an increase in the proportion of I $\geqslant$ 2 sec. In Parkinson's disease, the proportions of the three types of intervals were decreased.

References

1 DE LEE, C.: Modifications hormonales et affections cérébrales. Contribution à l'étude du sommeil et des mouvements oculaires; Acta neurol. belg. (in press).

2 HEUSER, G. and EIDELBERG, E.: Steroid-induced convulsions in experimental animals. Endocrinology *69:* 915–924 (1961).

3 HOYT, W. F. and DAROFF, R. B.: Supranuclear disorders of ocular control systems in man. Clinical, anatomical and physiological correlations; in BACH-Y-RITA and COLLINS Control of eye movements, pp. 175–235 (Academic Press, New York 1971).

4 JUNG, R. and KUHLO, W.: Neurophysiological studies of abnormal night sleep and the Pickwickian syndrome; in AKERT, BALLY and SCHADÉ Sleep mechanisms, pp. 140–159 (Elsevier, Amsterdam 1965).

5 PRECHTL, H. F. R. and LENARD, H. G.: A study of eye movements in sleeping newborn infants. Brain Res. *5:* 477–493 (1967).

6 SPRENG, L. F.; JOHNSON, L. C., and LUBIN, A.: Autonomic correlates of eye movement burst during stage REM sleep. Psychophysiology *4:* 311–323 (1968).

Author's address: Dr. C. DE LEE, Department of Developmental Neurology, Born-Bunge Research Foundation, *B-Antwerp* (Belgium)

Sleep: Physiology, Biochemistry, Psychology, Pharmacology, Clinical Implications. 1st Europ. Congr. Sleep Res., Basel 1972, pp. 563–571 (Karger, Basel 1973)

The Mood-regulating Function of Sleep

M. KRAMER and T. ROTH

Veterans Administration Hospital, and Department of Psychiatry, University of Cincinnati, Cincinnati, Ohio

One goal of research in the field of hypnology is to search for the functions of sleep. Our approach [1] to the problem has been (1) to conceptualize sleep as basically being made up of two interrelated components – its psychology, e.g. rapid eye movement (REM) period mental contents, and its physiology, e.g. various neurophysiologically defined stages; (2) to view sleep as part of the wake-sleep-wake continuum, and (3) to begin our investigations utilizing minimal manipulations of sleep in order to obtain leads about the natural relationships between sleep and wakefulness which could then be pursued with more vigorous experimental procedures.

Our first consideration was to select a relevant and measurable attribute of wakefulness which varied from day to day and within a day and which, on *a priori* grounds, might bear some relationship to both the physiology and psychology of sleep. We selected mood as the attribute of wakefulness that seemed to meet the necessary criteria for our wakefulness function [2].

In our first pilot effort, we sought to establish that mood was reliably measurable, that it varied from day to day, and especially that it varied systematically from night to morning. We obtained Clyde mood scales [3], an adjective check list of 48 items which yields scores on 6 components – unhappy, friendly, aggressive, clear-thinking, sleepy and dizzy, from 8 male subjects immediately before retiring and immediately upon awakening from sleeping in our laboratory for 15–20 consecutive nights.

All of the men were able to complete the forms at each test session. The range of scores we obtained was consistent with the levels reported by Clyde [2] for his mood scales and, for us, reflected that we were measuring mood as defined by the instrument we were using. To test for the independence of mood subscales, intercorrelations among the 6 subscales were performed and no significant correlations were obtained. Day-to-day variability in mood was checked by comparing the range of scores in the first third to the last third of the experimental period. As we found no difference in the mean and standard deviations of the mood subscale scores in the first *versus* the last third, we felt it reasonable to assume that stereotypy of response had not occurred and that day-to-day variability in mood was being reflected.

To explore the major issue of whether mood varied from night to morning and that our 6 mood components were different from each other, we performed analyses of variance on the means and standard deviations of our night and morning mood scores. As can be seen in table I, the mean and standard deviation of the mood subscale scores are indeed different from each other. More important, the mean and standard deviation of the night mood subscale scores are different from the morning subscale scores. And lastly, table I indicates that there is an interaction between mean mood subscale scores and when they are obtained. This interaction is illustrated in figure 1. It can be seen that mean subscale scores decrease from night to

Table 1. Results of analyses of variance on means and standard deviations of night and morning Clyde mood scores (8 subjects)

Variable	Means			Standard deviations		
	F	d.f.	p	F	d.f.	p
Mood	9.33	5,35	<0.001	8.40	5,35	<0.01
Time	24.31	1,7	<0.001	9.00	1,7	<0.05
Mood × time	3.64	5,35	<0.01	0.74	5,35	>0.10

morning except in the case of ' sleepy ' which increases. The standard deviation measure does not show an interaction, all subscales show a lower variance in the morning than at night.

We were encouraged that mood, the waking function we had selected to relate to sleep, showed a difference between night and morning, thus warranting an attempt to relate it, mood, to sleep. If correlations with REM content and sleep physiology are found, then the possibility that the night to morning difference in mood is related to sleep would be supported and worth pursuing. Then sleep, in both or either of its aspects, might subserve a mood-regulating function.

In our second pilot effort, we sought a possible relationship between mood and the psychology of sleep. For our present purposes, we arbitrarily

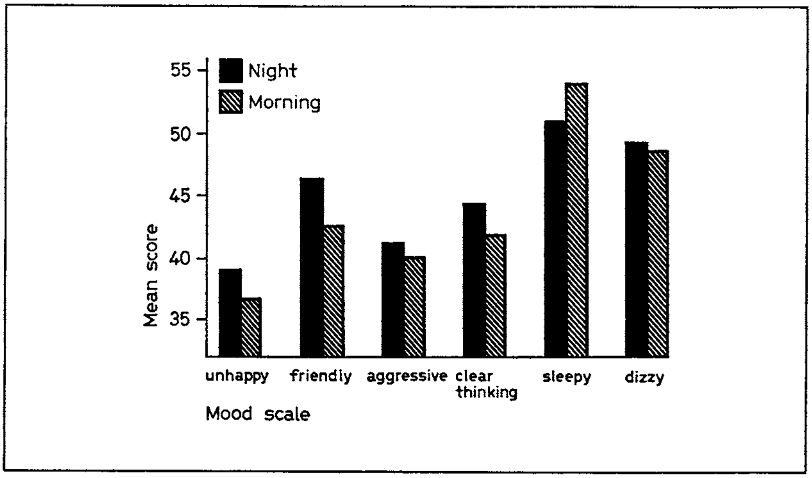

Fig. 1. Mean Clyde mood subscale scores, night and morning (Ss = 8).

chose to consider only the mental contents recovered from the end of the REM period for exploration. We studied two men for 20 consecutive nights in our laboratory. They were two of the 8 men in our first pilot effort. Using the standard EEG-EOG criteria [4], they were awakened at the end of each of the first four REM periods to report their dreams which were tape-recorded. They were then permitted to return to sleep following the fourth awakening for a minimum of 30 min, but without being allowed to have another REM period before being awakened for the day. The dream reports were transcribed, coded, and scored ' blind ' utilizing the characters (12 subscales), activities (9 subscales) and descriptive elements (10 subscales) scales of the HALL and VAN DE CASTLE [5] dream content scoring system. The subjects had filled out the Clyde mood scale before and after each night of sleep.

In order to explore for the possible relationship between mood and REM content, we correlated for each subject the rank order of his difference score (night mood minus morning mood) on each of the 6 Clyde mood subscales for the 20 nights with the rank order of the subject's content score on each content subscale (12 character subscales plus 9 activities subscales plus 10 descriptive elements subscales) for the 20 nights. We did 372 correlations (6 mood subscales $\times 31$ content subscales $\times 2$ subjects). Table II summarizes the results of our correlation matrix indicating the relationship between mood change and dream content as indexed by the number of significant correlations obtained as compared to what would have been expected by change.

Overall, we obtained three times the number of significant correlations between mood change and dream content that we could have expected by chance (54 as compared to 18.6 expected by chance). Specifically, we obtained our largest number of correlations in relationship to the ' unhappy ' mood subscale (23 compared to 3.1) and the character subscales (27 compared to 7.2). As might then be expected, the largest number of correlations were between the change score on the ' unhappy ' mood subscale and the character subscales (14 compared to 1.2). The nature of this interrelationship being that the more characters, regardless of type, the greater the decrease in the ' unhappy ' subscale score from night to morning.

It seemed likely to us, from this second pilot study, that the difference in mood from night to morning did relate to REM content. And, that who and, to a lesser degree, what we dream about could make a difference in how we feel. We are aware that correlation is not causation. Is it as likely that the difference in mood, night to morning, affected the dreams as

Table II. Significant correlations between mood changes and dream content (2 subjects)

	Both Ss	S1	S2
I. *Overall*			
A. Number of correlations done	372.0	186.0	186.0
B. Expected (5% level)	18.6	9.3	9.3
C. Observed	54.0	20.0	34.0
II. *With ' unhappy ' mood subscale*			
A. Number of correlations done	62.0	31.0	31.0
B. Expected (5% level)	3.1	1.6	1.6
C. Observed	23.0	8.0	15.0
III. *With character scales*			
A. Number of correlations done	144.0	72.0	72.0
B. Expected (5% level)	7.2	3.6	3.6
C. Observed	27.0	8.0	19.0
IV. *Between ' unhappy ' and character*			
A. Number of correlations done	24.0	12.0	12.0
B. Expected (5% level)	1.2	0.6	0.6
C. Observed	14.0	5.0	9.0

that the dreams ' caused ' the difference in mood? Or, that both the difference in mood and the night-to-night differences in dream content are related to yet a third factor affecting both? Our pilot effort could not permit us to choose between these alternative explanations, but did support, we believe, the hypothesis of a relationship between ' change ' in mood night to morning and the mental content that intervenes.

In our third pilot effort, we were interested in exploring the possibility that the physiological parameters of sleep, the ' other ' aspect of sleep, might show relationships to the difference in mood between night and morning. We studied 6 men (the remaining 6 from pilot study 1) for 15 consecutive nights in the laboratory. Clyde mood scales were collected before and after sleep and standard continuous EEG-EOG recordings were taken each night. The sleep records were scored using the Rechtschaffen-Kales classification system. To explore for the possible relationship between mood and sleep physiology, we correlated for each subject the rank order of his mood difference score (night mood minus morning mood) on each of the 6 Clyde mood subscales for the 15 nights with the rank order of the subject's scores on five physiological parameters of sleep (total sleep time, REM time,

Table III. Significant correlations between mood changes and sleep measures (6 subjects)

	All s
I. *Overall*	
A. Number of correlations done	180.0
B. Expected (5% level)	9.0
C. Observed	26.0
II. *With 'sleepy' mood subscale*	
A. Number of correlations done	30.0
B. Expected (5% level)	1.5
C. Observed	7.0
III. *With total sleep time*	
A. Number of correlations done	36.0
B. Expected (5% level)	1.8
C. Observed	11.0
IV. *Between 'sleepy' and total sleep time*	
A. Number of correlations done	6.0
B. Expected (5% level)	0.3
C. Observed	4.0

stage 3–4 time, stage 2 time and number of awakenings). We did 180 correlations (6 mood subscales ×5 physiological parameters ×6 subjects). Table III summarizes the results of our correlation matrix by indicating the relationship between mood 'change' and the physiological parameters of sleep as indexed by the number of significant correlations obtained as compared to what would have been expected by chance.

Overall, we obtained almost three times the number of significant correlations between mood 'change' and the physiological parameters of sleep that we could have expected by chance (26 as compared to 9). Specifically, we obtained our largest number of correlations in relationship to the 'sleepy' mood subscale (7 as compared to an expected 1.5) and to the measure of total sleep time (11 as compared to 1.8). As then might be expected, the largest number of significant correlations was between the 'sleepy' mood subscale and total sleep time (4 obtained as compared to 0.3 expected by chance). The nature of this interrelationship being that the more total sleep time the more sleepy one feels. Although this seems absurd on a logical basis, it must be remembered that this interrelationship is based on total sleep time only within the normal range (6–8 h).

Table IV. Percentage of significant correlations obtained with each mood subscale from the dream and sleep pilot studies[1]

	Dream	Sleep
Unhappy	43	15
Friendly	15	7
Aggressive	16	7
Clear-thinking	6	19
Sleepy	9	27
Dizzy	11	25

1 χ^2 performed on actual frequencies shows distributions to be significantly different, $p < 0.05$.

Our third pilot effort lends credence to the possibility that mood ' change ' across the night does relate to the physiological parameters of sleep. And that the amount and, to a lesser degree, perhaps the type, i.e. neurophysiologically defined, of sleep one obtains during the night does make a difference in how one feels. Again it would be premature, even if this relationship were more firmly established, to make the leap to causation. As we discussed in the case of our second pilot effort, relating mood difference to REM content, alternative explanations are possible and cannot be chosen between from this pilot effort. Nevertheless, the proposition that sleep, now in both of its aspects, may subserve a mood regulatory function seems to be a viable explanation worthy of further exploration.

As we obtained leads from our pilot studies that there appears to be a relationship between mood difference night to morning and the physiology and psychology of sleep, we wondered if these two aspects of sleep might be differentially related to the various aspects of mood (the 6 mood subscales of the Clyde mood scale). We compared the distribution of our significant mood subscale correlations from pilot study 2 (the ' dream ' study) with those we obtained from pilot study 3 (the sleep physiology study). Utilizing a χ^2 analysis to test for a difference between the distribution of the number of significant correlations on the 6 mood subscales, we did indeed find that the two distributions were different statistically. Table IV shows the distribution of significant correlations obtained from the dream and sleep physiology pilot studies reported as percentages, although the statistical analysis was performed on the actual frequencies. The unhappy, friendly and aggressive subscales appear to be more related to the psychology of sleep while the clear-thinking, sleepy and dizzy subscales may bear more of a relationship to the physiology of sleep.

Our pilot efforts have left us with the conviction that the systematic exploration of the relationship between mood and sleep might well lead us to an understanding of one significant function of sleep: its mood-regulatory capacity. In order to establish the mood-regulatory function of sleep, it will be necessary to establish definitely that mood, as we measure it, varies systematically night to morning. Secondly, it will be necessary to test additional explanations for our finding, such as the possibility that the difference in mood scales is also due to time, i.e. any 8-hour period might show the same ' change ', or due to social isolation, i.e. as sleep is a period of limited interpersonal contact does this isolation, in addition to sleep, account for some of the mood difference? Both of these possibilities must be tested to enhance our findings being related to sleep processes. Thirdly, it will be necessary to show that the correlations between mood difference night to morning and the physiological and psychological aspects of sleep can be demonstrated in signficant numbers of men and women. Fourthly, it will be necessary to explore the mood-sleep relationship in exaggerated and pathological states in which mood is known to be altered, e.g. sleep deprivation and psychological depression. And, lastly, it will be necessary to examine the possible differences that the sex or age of people make in their ability to regulate mood by sleep. All of these experimental explorations are necessary if we are to establish the mood-regulatory function of sleep.

We are of the opinion that our present work, in addition to suggesting a possible avenue for exploration to achieve the goal of every sleep researcher, i.e. finding one of the functions of sleep, has suggested two other points of importance. Firstly, that exploring sleep function in relationship to wakefulness or as part of the wake-sleep-wake continuum is a useful approach. And, secondly, that the systematic examination of the sleep-wake relationship in as naturalistic a manner as possible, i.e. with minimal manipulation, may be an important starting-point to explore any hypothesis related to sleep function.

References

1 Kramer, M.: Review of Hartmann Sleep and dreaming. The value of sleep, Contemp. Psychol. *16* (8): 510–512 (1971).
2 Hartmann, E.: Sleep and dreaming (Little Brown, Boston 1970).

3 CLYDE, D.: Manual for the Clyde mood scale (Biometric Laboratory, University of Miami, Coral Gables 1963).
4 RECHTSCHAFFEN, A. and KALES, A.: A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects (UCLA Brain Information Service, 1968).
5 HALL, C. and CASTLE, VAN DE, R.: The content analysis of dreams (Appleton-Century-Crofts, New York 1966).

Author's address: Dr. M. KRAMER, Sleep Lab. VA Hospital, *Cincinnati, Ohio* (USA)

Sleep: Physiology, Biochemistry, Psychology, Pharmacology, Clinical Implications. 1st Europ. Congr. Sleep Res., Basel 1972, pp. 571–577 (Karger, Basel 1973)

The Relationship between Weight Change and Sleep

A Study of Psychiatric Outpatients[1]

A. H. CRISP and E. STONEHILL

Central Middlesex Hospital, London and St. George's Hospital Medical School, London

Introduction

This paper describes a small part of a research project which began about 7 years ago with the observation that patients with anorexia nervosa report a disturbance of their normal sleep pattern [CRISP, 1967]. This observation was subsequently confirmed in a systematic study of these patients in which early morning waking, as measured by patients' self-reports, nocturnal motility and all-night EEG recordings, was found to be closely related to weight change independent of mood [CRISP and STONEHILL, 1971, CRISP *et al.*, 1971]. Interrupted sleep was also a feature but not sleep disturbance at the beginning of the night. In a further study, obese subjects losing weight were found to sleep less, especially in the second half of the night, although this finding was less consistent [CRISP and STONEHILL, 1970; CRISP *et al.*, 1972].

A further study [STONEHILL, 1972; CRISP and STONEHILL, 1972; STONEHILL and CRISP, 1972], part of which is described here, was designed to test a hypothesis that a relationship exists between changes in nutri-

1 The work was supported by a grant from the Medical Research Council.