Industrial Health 2017, **55**, 423–436

Original Article

The impact of meal timing on performance, sleepiness, gastric upset, and hunger during simulated night shift

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Received March 24, 2017 and accepted July 18, 2017
Published online in J-STAGE July 25, 2017

Abstract: This study examined the impact of eating during simulated night shift on performance and subjective complaints. Subjects were randomized to eating at night (n=5; 23.2 ± 5.5 y) or not eating at night (n=5; 26.2 ± 6.4 y). All participants were given one sleep opportunity of 8 h (22:00 h-06:00 h) before transitioning to the night shift protocol. During the four days of simulated night shift participants were awake from 16:00 h-10:00 h with a daytime sleep of 6 h (10:00 h-16:00 h). In the simulated night shift protocol, meals were provided at ≈0700 h, 1900 h and 0130 h (eating at night); or ≈0700 h, 0930 h, 1410 h and 1900 h (not eating at night). Subjects completed sleepiness, hunger and gastric complaint scales, a Digit Symbol Substitution Task and a 10-min Psychomotor Vigilance Task. Increased sleepiness and performance impairment was evident in both conditions at 0400 h ($p<0.05$). Performance impairment at 0400 h was exacerbated when eating at night. Not eating at night was associated with elevated hunger and a small but significant elevation in stomach upset across the night ($p<0.026$). Eating at night was associated with elevated bloating on night one, which decreased across the protocol. Restricting food intake may limit performance impairments at night. Dietary recommendations to improve night-shift performance must also consider worker comfort.

Key words: Shift-work, Performance, Circadian misalignment, Sleep loss, Psychomotor vigilance, Sleepiness, Timed eating, Hunger

Introduction

There are currently more than 1.5 million shift workers in Australia[1)] and 29% of workers in the United States, do not work regular daytime shifts, with numbers likely to rise with increasing demand by companies to extend working hours[2)]. Shift workers are often required to work throughout the night, for consecutive nights, when circadian processes typically promote sleep[3)]. Furthermore, night workers are required to sleep during the day, when sleep quality is reduced and sleep length can be between two and four hours shorter, resulting in sleep loss[4)]. The

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association between circadian misalignment, shortened sleep and impaired glucose has been well established[5, 6]. This impairment over a long period of time, is associated with the development of a number of health problems including obesity[7, 8], type 2 diabetes[9] and metabolic syndrome[10]. However shift work does not only have an impact on health but also performance output.

It has been well established that performance varies across the day, with declines in functioning post habitual bedtime[11]. This decline worsens over the course of the night with poorest performance in the early morning hours (approximately 0500 h) during the circadian nadir, reflected by minimum body temperature[12–14]. Furthermore, restricted sleep or sleep loss, as seen in shift work [4], affects many aspects of cognitive performance, particularly alertness[15]. The psychomotor vigilance task (PVT), a tool sensitive to sleep loss[16, 17], is used to assess vigilant attention/alertness. Many sleep restriction studies have shown that sleep loss increases the number of PVT lapses and errors[18–21]. In addition, sleep restriction also leads to increased subjective sleepiness[21] and impairments in cognitive processing[22]. Impairments in cognitive processing due to sleep restriction have also been reported using the digit symbol substitution task (DSST)[23].

In addition to the effects of sleep loss, a number of studies have shown that performance may be impaired following food intake[24–26]. For example, Smith and Miles[27], found that the ability to maintain attention and to react quickly to a visual stimulus was impaired post-lunch compared to when a meal was not provided. Furthermore, Smith, *et al.*[28] reported that food intake, regardless of composition, influenced mood, with subjects reporting increased lethargy, clumsiness, dreaminess and decreased mental sharpness post-lunch. Studies examining the effect of food intake on performance during the night are very limited, however another study by Smith and Miles[29], looked at the impact of food intake during the day (1230 h-1330 h) and during the night (0130 h-0230 h) on cognitive vigilance and found impairments in the number of correct responses at both time points.

Night shift workers often redistribute food intake from the daytime to the night hours[30, 31], which results in food consumption during a time when the body's normal biological processes are primed for sleep[14, 32, 33]. Indeed, nutrient absorption, metabolism[34, 35], enzyme activity and gastrointestinal motility[36, 37] are lower at night. It is perhaps not surprising then, that night shift work is associated with increased gastrointestinal complaints[38, 39], including abdominal pain, diarrhoea, constipation, heartburn, and indigestion[40].

Previous studies reporting the effects of sleep loss on performance have not reported the timing of food intake and its potential impact on performance or gastric upset. Given performance impairments are greatest at night[4] and the previously mentioned effects of food intake, reducing food intake during this time may be a suitable countermeasure to reduce performance impairments and improve gut reaction reported during a night shift. Therefore the aim of this study was to investigate the impact of eating at night vs not eating at night on performance, sleepiness, hunger and gastric upset following four nights of circadian misalignment in healthy young men. We hypothesised that consuming a meal at night would lead to impaired performance and increased sleepiness compared to not eating at night. To test the hypothesis we assessed vigilant attention, cognitive processing, sleepiness, hunger and gastric upset under both conditions, during four consecutive simulated night shifts.

Subjects and Methods

This study was approved by the University of South Australia Human Research Ethics Committee (0000033621) and was conducted in accordance with the Recommendations from the Declaration of Helsinki. This study is registered with the Australian New Zealand Clinical Trials Registry (ACTRN12615001107516). Subjects provided written informed consent prior to entering the laboratory and were provided with an honorarium. The metabolic[41] and further performance[42] data has been published elsewhere.

Subjects

A total of 13 healthy males, 18–45 y (mean age 24.7 ± 5.6 y, BMI 22.7 ± 1.9 kg/m^2), were recruited from the community via local noticeboards and social media. A detailed screening process was conducted to determine eligibility. A general health questionnaire, blood analysis, clinical history, and Beck Depression Inventory (score ≥14)[43] were used to determine good physical and mental health. Additionally, subjects reported regular sleep patterns, with the absence of daytime napping and sleepiness or known sleep disorders, confirmed using the general health questionnaire, the Pittsburgh Sleep Quality Index (score >6)[44], and the composite morningness- eveningness questionnaire (score of <31 or >69)[45]. Subjects were excluded if they reported specific food allergies or food habits, such as vegetarianism, BMI was >30 kg/m^2 or currently used pre-

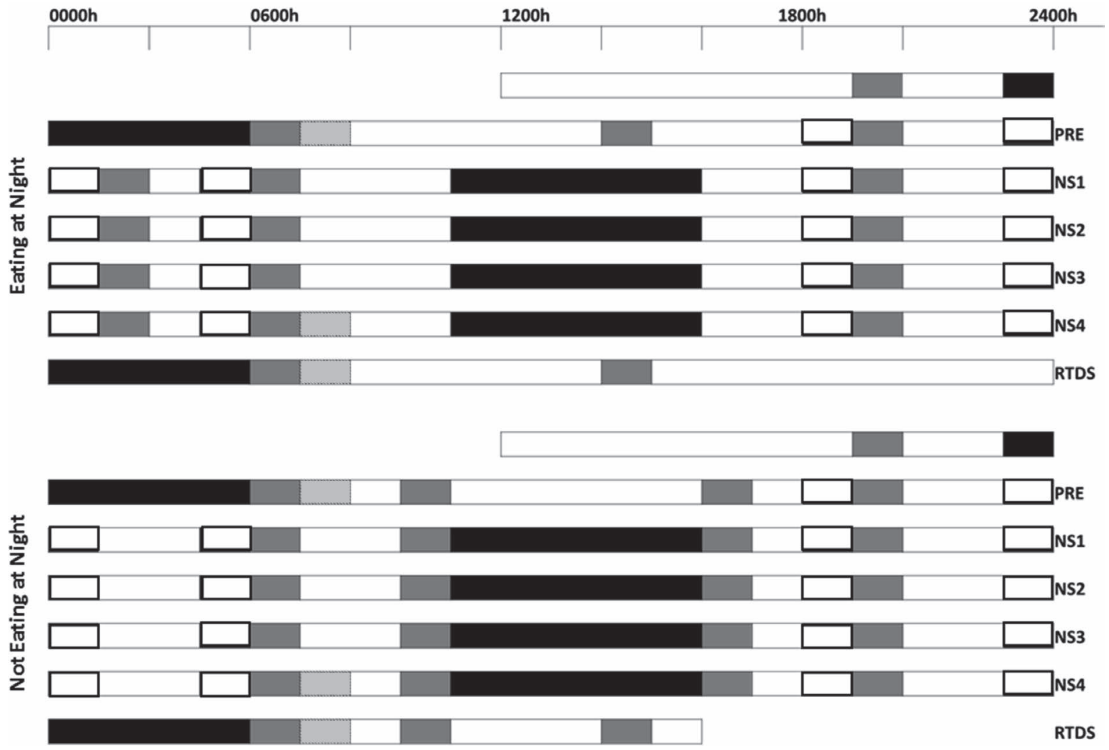

Fig. 1. Protocol schematic.

Black bars; scheduled sleep opportunities, white bars; periods of wake, patterned box; blood testing in response to breakfast meal, grey box; meal times, open black bar; neurobehavioral test battery, PRE; pre night shift, NS1-4; night shift work days, RTDS; return to day shift.

scription or over-the-counter medications. Subjects were not smokers, and had not engaged in shift work or transmeridian travel in the 2 months prior to the study. Participants were also asked to abstain from alcohol and caffeine for the week prior to the study. Females were not eligible for this study, the menstrual cycle and use of oral contraceptives has been shown to alter cognitive function during sleep loss[46].

Study design

This study was conducted in the sleep laboratory at the Centre for Sleep Research at the University of South Australia, Adelaide, Australia. The parallel study design was conducted from January to July 2015. The laboratory is windowless and sound attenuated and was kept at an ambient temperature 23 ± 1°C, and light intensity maintained at < 50 lux during wake periods and < 0.03 lux (complete darkness) during sleep periods. Access to clock and social time cues (i.e., mobile phones, internet, and live television) was restricted.

Randomization was completed at the group level (2–4 participants were in the laboratory at one time, one condition at a time). Subjects were assigned to either the eating at night (control) (n = 5; age 23.2 ± 5.5 y; BMI 22.2 ± 1.2 kg/m^2) or the not eating at night condition (intervention) (n = 5; age 26.2 ± 6.4 y; BMI 23.2 ± 1.3 kg/m^2).

Subjects remained in the sleep laboratory for the duration of the six day protocol. Subjects entered the laboratory and were familiarised with the laboratory environment and cognitive tasks. The protocol (Fig. 1) included a night-time sleep 8 h time in bed (TIB) (2200 h-0600 h), pre simulated night shift (PRE). This was followed by four consecutive nights of simulated night shift with a daytime sleep of 6 h TIB (NS1-NS4; 1000 h-1600 h). During each night of simulated night shift subjects completed a neurobehavioral test battery (NTB) at 1830 h, 2130 h, 2400 h and 0400 h in their individual bedrooms. The NTB consisted of, in order of presentation, a Karolinksa Sleepiness Scale (KSS)[47], mood scales, DSST [48], and a 10 minute PVT [49]. Participants were given a 15 min shower opportunity between 16:00–16:30 h on NS1–2 and NS4. A shower was not possible on NS3 due to cannulation. Subjects were then given a nocturnal return to daytime schedule sleep (RTDS) of 8 h TIB (2200 h-0600 h).

Compliance was monitored throughout the protocol by study personnel. When not completing scheduled tasks,

Table 1. Food intake

	Meal 1 (≈07:00 h) ≈30% EER	Meal 2 (09:30 h) 20% EER	Meal 3 (14:10 h) 10% EER	Meal 4 (19:00 h) 30% EER eating at night 40% EER not eating at night	Meal 5 (01:30 h) 40% EER
Eating at Night Condition	White bread Margarine Strawberry jam Reduced fat milk Cornflakes (cereal) Orange juice	No meal	No meal	Roast beef Sandwich: Roast beef Wholemeal bread Lettuce Reduced fat cheese Tomato Apple Small packet of potato crisps Granola bar	Chicken Salad: Chicken breast Tomato Lettuce Cucumber Parmesan cheese White bread roll Margarine Caesar salad dressing (reduced fat) Egg (hard boiled) Apple juice Small packet of potato crisps
Not Eating at Night Condition	White bread Margarine Strawberry jam Reduced fat milk Cornflakes (cereal) Orange juice	Shortbread cookie Apple	Apple juice Small bag of potato crisps	Chicken Salad: Chicken breast Tomato Lettuce Cucumber Parmesan cheese White bread roll Margarine Caesar salad dressing (reduced fat) Egg (hard boiled) Apple juice Small packet of potato crisps Granola bar	No meal

*EER: estimated energy requirement

subjects were free to read, watch DVDs, play board games, talk, or listen to music. Vigorous exercise was not permitted in the laboratory.

Food intake

Food timing and composition were strictly controlled throughout the study. Subjects in the eating at night condition consumed "breakfast" at ≈0700 h, "meal 4" at 1900 h and "meal 5" at 0130 h each day, and provided approximately 30%, 30% and 40% of the daily energy requirement, respectively. Subjects in the not eating at night condition consumed "breakfast" at ≈0700 h, "meal 2" at 0930 h, "meal 3" at 1410 h and "meal 4" at 1900 h, providing approximately 30%, 20%, 10%, 40% of the daily energy requirement, respectively. Individual daily energy requirements (kilojoules) were calculated using the Harris Benedict equation, a validated tool [8], with a light/sedentary activity level (laboratory condition). The individual energy requirement was further reduced by 15% to allow for extreme sedentary laboratory conditions. The energy content was increased by 30% on NS1 to allow for the increased time spent awake when transitioning to the night-shift. Macronutrient content of meals was based on the average Australian diet[50]. An example of the diet provided is shown in Table 1.

Subjects abstained from caffeine and alcohol intake while in the laboratory. Access to foods and beverages, other than water, was restricted outside of specified meal times.

Cognitive Performance

Psychomotor vigilance task

The PVT is a reaction time (RT) task which is considered a sensitive measure to assess the effects of sleep loss[17]. Subjects were instructed to respond to a visual stimulus (millisecond counter) displayed on the computer screen as quickly as possible by pressing a button

with their dominant thumb. They were asked to respond to the stimulus as quickly as possible without anticipating when the stimulus may appear. The stimulus appeared at random intervals between 2–10 s (inter-stimulus interval). Outcome measures for the PVT include; number of PVT lapses (RT > 500 ms), PVT errors (false starts; RT < 150 ms or clicking the button when no stimulus) and PVT median response time.

Digit symbol substitution test

The DSST was a computerised version of the cognitive performance test in the Wechsler Adult Intelligence Scale[48]. Subjects were instructed to match numbers (1–9) with a series of randomly presented symbols based on a code displayed on the screen. Subjects had 3 min to respond as quickly and accurately as possible. The outcome measure was number of correct responses.

Sleepiness, Hunger and Gut Reaction

Karolinksa Sleepiness Scale, hunger and gut reaction scales

The nine point Likert-type scales were completed electronically, with subjects providing a whole number response rather than marking their response on a line. The KSS is a standard subjective measure of sleepiness[47]. Subjects were asked to rate their sleepiness, using the nine point Likert-type scale, from 1 ("extremely alert") to 9 ("very sleep, great effort to stay awake") at the beginning of each NTB. Following the KSS, subjects were asked to complete eight Likert-type mood scales. Subjects rated their hunger (hunger, thoughts of food, fullness and urge to eat) and gut reaction (gassy, bloated, upset stomach and dizziness) on the scale from 1 to 9.

Sleep EEG

Sleep was recorded on PRE: 22:00 h-06:00 h, NS2: 10:00 h-16:00 h and RTDS: 22:00 h-06:00 h, using Compumedics GRAEL recorders (Melbourne, Australia). Data were collected from the F3, F4, C3, C4, O1 and O2 sites with reference to a contralateral mastoid (M1, M2). An experienced sleep technician used an infrared camera to monitor subjects overnight. Sleep studies were scored according to Rechtschaffen and Kales sleep staging criteria[51]. The sleep variables analysed included total sleep time, wake after sleep onset (WASO), sleep efficiency (SE), sleep onset latency (SOL) and time in minutes of rapid eye movement (REM), stage 1, stage 2, stage 3 and stage 4.

Statistical analyses

Data analyses were performed using SPSS Statistics Software Version 21.0 (IBM Corp, Armonk, NY, USA). Initially, fifteen subjects were enrolled in the study, two subjects then withdrew prior to entering the laboratory. After commencement of the study one subject withdrew due to illness. Data from two subjects were performance outliers and were withdrawn from analyses. In total data from 10 subjects was included in the final analyses (Fig. 2).

Mixed-effects ANOVAs were used to analyse the performance (PVT lapses, PVT errors, PVT median response time, DSST number correct responses), KSS, hunger (hunger, fullness, thoughts of food, urge to eat), and gut reaction (gassy, upset stomach, bloated, dizzy) variables with fixed effects of condition (eating at night, not eating at night), day (nightshift 1–4), and time (1830 h 2130 h, 2400 h, 0400 h) their interactions (condition*day, condition*time, condition*day*time), with a random effect of subject on the intercept. Significant interaction effects were explored using within-subjects planned comparisons, comparing all time points to 0400 h and all days to NS1. Basic repeated measures ANOVAs were run separately to estimate effect sizes, with small, medium and large effects determined with thresholds set at 0.01, 0.06, and 0.14, respectively[52].

Mixed-effects ANOVA were conducted for the sleep data with fixed effects of condition (eating at night, not eating at night) and day (PRE, NS4, RTDS), and their interaction (condition*day), and a random effect of the subject on the intercept.

Denominator degrees of freedom were corrected with the Satterthwaite approximation and are reported to the nearest whole number. In all analyses, results were considered statistically significant if $p < 0.05$.

Results

Cognitive performance

PVT lapses

There was a significant main effect of time ($p < 0.001$, $\eta^2_{(partial)} = 0.561$, large effect), such that performance was significantly worse at 0400 h ($p < 0.001$) (Table 2). There was a significant interaction effect of condition*time ($p < 0.001$, $\eta^2_{(partial)} = 0.289$, large effect), such that performance was significantly worse at 0400 h in both conditions, but the effect was stronger in the eating at night condition (Fig. 3).

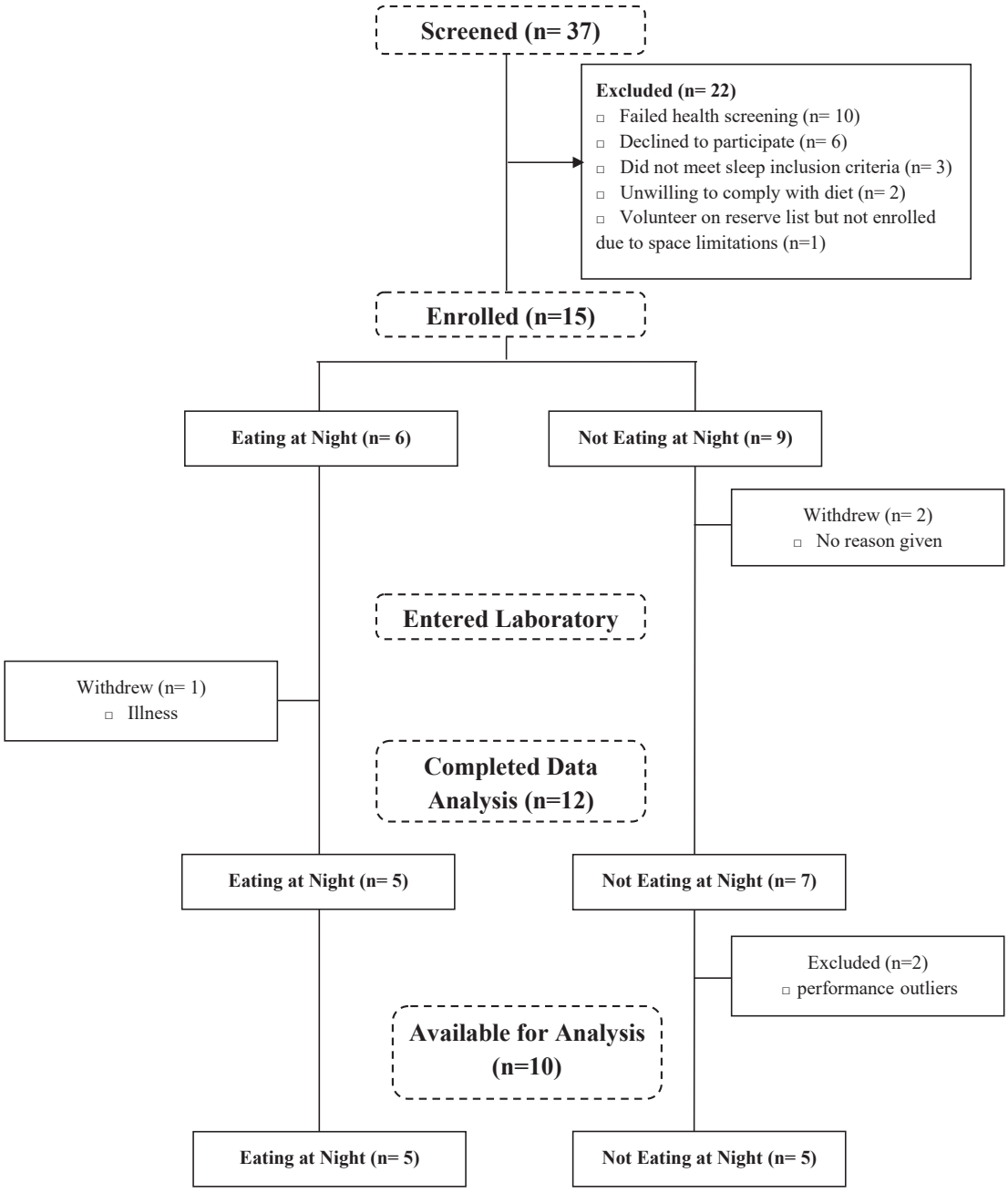

Fig. 2. Consort diagram of participants who were screened, allocated a condition, completed the laboratory protocol and data analysed.

PVT errors

There was a significant main effect of day ($p=0.043$, $\eta^2_{(partial)}=0.124$, medium effect), such that performance significantly worsened on NS3 ($p=0.018$) and NS4 ($p=0.031$) compared to NS1 (Table 2). There was a significant main effect of time ($p=0.043$, $\eta^2_{(partial)}=0.231$, large effect), such that performance was significantly worse at 0400 h. There was a significant interaction effect of condition*time ($p=0.009$, $\eta^2_{(partial)}=0.300$, large effect), such that performance was significantly worse at 0400 h in the eating at night condition ($p<0.001$) but not in the not eating at night condition (Fig. 3).

PVT median response

There was a significant main effect of time ($p<0.001$, $\eta^2_{(partial)}=0.587$, large effect), such that response time was significantly worse at 0400 h ($p<0.001$) (Table 2). There was a significant interaction effect of condition*time

Table 2. Linear mixed models: performance, hunger and gut reaction outcome measures

Outcome Measure	Condition		Day		Time		Condition*Day		Condition*Time		Condition*Day*Time	
	F_{df}	$\eta^2_{(partial)}$	F_{df}	$\eta^2_{(partial)}$	F_{df}	$\eta^2_{(partial)}$	F_{df}	$\eta^2_{(partial)}$	F_{df}	$\eta^2_{(partial)}$	F_{df}	$\eta^2_{(partial)}$
Performance												
PVT Lapses	$0.20_{1,8}$	0.024	$0.09_{3,120}$	0.013	$\mathbf{27.03_{3,120}}^{***}$	0.561	$1.76_{3,120}$	0.196	$\mathbf{8.60_{3,120}}^{***}$	0.289	$0.33_{9,120}$	0.078
PVT Errors	$1.36_{1,8}$	0.145	$\mathbf{2.80_{3,120}}^{*}$	0.124	$\mathbf{2.80_{3,120}}^{*}$	0.231	$2.37_{3,120}$	0.107	$\mathbf{4.00_{3,120}}^{**}$	0.300	$0.46_{9,120}$	0.112
PVT Median	$0.47_{1,8}$	0.056	$0.24_{3,120}$	0.028	$\mathbf{27.54_{3,120}}^{***}$	0.587	$\mathbf{2.76_{3,120}}^{*}$	0.248	$\mathbf{5.37_{3,120}}^{**}$	0.217	$0.56_{9,120}$	0.120
DSST correct	$0.00_{1,8}$	0.000	$\mathbf{4.79_{3,120}}^{**}$	0.328	$\mathbf{13.68_{3,120}}^{***}$	0.556	$0.67_{3,120}$	0.060	$1.08_{3,120}$	0.090	$1.46_{9,120}$	0.185
KSS	$1.19_{1,8}$	0.170	$\mathbf{8.61_{3,120}}^{***}$	0.472	$\mathbf{61.14_{3,120}}^{***}$	0.864	$1.26_{3,120}$	0.144	$1.20_{3,120}$	0.189	$1.23_{9,120}$	0.208
Hunger scales												
Hunger	$\mathbf{17.19_{1,8}}^{**}$	0.682	$\mathbf{2.56_{3,120}}$	0.168	$\mathbf{9.91_{3,120}}^{***}$	0.482	$\mathbf{2.86_{3,120}}^{*}$	0.184	$\mathbf{15.56_{3,120}}^{***}$	0.594	$0.54_{9,120}$	0.088
Fullness	$4.39_{1,8}$	0.052	$1.42_{3,120}$	0.086	$\mathbf{10.18_{3,120}}^{***}$	0.555	$2.28_{3,120}$	0.132	$\mathbf{10.92_{3,120}}^{***}$	0.572	$0.17_{9,120}$	0.030
Thoughts of food	$\mathbf{8.232_{1,8}}^{*}$	0.507	$\mathbf{4.85_{3,120}}^{**}$	0.295	$\mathbf{14.24_{3,120}}^{***}$	0.535	$1.21_{3,120}$	0.094	$\mathbf{14.56_{3,120}}^{***}$	0.540	$0.31_{9,120}$	0.055
Urge to Eat	$\mathbf{13.84_{1,8}}^{**}$	0.634	$\mathbf{2.54_{3,120}}$	0.171	$\mathbf{16.40_{3,120}}^{***}$	0.609	$0.26_{3,120}$	0.020	$\mathbf{15.83_{3,120}}^{***}$	0.601	$0.38_{9,120}$	0.062
Gut Reaction												
Gassy	$1.75_{1,7}$	0.200	$0.06_{3,105}$	0.007	$1.03_{3,105}$	0.081	$1.81_{3,105}$	0.172	$1.54_{3,105}$	0.117	$0.43_{9,105}$	0.081
Upset stomach	$0.41_{1,8}$	0.049	$0.36_{3,120}$	0.056	$\mathbf{7.15_{3,120}}^{***}$	0.353	$1.64_{3,120}$	0.214	$\mathbf{4.60_{3,120}}^{**}$	0.260	$0.85_{9,120}$	0.109
Bloated	$3.21_{1,7}$	0.314	$\mathbf{3.71_{3,105}}^{*}$	0.276	$1.93_{3,105}$	0.221	$\mathbf{3.95_{3,105}}^{*}$	0.288	$\mathbf{2.89_{3,105}}^{*}$	0.297	$0.46_{9,105}$	0.069
Dizzy	$0.055_{1,8}$	0.007	$1.11_{3,120}$	0.092	$\mathbf{20.02_{3,120}}^{***}$	0.608	$0.83_{3,120}$	0.071	$1.11_{3,120}$	0.079	$0.36_{9,120}$	0.062

$^{*}p=0.05$, $^{**}p=0.01$, $^{***}p<0.001$. KSS: Karolinska Sleepiness Scale, $\eta^2_{(partial)}$: Partial eta^2. Results shown are from linear mixed model analyses with main effects of condition: eating at night/not eating at night; time: 1830 h, 2130 h, 2400 h and 0400 h and day: Night shift 1–4 and their interactions (condition*day, condition*time and condition*day*time). Denominator df corrected with Satterthwaite approximation and reported to the nearest whole number (F value with degrees of freedom ($_{df}$) displayed as subscript). Basic repeated measures ANOVAs were run separately to estimate effect size, with small, medium and large effects valued at 0.01, 0.06, and 0.14, respectively[52].

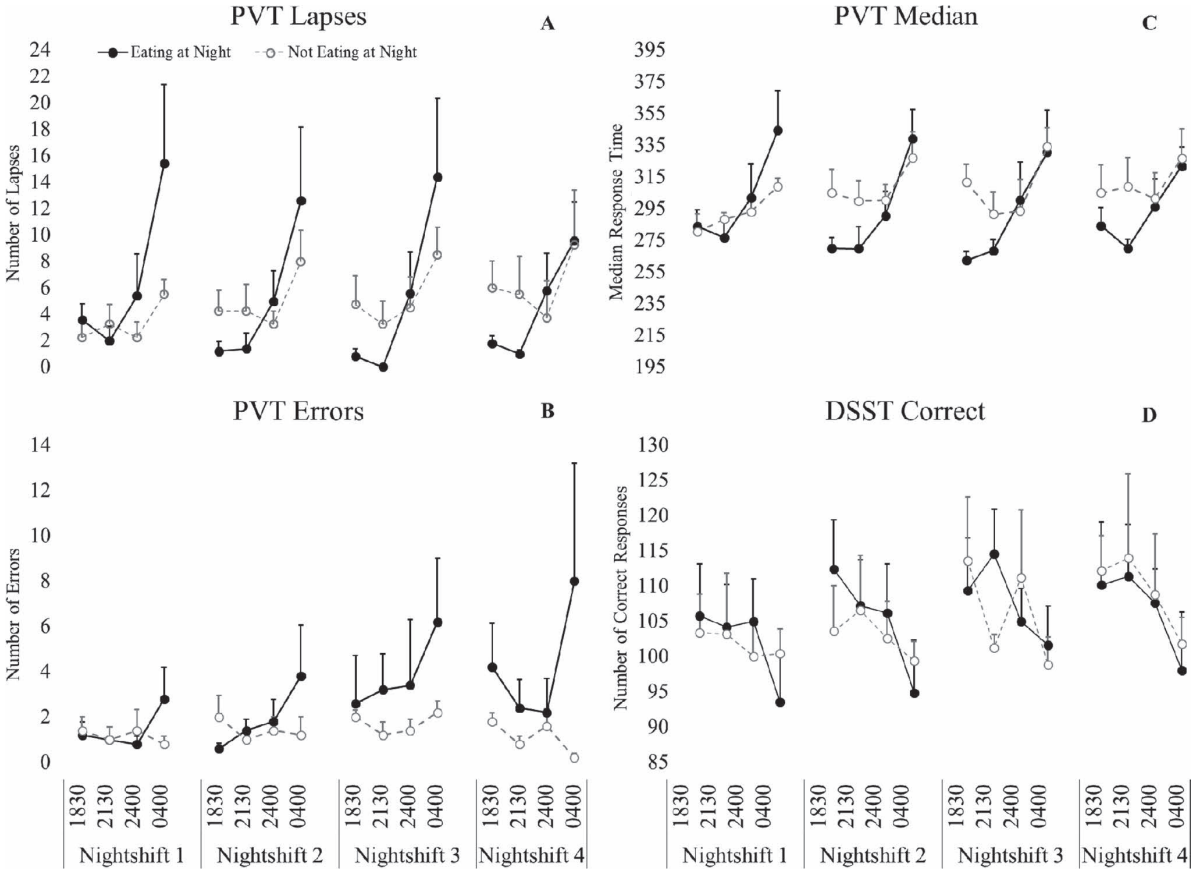

Fig. 3. Performance following simulated night-shift.

A-PVT Lapses, B- PVT Errors, C- PVT Median Response Time, D- Digit Symbol Substitution Task (DSST) number correct. Eating at night condition- solid black marker, not eating at night condition- open grey circle marker. Bars represent standard error.

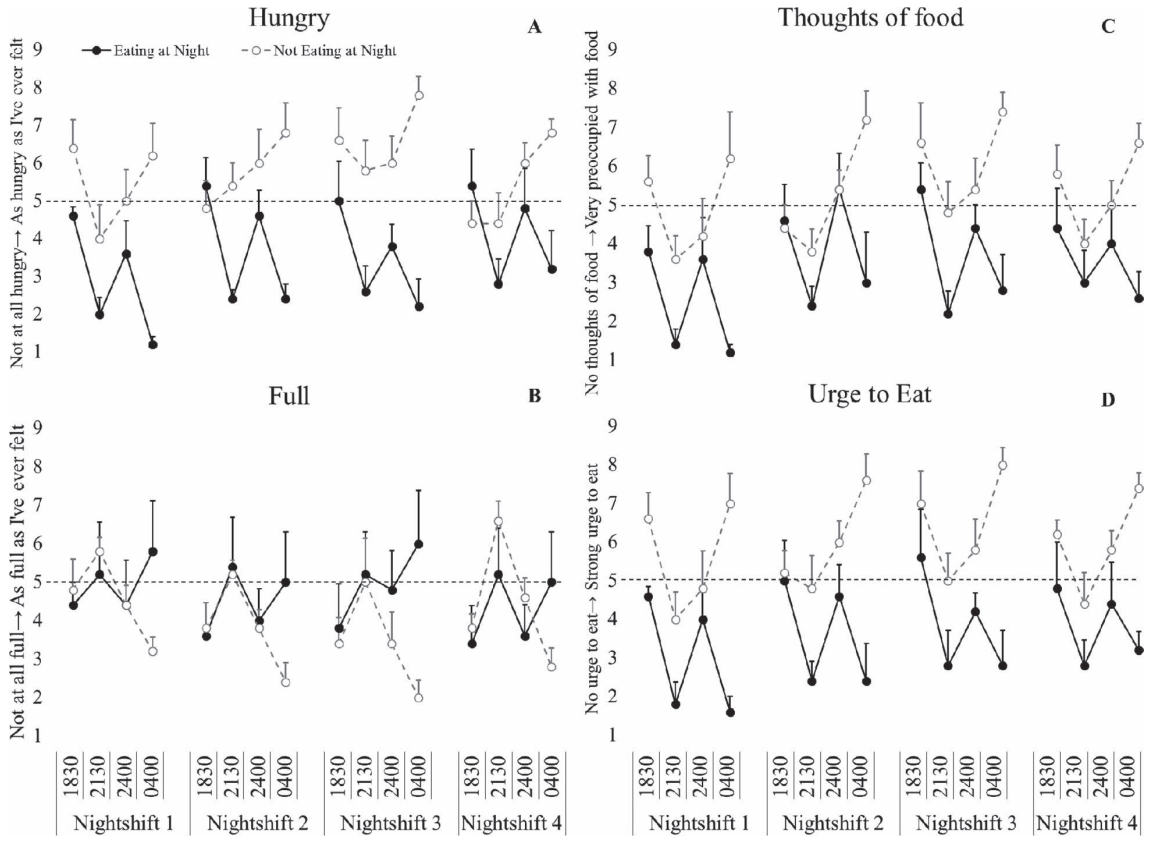

Fig. 4. Hunger ratings following simulated night-shift.

A-hungry, B- full, C- thoughts of food, D- urge to eat. Eating at night condition- solid black marker, not eating at night condition- open grey circle marker. Bars represent standard error. Dash line represents neutral on the rating scale.

($p=0.002$, $\eta^2_{(partial)}=0.217$, large effect), such that performance was significantly worse at 0400 h in both conditions, but the effect was stronger in the eating at night condition (Fig. 3).

DSST number correct

There was a significant effect of day ($p=0.003$, $\eta^2_{(partial)}=0.328$, large effect), such that performance significantly improved on NS3 ($p=0.006$) and NS4 ($p<0.001$) compared to NS1 (Fig. 3). There was a significant main effect of time ($p<0.001$, $\eta^2_{(partial)}=0.556$, large effect), such that performance was significantly worse at 0400 h ($p<0.001$) (Table 2).

Hunger and gut reaction

Hunger scales

There was a significant main effect of condition with hunger, thoughts of food and urge to eat significantly increased ($p<0.021$, $\eta^2_{(partial)}=0.507-0.982$, large effect), but no significant difference in fullness ($p=0.526$, $\eta^2_{(partial)}=0.052$, small effect) in the not eating at night condition. There was a significant main effect of day ($p<0.05$, $\eta^2_{(partial)}=0.295$, large effect) with thoughts of food significantly increased on NS2 ($p=0.010$), NS3 ($p<0.001$) and NS4 ($p=0.024$) compared to NS1. There was a significant effect of time ($p<0.001$, $\eta^2_{(partial)}=0.482-0.609$, large effect) with decreased fullness, and increased hunger, urge to eat and thoughts of food at 0400 h compared to 2130 h ($p<0.006$) and decreased hunger and urge to eat compared to 1830 h ($p<0.049$). There was a significant condition*day interaction for hunger ($p=0.040$, $\eta^2_{(partial)}=0.184$, large effect), with significantly increased hunger ratings on NS3 compared to NS1 ($p=0.012$) in the eating at night condition. In the not eating at night condition there was significantly increased hunger ratings on NS4 compared to NS1 ($p=0.009$). There was a significant condition*time interaction ($p<0.001$, $\eta^2_{(partial)}=0.540-0.601$, large effect), with significantly increased hunger ratings, thoughts of food, urge to eat and decreased fullness at 0400 h in the not eating at night condition compared to 1830 h ($p<0.006$), 2130 h ($p<0.001$) and 2400 h ($p<0.012$). In the eating at night condition there was significantly decreased hunger

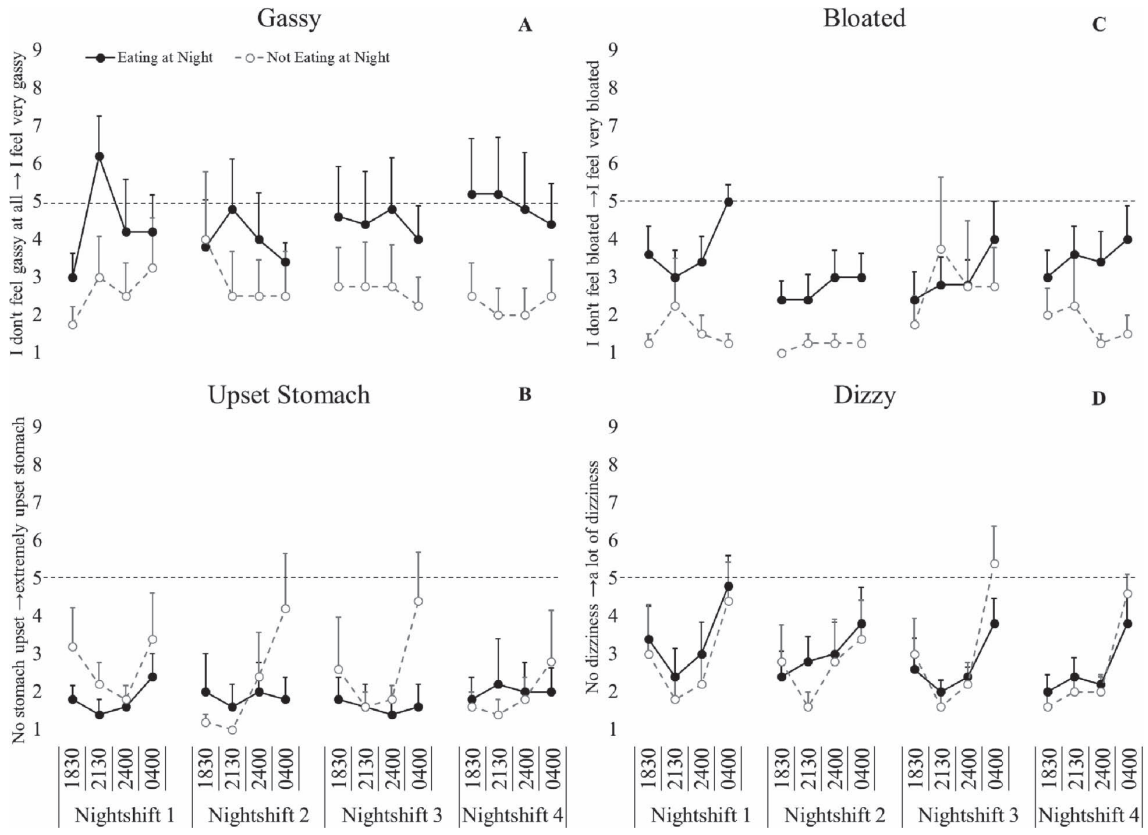

Fig. 5. Gut reaction following simulated night-shift.

A-Gassy, B- Upset stomach, C- Bloated, D- Dizzy. Eating at night condition- solid black marker, not eating at night condition- open grey circle marker. Bars represent standard error. Dash line- represents neutral on the rating scale.

ratings, thoughts of food and urge to eat and increased fullness at 0400 h compared to 1830 h ($p<0.001$) and 2400 h ($p<0.007$) (Fig. 4).

Gut reaction scales

There was a significant main effect of day for bloating ($p=0.014$, $\eta^2_{(partial)}=0.276$, large effect), with significantly increased bloating on NS1 compared to NS2 ($p=0.018$). There was a significant main effect of time with significantly increased stomach upset ($p<0.001$, $\eta^2_{(partial)}=0.353$, large effect) and dizziness ($p<0.001$, $\eta^2_{(partial)}=0.608$, large effect) but not bloating ($p=0.128$, $\eta^2_{(partial)}=0.221$, large effect) or gassiness ($p=0.382$, $\eta^2_{(partial)}=0.081$, medium effect) at 0400 h (Fig. 5). There was a significant interaction effect of condition*time ($p=0.004$, $\eta^2_{(partial)}=0.260$, large effect), with significantly increased stomach upset reported at 0400 h, compared to all other time points ($p<0.001$) in the not eating at night condition but not in the eating at night condition (Table 2). There was also a significant condition*day interaction for bloating ($p=0.010$, $\eta^2_{(partial)}=0.288$, large effect), with significantly increased bloating on NS1 ($p=0.009$) compared to NS2 in the eating at night condition. In the not eating at night condition there was significantly decreased bloating on NS1 compared to

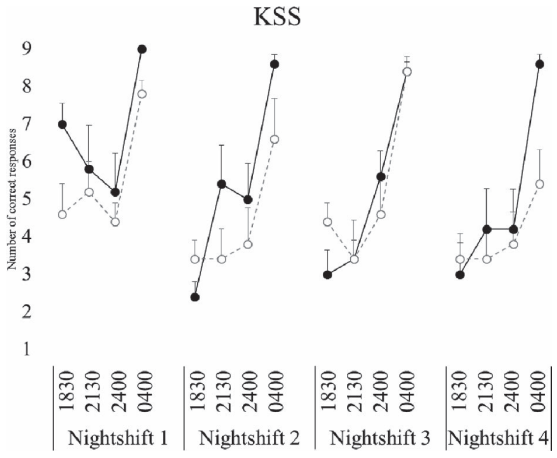

Fig. 6. Sleepiness following simulated night-shift.

KSS: Karolinska Sleepiness Scale. Eating at night condition- solid black marker, not eating at night condition- open grey circle marker. Bars represent standard error.

Table 3. Sleep length and architecture

Variable	Eating at night (mean ± SE)	Not Eating at Night (mean ± SE)	Condition		Day		Condition*Day	
			$F_{(df)}$	p_{value}	$F_{(df)}$	p_{value}	$F_{(df)}$	p_{value}
TST (min)	389.25 ± 7.25	387.72 ± 5.92	$0.03_{(1,24)}$	0.872	$40.58_{(2,24)}$	**<0.001**	$0.93_{(2,24)}$	0.407
WASO (min)	35.38 ± 8.33	33.42 ± 6.80	$0.03_{(1,8)}$	0.860	$1.60_{(2,16)}$	0.233	$0.22_{(2,16)}$	0.806
SE (%)	89.13 ± 1.71	88.67 ± 1.40	$0.04_{(1,24)}$	0.840	$3.26_{(2,24)}$	**0.056**	$0.716_{(2,24)}$	0.499
SOL (min)	13.13 ± 5.79	18.17 ± 4.73	$0.45_{(1,8)}$	0.519	$0.52_{(2,16)}$	**0.011**	$0.70_{(2,16)}$	0.514
REM (min)	74.88 ± 5.51	83.28 ± 4.50	$1.40_{(1,8)}$	0.271	$3.69_{(2,16)}$	**0.048**	$0.08_{(2,16)}$	0.925
Stage 1 (min)	31.92 ± 4.65	35.19 ± 3.80	$0.30_{(1,8)}$	0.600	$10.51_{(2,16)}$	**0.001**	$2.87_{(2,16)}$	0.086
Stage 2 (min)	201.33 ± 7.60	185.11 ± 6.20	$2.74_{(1,8)}$	0.137	$63.80_{(2,16)}$	**<0.001**	$0.33_{(2,16)}$	0.724
Stage 3 (min)	23.04 ± 2.65	19.69 ± 2.16	$0.96_{(1,8)}$	0.356	$5.40_{(2,16)}$	**0.016**	$0.72_{(2,16)}$	0.502
Stage 4 (min)	58.08 ± 7.13	64.44 ± 5.82	$0.48_{(1,8)}$	0.509	$19.87_{(2,16)}$	**<0.001**	$0.49_{(2,16)}$	0.625

Data presented (n=10). TST: total sleep time; min: minute; WASO: wake after sleep onset; SE: sleep efficiency; SOL: sleep onset latency; REM: rapid eye movement. Results shown are from linear mixed model analyses with main effects of meal timing; eating at night/not eating at night and day; pre night shift (PRE), the second consecutive night shift (NS2), and return to day shift (RTDS) and their interactions (condition*day). Denominator df corrected with Satterthwaite approximation and reported to the nearest whole number (F value with degrees of freedom ($_{df}$) displayed as subscript), and significance (p_{value}) values were presented.

NS3 ($p=0.008$).

Sleep data

Subjective sleepiness (KSS)

There was a significant effect of day ($p<0.001$), such that sleepiness was significantly worse on NS1 ($p<0.001$) compared to NS2–4 (Fig. 6). There was a significant main effect of time ($p<0.001$), such that sleepiness was significantly worse at 0400 h ($p<0.001$) (Table 2).

Sleep variables

There was no significant effect of condition for any of the sleep variables (Table 3). There was a main effect of day for TST, Stage 1, Stage 2, Stage 3 and Stage 4 ($p<0.05$), such that subjects had significantly less sleep during the day than at night. There was no significant condition*day interaction effect for any of the sleep variables.

Discussion

This study examined the effect of eating vs not eating during the nightshift on subjective sleepiness, vigilant attention, processing speed, hunger and gut reaction in a small, all male sample. Eating a meal at 0130 h resulted in poorer vigilant attention at 0400 h compared to not eating a meal at night. Subjective sleepiness was significantly greater at 0400 h compared to other time points, independent of meal condition. The number of DSST correct responses increased across night shifts which was consistent with anticipated learning effects seen in previous studies[53]. While restricting food intake limited performance impairments at night, subjects that did not eat at 0130 h reported increased hunger and stomach upset, although stomach upset did not rise above neutral on the Likert-type scale. The eating at night group reported elevated bloating and gassiness on the first night-shift, becoming less apparent across days. Even though both groups had increased energy content on NS1 when transitioning to the night-shift and extended wake time on NS1, this may have caused exacerbated gastric complaints when eating at night[39].

Regardless of meal timing, the largest impairments in subjective sleepiness, vigilant attention, and information processing were seen at 0400 h. This is consistent with a large number of previous studies showing decreased PVT performance at night, via increased errors of omission (lapses: when the subject fails to respond) and errors of commission (errors: when the subject responds prior to a stimulus appearing)[54–57]. The impaired performance at night has largely been reported as a reflection of the circadian low between 0200 h and 0600 h, when the body is primed for sleep[14]. However, when considering these findings it is important to note that, the previous studies assessing performance provided meals, whether ad libitum or structured, over the night time period and did not address the possible impact of food intake.

This study was the first study to address the impact of food intake on vigilant attention and information processing during the night. In this study we found that restricting food intake to only the daytime hours reduced the number of errors and lapses seen on the PVT. This is in agreement with previously reported findings from this study, in which driving performance (lane variability, time spent in the safe

zone, and number of crashes) was significantly impaired by eating at night but not when eating was restricted to only the daytime hours[42]. Previous literature has shown that there is increased circulating cortisol, decreased core body temperature and reduced glucose tolerance at night[13, 58, 59]. Changes in circulating cortisol, core body temperature and glucose tolerance have also been associated with poor performance outcomes. In addition, a number of key digestive functions, such as the rate of gastric emptying, colonic motility, intestinal absorption rates and enzymatic activities[60] are disturbed at night, although the effect of these changes on cognitive performance have not been tested. Further research is needed to determine the mechanisms involved in decreased postprandial performance at night.

Subjects reported increased sleepiness during the first nightshift compared to the following night shifts. This may be a reflection of the extended wake when transitioning from dayshift to nightshift. In this study we found subjective sleepiness was not affected by eating at night above the effects of remaining awake at night, with subjects in both conditions reporting increased sleepiness at 0400 h. This is in contrast to previous studies which have commonly reported enhanced subjective sleepiness following consumption of meals high in carbohydrate or fats[61–63]. However, each of these studies were conducted during the day, which may suggest the time of day (0400 h) effect on sleepiness may overwhelm any additional effect of food intake.

Subjects that did not eat at night reported feeling increased hunger and stomach upset during the night, although the mean rating for stomach upset did not rise above neutral on the Likert-type scale. Previous studies report mixed results regarding appetite during the night shift. Lowden, *et al.*[64] suggests hunger decreases at night during 24 h of constant conditions. Interestingly this study also found time of eating was more important than macronutrient composition (fat/carbohydrates) in determining hunger, although macronutrient content was not addressed in the current study. Decreased hunger at night was supported by a further study which suggested individuals lose their desire to eat when gut function is at its lowest (during the night)[65]. However, shortened sleep has been associated with elevated appetite and hunger due to significant reductions in the release of leptin and increases in ghrelin[66]. These hormones were not assessed in the current study, future studies should explore how these hormones change when timing of food intake is altered. It may also be that other factors such as habit and time pressure may play a part in food consumption during the night shift rather than hunger alone[67, 68]. While this study shows that performance may be better, they may report increased hunger, and stomach upset and bloating (although they did not rise above neutral) when food intake is restricted at night. It is important that future suggestions need to be balanced in the context of subjective symptoms and therefore future work needs to be done to determine whether different variations of food intake i.e. smaller snacks or differences in composition can be used as an intermediate, to reduce hunger, during the night shift.

This study has shown, in a controlled laboratory environment with healthy participants, that limiting food intake to the daytime hours may be an effective strategy to improve some aspects of performance. It is important to note this study only included healthy young men, females and older or shift working populations may be impacted differently. Sample sizes similar to those in this study are common in intensive sleep study protocols of this nature. Effect sizes have been added to assist in the interpretation of the data given the sample size, and are shown in Table 2. Previous findings relating to this study found that restricting food intake at night limited impaired glucose metabolism associated with night shift[41]. Together, these findings suggest the importance of food timing when discussing the impact of shift work on health and performance. Future studies are needed to address the most suitable balance between food intake, performance, mood, and health. These studies will need to address the varying effects of different meal times, meal content and meal size. Furthermore, although the subjects in this study were sleep restricted (6 h TIB), to simulate sleep restriction observed during shift work, they slept well during the day due to the ideal sleeping environment in the laboratory. Studies have shown that daytime sleep quality and quantity could be reduced due to social and environmental factors including family commitments, light, noise, caffeine and alcohol consumption in the home environment[69, 70].

The results of this study may be most generalisable in occupations such as air traffic control, train and truck driving where the majority of the workload is cognitive. Future studies should look at the relationship between food consumption at night and performance when there is an increase in physical demand. Additionally, future research is needed to assess the impact of macronutrient contribution and portion size on performance at night. Previous studies examining the effect of food consumption on performance during the day have shown adverse performance outcomes are more likely following foods with a high fat/carbohydrate content[71–73]. Furthermore, a study by Lloyd,

Green and Rogers[72], suggested that both low fat and high fat meals consumed during the day led to a slower reaction time, compared to a medium fat meal. However, the medium fat meal was closest in size and macronutrient content to the subject's habitual diet and therefore the change in response time may be a reflection of the disruption in normal intake rather than due to macronutrient content[74]. This suggests that macronutrient contribution and habitual food intake should also be considered in future studies used to determine dietary recommendations for shift workers.

Conclusions

The findings from this paper suggest eating at night leads to increased errors and delayed response time, leading to an increase in number of lapses. While redistributing food intake to the daytime hours showed some improvements in performance, subjects reported increased hunger and changes in gastric complaints. Therefore, to optimize performance during the night when performance is known to dip, night shift workers could use altered meal timing as an effective countermeasure to improve performance, however further research is needed, particularly into nightshift snacking options, in order to provide recommendations that are for the performance, safety and subjective wellbeing of night shift workers.

Acknowledgements

The authors would like to thank the research staff and students for their contributions. In particular, research assistants; Stephanie Centofanti, Emily Watson, Cassie Hilditch, Alex Agostini, Alex Chatburn, nursing staff; Katja Morsky and student; Kenji Sison. Thank you also to all the participants.

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