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Sleep during Ramadan intermittent fasting

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SUMMARY During the month of Ramadan intermittent fasting, Muslims eat exclusively between sunset and sunrise, which may affect nocturnal sleep. The effects of Ramadan on sleep and rectal temperature (Tre) were examined in eight healthy young male subjects who reported at the laboratory on four occasions: (i) baseline 15 days before Ramadan (BL); (ii) on the eleventh day of Ramadan (beginning of Ramadan, BR); (iii) on the twenty-fifth day of Ramadan (end of Ramadan, ER); and (iv) 2 weeks after Ramadan (AR). Although each session was preceded by an adaptation night, data from the first night were discarded. Polysomnography was taken on ambulatory 8-channel Oxford Medilog MR-9000 II® recorders. Standard electroencephalogram (EEG), electro-oculogram (EOG) and electromyogram (EMG) recordings were scored visually with the PhiTools ERA© software. The main finding of the study was that during Ramadan sleep latency is increased and sleep architecture modified. Sleep period time and total sleep time decreased in BR and ER. The proportion of non-rapid eye movement (NREM) sleep increased during Ramadan and its structure changed, with an increase in stage 2 proportion and a decrease in slow wave sleep (SWS) duration. Rapid eye movement (REM) sleep duration and proportion decreased during Ramadan. These changes in sleep parameters were associated with a delay in the occurrence of the acrophase of Tre and an increase in nocturnal Tre during Ramadan. However, the 24-h mean value (mesor) of Tre did not vary. The nocturnal elevation of Tre was related to a 2–3-h delay in the acrophase of the circadian rhythm. The amplitude of the circadian rhythm of Tre was decreased during Ramadan. The effects of Ramadan fasting on nocturnal sleep, with an increase in sleep latency and a decrease in SWS and REM sleep, and changes in Tre, were attributed to the inversion of drinking and meal schedule, rather than to an altered energy intake which was preserved in this study.

KEYWORDS core temperature, feeding, Islamic fasting, meal times

INTRODUCTION

As one of the five pillars of Islam, Ramadan is observed by millions of Muslims around the world. For 1 month each year, food, drinking and smoking abstinence is the rule from dawn to sunset. The faithful usually eat a large break-of-fasting meal after sunset (Ftur) and dinner before sleeping (Ichaa). A third meal (Suhur) can be taken just before dawn (Taoudi Benchekroun *et al.* 1999).

This nutritional inversion is thought to induce a loss in total energy intake (Angel and Schwartz 1975; Husain *et al.* 1987) with a drop in body weight (Bigard *et al.* 1998; Fedail *et al.* 1982; Hallak and Nomani 1988; Husain *et al.* 1987; Sweileh *et al.* 1992), although no change in body fat was reported in a recent study (Bigard *et al.* 1998). These metabolic modifications are accompanied by endocrine changes thought to be capable of altering sleep. Compared with non-fasting controls, cortisol secretion is significantly higher at 7 and 9 pm on the 28th day of Ramadan (Sliman

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et al. 1993). An early evening increase in cortisol may lead to alterations in sleep patterns, such as an increased sleep latency and decreased rapid eye movement (REM) sleep. It is known that an elevated adrenocortical activity at night is accompanied by a decrease in REM sleep (Buguet *et al*. 1998; Krieger and Glick 1974).

As a result of the delayed bedtime and shortened sleep, partial sleep deprivation can be expected to occur in Ramadan observants. The question remains as to whether such a sleep loss may induce modifications in sleep composition and favour daytime drowsiness (Roky *et al*. 2000) and possible napping. In Morocco during Ramadan, working hours are from 8 am to 3 pm, and the end of work coincides with the afternoon peak of somnolence (Lavie 1986). Therefore, Ramadan could be considered as a potential model to assess possible influences of the daily patterning of meals on sleep.

As sleep onset is triggered in the downward slope of core temperature (Murphy and Campbell 1997), events supposed to be thermogenetic are likely to delay sleep. This is observed after evening meals (Smith *et al*. 1994), exposure to bright light prior to sleep (Dijk *et al*. 1991) and after nocturnal exercise (Mizuno *et al*. 1998).

The purpose of this study was therefore to analyse the impact of Ramadan on nocturnal sleep patterns. The expected changes were examined in relation to the nocturnal rectal temperature pattern.

METHODS

Subjects

Eight healthy Muslims (20–28 years old) volunteered for the 2-month study and gave their informed consent to participate in the protocol, which was approved both by the University ethics committee and the Religious Committee of the Hassan II Foundation for Scientific and Medical Research on Ramadan. They were selected amongst 27 students after a thorough medical and biological examination and on their good and regular usual eating (breakfast at 07.00 ± 1 h, lunch at 12.00 ± 1 h and dinner at 20.00 ± 1 h) and sleeping habits (sleep between 23.00 and 07.00 ± 1 h). They were not regular nappers and belonged to the intermediate chronotype following their responses to the morningness vs. eveningness questionnaire of Horne and Östberg (1976). They were also selected on their eating habits (two nocturnal meals: break of fasting at 18.00 h; night meal between 22.00 and 24.00 h) and sleeping behaviour during Ramadan (sleep between 24.00 ± 1 h and 08.00 ± 1 h). They were non-smokers and were not addicted to caffeine or alcoholic beverages.

General protocol

The subjects reported at the laboratory on four occasions: (i) baseline recordings 15 days before Ramadan (BL); (ii) on the eleventh day of Ramadan (beginning of Ramadan, BR); (iii) on the twenty-fifth day of Ramadan (end of Ramadan, ER); (iv) and for recovery recording 2 weeks after Ramadan (AR). Polysomnographic recordings were taken continuously throughout each of the four experimental sessions. However, although the subjects slept at the laboratory during two consecutive nights (three nights in the BL condition), only data obtained from the second night recordings were considered to avoid any first night effect (Agnew *et al*. 1966). Except for the recording sessions, which were held at the laboratory, the subjects slept at home throughout the 2-month investigation. Everyday, they noted the time at which they bedded down and arose in the morning.

During the Ramadan sessions, they neither ate nor drank during the daytime. Meals schedule and composition were as follows: break of fasting (Ftur) between 18.00 h (at the beginning of Ramadan) and 18.30 h (at the end of Ramadan) with two bowls of soup, two hard boiled eggs, two small pancakes, two honey cakes, six dates, water, one glass of banana milkshake, one glass of milk tainted with coffee, a light snack with one glass of light mint tea with a croissant at 21.00 h; and the dinner (Ichaa) including a one fourth chicken with boiled vegetables, mixed tomato-cucumber-lettuce salad, one banana or apple at 22.30 h. Before and after Ramadan the overall energy intake and composition was similar over the 24-h period. Breakfast was taken at 08.00 h and included one glass of milk tainted with coffee, water, 150 g of bread with butter and honey. Lunch was served at 12.30 h with a one fourth of chicken with boiled vegetables, mixed tomato-cucumber-lettuce salad, one banana or apple. A light snack with croissant and mint tea was taken at 17.00 h. Dinner, at 20.30 h was composed of two bowls of soup, two hard boiled eggs, two honey cakes, six dates, water, one glass of milk and one banana. When at home, the subjects were asked to comply with this meal composition and with the schedule adopted during the laboratory investigations.

At the end of each session, body weight was taken with a personal scale (precision 200 g) and skinfold thickness was measured with a Harpenden skinfold caliper at the subscapular, tricipital and iliac sites and averaged over the three sites.

Polysomnography

Polysomnographic recordings of electroencephalogram (EEG), electro-oculogram (EOG) and electromyogram (EMG) were taken on ambulatory 8-channel Oxford Medilog MR-9000 II® recorders (Oxford Instruments, Oxford, Great Britain). The EEG was recorded with Oxford Instruments cup electrodes fixed on the scalp with collodion at sites C3, C4, A1, A2, Cz and Fz, the C3 and C4 electrodes being connected to the linked ears reference (A1–A2). The EOG was taken from two electrodes fixed at the outer canthi of the eyes, on diagonal to record both vertical and horizontal eye movements; chin EMG from two electrodes at the tip of the chin; anterotibial EMG was taken to record leg movements; ECG and thoracic respiratory movements were collected

from an XR-90 Oxford system plugged into the MR-9000 II recorder. No motor, cardiac or respiratory disturbances were noted. The quality of the recording was checked with the Oxford Mentor© software on a Toshiba T3200SXC. The analogic signals were stored on C-120 cassettes which were then transferred and converted to digital with the Oxford Instruments Vision© software.

Polygraphic traces were scored blind as to subjects and study phase using the ERA© software (PhiTools®, Grenoble, France). Concomitantly, two sleep scorers agreed to the classification of each 20-s scoring epoch, following classical criteria for wakefulness, stages 1, 2, 3 and 4 of non-rapid eye movement (NREM) sleep, stages 3 and 4 constituting slow wave sleep (SWS), and REM sleep (Rechtschaffen and Kales 1968). Complete sleep measures reports were automatically generated by the software according to Buguet *et al.* (1987). Time in bed, sleep onset (beginning of three consecutive 20-s epochs of stage 1 or 1 epoch of any other sleep stage, sleep period time (SPT = time from sleep onset to last awakening), time spent in wakefulness occurring during SPT (WASO), total sleep time (TST = SPT – WASO), and sleep latency (time from lights out to sleep onset) were calculated for each recording night. Latency to SWS and REM sleep (from sleep onset to the first epoch of SWS and REM sleep) were also computed. Each stage of sleep was analysed by measuring total duration, number of episodes, mean episode duration and proportion vs. TST. The first sleep cycle extends from sleep onset to the end of the first episode of REM sleep, the second sleep cycle from the end of the first REM sleep episode to the end of the second, etc., two REM sleep episodes being considered to differ if separated by more than 15 min. The distribution of wakefulness, stage 2, SWS and REM sleep was examined by calculating the relative percentage of each stage during each NREM–REM sleep cycle vs. the total overnight duration of the stage. Sleep instability was calculated using the ratio between the number of stage changes and TST, and the REM sleep stability index represented the percent of actual REM sleep within REM sleep episodes.

Core temperature measurements

Rectal temperature (Tre) was recorded during the 48-h laboratory sessions with a thermistor probe inserted 12 cm into the rectum and attached to a light (<150 g) portable belt recorder with a window displaying core temperature continuously. The one-channel recorder (Inovra, La Tronche, France) was set for 1-min temperature sampling intervals. Temperature values were downloaded into a PC with the Easylog© software (Lascar, Salisbury, UK). Due to either equipment failure or probe dropping out especially at night, a complete set of data was obtained from only six subjects for at least one 24-h recording. Temperature data were computed in 10- and 20-min averages and were analysed after being time-locked to lights out and sleep onset. The meal effect was observed during the first 3 h following meals and analysed as a difference with premeal values.

Circadian characteristics of Tre were calculated with an in-house software using a signal decomposition procedure in Fourier series, with the fundamental harmonic fixed on 24 h and a secondary harmonic on 12 h. The assessed variables were as follows: mesor (mean 24-h value), amplitude (the difference between the maximum and minimum values of the calculated fit of Tre in a complete cycle), acrophase (the time at which the maximum calculated value encountered in the cycle occurs) and bathyphase (the time at which the minimum calculated value encountered in the cycle occurs; Nelson *et al.* 1979).

Statistical analysis

The influence of Ramadan on all variables was analysed using a non-parametric Friedman analysis of variance (ANOVA), with the experimental nights as factor. When the ANOVA indicated significant differences, post-hoc multiple comparisons between sessions were performed using paired Wilcoxon *t*-tests. For the sleep measures, the first nights and the second nights of the successive sessions were compared separately, in order to avoid any possible adaptation effect within each session. When the Friedman ANOVA yielded significant differences in Tre time course, the effects of the experimental sessions were assessed for each 20-min moving average using Wilcoxon *t*-tests for matched pairs. Non-parametric tests were preferred because of their independence on normal distribution of the data and because of the small sample size.

RESULTS

Body weight changed over sessions ($P = 0.0024$) with a decrease during Ramadan and restoration afterwards (Table 1), along with similar modifications in skinfold thickness ($P = 0.0169$). Table 2 gives the sleep schedules followed by the subjects over the 2-month investigation when sleeping at home. Compared with both baseline and recovery, bedding down was delayed during Ramadan, whether it be during the first or second half of the month.

General sleep characteristics

Sleep values and stages distribution from the polysomnographic recordings are given in Tables 3 and 4, respectively. The main finding of the study was that sleep is delayed and its architecture modified during Ramadan. Sleep latency varied throughout the investigation (Fr = 28.958, $P = 0.0001$) and was longer than in BL during Ramadan (post hoc tests in Table 3), whether at the beginning (+41 min on BR) or at the end (+39 min, for ER). However, neither SWS latency nor REM sleep latency changed significantly.

Sleep period time also varied through sessions (Fr = 18.58, $P = 0.0098$). Compared with BL, it decreased in BR (−48 min) and ER (−32 min). Total sleep time showed a similar change (Fr = 24.74, $P = 0.0009$), being lower than in BL during Ramadan (BR, –41 min and ER, –39 min). The

Table 1 Body weight (kg) and skinfold thickness (mm) throughout the investigation, before Ramadan (baseline), at the end of the first 2 weeks of Ramadan (beginning), at the end of the last 2 weeks of Ramadan (end), and at the end of the 2-week recovery (mean ± SEM)

Variable	*Baseline*	*Beginning of Ramadan*	*End of Ramadan*	*Recovery*	*Friedman test*
Body weight	67 (2.8)	64 (2.4)	65 (2.7)	67 (2.7)	Fr = 14.426 $P = 0.0024$
Skinfold thickness	6.7 (0.3)	6.1 (0.3)	6.2 (0.4)	6.6 (0.4)	Fr = 10.200 $P = 0.0169$

Table 2 Sleep schedules noted by the subjects throughout the 2-month investigation before (baseline), during the first 2 weeks of Ramadan, during the last 2 weeks of Ramadan, and the 2-week recovery

Variable	*Baseline*	*First 2 weeks of Ramadan*	*Last 2 weeks of Ramadan*	*Recovery*	*Friedman Test*
Bedding time (h ± min)	23.48	00.36	00.41	23.52	Fr = 15.08
	7	11	13	15	$P = 0.0018$
Arising time (h ± min)	08.03	08.52	09.08	08.32	Fr = 19.05
	6	17	18	16	$P = 0.0003$

Table 3 Sleep characteristics of the eight subjects before (baseline), at the beginning and the end of Ramadan, and after Ramadan. Data from the second night recording are shown

Sleep variable	*Baseline (BL)*	*Beginning of Ramadan (BR)*	*End of Ramadan (ER)*	*After Ramadan (AR)*	*Night effect*
Sleep latency	19.2 (2.6)	60.5 (10.6)**	58.1 (14.8)**	58.9 (15.7)*	0.0001
Sleep period time	454 (4.7)	406 (11.9)**	422 (14.9)	421 (15.9)*	0.0098
Total sleep time	422 (9.0)	381 (11.6)*	383 (16.7)**	390 (14.1)*	0.0009
No. of sleep cycles	4.8 (0.3)	4.5 (0.2)	3.9 (0.4)	4.1 (0.4)	NS
Sleep cycle duration	92.1 (4.1)	91.1 (4.2)	102 (7.9)	88.0 (12.1)	NS
Intra-sleep awakening	34.9 (5.3)	24.8 (3.9)	38.7 (15.2)	30.5 (3.5)	NS
Stage 1	28.0 (6.8)	23.3 (3.4)	18.5 (2.8)	27.9 (2.9)	NS
(% TST)	6.6 (1.5)	6.1 (0.9)	4.9 (0.8)	7.2 (0.9)*	0.0269
Stage 2	212 (12.8)	194 (9.3)	211 (10.2)	187 (14.3)	NS
(% TST)	50.2 (2.5)	50.9 (3.0)	55.1 (2.6)*	47.9 (2.7)	0.0001
Slow-wave sleep	86.7 (6.8)	79.0 (6.4)	78.8 (6.4)**	78.6 (5.4)	NS
(% TST)	20.6 (1.7)	20.8 (2.3)	20.5 (1.3)	20.3 (1.5)	NS
SWS latency	11.8 (1.7)	17.7 (2.3)	30.5 (15.1)	27.7 (9.3)	NS
NREM	327 (9.7)	296 (6.7)*	309 (12.6)	294 (12.5)*	0.05
(%TST)	77.3 (1.4)	77.8 (1.3)	80.6 (0.9)*	75.5 (1.5)	0.0010
REM sleep	94.3 (5.2)	88.5 (6.6)	74.7 (5.3)*	96.4 (6.2)	0.0007
(% TST)	22.7 (1.4)	22.2 (1.3)	19.4 (0.9)	24.7 (2.1)*	0.0021
REM sleep phases	24.3 (1.5)	23.5 (1.9)	23.1 (1.7)	29.6 (2.8)	NS
REM sleep latency	80.0 (11.1)	77.9 (10.3)	90.6 (15.1)	73.0 (5.4)	NS
REM sleep stability	85.3 (1.0)	84.8 (1.8)	84.9 (2.7)	82.1 (2.5)	NS

Significant difference between the baseline session and other sessions, with *$P < 0.05$ and **$P < 0.01$; NS: non-significant difference.

modification in sleep duration was related to the increase in sleep latency as WASO did not vary and arising time was fixed.

Internal sleep structure

Stage 1 did not vary between sessions, except for an increase in recovery compared with both BL and Ramadan sessions (Table 3). However, there was a modification in the structure of NREM sleep. Stage 2 proportion (in percentage of TST) changed across sessions (Fr = 32.99, $P < 0.0001$), with an increase during Ramadan (Table 3). Conversely, the change in SWS duration (Fr = 20.99, $P = 0.0038$) was because of a decrease during Ramadan, although its proportion to TST was not affected (Table 3). These changes were not related to either the number of stage 2 and SWS episodes nor their mean duration.

Although NREM sleep duration did not change significantly, its proportion related to TST varied (Fr = 24.32, $P = 0.0010$), with an increase during Ramadan (Table 3). The REM sleep varied in the opposite direction, in duration (Fr = 25.14, $P = 0.0007$) as well as proportion (Fr = 22.56, $P = 0.0021$). Ramadan induced an overall decrease in both REM sleep duration (BR, −6 min and ER, −20 min) and proportion vs. TST (Table 3), the result of a tendency of a decrease in the duration of REM sleep phases (Fr = 12, $P = 0.09$), as their number (i.e. number of sleep cycles) remained constant.

Table 4 Distribution of the sleep stages related to total stage duration throughout the first four NREM–REM sleep cycles before, during the beginning, the end of Ramadan and after Ramadan. Data from the second night recording are shown

Sleep variable per cycle	*Baseline (BL)*	*Beginning of Ramadan (BR)*	*End of Ramadan (ER)*	*After Ramadan (AR)*	*Night effect*
First cycle					
Wakefulness	15.5 (3.6)	22.7 (5.0)	34.0 (8.5)	21.9 (5.5)	NS
Stage 1	18.2 (3.2)	26.6 (4.0)*	33.2 (4.0)*	22.4 (3.2)	0.0831
Stage 2	15.4 (2.5)	18.0 (2.2)	19.0 (3.5)	17.1 (2.0)	NS
Slow-wave sleep	46.5 (6.1)	45.2 (8.7)	37.3 (6.0)	34.5 (8.5)	NS
REM sleep	13.9 (2.4)	13.3 (2.3)	17.7 (5.0)	11.5 (2.5)	NS
Second cycle					
Wakefulness	16.5 (3.3)	21.7 (5.0)	12.6 (2.5)	17.6 (4.5)	NS
Stage 1	15.6 (4.7)	14.9 (4.2)	16.0 (4.3)	14.4 (4.6)	NS
Stage 2	19.5 (3.0)	19.7 (1.9)	18.0 (2.9)	22.7 (4.2)	NS
Slow-wave sleep	23.6 (6.8)	30.9 (5.0)	41.1 (6.6)	39.4 (8.9)	NS
REM sleep	18.1 (3.1)	29.6 (3.3)	23.1 (5.0)	19.7 (4.4)	NS
Third cycle					
Wakefulness	27.1 (4.7)	19.2 (1.5)**	26.9 (4.6)	35.7 (5.6)	0.0112
Stage 1	22.7 (3.9)	17.3 (2.8)	24.3 (3.5)	29.5 (6.1)	NS
Stage 2	24.8 (2.6)	24.0 (1.4)	27.5 (2.6)	28.6 (3.6)	NS
Slow-wave sleep	17.1 (5.4)	12.5 (3.6)	9.6 (4.1)	18.7 (6.0)	NS
REM sleep	26.8 (4.2)	24.0 (3.2)	38.9 (5.0)	34.0 (6.6)	NS
Fourth cycle					
Wakefulness	19.6 (2.9)	21.1 (2.3)	9.5 (3.2)	13.3 (4.1)	NS
Stage 1	17.9 (3.1)	21.0 (4.4)	11.6 (3.9)	17.1 (4.4)	NS
Stage 2	20.7 (1.8)	24.9 (1.4)	15.6 (4.6)	17.6 (4.0)	NS
Slow-wave sleep	12.2 (4.6)	5.3 (2.7)	10.5 (4.3)	6.0 (3.0)	NS
REM sleep	24.8 (5.0)	21.0 (2.3)	11.5 (4.8)	25.1 (7.2)	NS

Significant difference between the baseline session and other sessions, with *$P < 0.05$ and **$P < 0.01$; NS: no significant difference.

Overnight distribution of sleep parameters

The number of sleep cycles and the mean REM–NREM sleep cycle duration did not change across sessions (Table 3). However, the overnight distribution of stage 1 and wakefulness varied throughout the investigation. In the first cycle, stage 1 ($Fr = 12.58$, $P = 0.0831$) tended to increase during Ramadan (Table 4), and wakefulness decreased during the third cycle ($Fr = 18.18$, $P = 0.0112$). The distribution of the other stages was not affected by the experimental conditions.

Recovery after Ramadan

Two weeks after Ramadan, slight changes compared with BL were still present. The AR recording represented also the last experimental night of the 2-month study. The subjects' sleep latency increased, leading to a decrease in SPT (Table 3).

Rectal temperature

Figure 1 gives 20-min Tre moving averages during each 24-h session. Figure 2 gives the circadian characteristics of Tre during each session. The circadian rhythm of Tre during Ramadan was modified with a 2–3-h delay in both the acrophase (BL: 19.21 ± 1.12 h; AR: 19.63 ± 0.45 h; BR: 22.00 ± 0.21 h; ER: 23.15 ± 1.63 h; $Fr = 14.6$, $P = 0.0022$) and bathyphase (BL: 04.20 ± 0.30 h; AR: 04.08 ± 0.43 h; BR: 07.48 ± 1.40 h; ER: 08.44 ± 3.11 h; $Fr = 13.4$, $P = 0.00385$) and a decrease in the amplitude (BL: 1.10 ± 0.08°C; AR: 1.15 ± 0.14°C; BR: 0.79 ± 0.09°C; ER: 0.92 ± 0.11°C; $Fr = 10.52$, $P = 0.0146$). Using a post hoc Wilcoxon *t*-test, the acrophase and bathyphase values of Tre differed from BL during both Ramadan sessions ($P < 0.02$, Fig. 2). The post-Ramadan acrophase had returned to BL values, differing from both Ramadan sessions. The AR bathyphase differed only with BR ($P < 0.02$; $P = 0.0781$ for AR vs. ER). The amplitude of the Tre rhythm was lower in BR than in BL and AR ($P < 0.02$). On the contrary, the overall 24-h mean Tre (mesor) did not change throughout the investigation ($Fr = 3.40$, $P = 0.3340$).

At lights out (Table 5), Tre was increased during Ramadan compared with both BL and AR ($P = 0.043$). Rectal temperature recorded during sleep varied within sessions ($P = 0.05$), and relatively to sessions ($P = 0.05$), with an increase during Ramadan (post hoc statistics shown in Fig. 1). Compared with BL, Tre was higher between 03.00 and 05.00 h in BR ($P = 0.027$) and between 00.00 and 05.00 h in ER ($P = 0.046$). On the recovery AR session, almost all Tre measurements were lower compared with the other three experimental sessions. At sleep onset, Tre was similar in all sessions (Table 5).

Concerning the effect of meals on Tre during the three successive hours, compared with the same time during

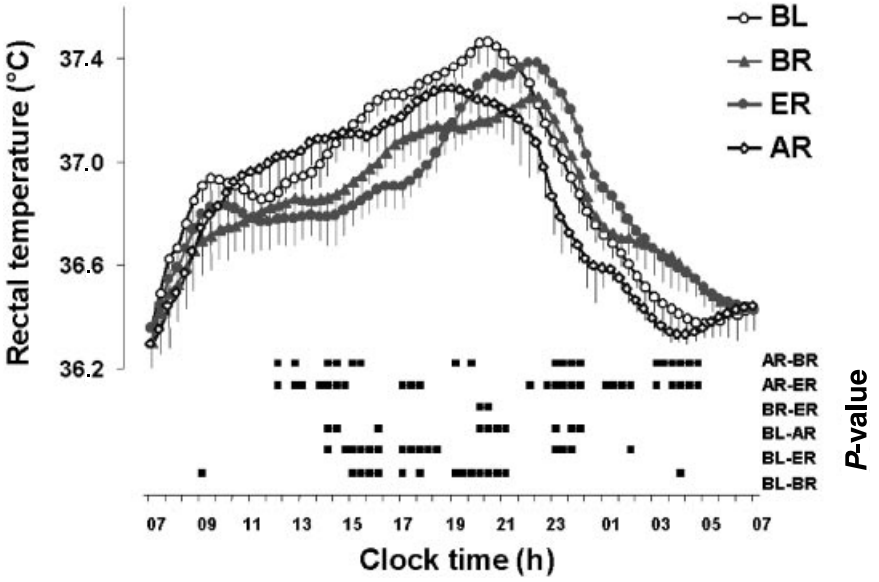

Figure 1. Circadian distribution of rectal temperature (Tre) (°C) during baseline session (BL), beginning (BR) and end of Ramadan (ER) and during the recovery session (AR). Each point represents a 20-min moving average ± SEM. At the bottom of the figure, *P*-values ($P < 0.05$) of the post hoc analysis are shown as dark squares.

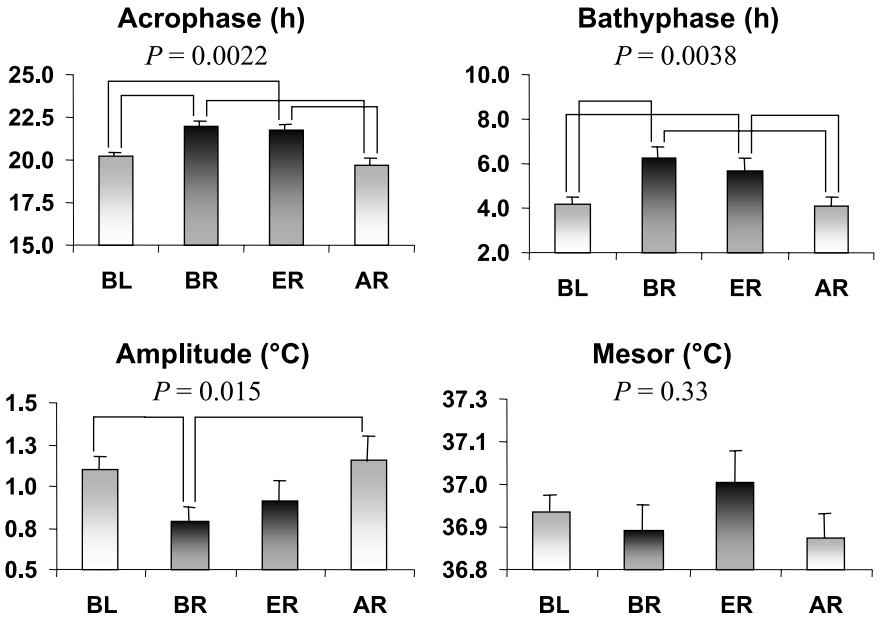

Figure 2. Circadian characteristics of rectal temperature (Tre) (acrophase, bathyphase, amplitude and mesor) during baseline session (BL), beginning (BR) and end of Ramadan (ER) and during the recovery session (AR). The significance of the overall change of the circadian parameters throughout the investigation is indicated by the corresponding *P*-value below the name of the parameter. When significant ($P < 0.05$, shown by liaison lines), the variation was further analysed with between sessions post hoc Wilcoxon paired *t*-tests.

Table 5 Nocturnal rectal temperature (Tre, in °C) characteristics of the six subjects before Ramadan (baseline), at the beginning of Ramadan, at the end of Ramadan and after Ramadan

Tre characteristics	*Baseline*	*Beginning of Ramadan*	*End of Ramadan*	*After Ramadan*
Mean Tre	36.51 (0.05)	36.63 (0.06)	36.65 (0.06)	36.49 (0.02)
Tre at lights off	36.96 (0.06)	37.23 (0.13)	37.40 (0.08)	36.75 (0.13)
Tre at sleep onset	36.81 (0.07)	36.64 (0.04)	36.86 (0.06)	36.49 (0.04)
Tre minimum	36.29 (0.05)	36.37 (0.05)	36.35 (0.06)	36.25 (0.04)

Ramadan (Fig. 3), significant differences between sessions were observed only for Ramadan evening meals ($Fr = 7.682$, $P = 0.0001$) with a more pronounced increase in ER ($P = 0.027$). Concerning the influence of midday meals, Tre increased steadily after the meal for the following 3 h in BL.

DISCUSSION

This is the first study on sleep pattern changes in intermittent fasting due to the religious compliance to Ramadan. Sleep onset was delayed and sleep alleviated.

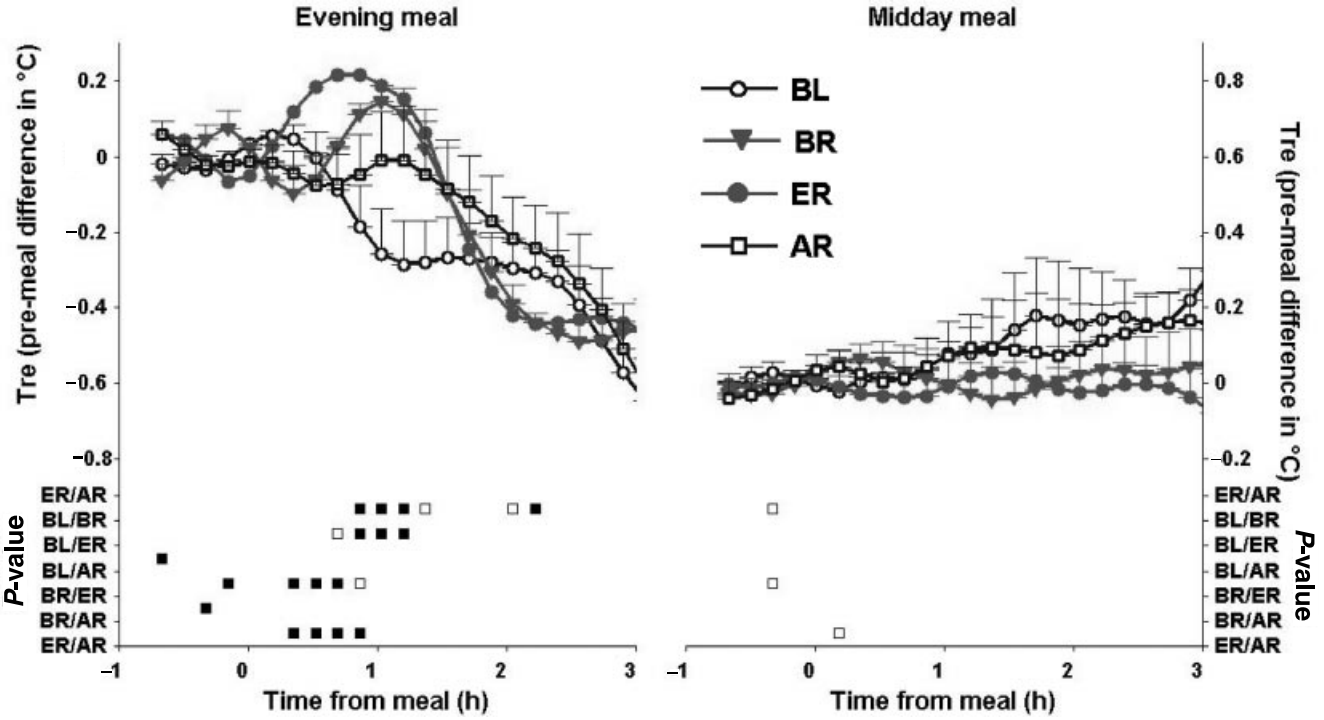

Figure 3. Post-meal changes in rectal temperature (Tre) during baseline session (BL), beginning (BR) and end of Ramadan (ER) and during the recovery session (AR) (see legend of Figure 1). Each point represents the difference with premeal values serving as control. The left panel shows the 10-min averages of the difference in Tre between post-meal values (throughout the first post-meal 3 h) and values 1 h before dinner (at 21.00 h) in all experimental conditions. The right panel shows Tre differences between values collected 1 h before lunch and values of the 3 h following lunch in BL and AR conditions, and at the same time in fasting conditions (BR and ER). At the bottom of the graph, significant *P*-values ($P < 0.05$, open square and $P < 0.01$, dark square) of the post hoc analysis are shown.

The delay in sleep onset was associated with an increase in nocturnal body temperature. This may be related to the late occurrence of meals. The increase in sleep latency during Ramadan is consistent with the findings that evening meals improve alertness at bed-time (Smith *et al.* 1994) and that sleep onset is delayed when body temperature is increased by nutritional factors such as spicy nocturnal meals (Edwards *et al.* 1992). Other evening thermogenetic environmental factors, such as a 3-h exposure to bright light prior to sleep (Dijk *et al.* 1991) and nocturnal exercise (Mizuno *et al.* 1998), also increase sleep latency. Moreover, sleep onset is believed to be triggered by a rapid decrease in core temperature, the maximum rate of decline being observed 60 min before sleep onset (Murphy and Campbell 1997). Our data show that sleep onset occurred at the same Tre in all experimental sessions, suggesting that sleep onset depends on the thermogenetic effect of ingesting the daily amount of food in a restricted period of time displaced in the evening.

Alternatively, the increased sleep latency could be explained by more specifically sleep-related mechanisms. Feeding has been shown to induce an increase in the extracellular concentration of histamine in the hypothalamus (Itoh *et al.* 1991; Oishi *et al.* 1987). In turn, the hypothalamic administration of histamine is able to increase wakefulness (Lin *et al.* 1994).

The alleviation of sleep regarded SWS and REM sleep exclusively. The decrease in SWS during Ramadan is consistent with previous findings showing that high carbohydrate bedtime meals decrease SWS (Porter and Horne 1981). However the decrease in SWS duration could be because of the decreased TST duration, as the proportion of SWS was not changed in the present study. It could also be related to stress-induced processes which are likely to be produced by daytime fasting and drinking restriction, at a time at which people need to be active and work. In fact, cortisol secretion rhythmicity changes during Ramadan, peaking both in the advanced morning and evening (Sliman *et al.* 1993) and at midnight (Al-Hadramy *et al.* 1988), when the acrophase of the cortisol secretory rhythm occurs normally in the early morning hours (Follenius *et al.* 1992). A stress reaction during the daytime is likely to provoke a diachronic decrease in SWS (Buguet *et al.* 1998).

The REM sleep was decreased during Ramadan both in duration and in proportion vs. TST. The high nocturnal body temperature observed during Ramadan may also intervene in this modification. The REM sleep duration is inversely proportional to core temperature (Dijk and Czeisler 1995; Jouvet *et al.* 1988; Krueger and Takahashi 1997) and nocturnal hyperthermia decreases REM sleep (Haskell *et al.* 1981b; Libert 1988). In normal conditions, REM sleep episodes are longer towards the end of the night, when body temperature is

minimal (Jouvet *et al*. 1988). Other factors such as cortisol and insulin may explain the decrease in REM sleep (Born *et al*. 1989; Sangiah and Caldwell 1988), as an increase in nocturnal cortisol (Al-Hadramy *et al*. 1988; Sliman *et al*. 1993) and insulin (Elati *et al*. 1995; Iraki *et al*. 1997) has been observed during Ramadan and after meals intake (Hansen *et al*. 1997). The REM sleep is also altered in stressful situations at night (Buguet *et al*. 1998). Furthermore, REM sleep always occurs concomitantly with a diminution in adrenocortical activity (Follenius *et al*. 1992).

During Ramadan, the circadian rhythm of Tre was disturbed, with a delay in the occurrence of the acrophase and bathyphase, and with a reduction in amplitude. This suggested that the circadian system could be affected by the exclusive evening meals. The influence of the thermogenetic effect of meals on the time course of Tre is supported by the absence of any early afternoon increase in Tre during Ramadan daytime fasting, contrarily to that observed in BL. The circadian increase in Tre occurred lately in the afternoon. Such an alteration in the expression of the circadian system has also been evoked by the shift in the chronotype towards the evening type in students observing Ramadan (Taoudi Benchekroun *et al*. 1999). The 24-h mean of Tre was not changed by Ramadan. This was expected, because daily meal composition was maintained constant in both fasting and non-fasting conditions. This observation is consistent with the finding that the average daily metabolic rate and energy intake during Ramadan remain comparable with those of non-fasting days (Elati *et al*. 1995).

The nocturnal decrease in metabolic rate is a result of both sleep and circadian oscillation, the contribution of the two components being equivalent (Fraser *et al*. 1989). At the beginning of the night, only the sleep-related decrease in body temperature was observed. The increased minimum of temperature may also be related to the decreased SWS. Oxygen consumption is at its lowest during SWS (Haskell *et al*. 1981b; Shapiro *et al*. 1984). Also, in SWS, body heat content diminishes. This is achieved by an increase in evaporative heat loss concomitant with SWS (Haskell *et al*. 1981a; Hénane *et al*. 1977; Ogawa *et al*. 1967) and by a decrease in core temperature, as demonstrated in a patient with an aplasia of sweat glands (Buguet *et al*. 1990). The lowering in metabolic brain activity during SWS would allow the occurrence of REM sleep, a state of high-energy consumption (Jouvet 1994), as REM sleep occurs when the brain is cool (Jouvet *et al*. 1988).

Although the subjects were asked to keep similar energy intakes during the month of Ramadan, they lost weight and body fat, but body weight and composition were quickly restored after 15 days of usual eating habits. The change in body weight and composition suggests that the daily energy intake may have been decreased. However, Ramadan diet does not compare with a 4-week restricted energy diet, which lowers body temperature (Karklin *et al*. 1994), although the effects on sleep were similar than during Ramadan, with an increase in sleep latency and a decrease in SWS. The opposite thermoregulatory effect suggests that the changes in body temperature and sleep during Ramadan are not related to the decreased energy intake but rather to the inversion of drinking and meals schedule.

In conclusion, the intermittent fasting of Ramadan delays sleep onset, impairs sleep structure, especially REM sleep, in relation to a delay in the acrophase and bathyphase of Tre and concomitant alterations in the metabolic and endocrine status of the individual.

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