

Normalizing effect of bright light therapy on temperature circadian rhythm in patients with eating disorders

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Abstract

OBJECTIVES: Light and food are important synchronizers of circadian rhythmicity. In eating disorders, the circadian rhythms of food intake and temperature are abnormal.

METHODS: We analyzed the effect of the morning light application on the circadian rhythm of tympanic temperature and its association with hunger and mood changes in the sample of 25 female patients hospitalized with DSM-IV diagnosis of eating disorders (14 bulimia nervosa and 11 anorexia nervosa) and in 6 healthy women.

RESULTS: Light therapy reduced interindividual variability of the temperature acrophase, synchronized the temperature and hunger rhythms and showed an antidepressant effect on patients with eating disorders. Bright light therapy normalized the circadian rhythm of body temperature in both anorexic and bulimic patients: phase advanced rhythm was delayed and phase delayed rhythm was advanced. In contrast with anorexic patients, the majority of bulimic patients had normal temperature rhythm before the therapy and this rhythm was not changed by the therapy.

CONCLUSION: The light therapy normalized temperature circadian rhythm in patient with eating disorders. We hypothesize that the light therapy can also contribute to improvement of pathological eating pattern because of the functional connections between light and food entrained oscillators. The light may help to restore the irregular circadian rhythmicity induced by chaotic food intake.

INTRODUCTION

Light therapy, a non-pharmacological therapeutic method originally used for the treatment of seasonal affective disorders (Thompson et al., 1997) or non-seasonal depressions (Yamada et al., 1995; Praško et al., 2002), was applied last decades in the treatment of bulimia nervosa to improve mood and

eating symptoms worsening in winter (Lam et al., 1994; Levitan et al., 1994; Blouin et al., 1996). Serotonin (5-HT) is supposed to be a mediator of light information transmission into the light-entrained oscillator (LEO) located in hypothalamic supra-chiasmatic nuclei (SCN). Light phase of diurnal

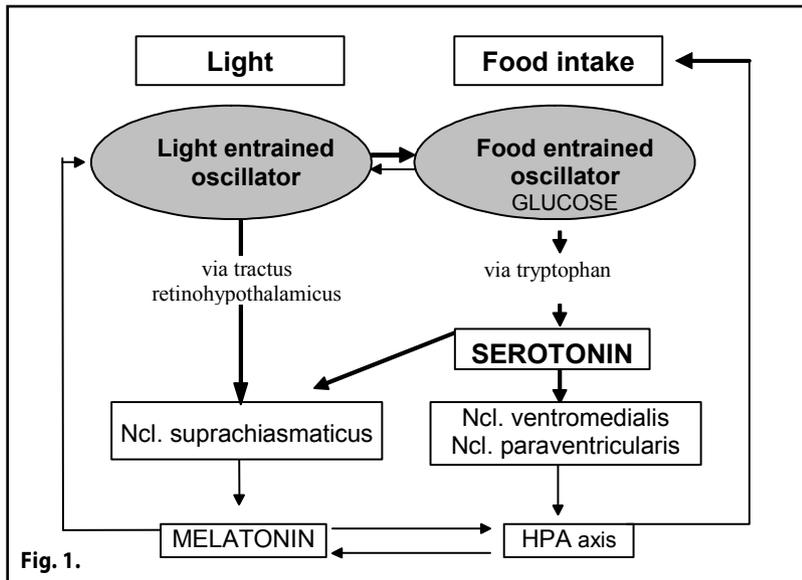


Fig. 1. Schematic representation of interaction between the light (LEO) and the food entrained oscillators (FEO). Mutual synchronization consists of the entrainment of circadian rhythm by food (non-photic synchronization) or by light (photic synchronization) or by their interaction. In patients with eating disorders the effect of FEO on LEO can be reduced as a result of dysregulation of serotonergic system.

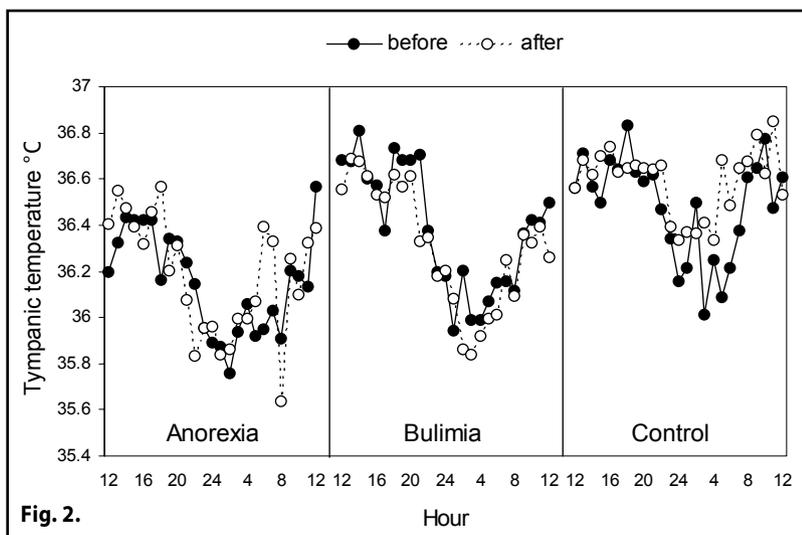


Fig. 2. Comparison of average circadian temperature curves before and after the light therapy in different groups.

rhythm induces *c-fos* expression in these nuclei, while serotonin depletion decreases the number of *c-fos* positive cells (Moyer, Kennaway et al., 2000).

Eating is another synchronizer influencing biological rhythms. The food entrained oscillator (FEO) is responsible for non-photic synchronization (Figure 1). Glucose is supposed to be „Zeitgeber“ of this oscillator (Stephan et al., 1998). Carbohydrate consumption causes an insulin-mediated fall in the plasma levels of the large neutral amino acids (LNAA) competing with tryptophan (TRP) for uptake into the brain. This elevates the ratio of the plasma tryptophan to large neutral amino acids (TRP/LNAA), and thus brain tryptophan rapidly accelerates brain serotonin synthesis and release (Fernstrom, 1990). Serotonin is supposed to play the key role in the regulation of both oscillators.

In people with a regular eating pattern, the food intake contributes to synchronization and stability of the circadian rhythms. We hypothesized that the restricted

eating pattern in anorexia and frequent and irregular overeating in bulimia can affect circadian rhythm monitored by body temperature rhythm.

The aim of the study was to test the effect of light therapy on temperature rhythm and its relation to hunger and appetite in females with eating disorders and in healthy women.

METHODS

Patients and Procedure

The study sample consists of 25 female patients hospitalized at the Special Unit for Eating Disorders in Department of Psychiatry of the 1st Faculty of Medicine of Charles University (Prague) with DSM IV diagnosis of eating disorders (14 bulimia: age = 24.3 ± 4.6 years, BMI = 20.0 ± 1.6 kg.m⁻²; 11 anorexia: age = 23.5 ± 4.1 years, BMI = 14.5 ± 1.2 kg.m⁻²; means ± S.D.). No patients had any severe somatic complication or IMAO

Fig. 3. The light therapy induced phase advance of temperature curves in subjects with delayed temperature minima (dashed columns) and induced phase delay in subjects with advanced temperature minima (black columns); $F_{(1, 29)} = 19.14$, $p = 0.0001$, vertical bars denote 0.95 confidence intervals.

Table 1. Description of anthropometric characteristics of patients and control subjects. Data are presented as means \pm SD.

	Anorexia N=11	Bulimia N=14	Control N=6	Kruskal- Wallis df(2,31)
Age (years)	23.5 \pm 4.1	24.3 \pm 4.6	31.8 \pm 12.2	$p = 0.311$
Weight (kg)	40.2 \pm 4.3	55.3 \pm 5.1	60.3 \pm 7.9	$p = 0.000$
Height (cm)	166.3 \pm 5.6	166.4 \pm 5.1	168.0 \pm 4.6	$p = 0.630$
BMI (kg/m ²)	14.5 \pm 1.2	20.0 \pm 1.6	21.4 \pm 3.0	$p = 0.000$

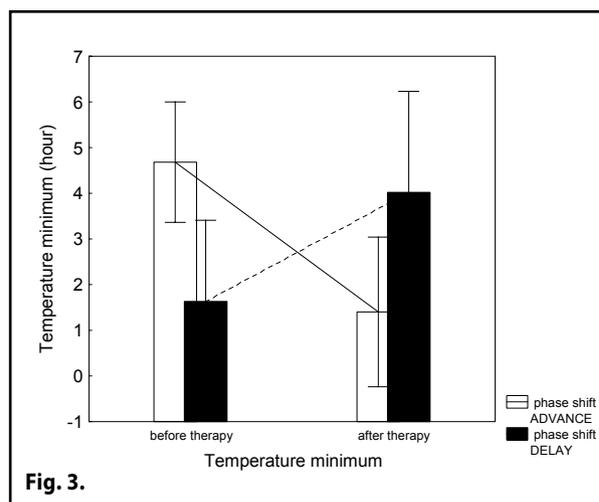


Fig. 3.

Table 2. The effect of light therapy on parameters of circadian temperature rhythm. Data are presented as means \pm SD. Wilcoxon matched pairs test was used for a statistical evaluation within each diagnostic category.

	Anorexia nervosa N=11		Bulimia nervosa N=14		Control N=6	
	before	after	before	after	before	after
Mesor (°C)	36.3 \pm 0.6	36.2 \pm 0.4 ^a	36.4 \pm 0.3	36.3 \pm 0.3	36.5 \pm 0.3	36.6 \pm 0.3 ^a
	$p = 0.859$		$p = 0.272$		$p = 0.116$	
Amplitude (°C)	0.33 \pm 0.23	0.29 \pm 0.20	0.36 \pm 0.10	0.36 \pm 0.17	0.24 \pm 0.12	0.30 \pm 0.16
	$p = 0.424$		$p = 0.937$		$p = 0.173$	
Acrophase (hour)	16.1 \pm 4.4	13.6 \pm 3.3	16.00 \pm 1.8	16.2 \pm 2.3 ^b	13.8 \pm 3.1	11.3 \pm 5.2 ^b
	$p = 0.155$		$p = 0.826$		$p = 0.248$	
HAMD	13.9 \pm 9.1	8.9 \pm 7.5	17.9 \pm 12.1	13.6 \pm 10.7	1.2 \pm 1.6	0.8 \pm 1.6
	$p = 0.008$		$p = 0.003$			

^a Kruskal-Wallis nonparametric test, $H(2,31) = 5.96$, $p = .0509$; significant difference between anorexia and control (Mann-Whitney U test, $p = 0.021$)

^b Kruskal-Wallis nonparametric test, $H(2,31) = 8.41$, $p = .0149$; significant difference between bulimia and control (Mann-Whitney U test, $p = 0.006$)

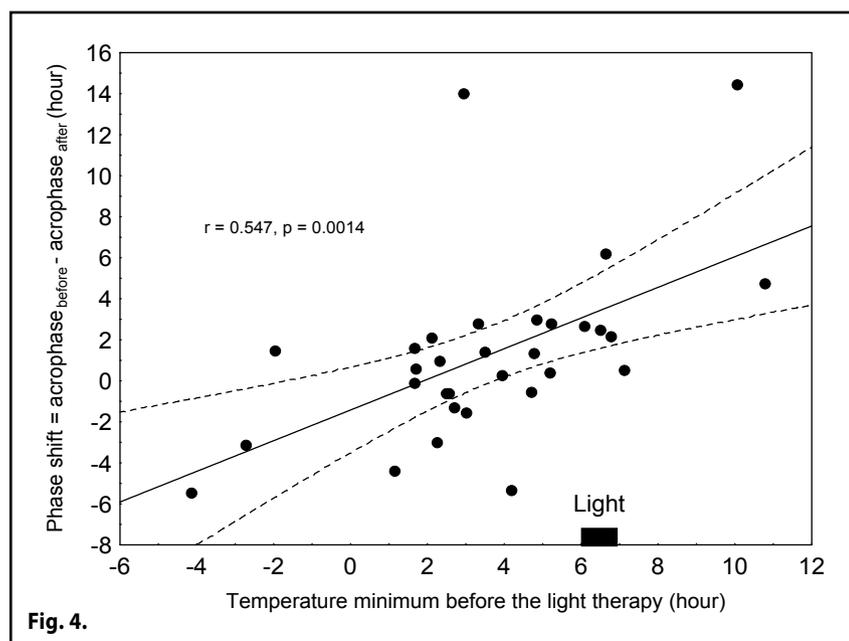


Fig. 4.

Fig. 4. Normalizing effect of the light therapy. Correlation shows individual shifts in acrophase of tympanic temperature rhythm (ordinate) in dependency on initial nadir/acrophase before the light therapy (abscissa). Positive phase shifts correspond to phase advance, negative phase shifts correspond to phase delay induced by light therapy.

treatment in the previous two weeks. Six healthy women were included in the control group. After the procedures had been fully explained, all participants signed an informed consent approved by the University Hospital Ethical Committee.

Morning light of an intensity 5000 lux was applied from a distance of 90 cm from 6 AM to 7 AM. Treatment was given for one week in January. The patients were assessed before and after phototherapy using the Hamilton Psychiatric Rating Scale for Depression (HAM-D). Hunger and appetite rhythms were assessed according to the patient's diary and consisted of reduced orexigram, i.e. the hours of maximum and minimum hunger. Tympanic temperature was measured before and after the phototherapy in one-hour intervals starting from 12 AM of the first day to 12 PM of the next day (ThermoScan, Braun).

Statistical Analysis

The individual temperature curves were analyzed by cosinor analysis. This analysis estimates amplitude, mesor and acrophase of the circadian rhythm. Parameters of fitted cosine curves were compared using nonparametric statistics. Group comparisons were performed using the Kruskal-Wallis test. The Mann-Whitney U test was used as a post-hoc test to compare anorexic patients, bulimic patients and control groups when indicated. Changes after phototherapy were analyzed by Wilcoxon matched pairs test. A $p < 0.05$ was considered significant. All data are expressed as mean \pm SD.

RESULTS

Description of anthropometric characteristics of patients and controls is summarized in Table 1. Controls have higher body mass index and body weight than anorexia and bulimia patients. They did not differ in age.

The results of cosinor analysis and the effects of light therapy on parameters of circadian temperature rhythm are shown in Table 2.

Temperature rhythm

Changes of the phase of temperature minimum induced by the light therapy are shown in Figures 2 and 3. In Figure 3, the effect of light therapy is expressed as a correlation between the time of the temperature minimum before light therapy (abscissa) and induced phase shift (ordinate) after therapy. Significant positive correlation ($r = 0.547$, $p = 0.0014$) indicates, that subjects with the normal temperature rhythm, i.e. temperature minimum within the range from 2 AM to 4 AM, have only minimal phase shifts in both directions. On the other hand, subjects with originally advanced rhythms are delayed after the light therapy and subjects with originally delayed rhythms are advanced as demonstrated in Figure 4.

Hunger rhythm

In controls, minimum hunger occurs at 8.8 ± 4.7 and maximum hunger at 16.0 ± 2.9 ($p < 0.05$). Reduced orexigram differs in ED subgroups; bulimic patients in contrast with anorexic ones and controls do not discriminate between the time of maximum and minimum hunger (hours 13.6 ± 5.8 and 10.2 ± 5.4 , respectively). In anorexia patients only, the time of maximum hunger precedes the time of minimum hunger (10.6 ± 4.3 and 13.8 ± 2.4 , respectively, $p < 0.05$).

After the light therapy, the hours of maximum hunger correlated positively with the hours of temperature maximum in patients with anorexia and bulimia nervosa ($r = 0.57$, $p < 0.05$). This correlation was not significant before the therapy ($r = -0.05$).

Affective symptoms

After the light therapy, there was a significant reduction in the general score of depression in the HAM-D scale in both groups of patients: in anorexic patients 13.9 ± 9.1 at the beginning and 8.9 ± 7.5 at the end of light therapy ($p = 0.0009$); in bulimic patients 17.9 ± 12.1 at the beginning and 13.6 ± 10.7 at the end of light therapy ($p = 0.003$).

DISCUSSION

Our study shows that light, beside its antidepressant and modulating effects on circadian rhythm, can serve as a normalizing factor on body temperature phase. Light therapy in general reduced the inter-individual variability of the temperature minimum phase in eating disorders and controls, so that their temperature curves became more consistent.

Temperature rhythms

The effect of one-week light therapy did not affect any parameter of temperature curves when anorexic and bulimic patients were analyzed separately. However, when both patient groups were pooled together, the normalizing effect of the light became more evident. This effect consists of different phase shifts. The degree of phase shift after the light therapy depends on the temperature phase before the therapy: normal rhythm is not affected, but phase advanced rhythm is delayed and phase delayed rhythm is advanced. Whether the observed phase changes can be considered as a return to a healthy state remains to be verified on a larger sample of patients. In this type of experiment we cannot exclude a possible additive or intervening effect of intermittent sleep deprivation induced by temperature measurement.

In our sample of eating disorder patients, we confirmed the literature findings of improvement of depressive symptoms after the light therapy (Lam et al., 1994; Blouin et al., 1996).

Hunger rhythms

The light and regular eating can synchronize circadian rhythms. In anorexia nervosa, the synchronizing effect of food can be significantly reduced; they are less seasonal and less circadian than bulimic patients (Teicher et al., 1997) due to dysregulated serotonergic system (Jimerson et al. 1997; Kaye et al. 1998). The light therapy induced synchronization of temperature and hunger rhythms. The hours of maximum hunger correlated after the light therapy with the hours of temperature maximum. The synchronization of these rhythms facilitates then the maximum food intake at the time of culminating metabolism.

The time of maximum and minimum hunger differs in anorexic and bulimic patients and indicate that common two types of circadian typology do exist in eating disorders: phase advanced "the morning type" in anorexic patients and phase delayed "the evening type" in bulimic patients. This is in agreement with clinical observation of morning anorexia in bulimic, night eating and overeating patients. Further studies will clarify these relations.

We conclude that the light therapy normalized temperature circadian rhythm in patient with eating disorders. Our results show the effect of mutual synchronization between light and food entrained oscillators on hunger feeling associated with temperature rhythm in eating disorders. These findings contribute to our understanding of the importance of the regular eating pattern in the treatment of eating disorders.

Further studies are necessary to clarify the long term synchronizing effect of light on pathological eating patterns.

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REFERENCES

- 1 Blouin AG, Blouin JH, Iversen H, Carter J, Goldstein C, Goldfield G, Perez E. Light therapy in bulimia nervosa: a double-blind, placebo-controlled study. *Psychiatry Res* 1996; **60**:1–9.
- 2 Fernstrom JD. Aromatic amino acids and monoamine synthesis in the central nervous system: influence of the diet. *J Nutr Biochem* 1990; **10**: 508–517.
- 3 Jimerson DC, Wolfe BE, Metzger ED, Finkelstein DM, Cooper TB, Levine JM. Decreased serotonin function in bulimia nervosa. *Arch Gen Psychiatry* 1997; **54**:529–534.
- 4 Kaye W, Gendall K, Strober M. Serotonin neuronal function and selective serotonin reuptake inhibitor treatment in anorexia and bulimia nervosa. *Biol Psychiatry* 1998; **44**:825–838.
- 5 Lam RW, Goldner EM, Solyom L, Remick RA. A controlled study of light therapy for bulimia nervosa. *Am J Psychiatry* 1994; **151**:744–750.
- 6 Levitan RD, Kaplan AS, Levitt AJ, Joffe RT. Seasonal fluctuations in mood and eating behavior in bulimia nervosa. *Int J Eat Disord* 1994; **16**:295–299.
- 7 Moyer RW, Kennaway DJ. Serotonin depletion decreases light induced *c-fos* in the rat suprachiasmatic nucleus. *Neuroreport* 2000; **11**:1021–1024.
- 8 Prasko J, Horacek J, Klaschka J, Kosova J, Ondrackova I, Sipek J. Bright light therapy and/or imipramine for inpatients with recurrent non-seasonal depression. *Neuro Endocrinol Lett* 2002, **23**:109–113.
- 9 Stephan FK, Davidson AJ. Glucose, but not fat, phase shifts the feeding-entrained circadian clock. *Physiol Behav* 1998; **65**:277–288.
- 10 Teicher MH, Glod CA, Magnus E, Harper D, Benson G, Krueger K, McGreenery CE. Circadian rest-activity disturbances in seasonal affective disorder. *Arch Gen Psychiatry* 1997; **54**:124–130.
- 11 Thompson C, Childs PA, Martin NJ, Rodin I, Smythe PJ. Effects of morning phototherapy on circadian markers in seasonal affective disorder. *Br J Psychiatry* 1997; **170**:431–435.
- 12 Yamada N, Martin-Iverson MT, Daimon K, Tsujimoto T, Takahashi S. Clinical and chronobiological effects of light therapy on non-seasonal affective disorders. *Biol Psychiatry* 1995; **37**:866–873.