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MORNINGNESS-EVENINGNESS, CIRCADIAN PHASE AND THE TIMING OF SLEEP IN PATIENTS WITH SEASONAL AFFECTIVE DISORDER

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Introduction. We revisited an old question, never clearly resolved: is seasonal affective disorder (SAD) characterized by delayed circadian phase in winter? Response to morning light therapy is correlated with the size of phase advances in melatonin onset, yet patients who are relatively delayed at baseline do not show preferential response.[1] Several studies show that the Horne-Östberg scale[2] correlates strongly with objective measures of circadian phase. Avery et al.[3] found a prevalence of evening types in hypersomnic SAD patients, and significant summertime increases in morningness (retrospectively assessed in winter) as well as increases after morning light therapy. However, many SAD patients are not hypersomnic, and it is unclear whether those findings describe the SAD population as a whole. In this study, we sought to describe the population by diurnal type irrespective of sleep duration and to determine the extent of spontaneous springtime change.

Setting, Patients. Patients were 71 research volunteers (56 women, 15 men), ages 23 to 64 (mean age ± SD, 40.4±10.7 y). DSM-IV diagnoses were: Major Depressive Disorder, Recurrent (code 296.32), 69 (97.2%), and Bipolar II Disorder, Depressed (code 296.89), 2 (2.8%), both with seasonal pattern, winter type. All were medically healthy and without other Axis I disorders. Patients entered in late fall or winter for treatment trials of light therapy or negative air ionization. These trials are ongoing and blinded; the present analysis is restricted to winter baseline measures and springtime follow-up measures, during the period of spontaneous remission.

Procedure. The *Morningness-Eveningness Questionnaire* (MEQ)[2] was administered at both time points. Subjects maintained daily logs noting times of nocturnal sleep onset and offset; 7-day means were used for analysis of onset, midpoint, offset and duration. On the last evening of log entries subjects deposited saliva samples (30-min intervals, 4.5 h preceding sleep) under dim light conditions at home. Saliva was assayed for melatonin concentration using the Bühlmann RIA. The dim light melatonin onset (DLMO) at 3 pg/mL was used as the circadian phase anchor point. At this writing, sample sizes for DLMO and springtime sleep log and MEQ are smaller than for baseline MEQ.

Statistical Analyses. The Pearson *r* was used to compare continuous measures within and across seasons. Differences between means were evaluated using paired *t*-tests (2-tailed, α=.05) and the measure of effect size, *d* (0.3, small; 0.5, medium; 0.8, large).

Results. The frequency of morning types [MEQ scores >58, 22.5% (16/71)] and evening types [scores <42, 21.1% (15/71)] was nearly equal in winter, with most patients of intermediate type [scores 42-58, 56.3% (40/71)]. Although the average MEQ score increased in spring toward morningness (winter, 51.7±13.1; spring, 56.3±11.5; n=41, *P*=.02, *d*=0.37), evening types increased by 6.9±7.6 points while morning types showed no change (–0.4±4.7 points, *P*=.03, *d*=1.15). The springtime shift toward morningness was also reflected in significantly earlier DLMO (0.94±0.78 h, n=10, *P*=.004, *d*=0.82), with the largest phase shifts in the patients who were most delayed at baseline. Winter MEQ scores were highly correlated with the DLMO (*r*=–.81, n=35, *P*<.001) and sleep midpoint (*r*=–.80, n=71, *P*<.001), demonstrating close agreement among these phase indicators. However, the correlation of sleep duration with MEQ score and DLMO was nearly zero. Although the DLMO preceded sleep onset by 2.05±0.76 h on average, the interval depended on DLMO phase (*r*=–0.71, n=35, *P*<.001). While evening types fell asleep 2.01 h later than morning types, the interval between DLMO and sleep onset was significantly shorter for evening types (1.66±0.53 h) than for morning types (2.48±0.27 h, *P*<.03, *d*=1.91).

Conclusion. Our patients showed a symmetrical distribution of morningness-eveningness in winter. Increased morningness during spring was robust in evening types but absent in morning types. Evening types also had later DLMO's, yet fell asleep sooner than morning types relative to the DLMO. This implies that the phase relationship between melatonin onset and opening of the "sleep gate" is not a constant. Although morning types showed least seasonal change, response to morning light therapy is unrelated to baseline phase position when treatment is administered at the end of habitual sleep.[1] We hypothesize that morning types show greatest stability of circadian phase because the advancing springtime sunrise encroaches least on their subjective night.

(1) Terman JS, Terman M, Lo ES, Cooper TB. Circadian time of morning light administration and therapeutic response in winter depression. Arch Gen Psychiatry 2001;58:69-75. *(2)* Horne JA, Östberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. Int J Chronobiol 1976;4:97-110. *(3)* Avery D, Hellekson C, Bolte MA. The Morningness-Eveningness Questionnaire in SAD in the summer, winter, and after light treatments. Soc Light Treatment Biol Rhythms Abst 1997;9:18.

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