. We can conclude that the global distress of all patient shows a clinically significant improvement (Table 1).

Anxiety

The anxiety dimension consists of a set of symptoms that are clinically associated with manifest anxiety. Indicators such as nervousness, restlessness tension, and trembling as well as feelings of free floating anxiety and panic are included. The total group shows an improvement from moderate to below moderate. Remarkably, patient 4 shows an adverse effect, which influences the overall score negatively. The patient had been diagnosed with Generalized Anxiety Disorder. Immediately after the light intervention she suffered a severe panic attack and developed a panic disorder (Table 2).

Sleep

In the Dutch SCL-90 version one dimension is called 'sleeping disorder'. We see a slight improvement from average to below average between pre-test and follow-up. The results were supported by the analysis of the sleep/wake diary. Light therapy is often used for the treatment of sleeping disorders and synchronizes the circadian rhythms. In addition to light therapy, it is important that patients improve their sleep hygiene (Table 3).

Tab. 1. SCL-90: global distress index.

patient	T1	T2	T3	improvement fu
1	289	243	204	
2	277	206	128	
3	179	131	119	
4	161	157	161	
5	295	293	266	
average	**240**	**206**	**174.5**	**43%**

Tab. 2. SCL-90: anxiety.

patient	T1	T2	T3
1	38	34	28
3	33	21	16
4	17	11	10
4	10	14	26
6	35	32	28
average	**26.6**	**22.4**	**21.6**

Tab. 3. SCL-90: sleep.

patient	T1	T2	T3
1	14	5	5
2	9	15	6
3	6	5	3
4	3	3	3
5	10	7	15
average	**8.4**	**7**	**6.3**

Tab. 4. BDI.

patient	T1	T2	T3	improvement fu
1	37	31	29	
3	19	13	3	
4	18	7	8	
4	26	13	26	
6	48	48	49	
average	**29.6**	**22.4**	**23**	**23%**

Depression

For measuring depression we used the BDI. As expected on the short term four patients showed a decrease of depression on short term and stabilized at follow up. On average there was an improvement of 23% between pretest and follow-up. Only one patient returned to baseline. Patient 6 did not seem to benefit at all (Table 4).

Eating pathology

The eating pathology was measured by the EDI-II core subscales 'drive for thinness, bulimia, body dissatisfaction and interoceptive awareness. On all subscales there was a slight improvement immediately after the light intervention and at follow-up.

The subcale 'drive for thinness" assesses the concern of an individual with dieting, preoccupation with weight and fear of weight gain. Two patients show a clinically important improvement, whereas there was no effect with the other patients. The mean improvement rate was 13% (Table 5).

The subscale bulimia evaluates eating behavior. The subscale is defined as a tendency to eat huge amounts and purging. Only two patients benefit slightly. These results were not surprising, because all patients had the diagnosis anorexia nervosa, restrained subtype (Table 6).

Body dissatisfaction is one of the major cognitive symptoms of eating disorders. Patients are dissatisfied with the overall size and regions of the (body, stomach, hips, thighs and buttocks) Two patients showed a clinically important improvement on this subscale between pretest and follow-up. At mean there was an 10% improvement at follow-up (Table 7).

Interoceptive awareness is the ability to discriminate between sensations and emotions and in eating disorder patients between sensations of hunger and satiation. On group level there was 11% improvement, but 2 patients had a clinically important improvement. The mean of all patients in this study is below average of all eating disorders. This was expected as anorectic patients have a lower interoceptive awareness than patients with bulimia nervosa or binge eating disorder (Fassino *et al.* 2004; Table 8).

Menstrual cycle

The effect on the menstrual cycle could only be measured in two patients. Both started to menstruate immediately after the interventions. The effect was maintained at follow up (Table 9).

DISCUSSION

Light therapy seems to be a new and promising intervention for chronic anorectic patients. On average, half of all patients benefit on the short term. It is remarkable that although there was no weight gain after treatment, 2 patients had a return of their menstrual cycle. As expected, most patients had a fair decrease of their

Tab. 5. EDI II drive for thinness.

	T1	T2	T3	improvement fu
1	41	35	41	
3	34	31	25	
4	28	25	15	
5	24	20	24	
6	35	38	36	
average	32.4	29.8	28.2	13%
ed patients	35.1			
students	**16.2**			

Tab. 6. EDI II bulimia.

	T1	T2	T3	improvement fu
1	15	15	15	
3	16	14	12	
4	14	16	10	
5	39	37	38	
6	33	31	36	
average	**23.4**	**22.6**	**22.2**	**6%**
ed patients	**26.4**			

Tab. 7. Body Dissatisfaction.

	T1	T2	T3	improvement fu
1	50	47	54	
3	35	31	28	
4	40	36	37	
5	30	21	23	
6	40	44	37	
average	**39**	**35.8**	**35.8**	**10%**
ed patients	**44.5**			
students	**31.2**			

Tab. 8. Interoceptive awareness.

	T1	T2	T3	improvement fu
1	33	25	34	
3	30	25	16	
4	19	20	18	
5	27	23	20	
6	33	35	36	
average	**28.4**	**25.6**	**24.8**	**11%**
ed patients	**33**			
students				

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Tab. 9. Menses.

	T1	T2	T3
1	None (hysterectomy)	none	none
3	none	menses	regular
4	irregular	irregular	Irregular
5	none	menses	regular
6	regular (AC)	regular	regular

depressive symptoms and a small improvement on core eating disorder symptoms as body dissatisfaction, drive for thinness and interoceptive awareness immediately after treatment. At the follow-up after 3 months the benefits were partly lost, but the global distress diminished even further. It is therefore recommended to repeat treatment after two or three months.

This pilot study had several limitations. The number of patients included was limited. It was impossible to find any significance on group level and we were obliged to analyze the data on an individual basis. A second limitation was the short period of exposure. In practice, patients are exposed to light therapy for at least 2 weeks. We would probably have seen a better result if we had expanded the exposure period.

A third limitation was the recruitment of chronic patients. Because there was no knowledge of the effect of bright light therapy on anorexia nervosa, we decided to select chronic patients as defined before. Nevertheless, the results are promising. We see that in the short term, important physiological and psychological parameters improve.

Based on our results we expect that bright light therapy could be an effective intervention as pre-therapy to prepare patients for treatment as usual, as an additional treatment supporting intervention or as maintenance therapy for chronic anorectics. More research in this field is necessary.

REFERENCES

1 Agras WS, Crow SJ, Halmi K.A, Mitchell JE, Wilson GT, Kraemer HC (2000). Outcome predictors for the cognitive behavior treatment of bulimia nervosa: data from a multisite study. Am J Psychiatry. **157**: 1302–1308.
2 American Psychiatric Association (1994). Diagnostic and statistical manual of mental disorders. Washington: American Psychiatric Association.
3 Ash JB, Piazza E, Anderson JL (1998). Light therapy in the clinical management of an eating-disordered adolescent with winter exacerbation. Int J Eat Disord. **23**: 93–97.
4 Beck TB, Steer RJ, Brown GK (1996). Beck depression Inventory-II. The Psychological Corporation. Dutch Version: Does AJW. BDI-II. Handleiding. Lisse: Harcourt Publishers. 2002.
5 Blouin AG, Blouin JH, Iversen H, Carter J, Goldstein C, Goldfield G *et al.* (1990). Light therapy in bulimia nervosa: a double blind, placebo controlled study. Psychiatry Res. **60**: 1–9.
6 Braun DL, Sunday SR, Fornari VM, Halmi KA (1999). Bright light therapy decreases winter binge frequencies in women with bulimia nervosa: a double blind, placebo controlled study. Compr Psychiatry. **40**: 442–448.
7 Breiling B, Argisle B (1996). Light years ahead: the illustrated guide o full spectrum and coloured light in mind-body healing. Berkeley, Celestial Arts.
8 Brewerton TD, Jimerson DC (1996). Studies of sertonin function in anorexia nervosa. Psychiatry Res. **62**: 31–42.
9 Brinkhuisen M, Koenegracht F, Meesters Y (2003). Symptoms of seasonal affective disorders and of compulsive disorder reduced by light therapy. J Affect Disord. **74**: 307–308.
10 Bulik CM, Berkman ND, Brownley KA, Sedway JA, Lohr KN (2007). Anorexia nervosa treatment: a systematic review of randomized controlled trials. Int J Eat Disord. **40**: 310–320.
11 Bulik CM, Sullivan PF, Wade T, Kendler KS (2000). Twin studies of eating disorders: a review. Int J Eat Disord. **27**: 1–20.
12 Button EJ, Benson E, Nollet C, Palmer R (2005). Don't forget EDNOS (eating disorder not other specified): patterns of service use in an eating disorders service. Psych Bull. **29**: 134–136.
13 Cooper Z, Shafran R (2008). Cognitive behaviour therapy for eating disorders. Behavioural and Cognitive Psychotherapy. **36**: 713–722.
14 Daansen P, De Jong M (2009). Voldoet Fairburns Enhanced cognitive behavioral therapy voor eetstoornissen? (Is Fairburn's Enhanced cognitive behavioral therapy a sufficient treatment for eating disorders? Tijdschrift voor Psychotherapie. 262–278.
15 Derorgatis LR (1977). The SCL-90 manual. Administration, scoring and procedures for the SCL-90. Baltimore: Clinical Psychometric Research. Dutch Version: Arrindell, W.A. & Etteman, H. SCL-90 (1990). Handleiding bij een multidimensionele psychopathologie-indicator. Groningen: Swets.
16 Eddy KT, Crosby RD, Keel P, Wonderlich SA, Le Grange D, Hill L *et al.* (2009). Empirical identification and validation of eating disorder phenotypes in a multisite clinical sample. J Nerv Ment Dis. **197**: 41–49.
17 Fairburn CG (2008). Cognitive behavior therapy and eating disorders. New York: Guilford Press.
18 Fairburn CG, Harrison PJ (2003) Eating disorders. Lancet. **361**: 407–416.
19 Fassino S, Pierò A, Gramaglia C, Abbate-Daga G (2004). Clinical, psychopathological and personality correlates of interoceptive awareness in anorexia nervosa, bulimia nervosa and obesity. Psychopathology. **37**: 168–174.
20 First MB, Spitzer RL, Gibbon M, Williams JBW (2002). Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Non-Patient Edition (SCID-I/NP). New York, Biometrics Research. New York State Psychiatric Institute.
21 Garner DM (1991). Eating Disorder Inventory-II, Manual. Odessa: Psychological Assessment Resources. Dutch Version: T.van Strien: EDI-II, Lisse: Swets
22 Grice DE, Halmi KA, Fichter MM, Strober M, Woodside DB, Treasure JT *et al.* (2002). Evidence for a susceptibility gene for anorexia nervosa on Chromosome 1. Am J Hum Gen. **70**: 787–792.
23 Haffmans PMJ, Loonen AJM, & Hoencamp E (2006). A practical approach to chronobiology and mood disorders. Therapeutic Values. **1**: 1–8.
24 Halmi KA, Eckert E, Marchi P, Sampugnaro V, Apple R, Cohen J (1991). Comorbidity of psychiatric diagnosis in anorexia nervosa. Arch Gen Psychiatry. **48**: 712–718.
25 Halvorsen I, Andersen, A, Heyerdahl S (2004). Good outcome of anorexia nervosa after systematic treatment: Intermediate to long term follow-up of a representative county sample. Eur Child Adolesc Psychiatry. **13**: 295–306.
26 Herpertz-Dahlmann B, Muller B, Herpertz S, Heussen N, Hebebrand J, Remschmidt H (2001). Prospective 10-year follow-up in adolescent anorexia nervosa – course, outcome, psychiatric comorbidity and psychosocial adaptation. J Child Psychology and Psychiatry. **42**: 603–612.
27 Herzog DB, Keller MB, Sacks NR, Yeh CJ, Lavori PW (1992). Psychiatric comorbidity in treatment-seeking anorexics and bulimics. J Am Acad Child Adolesc Psychiatry. **31**: 810–818.

28 Howard WT, Evans KKRN, Quintero-Howard, CV, Bowers WA, Anderson AE (1999). Predictors of success or failure for inpatients with anorexia nervosa. Am J Psychiatry. **156**: 1697–1702.
29 Ivarsson T, Rastam M, Wentz E, Gillberg I, Gillberg C (2000). Depressive disorders in teenage-onset anorexia nervosa: A controlled longitudinal, partly community-based study. Compr Psychiatry. **42**: 398–403.
30 Keski-Rahkonen A, Hoek HW, Susser ES, Linna, MS, Sihvola E, Raevuori A *et al.* (2007). Epidemiology and course of anorexia nervosa in the community. Am J Psychiatry. **164**: 1259–1265.
31 Lam RW, Goldner EM, Solymon L, Remnicj RA (1994). A controlled study of light therapy for bulimia nervosa. Am J Psychiatry. **151**: 744–750.
32 Lewandowski LM, Gebing A, Anthony JL, O'Brien WH (1997). Meta-analysis of cognitive-behavioral treatment studies for bulimia. Clin Psych Rev. **17**: 703–718.
33 Milos G, Spindler A, Schnyder U, Fairburn CG (2005). Instability of eating disorder diagnosis: prospective study. Brit J Psychiatry. **187**: 573–578.
34 National Institute for Clinical Excellence (2004). Eating disorders: core interventions in the treatment and management of anorexia nervosa, bulimia nervosa and related eating disorders. Nice Clinical Guideline No 9. London: NICE 2004.
35 Neumeister A, Rieder N, Hesselmann, B, Rao ML, Gluck J, Kasper S (1997). Effects of trypthophan depletion on drug free-patients with seasonal affective disorders during stable response to bright light therapy. Arch Gen Psychiatry. **54**: 133–138.
36 Noordenbos G, Seubring A (2006). Criteria for recovery from eating disorders according to patients and therapists. Eating Disorders: Journal of Treatment & Prevention. **14**: 41–54.
37 Pike MK (1998). Long term course of anorexia nervosa: response, relapse, remission and recovery. Clin Psych Rev. **18**: 447–475.
38 Richard M (2005). Effective treatment of eating disorders in Europe. Treatment outcome and its predictors. Eur Eat Dis Rev **13**: 169–179.
39 Rotenberg VS (2000). Anorexia nervosa: Old contradictions and a new theoretical approach. International J Psychiatry Clin Pract. **4**: 89–92.
40 Steinhausen HC (2002). The outcome of anorexia nervosa in the 20th century. Am J Psychiatry. **159**: 1284–1293.
41 Thompson-Brenner H, Glass S, Westen D (2003). A multidimensional meta-analysis of psychotherapy for bulimia nervosa. Clinical Psychology: Science and Practice. **10**: 269–287.
42 Wilson GT, Grilo CM, Vitoussek KM (2007). Psychological treatment of eating disorders. Am Psychol. **62**: 199–216.
43 Yamamotova A, Papezova H, Vevera J (2008). Normalizing effect of bright light therapy on temperature circadian rhythm in patients with eating disorders. Neuroendocrinol Lett. **29**: 168–172.

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