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Electrodermal activity during total sleep deprivation and its relationship with other activation and performance measures

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SUMMARY The present study analyses the variations of the skin resistance level (SRL) during 48 h of total sleep deprivation (TSD) and its relationship to body temperature, self-informed sleepiness in the Stanford Sleepiness Scale (SSS), and reaction time (RT). All of the variables were evaluated every 2 h except for the SSS, which was evaluated every hour. A total of 30 healthy subjects (15 men and 15 women) from 18 to 24 years old participated in the experiment. Analyses of variance (ANOVAS) with TSD days and time-of-day as factors showed a substantial increase of SRL, SSS, and RT, and a decrease in body temperature marked by strong circadian oscillations. The interaction between day by time-of-day was only significant for RT. Furthermore, Pearson's correlations showed that the increase of SRL is associated to the decrease in temperature (mean $r = -0.511$), the increase of SSS (mean $r = 0.509$), and the deterioration of RT (mean $r = 0.425$). The results support previous TSD reports and demonstrate the sensitivity of SRL to TSD. The non-invasive character of SRL, its simplicity, and its relationships with other activation parameters, widely validated by previous literature, convert SRL into an interesting and useful measure in this field.

KEYWORDS body temperature, correlations, electrodermal resistance, reaction time, sleepiness, total sleep deprivation

INTRODUCTION

Disturbances in the sleep–wake rhythm and/or sleep deprivation are frequent events in modern life. Many subjects can be chronically sleep deprived as a result of current life style expectations as occurs in the case of night shift workers, on-call doctors, truck drivers and airplane pilots, situations of prolonged athletic competition, insomnia or sleep apnea (Bonnet and Arand 1995). Such deteriorations in activation are frequently the cause of industrial or automobile accidents found in some sectors, and are simply added to the list of 'human errors' (Kuhn 2001; Philip *et al.* 1999).

Total sleep deprivation (TSD) has been shown to negatively impact a wide range of cognitive, behavioural, physiological, and emotional measures (Dinges 1992; Horne 1988, 1992; Pilcher and Huffcutt 1996). One of the most prominent and consistent effects of sleep loss is deterioration in vigilance and activation which has been reported in a wide variety of objective and subjective measures. The basal level of electrodermal activity (EDA) is an accepted vigilance index used in diverse psychophysiological fields (Freixa i Baqué 1990). It is quite surprising that this measure has been ignored in previous sleep deprivation studies.

During normal wakefulness, as a subject relaxes and nears a presomnolence state, his/her skin resistance level (SRL) progressively increases. In this situation an inverse relation between the vigilance level and the SRL is found (Freixa i Baqué 1990). The circadian variations in body temperature have been reported to positively correlate with skin conductance levels (SCL) and to negatively correlate with SRL (Buela-Casal 1990). In studies carried out during sleep, it has been found that different EDA recording procedures do not provide compatible information. Throughout the slow sleep phases an increase in SCL recordings is found until it reaches a maximum

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value in stage 4, while a strong decrease in SCL was experienced during rapid eye movement (REM) sleep (Freixa i Baqué 1990). However, in SRL studies (SRL is the inverse of SCL) non-rapid eye movement (NREM) sleep is always associated with an increased SRL, with the exception of transient changes associated to the presence of K-complexes in stage 2 sleep (Noll *et al*. 1994).

In relation to the sleep deprivation effect on EDA, there exist several earlier studies, all revised by Horne (1978), that analysed the changes in SCL or skin conductance response (SCR) at different periods of TSD. Of the investigations collated by Horne (1978), SCL decreased in four studies (24–205 h of TSD), and increased in another four studies (24–264 h). SCR decreased in two investigations (123 and 264 h of TSD), and increased in another study of 54 h of TSD. There are also two investigations that did not find any modifications in these parameters. As Horne (1978) suggested in his classical review these results are quite equivocal. In fact, many of these studies suffered from methodological limitations. For example, they were single case studies, they used measurements taken only once or twice during the whole TSD period, or they did not include statistical analysis.

From the most recent investigations, to our knowledge, there exist no studies about SRL and TSD. Dikaya (1990) investigates the impact of the Shultz relaxation technique on performance during 72 h of TSD. Although electroencephalogram (EEG), heart rate and electrodermal activity were recorded, the author does not comment on the EDA. McCarthy and Waters (1997) analyse the effect of 36 h of TSD on skin conductance-orienting response (OR), a measure of attentional shift or capture. The OR latency was significantly delayed following TSD, the OR amplitude decreased, and the habituation of OR was quicker. All of this indicates slowness in attentional change to new stimuli, decreasing allocation to stimuli, and a more rapid loss of attention to repeated stimuli, respectively.

The present study analyses the variations in skin resistance level (SRL) during 48 h of TSD. Body temperature and other important activation and vigilance variables in the TSD studies, such as self-informed sleepiness and simple reaction time, are also recorded in order to determine the relationship between SRL and each one of these variables.

METHODS

Subjects

Volunteers were recruited from an announcement posted in the School of Psychology, at the University of Granada. A total of 30 healthy subjects (15 men and 15 women) from 18 to 24 years old (mean age = 20.06 and standard deviation (SD) = 4.38) participated in the study.

The final sample was selected by means of a questionnaire elaborated for this purpose. The questionnaire explored such areas as physical and psychological health, consumption of tobacco, medication, alcohol and other drugs, ingestion of coffee, tea and other stimulants, menstrual cycle regularity in the case of women, regularity in sleep schedules, and the possible existence of sleep disorders. The Horne and Östberg Morningness–Eveningness Scale for determining each subject's circadian type was administered along with the selection questionnaire. Subjects who obtained scores superior to 21 ('clearly morning-type') or scores inferior to 8 ('clearly evening-type') were not admitted. The Beck Depression Inventory (BDI), the State-Trait Anxiety Inventory: Trait Scale (STAI), and the Eysenck Personality Questionnaire (EPQ-A) were also administered. The basis for exclusion was a score superior to 9 on the BDI or a direct score on the STAI and in the neuroticism and psychoticism dimensions of the EPQ-A over the centil 70. These last exclusions were established to assure that the subjects did not experiment extreme variations in mood, or exhibit personality characteristics that could indicate the presence of a possible behaviour disturbance, or an uncommon response to the TSD.

All of the selected participants presented good health and did not consume any type of medication. Each subject had a regular sleep pattern of 7–9 h, with a common bedtime hour between 11:30 p.m and 2:30 a.m and a wake time hour between 7:30 a.m and 10:30 a.m. Those subjects who did not consume more than three cups of coffee per day were admitted. Those subjects who presented high tobacco consumption (more than 15 cigarettes per day) or low tobacco consumption (five cigarettes or less per day) were excluded in an attempt to even out the effects of the nicotine by impeding an excessive or minimal nicotine habituation. The final sample included 10 smokers (five women and five men). Finally, the women were given a date that did not coincide with their premenstrual, menstrual, or ovulation periods. That is, 1 week between the last day of their menstruation and the following 7–10 days (preovulation phase).

Experimental protocol

All subjects received precise instructions concerning the week prior to the investigation, indicating them to sleep according to their normal schedules. The day before the experiment the subjects were familiarized with the experimental procedure. In the first day of the study, the subjects (always a mixed group of 5–7) arrived at the laboratory at 8:30 a.m., were reminded of the participation rules of the experiment, and signed a written consent binding them to such norms. Subjects were free to withdraw from the experiment at any time. They were not allowed to consume coffee, tea or cola refreshments, alcohol, nor any medication. They could not shower (they could freshen up and change clothing) or do any physical exercise that could facilitate activation. Smoking was only allowed immediately after finishing each data recording (data recordings were performed every 2 h) to prevent a nicotine effect on psychophysiological variables.

The investigation began at 9:00 a.m. on Tuesday morning and continued until 9:00 p.m. Thursday evening, at which time subjects were de-briefed and accompanied home. Throughout the 60 h of TSD the subjects remained in a large room that

could only be abandoned in order to go to an adjacent laboratory where the psychophysiological recordings were performed every 2 h. A total of 30 recordings were completed, always at the same time of day: 9:00 a.m., 11:00 a.m., 1:00 p.m., 3:00 p.m., etc. Sleepiness was evaluated every hour (at 9:00 a.m., 10:00 a.m., 11:00 a.m., etc.). The room temperature in both areas was maintained at a thermoneutral level (22–25 °C) by means of an air conditioner with thermostat, the lighting was constant, and there was isolation from any noise. Each recording session in the laboratory consisted of a reaction time task, which lasted about 3 min in length. Following the reaction time (RT) task, a slightly longer vigilance task was initiated (5–7 min in length) in which the SRL (the average value of the second minute) and the body temperature were recorded. The electrodes for the SRL recording were placed on the middle phalanges of the index and middle fingers of the left hand, previously washed with soap, and the thermistor was placed in the left axilla, after cleaning the registered area with alcohol (all of the subjects were right handed).

When subject 1 completed a data recording session in the laboratory and returned to the room, subject 2 arrived, and this process was repeated with all subjects until the entire group had completed the recording process. Once all of the subjects returned to the room, several tests of cognitive performance and mood state (which will be discussed elsewhere) were administered. The remaining free time between data recordings and tests could be spent freely on reading, listening to music, watching television, playing board games, etc. The subjects were under the constant supervision of two laboratory technician to prevent any subject from sleeping. Technicians worked 8-h shifts. Breakfast was provided at 8:30 a.m and the main meal at 2:30 p.m., with the same menu for all subjects. Dinner was provided at 8:30 p.m and a snack was available at 2:30 a.m., which consisted of sandwiches, potato chips, etc.

Apparatus and test

A BIOCIBER, model DERMBACK CY-15, polygraph module for EDA provided the measure for the SRL. This instrument reads four data per second (resolution 0.5 kΩ) and averages a total of 60 s (240 SRL values in 1 min), visually providing this information in a digitalized display (that the subject was prevented from viewing) or in a computerized recording by means of a Windows 95 software program. San-ei conductive gel (ClNa 0.29 g/100 mL) facilitated contact with Ag–AgCl electrodes (1 cm^2), fixed to the subjects by velcro adhesive bands. A KEITO, model KT-70, digital thermometer (range 32 °C–43.9 °C, resolution 0.1 °C) recorded the axillary body temperature.

The Stanford Sleepiness Scale (SSS) (Hoddes *et al.* 1973) assessed the activation-sleepiness state with seven items referring to different subjective somnolence descriptions. It consists of a self-applied scale in which the subject must mark the statement that best describes his/her activation state at that moment. The scores range from 0, indicating the lowest somnolence, to 7 indicating the highest sleepiness level.

A computer task (Buela-Casal 1990) was employed for measuring reaction time. The subject was instructed to press the 'enter' key as quickly as possible after the presentation of a stimulus, a white letter 'A' (4 mm in height) in the centre of the screen (12 inches) on a black background. The subject was placed in front of the computer monitor at a distance of about 50 cm, with his/her eyes at the same horizontal level as the stimulus. The stimulus was presented 50 times in each recording session, with an intertrial interval, which ranged randomly from 0.5 to 3 s. A filter was incorporated into the software program to detect and eliminate any anticipatory responses (those responses produced before the stimulus is presented and up to 130 ms. after its presentation). In this case, the trial was considered null and void and the presentation of that stimulus was repeated. The computer program also provided a self-correction mechanism, which for each trial subtracted the time elapsed for transmitting the command from the keyboard to the central processing unit (CPU).

Data analysis

The skewness and kurtosis indexes of the data distribution, as well as the significance level of the W statistic, showed that the normality assumption was satisfactory, so that parametric analysis techniques were employed. Two-way analyses of variance (ANOVA) for repeated measures were performed to assess the effects of sleep deprivation and time of day as well as their interaction on each parameter. Within-subject ANOVAS had days of TSD (day 1 and day 2) as one factor, and time of day (12 times for SRL, temperature and RT, and 24 times for SSS) as the other factor, with significance levels corrected for sphericity by Greenhouse–Geisser epsilon. The expectation was for decreased activation and performance over the 2 days of sleep loss and for changes over the hours of the day within each day. When appropriate, *posthoc* paired tests were used to compare outcomes at discrete time points.

Polynomial contrasts for linear, quadratic, and cubic trends were further performed on data for which there were significant variation across the TSD days. This analysis was applied to the 24 measures of the 2 days of TSD (48 for the SSS), and it yielded information about the nature of the relationship between TSD and each parameter. The data collected during TSD are subjected to inherent rhythmic oscillations along with effects related to the sleep loss *per se*. It has been established that a linear or monotonic function would be related to the cumulative effect of TSD, while the circadian and/or ultradian rhythmicity would be specified in other types of functions (Babkoff *et al.* 1991a). In addition, the percentage of variance accounted for by each significant trend was estimated.

It must be noted that when variables are measured repeatedly throughout the first and the second days, but only during the first half of the third day for a total of 60 h of TSD, there is no equivalent control for the results of day 3 with respect to

time of day. For this reason only results from day 1 and day 2 (48 h of TSD) were considered for all these analyses described above.

Finally, the relationship between the variables showing significant modifications during the TSD is obtained from a partial intercorrelation matrix between such measures. Each Pearson's correlation coefficient was based on 30 pairs of scores for each one of the 30 subjects during the 3 days of TSD (for this analysis it was not necessary to disregard the last 12 h). The correlations were performed within each subject separately, and then we assessed the average correlation for each pair of variables determining the 95% confidence limits around the *r*.

RESULTS

Electrodermal resistance

The results of the ANOVA for repeated measures indicated a significant main effect of the day ($F_{1,29} = 55.96$, $P < 0.001$), and of the time of day ($F_{11,319} = 15.03$, $P < 0.001$), but no interaction between these factors. A summary of these results with Greenhouse–Geisser corrections can be seen in Table 1. The mean values of SRL increased significantly from day 1 (190.74 ± 139.93 kΩ) to day 2 (298.79 ± 209.81 kΩ) indicating decreased activation and reduced alertness after sleep deprivation. This large overall increase of SRL was also marked by circadian oscillations (see Fig. 1). Skin resistance level changed within both days from the trough or minimum value at around 7:00–9:00 p.m. to the peak value between 5:00 and 7:00 a.m.

Table 2 shows a summary of the trend analysis for the SRL in relation to the 48 h of TSD. The trends found to be significant are the linear ($F_{1,29} = 74.05$, $P < 0.001$), and the cubic ($F_{1,29} = 13.01$, $P < 0.001$). The polynomial trend that explains a greater percentage of the variance (62.74%) in SRL during the TSD is an ascending linear function related to the accumulated sleep loss effect (see Fig. 1). Trends of greater order must be understood as approximating to the rhythmicity of the data (see Table 2).

Body temperature

The ANOVA for repeated measures revealed a significant main effect of day ($F_{1,29} = 27.08$, $P < 0.001$), and of time of day ($F_{11,319} = 28.55$, $P < 0.001$). There was no significant day by time-of-day interaction. As in the case of SRL, the lack of interaction points toward orthogonal influences of sleep loss and diurnal oscillations on body temperature. The average temperature showed a small downward trend from day 1 (36.44 ± 0.40 °C) to day 2 (36.30 ± 0.37 °C) of TSD, marked by strong rhythmic oscillations (see Fig. 2). Body temperature changed within both days from the peak between 7:00 and 9:00 p.m. to the trough during the early morning hours at around 5:00–7:00 a.m.

The trend analysis (see Table 2) was significant for the linear ($F_{1,29} = 41.24$, $P < 0.001$), quadratic ($F_{1,29} = 8.59$, $P < 0.01$), and cubic trends ($F_{1,29} = 17.23$, $P < 0.001$). The amount of variance accounted for by the trends associated to rhythmic aspects (7.81% the quadratic and 12.26% the cubic, a total of 20.07%) was very similar to the variance accounted for by the linear trend (25.86%). This finding provides quantitative support for the visual impression of strong rhythmic oscillations with a slight overall decrease in temperature during TSD (see Fig. 2).

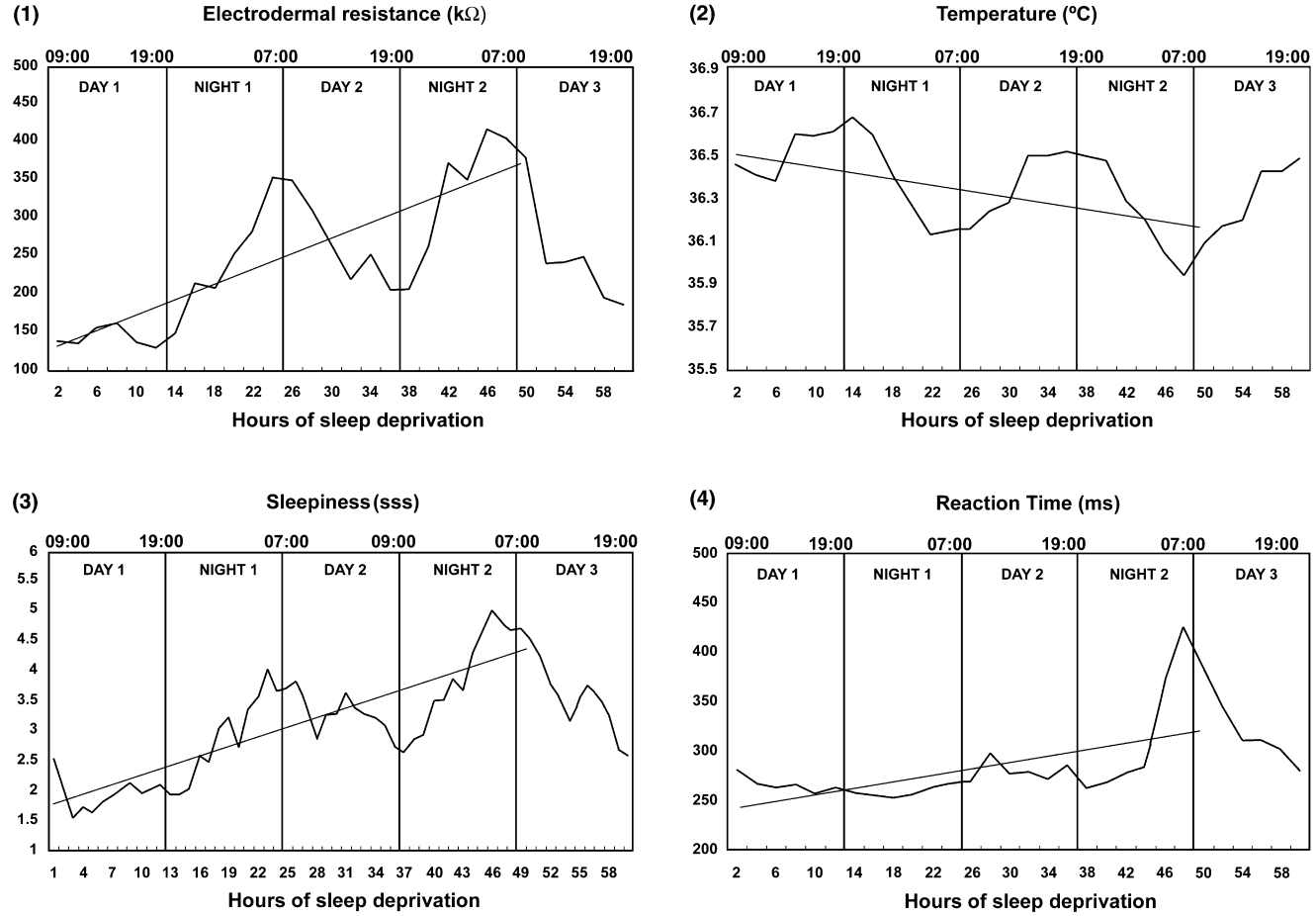
Sleepiness

The results of the ANOVA indicated that for sleepiness there were significant differences across the 2 days of TSD ($F_{1,29} = 42.18$, $P < 0.001$), and across hours of each day ($F_{23,667} = 21.01$, $P < 0.001$). There was no significant interaction between these factors. The mean values of self-reported sleepiness increased from the first (2.40 ± 1.20) to the second day (3.57 ± 1.43) of sleep deprivation. In addition, the lowest sleepiness ratings occurred between 11:00 a.m. to 1:00 p.m., whereas the highest ratings occurred between 6:00 and 8:00 a.m. (see Fig. 3).

The trend analysis (see Table 2) was significant for the linear ($F_{1,29} = 72.44$, $P < 0.001$), and cubic trends ($F_{1,29} = 11.75$,

Variable	*d.f.*	*d.f.*^{c}	*F* & *F*^{c}	*P*	*P*^{c}
SRL					
Day	(1, 29)	(1, 29)	55.96	0.001	0.001
Time of day	(11 319)	(5.10, 148.14)	15.03	0.001	0.001
Day *x* time of day	(11 319)	(5.28, 153.32)	1.71	NS	NS
TEMP					
Day	(1, 29)	(1,29)	27.08	0.001	0.001
Time of day	(11, 319)	(5.09, 145.26)	28.55	0.001	0.001
Day *x* time of day	(11, 319)	(6.84, 198.38)	1.33	NS	NS
SSS					
Day	(1,29)	(1,29)	42.18	0.001	0.001
Time of day	(23, 667)	(8.44, 244.86)	21.01	0.001	0.001
Day *x* time of day	(23, 667)	(8.76, 254.08)	1.47	0.072	NS
RT					
Day	(1, 29)	(1,29)	29.30	0.001	0.001
Time of day	(11, 319)	(3.08, 87.24)	12.78	0.001	0.001
Day *x* time of day	(11, 319)	(2.70, 78.41)	4.78	0.001	0.01

Table 1 Summary of two-factor analysis of variance for repeated measures with assumed sphericity and corrected by Greenhouse–Geisser epsilon. The table shows the degrees of freedom, the *F*-ratios, and the *P*-values with correction (d.f.^{c}, F^c, P^c) and without correction for skin resistance level (SRL), body temperature (TEMP), Stanford Sleepiness Scale (SSS), and reaction time (RT)



Figures 1, 2, 3 and 4. The mean scores of skin resistance level (SRL), body temperature (TEMP), Stanford Sleepiness Scale (SSS), and reaction time (RT) are plotted on the ordinate as a function of 60 h of total sleep deprivation (TSD) on the abscissa. At the top are given some indications of the hours of the day that correspond to the accumulated hours of TSD. The graphs also show the lineal trend adjustment for each variable. Please note that the ANOVAS and the trend analysis were only conducted on 48 h of TSD (two complete circadian cycles).

Table 2 Summary of the trend analysis for skin resistance level (SRL), body temperature (TEMP), Stanford Sleepiness Scale (SSS), and reaction time (RT) in relation to 48 h of TSD. The determination coefficients (r^2) of polynomial regression express the variability accounted for by each one of the significant trends

	Trends	r^2 *(%)*
SRL	Linear**	62.74
	Cubic**	9.88
TEMP	Linear**	25.86
	Quadratic*	7.81
	Cubic**	12.26
SSS	Linear**	69.60
	Cubic**	8.03
RT	Linear**	31.80
	Quadratic**	27.67
	Cubic**	9.01

*$P < 0.01$; **$P < 0.001$.

$P < 0.001$). An ascending linear trend attributable to the accumulated wakefulness accounted for a 69.60% of the variance of SSS during the TSD. The cubic trend approximated to the rhythmicity of the data, and accounted for a 8.03% of the SSS variability during the TSD.

Reaction time

The result of the ANOVA showed a significant main effect for day ($F_{1,29} = 29.30$, $P < 0.001$), with RT higher on day 2 (298.07 ± 119.83 ms) than on day 1 (262.34 ± 23.98 ms), and for time of day ($F_{11,319} = 12.78$, $P < 0.001$). In addition, the interaction between day and time-of-day was significant ($F_{11,319} = 4.78$, $P < 0.001$). This implies that the change of RT over the hours of the day differs from day 1 to day 2. The interaction should be reflected in the exaggerated peak of RT on the second day (at 7.00 a.m), as is clearly visible in the curve (see Fig. 4). *Post-hoc* paired test confirmed the visual impression that the peak-to-trough amplitudes on day 1 (mean = 52.08 ± 37.84) are significantly lower than the amplitudes on day 2 (mean = 226.65 ± 185.24) ($t = -4.76$, $P < 0.001$).

The trend analysis, applied to the 48 h of TSD, indicated a significant adjustment to the linear ($F_{1,29} = 24.75$, $P < 0.001$), quadratic ($F_{1,29} = 24.79$, $P < 0.001$), and cubic trends

($F_{1,29} = 10.13$, $P < 0.001$). This highlights the percentage of variance accounted for by an ascending linear trend (31.8%) attributable to the accumulated sleep loss effect, and by a quadratic trend (27.67%) associated to the large peak on day 2 of TSD (see Table 2).

Relationships between variables

Table 3 shows the mean partial correlations computed between the different variables under examination during the entire period of TSD (60 h). Skin resistance level was significantly correlated to the changes in temperature during the TSD (mean $r = -0.511$). Increments in SRL are associated with decreases in body temperature. Furthermore, SRL significantly correlated with both SSS (mean $r = 0.509$) and RT (mean $r = 0.425$). That is, when the SRL values are high, indicating decreased activation, the SSS ratings are also high, and the RTs increase showing an impairment of performance in this task.

Significant correlations were obtained between temperature and SSS (mean $r = -0.46$), and between temperature and RT (mean $r = -0.367$). This suggests that the increase in body temperature is associated with the decrease in SSS ratings, and with the improvement of performance in the RT task. Finally, the SSS values significantly correlated with the RT scores (mean $r = 0.584$). Higher sleepiness self-evaluations are associated with higher reaction times, and therefore worse performance on such task.

DISCUSSION

A TSD of 48 h produces a substantial increase of SRL, SSS and RT, and a decrease in body temperature, marked by strong rhythmic oscillations. Accumulating hours of wakefulness were reflected in all cases by the significant main effect of the day factor in the ANOVA, and by the significant adjustment to the linear function in the trend analysis. The SRL, SSS and RT were adjusted to an ascending linear trend, which accounted for 62.74, 69.60 and 31.08% of its variance, respectively. Temperature adjusted to a descending linear trend, which explained 25.86% of its variance during the TSD.

The large increase in SRL suggest a decrease in the sympathetic activation of the autonomous nervous system. The average of SRL increased from 190.74 kΩ on day 1 to 298.79 kΩ on day 2, and the highest SRL values were reached on the second night (e.g. 413.80 kΩ at 5.00 a.m). Several individual scores were even higher during this second night, reaching more than 1000 kΩ. These results extend the findings of studies on normal waking without TSD, which establishes that when a subject experiences sleepiness their SRL values increase (Freixa i Baqué *et al.* 1990). To the best of our knowledge there are no investigations about TSD, which can be directly related to our results. The only current TSD research, where EDA is recorded, focuses on parameters of conductance (McCarthy and Waters 1997). The same thing happens with earlier studies that also suffer from methodological problems (for review, see Horne 1978).

Table 3 Mean correlations between the different analysed variables and 95% confidence boundaries around each correlation

	SRL	*TEMP*	*SSS*	*RT*
SRL				
TEMP	−0.511 (±0.153)*			
SSS	0.509 (±0.128)*	−0.462 (±0.178)*		
RT	0.425 (±0.210)*	−0.367 (±0.113)*	0.584 (±0.235)*	

*$P < 0.05$.

The slight decrease found in body temperature is consistent with the majority of previous reports (Babkoff *et al.* 1991a; Horne and Pettitt 1984; Landis *et al.* 1998). The temperature decreased from 36.44 °C on day 1 to 36.30 °C on day 2 (a total of 14 °C). The similarity between our results and the data of Babkoff *et al.* (1991a) is remarkable. These authors, in a study of 72 h of TSD, with oral temperature measurements every 2 h, found that temperature decreased from 36.39 °C on day 1 to 36.25°C on day 3. Furthermore, Babkoff *et al.* (1991a) observed that temperature adjusted to a descending linear trend, which accounted for 19% of their variance throughout TSD. Whereas in our case the same trend explains 25.86% of the temperature changes.

We found, from the subjects' anecdotal reports, that they felt cold in the early morning hours, although the room temperature was not modified, as has been reported previously (Horne and Pettitt 1984). It has been suggested that these changes could reflect an affectation of thermoregulation (Landis *et al.* 1998). In fact, excessive body heat loss is one of the signs of lethal outcome in uninterrupted studies of TSD with rats (Rechtschaffen and Bergmann 1995). However, in human beings it seems that the clinical significance of temperature decrease is too small to suggest this kind of alterations. It is probably more related to a generalized decrease in activation and perhaps of body metabolism (Horne 1988, 1992).

On the other hand, as expected, the marked increase in SSS and in RT confirms, as with the previous variables, that the deterioration in activation and vigilance are found amongst the most prominent effects of TSD. Sleepiness increased from 2.40 on day 1 to 3.57 on day 2, and the highest SSS ratings were reached on the second night (e.g. 5.13 at 5:00 a.m). These results are consistent with other investigations that include the SSS during periods of 24–72 h of TSD (Babkoff *et al.* 1991b; Patat *et al.* 2000; Webb and Levy 1982; Wilhelm *et al.* 1998).

RT increased from 262.34 ms on day 1 to 298.07 ms on day 2. The worst RTs were produced on the second night (e.g. 427.97 ms at 7:00 a.m). At those times some subjects presented RTs higher than 1000 ms, which has been called lapsus (Dinges 1992). It has been demonstrated that deterioration in RT is independent from lapsus although both processes combined produce the greatest deterioration (Dinges and Powell 1988). This increase in RT coincides well with other

investigations between 24 and 60 h of TSD (Gillberg *et al.* 1994; Lorenzo *et al.* 1995). However, in our study RT remained almost unaffected throughout the first 24 h of TSD, and only deteriorated during the second day, while in general, activation measures show changes from the first night of TSD (which also occurred in SRL, temperature and SSS). It is important to note that our RT task only lasted 3 min and the subjects received feedback about their performance (the result of this task was visible on the computer monitor). These factors may explain that deterioration was minimized during the first 24 h of TSD (Dinges 1992; Jaskowski and Wlodarczyk 1997).

It must be noted that the changes found throughout TSD are marked by rhythmic oscillations. The significant main effect of the time-of-day factor in the ANOVA of all the variables, as well as the significant adjustment to quadratic and/or cubic trends, reflects the rhythmicity of the data. While time-of-day effects were significant in SRL, temperature and SSS, there was no evidence of an interaction between TSD days and time-of-day for these variables. That is, the rhythmic oscillations do not change over the 2 days of TSD. SRL, temperature and SSS changed within both days from the minimum of activation between 5:00 and 7:00 a.m to the maximum between 7:00 and 9:00 p.m. (at 11:00–1:00 p.m. for SSS), which coincides with previous data (Mitler and Miller 1996). Other reports support the finding that body temperature is an example of an orthogonal relationship between sleep loss and diurnal oscillations (Babkoff *et al.* 1991a, b; Horne 1988). In contrast, the interaction of TSD days and rhythmic oscillations for RT was significant. The change of RT over the hours of the day appears to enhance from the first to the second day of TSD. Several studies have indicated that TSD alters the characteristics of performance rhythms, reflecting the change a stronger rhythmicity during long-term sleep loss as the amplitude of the rhythm is enhanced (Adan and Sánchez-Turet 2000; Babkoff *et al.* 1991a, b). Perhaps physiological functions show orthogonal relationships between monotonic and rhythmic factors during TSD, whereas performance functions show interactive relationships. However, further studies are needed to test this hypothesis.

Finally, we found considerable parallelisms between the objective and subjective activation indicators. The increase in SRL is associated with a decrease in body temperature (mean $r = -0.511$), an increase of subjective sleepiness (mean $r = 0.509$), and an impairment of performance in the RT task (mean $r = 0.425$). There was no similar data available with which to compare these results. Although it appear to be coherent with reports that during normal wakefulness the SRL is negatively correlated to peripheral temperature (Buela-Casal 1990). Similarly, changes in temperature are negatively correlated to the SSS (mean $r = -0.462$) and with RT scores (mean $r = -0.367$). This finding is consistent with other studies with the SSS (Leproult *et al.* 1997; Moses *et al.* 1978). For example, in the 40 h TSD study by Moses *et al.* (1978) the lowest points of the temperature curve correlated with the maximum SSS ratings ($r = -0.25$). The correlation between SSS and RT (mean $r = 0.584$) is also similar to previous results (Gillberg *et al.* 1994). In general, the subjective sleepiness ratings relate well to performance when vigilance is seriously deteriorated (Herscovitch and Broughton 1981; Johnson *et al.* 1991). These questions are important because in many daily sleepiness situations, the only indication that a subject has about their state of activation is their subjective judgement.

In short, 48 h of TSD produced a marked deterioration in activation and vigilance measures. These kind of findings, along with the presence of performance problems in complex cognitive tasks, memory, verbal fluency, etc. led some authors to propose that if there is a restorative function associated with sleep it could be primarily cerebral (Horne 1988, 1992). It has been demonstrated that TSD generates reversible deficits in neuropsychological tests of the prefrontal cortex (CPF) (Harrison and Horne 1998, 2000). This notion is consistent with the role of CPF in vigilance maintenance, in the attention to a given task, in the goal-directed behaviour, in taking decisions, or in the temporal sequencing and integration of neural information (Horne 1988). Moreover in several studies with neuroimaging techniques the most significant decreases in glucose uptake during TSD are seen in the CPF (Drummond *et al.* 1999).

In conclusion, we have found a marked increase in SRL that was accompanied by a decrease in body temperature, a severe increase in sleepiness and deterioration in the performance of a RT task. In spite of the fact that there are no reports which document changes of SRL during sleep deprivation, its sensitivity to TSD, its non-invasive character, its simplicity and its relationship with other activation parameters makes it a promising and very useful measure in sleep deprivation studies.

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