Research Reports

Spicy meal disturbs sleep: an effect of thermoregulation?

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Tabasco sauce and mustard taken with the evening meal markedly disturbed sleep of six, young, healthy male subjects; reducing slow wave and stage 2 sleep, increasing total time awake and tending to increase sleep onset latency. Whilst post meal effects on temperature and oxygen consumption were not significantly different from control meals the spicy food condition elevated body temperature during the first sleep cycle. The possibility that the spice principle capsaicin affects sleep via changes in body temperature is discussed.

While the thermic effect of food is a well established phenomenon (Rothwell and Stock, 1981), it has only recently been reported that pungent spices in food result in a higher post meal metabolic rate (Henry and Emery, 1986). Other work has shown that capsaicin, the principle active ingredient of the pungent spice Tabasco sauce, stimulates energy metabolism (Kawada et al., 1986; Cameron-Smith et al., 1990). Isothiocyanate from mustard is known to stimulate heat production in isolated brown adipose tissue of the rat (Yoshida et al., 1988). In conjunction with a programme of research into the metabolic effects of spicy foods, we assessed their effects on sleep. Our hypothesis was that pungent spices taken with the evening meal would increase metabolic rate, and thus result in a disturbance of sleep, a connection indicated by Roehrs et al. (1989).

Dietary factors known to affect sleep include caffeine and alcohol which have clearly defined detrimental sleep effects (e.g., Zarcone, 1989). Variation in carbohydrate intake is known to affect rapid eye movement (REM) sleep (Phillips et al., 1975; Lacey et al., 1975) while a bedtime high carbohydrate supplement resulted in reduced sleep stage 1 (ST1) over the whole night and increased REM over the first half of the night, and a whole night decrease in stage 4 (ST4) of sleep (Porter and Horne, 1981). Other bedtime supplements have been studied (Adam, 1980; Brezinova and Oswald, 1972) but an effect of spices on human sleep has not yet been reported.

Six healthy subjects, free from sleep disturbances, were recruited from a subject pool of aerobically fit men experienced in the sleep laboratory. Subjects had a mean age of 22 (S.D. 3.3) and body mass index 20.9 (S.D. 1.5). Subjects were told they would receive spices that could and could not be tasted but not told when nor what effects were anticipated. They were instructed to refrain from alcohol and caffeine; vigorous or prolonged exercise; day-time naps,

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hot baths and showers; and to have a bland lunch of approximately the same caloric content at the same time on each experimental day. Subjects were studied in the sleep laboratory on five non-consecutive occasions within 2 weeks, an adaptation night, then two standard meal (A) and spicy meal (B) nights in an ABBA design.

Subjects reported to the laboratory at 19.00 h, placed a rectal temperature probe in position and rested supine for 20 min after which oxygen consumption was determined for 20 min. They then had their meal within 40 min and returned to the supine position for a 60 min oxygen consumption determination following which subjects arose to have the sleep electrodes fitted. Subjects were typically ready for bed by 22.30 p.m. and another 20 min oxygen consumption measure and temperature (REST) was taken before removing the oxygen mask and checking electrode placement prior to lights out at 23.00 h. During the night standard sleep measures, EEG(C3/A2), EMG and EOG (Rechtschaffen and Kales, 1968), were taken in addition to the monitoring of rectal body temperature. Subjects were woken at approx. 07.00 h. Sleep measures were recorded directly onto chart paper using a Beckman Dynagraph Model 411. Records were scored blind by two experienced scorers and disagreements were resolved by discussion. Scoring of sleep cycle data

was conducted according to the rules of Trinder et al. (1982).

Meals were standard portions of the same commercial canned soup, frozen TV dinners and frozen muffins comprising 3940 kJ and provided a protein, carbohydrate and fat content similar to the Australian average diet (National Dietary Survey 1983 published by the Department of Community Services and Health). The spicy sauce comprised 3.0 g each of commercially available Tabasco sauce (McIlhenny and Co.) and hot English mustard (Keens prepared English Mustard) and was similar to that used by Henry and Emery (personal communication).

The rectal temperature probe (Yellow Springs, YSI series 401, calibrated to an accuracy of 0.01°C at the beginning of each session) was inserted to a depth of 10 cm and fixed by surgical tape. The signal was fed, via a cable long enough to allow movement around the laboratory, into a PDP 11/23 computer and sampled at 1 min intervals.

Oxygen consumption was measured at 23–25°C with the subject supine on a bed with eyes open and the room light on. Expired air was collected by a face mask attached to a two-way breathing valve. Breath by breath computation of oxygen consumption was achieved using a Fleisch Pneumotachograph and Bould PM 15E Pressure Transducer for ventilation and an Applied Elec-

TABLE I

Effect of a spicy meal on length of sleep stages and sleep efficiency

Data are the means ± S.D. of values for all six subjects on control and spicy food nights. Results are in min except for sleep efficiency. Significance levels obtained by analysis of data as a one way ANOVA within subjects design are displayed on the right. ns, not significant; SOL, sleep onset latency; REM, rapid eye movement sleep; SWS, slow wave sleep (stage 3 + stage 4 sleep); TIB, time in bed; TST, total sleep time; MT, movement time; ST1, stage 1 sleep; TTA, total time awake.

Sleep variable	Control	Spicy	Significance
SOL	16.1 ± 10.4	34.8 ± 26.6	$0.1 > P > 0.05$
Stage 1	24.2 ± 8.3	28.0 ± 6.5	ns
Stage 2	265.2 ± 26.6	257.0 ± 30.5	$P < 0.05$
Stage 3	30.7 ± 5.0	29.1 ± 4.3	ns
Stage 4	33.2 ± 14.8	23.0 ± 20.2	$P < 0.06$
SWS (total)	63.9 ± 14.0	52.1 ± 17.9	$P < 0.05$
REM	89.2 ± 18.7	83.7 ± 22.3	ns
TST	445.2 ± 39.0	423.3 ± 39.3	$P < 0.002$
TTA	25.3 ± 25.2	43.1 ± 30.2	$P < 0.03$
Disturbance TTA + MT + ST1	52.3 ± 34.5	73.6 ± 28.9	$P < 0.03$
Sleep efficiency TST/TIB	0.94 ± 0.05	0.90 ± 0.07	$P < 0.03$

trochemistry S-3A Oxygen Analyzer for expired oxygen levels with signals fed via a Beckman R411 Recorder to the PDP 11 computer as detailed by Colrain et al. (1988).

Data were first averaged over replications within conditions and subjects before use in all subsequent analyses, tables and figures. Replications were designed to reduce within subject variability and were not significantly different. Data were analysed by a one-way ANOVA within subjects design with two levels (bland food versus spicy food) on the CSS statistical package using an IBM 386 computer.

Sleep data are displaced in Table I. Slow wave (SWS) and stage 2 (ST2) sleep are reduced and sleep onset latency (SOL) tends to increase with the spicy meal. Thus, the sleep disturbance factor, which is defined as total time awake during the night (TTA) plus ST1 plus movement time during the night (MT), increases. Sleep efficiency, defined by total sleep time (TST) as a fraction of time in bed (TIB), is significantly reduced with the spicy meal.

Oxygen consumption showed no significant differences between the two conditions across all subjects but four of the subjects showed a tendency towards increased oxygen consumption in the spicy food condition. Average oxygen consumption per min over the total post meal measurement period was significantly greater than in the pre meal measurement period ($F(1,5) = 36.5$; $P < 0.005$) indicating the expected thermic effect of food. Temperatures over the same time frame showed no significant difference, although temperature data could not be collected to check for an immediate post meal effect.

Body temperature data collected throughout the night was analysed using a repeated measures ANOVA comparing the two conditions at equivalent sleep stages: at rest during the last metabolic rate determination (REST), then the 5 min before and after lights out (LO), sleep (ST2) onset, slow wave sleep (ST3) onset and REM onset for the first sleep cycle and a similar analysis for onset of second sleep cycle ST2, ST3, and REM and are graphed in Fig. 1. There is a significant diet by sleep stage interaction during both the first sleep cycle ($F(8,40) = 2.21$; $P < 0.05$) and the
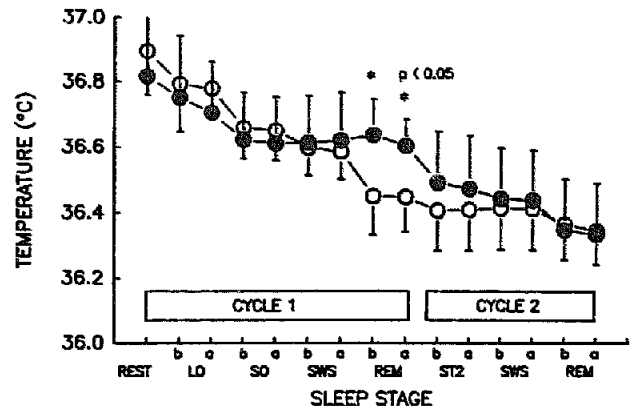

Fig. 1. Effect of a spicy meal on core temperature at various stages of arousal. Temperatures are 5 min averages during the last metabolic rate assessment (REST) and before (b) and after (a) lights out (LO), sleep onset (SO) and the onset of first and second cycle sleep stages of slow wave sleep (SWS), rapid eye movement sleep (REM) and stage two sleep (ST2). Values given are mean±S.E. for all six subjects on control (O) and spicy (●) food nights. * Denotes points significantly different by t-test ($P < 0.05$).

second sleep cycle ($F(5,25) = 3.50$; $P < 0.05$). Related measures t-tests indicate significant differences between conditions during the 5 min before ($t = 3.0$; $P < 0.05$) and after ($t = 2.4$; $P < 0.05$) the first REM stage.

It appears that the period of maximum effect of the spices on sleep is in the first and second sleep cycles as there is no apparent difference between the two conditions at the conclusion of the second sleep cycle. This is surprisingly about 4 to 7 h after ingestion of the spices but there is probably an effect on thermoregulation which is only apparent under the physiological conditions of sleep or REM in particular. Thermoregulation is impaired during REM sleep in mammals (Parmeggiani, 1990) but this alone would fail to explain the clear effects on length of other sleep stages. Certainly rats desensitized to capsaicin have their sleep affected in a non-thermoneutral environment (Benedek et al., 1980) but our laboratory is thermoneutral.

The effect of the spicy meal could be manifest through two broad mechanisms. Firstly capsaicin and isothiocyanate could increase oxygen consumption by direct stimulation of heat producing tissues (Henry and Emery, 1986; Kawada et al.,

1986; Yoshida et al., 1988). Secondly as capsaicin is concentrated in peripheral and central nervous tissue (Donnerer et al., 1990) and directly stimulates sensory C fibres (Buck and Burks, 1986) it could trigger brain reflexes. This could affect sleep directly or again by increasing heat production. Accordingly it seems likely that the spicy diet affects sleep either directly through action on arousal or via changes to body temperature and/or body temperature rhythm which in turn affects sleep.

Our subjects were all endurance athletes and such subjects have efficient thermoregulation and a reduced thermic effect of food (LeBlanc, 1986; LeBlanc et al., 1984). Also four of them report being accustomed to eating spicy food at some stage in the past (though not currently). Thus, we may have seen a more limited response to the spices than might be expected from a sedentary and/or unaccustomed group of subjects.

From this study it is clear that spicy foods affect sleep in healthy good sleepers. Spices may affect those with lighter or more vulnerable sleep to a greater degree. It would be of interest for a future study to assess the effect of spicy foods on insomniacs.

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