Insomnia, metabolic rate and sleep restoration

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Studies have shown occasional evidence of increased physiological activity in patients with primary insomnia. We hypothesized that metabolic rate, as measured by overall oxygen use ($\text{VO}_2$), might be a more general index of increased physiological activity. An initial experiment found elevated $\text{VO}_2$ both at night and during the day in patients with primary insomnia as compared with matched normal sleepers. A second experiment found significant but more modest increases in $\text{VO}_2$ in patients with Sleep State Misperception Insomnia [who complain of poor sleep but who had normal sleep by electroencephalographic (EEG) criteria]. In a third experiment, normal young adults were given caffeine 400 mg three times per day (TID) for 1 week as a means of increasing $\text{VO}_2$ and possibly producing other symptoms of insomnia. Participants developed many symptoms consistent with those seen in patients with primary insomnia (poor sleep, increased latency on the Multiple Sleep Latency Test, increasing fatigue despite physiological activation, and increased anxiety on the Minnesota Multiphasic Personality Inventory (MMPI)). In a final experiment, physiological arousal was again produced by caffeine to determine if sleep with elevated arousal would be less restorative. All subjects (Ss) slept for 3.5 h after being given 400 mg of caffeine. During 41 h of sleep deprivation that followed, there was no significant condition difference for the Multiple Sleep Latency Test or mood measures. The results provided only weak support for the idea that sleep is less restorative after physiological arousal.

Keywords: insomnia, metabolic rate, mood, Multiple Sleep Latency Tests, personality, psychomotor performance, sleep.

Introduction

Patients with insomnia frequently have a symptom complex that includes tension, fatigue and irritability [1] as well as some level of depression or anxiety [2]. As a result, many investigators hypothesize that insomnia is the result of internalization of emotions producing emotional arousal. Others believe that insomnia can develop entirely from physiological activation, as in phase shift insomnia, and could be based upon elevated trait levels of central nervous system arousal. Several studies have found significantly increased physiological activation in patients with insomnia. For example, Monroe [3] found significantly increased rectal temperature, heart rate, basal skin resistance and phasic vasoconstrictions 30 min prior to and during sleep in insomniacs as compared with normal sleepers. Other studies have shown that patients with sleep-onset insomnia had increased frontalis and mentalis electromyography (EMG) [4, 5], increased heart rate [6, 7], increased finger temperature, and more beta and less alpha frequencies in the electroencephalography (EEG) [4, 5]. Poor sleepers also had increased secretion of
corticosteroids [8, 9] and adrenaline [8]. However, several studies have not found increases in physiological measures [9, 10]. The inconsistent results in some of these physiological activation studies may indicate that physiological activation is not a major factor in at least some insomniacs [11] or that wide variability and small sample sizes may make it difficult to show clear physiological differences. It is also possible that the involved physiological system(s) differ from patient to patient and that a global measure, such as whole body oxygen use, would more consistently show differences.

Other research has examined the daytime functioning of insomniacs to determine the existence of subjectively reported deficits in performance, mood and alertness. The cumulative partial sleep deprivation that should arise from chronic insomnia should produce daytime sleepiness or increased susceptibility to acute sleep loss in insomniacs. However, studies have consistently found that insomniacs are not sleepier than normal controls on Multiple Sleep Latency Tests (MSLT) [10, 12] or after sleep loss [13] and may actually have longer MSLT latencies [14–16].

Study 1: Metabolic rate in insomnia

The presence of both poor sleep and daytime dysphoria and fatigue in insomniacs implies the presence of a 24-h dysfunction. We hypothesized that insomniacs have increased metabolic rates secondary to physiological arousal. To test this hypothesis, metabolic data were collected across the night and day in groups of insomniacs and carefully matched control subjects for a 36-h period in the laboratory under standard conditions. A second study was planned to examine metabolic data in patients with sleep state misperception (SSM), as it is also known that metabolic rate is increased during periods of wake compared with sleep. These patients have a complaint of insomnia but are found to have EEG-defined sleep that is similar to that seen in normal sleepers. Finally, the role of physiological arousal in the production of insomnia and in sleep restoration was examined in two additional experiments.

Method

All subjects (Ss) were required to be healthy, 18–50-year-old males and females.

Insomniacs

Individuals completing a screening questionnaire indicating that they had a sleep problem, and that it took them 45 min or more to fall asleep at least four nights each week or that they were awake for 60 min or more each night after falling asleep for at least four nights each week and that this condition had existed for at least 1 year were considered further.

Normals

Ss with reported normal sleep were selected to match a qualified insomniac by sex, age (within 5 years), weight (within 25 pounds) and general time in bed characteristics.

Exclusions

Potential subjects who indicated excessive caffeine consumption (more than 250 mg day⁻¹), who were using psychoactive medication or drugs, or who had completed a drug or alcohol abuse programme within the previous year were excluded. Ss with a history of depression or psychiatric hospitalization were excluded. Potential Ss who had histories strongly suggestive of circadian desynchrony (e.g. shift workers), sleep apnoea, or periodic leg movements were excluded.

Design

Subjects spent two nights and the intervening day in the laboratory. On both nights, a standard clinical polysomnogram was performed. On the second night, metabolic measures were recorded. On the day spent at the laboratory, Ss had a metabolic observation after awakening, performed computer tests, took an MSLT test followed by daytime resting metabolic observations, and completed a Minnesota Multiphasic Personality Inventory (MMPI) and a sleep history. They were fed the same daily menu of food prepared at the lab. Caffeinated beverages were not available.

Metabolic measurements

All metabolic measurements were performed with a SensorMedics Deltatrac Metabolic Monitor (SensorMedics, Yorba Linda, CA, USA). The Deltatrac...
generates a constant flow of 40 l min\(^{-1}\) through a canopy or mask and into the metabolic cart. The high flow pulls all expired air and a significant amount of room air from an external inlet into the machine. The metabolic monitor then calculates the difference between the flow of air from the subject and a separate measure of pure room air to determine oxygen use and carbon dioxide production by the subject. For this report, only \(\text{VO}_2\) data are summarized. The metabolic data from each pair of subjects were matched minute by minute throughout daytime and nighttime metabolic observation periods starting at the first morning metabolic observation.

To qualify for the study as an insomniac, patients were required to have EEG sleep latencies greater than 30 min on both lab nights or to have a sleep efficiency of less than 85% on both nights. Subjects with an apnoea/hypopnoea index greater than 10 or a periodic leg movement arousal index greater than 10 were disqualified.

To qualify for the study as a normal, subjects were required to have EEG sleep latencies of less than 30 min on both lab nights and to have a sleep efficiency greater than 90% on both nights. Ten normal Ss were matched with insomniacs on the basis of age, weight and sex.

**Analyses**

Paired t-tests or repeated measures analysis of variance was performed on the paired data. First, for each subject pair, bedtimes were matched, and all \(\text{VO}_2\) values were compared with a paired t-test. It is possible that the insomniacs had higher \(\text{VO}_2\) during the night simply because they were awake more and had more body movements and arousals (all of these events would increase \(\text{VO}_2\)). To examine this issue, the metabolic data were matched with the sleep data so that each metabolic observation was assigned the lightest stage of sleep for the two 30-s sleep epochs that made up the 60-s metabolic observation. Then, all metabolic observations containing EEG scored wake, movement, or arousals greater than or equal to 3-s were eliminated from the data sets. The following two metabolic observations were also eliminated from the data sets, because arousal or awakening can increase metabolic rate for more than 1 min. The remaining metabolic data were matched again within subject pairs and the t-tests were repeated. As a final test, metabolic data from SWS (stages 3 and 4) were also compared within subject pairs.

**Results**

The groups did not differ in age, weight, or time usually spent in bed per night. As expected by selection criteria, insomniacs reported significantly longer sleep latencies and shorter total sleep per night. Additional details of the study are published elsewhere [16].

Sleep data from the two groups are presented in Table 1. It can be seen from the table that sleep was substantially different in the two groups based upon the inclusion criteria.

Metabolic data: In the analysis containing all of the data, all 10 of the t-values were in the same direction and nine of 10 were statistically significant (\(P < 0.01\)). The average t-value was 13.10 with 475 degrees of freedom (df) (\(P < 0.0001\)). The respective mean values of \(\text{VO}_2\) for insomniacs and normals were: 296 and 266 mL min\(^{-1}\). In the set of 10 t-tests with awakenings, movements and arousals eliminated, nine t-values were in the predicted direction and eight of 10 were statistically significant (\(P < 0.02\)). The average t-value was 13.38 with an average of 140 df (\(P < 0.0001\)). The overall mean sleep metabolic rates for the insomniac and normal groups respectively were: 280 and 256 mL min\(^{-1}\). When slow wave sleep (SWS) was examined, it was found that two subjects (one insomniac and one normal) had no stage 3 or stage 4 sleep. As the two subjects were in different

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Sleep parameters in insomnia patients and normals: mean values with (standard deviation)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Insomniacs</td>
</tr>
<tr>
<td>Total sleep (min)</td>
<td>342 (75)</td>
</tr>
<tr>
<td>% Stage 1</td>
<td>14.6 (9.8)</td>
</tr>
<tr>
<td>% Stage 2</td>
<td>32.8 (14)</td>
</tr>
<tr>
<td>% Stage 3</td>
<td>7.8 (6.4)</td>
</tr>
<tr>
<td>% Stage 4</td>
<td>5.2 (5.8)</td>
</tr>
<tr>
<td>% REM</td>
<td>14.4 (5.4)</td>
</tr>
<tr>
<td>% Wake</td>
<td>25 (14)</td>
</tr>
<tr>
<td>Sleep latency (min)</td>
<td>20.5 (13)</td>
</tr>
<tr>
<td>Sleep efficiency</td>
<td>75 (14)</td>
</tr>
<tr>
<td>REM latency (min)</td>
<td>132 (69)</td>
</tr>
<tr>
<td>Arousal index</td>
<td>16.7 (16)</td>
</tr>
</tbody>
</table>

NS, not significant; REM, rapid eye movement.
pairs, those pairs were eliminated, and the SWS analysis proceeded on the eight pairs of subjects who all had SWS. In the set of eight t-tests comparing metabolic values from SWS observations between matched insomniacs and normals, seven t-values were in the predicted direction (binomial probability = 0.035), and five of eight were statistically significant. The average t-value was 5.57 with an average of 51 df ($P < 0.0001$). The overall mean sleep metabolic rates for the insomniac and normal groups respectively were: 266 and 250 mL min$^{-1}$.

Finally, the equation of the best-fit third-degree polynomial was determined for the complete VO$_2$ data set for each subject. The equations were averaged for the normal subjects and for the insomniacs. The average equation for each group has been plotted in Fig. 1. It can be noted from the figure that both groups had a characteristic circadian metabolic curve. The primary difference between the groups appears to be that the entire curve for the insomniacs is elevated about 30 mL min$^{-1}$.

**Study 2: Metabolic rate in sleep state misperception**

Study 2 had exactly the same design and measurements as study 1 except that the patient group was made up of individuals with a complaint of insomnia who were found to have relatively normal sleep on their sleep evaluation. Group selection criteria were the same as in the first study. To be considered for the sleep state misperception (SSM) group, patients with a complaint of insomnia were required to demonstrate normal sleep (sleep latency < 30 min and sleep efficiency > 90%) despite their claim of insomnia, to overestimate their sleep latency by at least 100% on both nights, and to have sleep latency estimates of 20 min or more on both nights.

**Results**

Eleven patients with SSM were identified. It was possible to match nine of these patients with normal sleepers having the same age, weight and sex [17]. Demographic data are presented in Table 2. Two of the nine subject pairs were female. As expected, the SSM patients judged their usual sleep latencies to be longer, their total sleep time to be less and their total wake time to be longer than the matched normals. However, the SSM patients did not differ significantly from the normals on any EEG sleep variable. In fact, total sleep time was actually nonsignificantly longer in the patients as
compared with the normal sleepers. As expected, the SSM patients estimated that their sleep latency was significantly longer than the normals (52 min vs. 24 min), that their sleep time during the night was significantly shorter than the normals (6.8 h vs. 7.5 h), and that their sleep quality was significantly worse than the normals.

The mean \( V\overline{O}_2 \) for each of the eight waking metabolic measurements and the mean \( V\overline{O}_2 \) for each hour during the sleep period were entered into a repeated-measures ANOVA with repeated terms for the matched subjects (1 df) and time (15 df). The time by group interaction was not significant \((F_{15,120} = 1.13, \text{ NS})\), but both of the main effects were significant. \( V\overline{O}_2 \) overall was elevated in the SSM patients as compared with their matched controls \((F_{1,143} = 45.22, \ P < 0.001)\). The overall mean values for the groups were 304 mL min\(^{-1}\) (±26 SD) and 286 mL min\(^{-1}\) (±34 SD). The \( V\overline{O}_2 \) data are plotted in Fig. 2.

### Study 3: Production of insomnia by physiological arousal

Observation of elevated metabolic rate in both subjective and objective insomnia patients leads to the hypothesis that insomnia could be caused by elevated arousal. The experimental question implied is that normal young adults placed in a situation involving physiological arousal might develop both the poor sleep and the secondary daytime symptoms reported by patients with psychophysiological insomnia. The typical secondary symptoms reported by patients with insomnia are listed in the left-hand column of Table 3. We hypothesized that administration of caffeine at a rate of 400 mg three times per day (TID) to normal young adults with a history of moderate caffeine use would produce significant physiological activation. Treatment at this dose level for a week might allow for the development of symptoms similar to those seen in insomnia patients.

Twelve healthy 18–30-year-old male subjects, weighing between 140 and 200 pounds and without significant history of sleeping problems, shift work, or frequent naps [18] were included in the

![Fig. 2 Average best fit VO2 in groups of Sleep State Misperception Insomniacs and Normals as a function of time of day. The data represent averaged best fit third-degree polynomials. A marker has been added at the right and left margin of both lines to indicate the group.](image)

### Table 2  Demographic data: mean and (standard deviation) in sleep state misperception patients and normal controls

<table>
<thead>
<tr>
<th></th>
<th>Misperception</th>
<th>Normals</th>
<th>( t )</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>9</td>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>31.7 (8.4)</td>
<td>32.8 (6.2)</td>
<td>0.921</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (lbs)</td>
<td>169 (16)</td>
<td>172 (16)</td>
<td>0.667</td>
<td>NS</td>
</tr>
<tr>
<td>Usual sleep Latency (min)</td>
<td>98 (88)</td>
<td>18 (6.2)</td>
<td>2.753</td>
<td>0.025</td>
</tr>
<tr>
<td>Usual time Awake (min)</td>
<td>68.5 (55)</td>
<td>3.33 (10)</td>
<td>3.174</td>
<td>0.016</td>
</tr>
<tr>
<td>Usual total Sleep (h)</td>
<td>5.3 (0.97)</td>
<td>7.4 (0.86)</td>
<td>-4.942</td>
<td>0.001</td>
</tr>
</tbody>
</table>

NS, not significant.

### Table 3  Symptoms in patients with insomnia compared with normal sleepers given caffeine 400 mg TID

<table>
<thead>
<tr>
<th></th>
<th>Patients with insomnia</th>
<th>Normals with caffeine 400 mg TID</th>
</tr>
</thead>
<tbody>
<tr>
<td>MSLT</td>
<td>Increased</td>
<td>Increased</td>
</tr>
<tr>
<td>Metabolic rate PM</td>
<td>Increased PM</td>
<td>Increased</td>
</tr>
<tr>
<td>Metabolic rate AM</td>
<td>Increased AM</td>
<td>Increased</td>
</tr>
<tr>
<td>Body temperature</td>
<td>Increased</td>
<td>Not measured</td>
</tr>
<tr>
<td>Heart rate</td>
<td>Increased</td>
<td>Not measured</td>
</tr>
<tr>
<td>Tension amp; confusion</td>
<td>Increased</td>
<td>Increased</td>
</tr>
<tr>
<td>Vigour</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Fatigue</td>
<td>Increased</td>
<td>Increased</td>
</tr>
<tr>
<td>Personality disturbance</td>
<td>Increased</td>
<td>Increased</td>
</tr>
<tr>
<td>Subjective sleep latency</td>
<td>Overestimated</td>
<td>No change</td>
</tr>
</tbody>
</table>

TID, three times per day.
study. Potential subjects using more than 250 mg of caffeine equivalent per day were excluded. Subjects participating in this study had two baseline nights (with placebo administration) followed by 7 days of caffeine TID administration followed by two withdrawal nights (with placebo administration). In the morning, all subjects followed the same schedule of alternating MSLTs, metabolic observations, performance test blocks, meals and breaks during each day. All subjects received pills at morning awakening, 8 h later, and 15 h later (the times were approximately 08:00, 16:00 and 23:00 hours).

Results

The 12 subjects selected for this study were 20.6 ± 1.5 years of age, 170 ± 23 pounds, and consumed 86 ± 74 mg of caffeine per day prior to entry into the study. Significant group differences indicative of poor sleep were seen during caffeine administration. As expected, acute caffeine administration resulted in decreased total sleep time and increased sleep latency. Additionally, stage 2 and stage 4 sleep were significantly reduced whilst awakenings and brief arousals were increased. On the final caffeine administration night, the degree of sleep disturbance was decreased compared to the acute caffeine condition, but stage 4 sleep was still significantly reduced in comparison with baseline, and brief arousals were still significantly elevated. With an average sleep latency of 28 min and sleep efficiency below 90%, these young adults could still receive a tentative diagnosis of insomnia.

In the ANOVA that compared the six daily metabolic observations, a significant condition by time interaction was found (\( F_{10,90} = 2.78, P = 0.005 \)). The interaction reflected the fact that metabolic rates did not differ before caffeine administration (08:00 hours), that metabolic rate on the initial caffeine day was significantly elevated 2, 4 and 6 h after caffeine administration, and that metabolic rate was not significantly elevated 8 h after caffeine administration (16:00 hours). Metabolic rate was increased an average of 12% over the baseline level during the 8 h after caffeine administration on the first caffeine day and an average of 3% over the baseline value on the final day of administration. Within-subjects correlations across the study showed that all Ss except one had a negative correlation between metabolic rate at bedtime and sleep efficiency for the sleep period that followed (average \( r = -0.58 \), binomial probability = 0.0059). Within-subjects correlations between each paired MSLT value and the following average VO\(_2\) value from the metabolic observation showed that all 12 Ss had positive correlations between sleep latency and metabolic rate (binomial probability = 0.0002).

The entire MMPI was administered before caffeine use and at the end of the caffeine administration. All the MMPI values remained characteristic of young adults, but there was movement towards increased pathology on all the clinical scales except MF, and the change was statistically significant for the psychasthenia (PT) (anxiety) scale.

Data from the Profile of Mood States (POMS) suggested increasing dysphoria as caffeine administration progressed (see Table 4). Significant main effects for Condition were found for all six POMS scales. For the scales Vigour and Tension (anxiety) initial caffeine administration resulted in an immediate significant increase followed by a decrease as caffeine administration continued (significant for

| Table 4 Profile of mood states after acute and chronic caffeine use |
|---------------------|---------|---------|---------|---------|--------|---|-----------------|
|                    | Baseline | Early caffeine | Late caffeine | Withdrawal | \( F_{3,132} \) | \( P \) | Differences |
| Fatigue            | 4.8      | 5.8      | 6.8      | 7.8      | 4.23   | 0.01 | B < CL, B = CE < WDL |
| Tension            | 5.9      | 10.2     | 8.6      | 9.8      | 9.56   | 0.001| B < all |
| Depression         | 3.7      | 4.1      | 5.9      | 8.4      | 7.84   | 0.001| WDL > all |
| Anger              | 4.4      | 5.7      | 7.2      | 8.6      | 5.05   | 0.005| B < CL = WDL, CE < WDL |
| Vigour             | 15.5     | 20.1     | 13.7     | 13.0     | 10.89  | 0.001| WDL = CL < B < CE, WDL > WDL |
| Confusion          | 4.0      | 4.3      | 5.1      | 6.4      | 8.42   | 0.001| WDL > all |

B = baseline; CE = early caffeine; CL = late caffeine; WDL = withdrawal.

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Fatigue was significantly increased at the end of caffeine administration compared with placebo.

Analysis of the MSLT data revealed that objective alertness was significantly improved throughout caffeine administration as compared with baseline and withdrawal, which did not differ ($F_{3,165} = 39.29, P < 0.0001$). The mean latency after early caffeine use was significantly longer than the latency after chronic caffeine use. Respective mean values for baseline, early caffeine, late caffeine and withdrawal were 10.7, 17.9, 13.4 and 11.3 min.

In addition to poor sleep, it has generally been established that many patients with insomnia will (a) report daytime fatigue or dysphoria; (b) have normal or longer than normal MSLT values; (c) report increased stress; (d) possibly have abnormal MMPI values; (e) subjectively misperceive their sleep process; and (f) have few notable differences from normals on psychomotor performance tasks. Ss receiving caffeine in this study were found to develop a similar symptom complex. Table 3 shows common secondary symptoms seen in insomnia patients (left column) and changes found in the current study (right column). Ss given caffeine had significant changes in the direction of chronic insomnia patients on MSLT, metabolic rate, negative moods and personality.

### Study 4: Is sleep at an elevated level of physiological arousal less restorative than normal sleep?

Previous studies have shown that insomnia patients and normal young adults given caffeine have poor sleep and consistent subjective complaints during the day. Normal sleepers also have a higher metabolic rate on nights of poor sleep than on baseline nights, and their baseline sleeping metabolic rate is significantly higher than during recovery sleep after a night of sleep disturbance [19]. It is therefore possible that increased physiological activity itself may reduce the ability of sleep to restore function, and if so, it may be that this less restorative sleep results in the reports of fatigue and poor function.

A final study sought to examine the recuperative potential of sleep by holding total sleep time constant and varying the level of physiological arousal at the trough of the circadian curve [20]. Specifically, it was hypothesized that performance and alertness following sleep in a shortened nocturnal sleep period at an increased metabolic rate would be degraded, as compared with performance and alertness following a similar amount of non-manipulated nocturnal sleep.

As in the previous study, a group of normal young adults participated. The study proper involved spending two sessions of four consecutive nights in the laboratory. The initial night of each of the two periods was a baseline sleep night. On the second night, Ss received either placebo on 1 week or 400 mg of caffeine (on the alternate week), and were put in bed at 02:15 hours. Subjects were allowed to accumulate a total of 210 min of sleep and were awakened a little after 06:00 hours. Metabolic data were collected during the 210 min of sleep (in 10 of the 12 subjects) to document the impact of caffeine on metabolic rate. Subjects then remained awake for approximately 41 h before being allowed a normal night of recovery sleep. Whilst awake, Ss performed computer tests and MSLTs and were fed standard meals.

### Results

Twelve subjects participated in the study. The actual total sleep time for both conditions was 213 min. There was a significant increase in stage 1 sleep (17% vs. 12%) and a decrease in stage 4 sleep (9.8% vs. 16%) in the caffeine condition compared with the placebo condition. Characteristic postdeprivation recovery sleep changes were seen on the recovery night, but there were no significant differences between conditions.

Average $\dot{V}O_2$ during the 210-min nap was 82.7% of baseline after the placebo and 87.1% of baseline after caffeine administration. These values differed significantly: $t_9 = 2.802, P = 0.021$.

No significant condition difference was found on the Multiple Sleep Latency Test. Latencies were slightly longer following the caffeine administration. No significant effects were found for oral temperature, and the data were similar for both conditions.

Results from the Addition Test (number of problems attempted) showed a significant group by time interaction ($F_{6,66} = 2.46, P < 0.05$). Significantly more problems were attempted following placebo at 22:00 and 05:00 hours than following caffeine. About 15% fewer problems were attempted at those times following caffeine use. This compares with
about a 41% drop in attempted problems in Ss who have not slept at all [21].

Vigour was rated as significantly higher in the caffeine group in the complete study data, and the mean Vigour rating remained nonsignificantly higher in the caffeine group during the second half of the study. For fatigue, ratings were nonsignificantly greater in the placebo group for the entire study and nonsignificantly greater in the caffeine group for the second half of the study.

In this experiment, there was a significant increase in metabolic rate during a 3.5-h nap period as a function of caffeine administration. This 5% increase in metabolic rate can be compared with a 11% difference between chronic insomniacs and matched normals in the first study and a 5% difference in the study of sleep state misperception insomniacs. Arousal produced by the caffeine was sufficient to increase stage 1 sleep and decrease stage 4 sleep significantly. This sleep and metabolic data support the contention that the caffeine administration did have an impact on the sleep process.

One of the primary functions of sleep is the reversal of accumulated sleepiness. Several means to measure sleepiness have been developed and typically include changes in mood, objective sleep latency and ability to perform various psychomotor performance tests. In most experiments, these three groups of dependent variables all move in consistent directions to reflect sleepiness or alertness. In the current study, the psychomotor tests consistently indicated decreased performance ability compared with baseline, following the caffeine administration and nap. However, the mood data did not clearly support degraded or improved mood after caffeine or placebo administrations. In the second half of the study, both POMS Vigour and Fatigue were nonsignificantly higher following the caffeine nap, and subjective alertness tended ($P < 0.1$) to be higher after placebo. The MSLT data weakly (i.e. nonsignificantly) supported increased alertness following the caffeine administration. As such, the complete data set provide only weak support for the major hypothesis that sleep during physiological activation is less beneficial than normal sleep.

**Discussion**

These four studies have shown that there are characteristic increases in oxygen use in patients with a complaint of insomnia whether their sleep is degraded or not. At one level, these data support many other studies that have shown evidence of increased physiological arousal in insomnia patients. An important question in understanding insomnia is whether it is this physiological arousal which produces personality and mood symptoms or whether personality or other behavioural changes produce the poor sleep. Administration of caffeine to normal young adults was successful in producing physiological activation, poor sleep and the mood changes commonly seen in insomnia patients indicating that trait arousal/metabolic rate could be responsible for the symptoms typically reported by patients. Also, because the caffeine produced both arousal and poor EEG sleep, one could question whether the secondary symptoms were related to the arousal or to the poor sleep. One study has examined this issue by using a yoke-control design to produce the same sleep and arousal pattern in good sleepers that is seen in insomnia patients for a week to examine the impact of these sleep changes on insomnia symptoms [22]. The young adults in this design became mildly sleep-deprived but did not develop the MSLT, mood, or personality changes seen in insomnia patients. In a second study, the sleep of insomnia patients was made significantly worse for a week [23]. In this study, despite very poor sleep, insomnia patient symptoms did not become worse. These studies imply that EEG sleep is perhaps less important in controlling insomnia symptoms than other variables such as arousal.

If level of physiological arousal is an important variable in producing poor sleep and subjective dysphoria, does it also make sleep less restorative? The final study sought to examine sleep restoration independent of insomnia status by comparing comparable amounts of EEG sleep with or without arousal produced by caffeine and the ability to maintain function during a night and day of total sleep deprivation. Only meagre differences were found after the ‘aroused’ sleep. Again, the implication from this study is that sleepiness is strongly determined by the sleep system, i.e. the amount of time awake and the total amount of prior sleep. Modifications of the arousal system seem to have little impact on the ability of the sleep system to restore alertness. Thus patients with insomnia may have moderately poor sleep without the development of overwhelming sleepiness. On the other
hand, chronic elevations in arousal, whilst not having large impact on the sleep system, can have a significant impact on mood and personality variables and are related to reports of poor sleep (which may not be objectively verifiable by traditional EEG measures).

Conflict of interest statement
No conflict of interest was declared.

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References

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