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Impacts of shift work on sleep and circadian rhythms

Les impacts du travail posté sur le sommeil et les rythmes circadiens

D.B. Boivin *, P. Boudreau

Department of Psychiatry, Centre for Study and Treatment of Circadian Rhythms, Douglas Mental Health University Institute, McGill University, 6875, LaSalle Boulevard, 1115, H4H 1R3 Montreal, QC, Canada

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ABSTRACT

Shift work comprises work schedules that extend beyond the typical "nine-to-five" workday, wherein schedules often comprise early work start, compressed work weeks with 12-hour shifts, and night work. According to recent American and European surveys, between 15 and 30% of adult workers are engaged in some type of shift work, with 19% of the European population reportedly working at least 2 hours between 22:00 and 05:00. The 2005 International Classification of Sleep Disorders estimates that a shift work sleep disorder can be found in 2–5% of workers. This disorder is characterized by excessive sleepiness and/or sleep disruption for at least one month in relation with the atypical work schedule. Individual tolerance to shift work remains a complex problem that is affected by the number of consecutive work hours and shifts, the rest periods, and the predictability of work schedules. Sleepiness usually occurs during night shifts and is maximal at the end of the night. Impaired vigilance and performance occur around times of increased sleepiness and can seriously compromise workers' health and safety. Indeed, workers suffering from a shift work sleep-wake disorder can fall asleep involuntarily at work or while driving back home after a night shift. Working on atypical shifts has important socioeconomic impacts as it leads to an increased risk of accidents, workers' impairment and danger to public safety, especially at night. The aim of the present review is to review the circadian and sleep-wake disturbances associated with shift work as well as their medical impacts.

Mots clés :
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RÉSUMÉ

Le travail posté exige des périodes de travail qui sortent du cadre conventionnel des journées "de neuf à cinq". Les horaires de travail comportent souvent un début de travail précoce, des horaires comprimés avec quarts de 12 heures ainsi que des périodes de travail de nuit. Des enquêtes récentes américaine et européenne rapportent qu'entre 15 et 30 % des travailleurs adultes travaillent sur un horaire non conventionnel, incluant 19 % de la population européenne qui rapporte travailler au moins 2 heures entre 22:00 et 05:00. La classification internationale des troubles de sommeil de 2005 estime qu'un désordre de sommeil lié au travail posté peut être observé chez 2–5 % des travailleurs. Ce désordre se caractérise par de la somnolence excessive et/ou des perturbations du sommeil pendant au moins un mois en relation avec l'horaire atypique de travail. La tolérance d'un individu au travail posté demeure un problème complexe qui dépend du nombre d'heures et de quarts consécutifs de travail, des périodes de repos et de l'aspect prévisible de l'horaire de travail. La somnolence survient généralement la nuit et est maximale en fin de nuit. Les perturbations de vigilance et de performance surviennent lors des périodes de somnolence accrue et peuvent compromettre sérieusement la santé et sécurité des travailleurs. En effet, les travailleurs souffrant du désordre de sommeil lié au travail posté peuvent s'endormir involontairement au travail et en conduisant lors du retour à domicile après un quart de nuit. Le travail posté a d'importantes implications socioéconomiques en raison du risque accru d'accidents, de

* Corresponding author.
E-mail address: diane.boivin@douglas.mcgill.ca (D.B. Boivin).

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l'altération des capacités des travailleurs et des dangers de sécurité publique qu'il comporte, en particulier la nuit. Le but du présent ouvrage est de revoir les troubles des rythmes circadiens et du cycle veille-sommeil liés au travail posté ainsi que leurs implications médicales.

1. Circadian rhythms

Near 24-hour rhythmicity regulates human physiology and behavior, and was observed in most parameters studied, such as hormone secretion [1], sleep propensity and architecture [2], and subjective and electroencephalographic (EEG)-estimated alertness [3]. The endogenous circadian nature of these rhythms is demonstrated by their persistence in the absence of external time cues and their disappearance after ablation of the suprachiasmatic nucleus (SCN) of the anterior hypothalamus, considered the necessary master component of endogenous circadian rhythms [4].

In a subject living on a conventional day-oriented schedule, melatonin secretion starts in the evening, levels peak in the middle of the night, and slowly decline thereafter to reach their lowest levels at the end of the morning (Fig. 1). Cortisol levels also vary throughout the day. They peak at the habitual wake time and reach their nadir in the first hours of the nocturnal sleep episode. Core body temperature (CBT) varies across the circadian day. Its circadian variation is such that it reaches its nadir 1–2 hours before the regular wake time and reaches its crest 1–2 hours before the regular bedtime. Circadian phase of these rhythms is usually assessed with harmonic regression models in order to find the best fit for melatonin and cortisol peak levels, and CBT minimum. Melatonin phase can also be assessed by the so-called dim light melatonin onset (DLMO) in the evening [5].

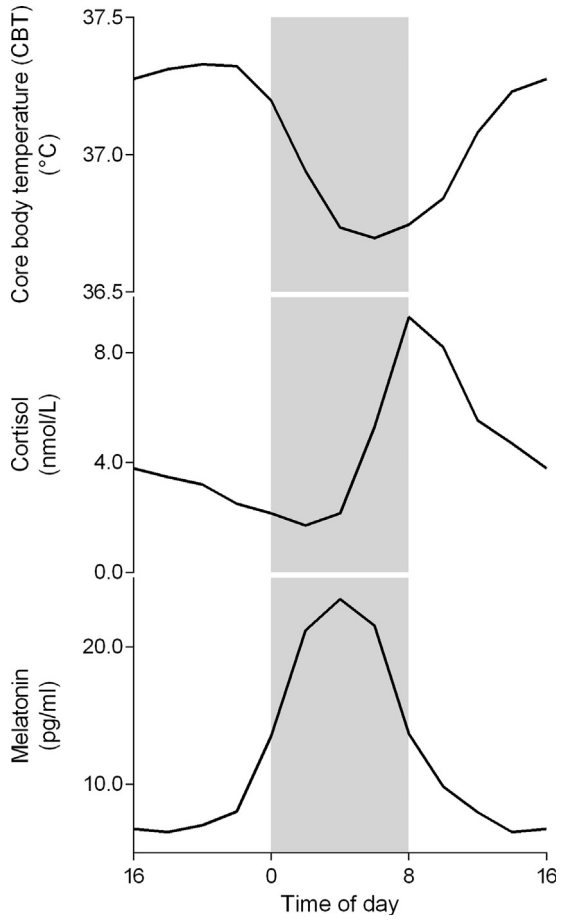

Fig. 1. Rhythms of core body temperature (CBT), cortisol and melatonin secretion in humans illustrated relative to a nocturnal sleep period (shaded area). The illustration is based on the author's data.

A set of clock genes, first identified in the SCN and later on in most tissues studied, are involved in negative and positive regulatory intracellular feedback loops, and are responsible for the generation of circadian rhythms [6]. Besides the SCN which is considered the main circadian pacemaker in mammals, cycling of clock genes were documented in various human tissues such as skin, adipose tissue, oral mucosa, peripheral blood mononuclear cells (PBMCs), bone marrow, colon cells, hair follicles, and non-SCN brain region. Oscillations generated by clock genes are thought to be transmitted to the expression of clock-controlled genes that ultimately leads to rhythms in tissue function.

In the absence of external time cues, circadian rhythms oscillate with an intrinsic period slightly different from 24 hours [7]. This endogenous period is entrained to that of the environment through daily exposure to external synchronizers, the most powerful being the light-dark cycle. The light information necessary to entrain circadian rhythms is detected by a population of specialized retinal ganglion cells containing melanopsin [8], although retinal rods and cones also play a role. Retinal light exposure is then transmitted to the SCNs via the retinohypothalamic tract, a direct and powerful monosynaptic pathway.

The light exposure schedule, its intensity, spectral composition, and the prior history of light exposure influence the size and direction of circadian phase shifts induced by light. In humans, exposure to bright light in the early morning advances circadian rhythms earlier. In contrast, exposure to bright light early at night delays these rhythms [7]. The pattern of light exposure can be planned to rapidly reset the central circadian pacemaker to earlier or later phases [9]. The central circadian clock is especially sensitive to light in the 440–480 nm spectrum such that shorter wavelength light (e.g. in the blue visible light range) can more efficiently shift the phase of the temperature and melatonin rhythms than light of longer wavelengths [10]. At lower irradiances, a switch of sensitivity towards light in the green visible spectrum of 555 nm was reported [11].

Exogenous melatonin can exert a resetting effect opposite to that of light, with evening and morning administration advancing and delaying circadian rhythms, respectively [12]. The effects of other non-photic synchronizers such as exercise, social interactions, and meal timing are less well characterized and require further experimental testing.

2. Circadian variation of sleep and waking

Sleep-wake behaviors have been shown to be regulated by a complex interaction of two processes called the homeostatic or S process, and the circadian or C process [13,14]. The homeostatic process corresponds to the build-up of sleep pressure throughout a wake period and its reduction throughout a sleep period. The S process is quantified by the amount of slow wave sleep (SWS) and slow wave activity (SWA) during non-rapid-eye movement (REM)