HHS Public Access

Author manuscript

Sleep Med. Author manuscript; available in PMC 2021 March 01.

Published in final edited form as:

Sleep Med. 2020 March ; 67: 66–70. doi:10.1016/j.sleep.2019.11.1248.

Impaired Neurobehavioral Alertness Quantified by the Psychomotor Vigilance Task is Associated with Depression in the Wisconsin Sleep Cohort Study

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Abstract

Background: Excessive daytime sleepiness plays an important role in the presentation and course of mood disorders. Standard objective measures of daytime sleep propensity are of little to no value in depressive illness. This study examined the psychomotor vigilance task (PVT), an objective measure of neurobehavioral alertness, and its cross-sectional and longitudinal associations with depressive symptomatology in the Wisconsin Sleep Cohort Study.

Methods: The sample consisted of 1569 separate 10-minute PVT assessments conducted in 942 unique individuals. Cross-sectional and longitudinal conditional logistic regression models were used to estimate associations between the primary outcome of depression symptomatology (adjusted Zung scale≥50) and six separate PVT variables: mean reciprocal reaction time (1/RT); total lapses (RTs≥500msec; LAPSE); total false responses (FALSE); reciprocal of the mean of the 10% fastest (FAST) and 10% slowest (SLOW) RTs; and slope of the linear regression line for all transformed 1/RTs (SLOPE).

Results: In fully-adjusted cross-sectional models, 1/RT, LAPSE, FAST, and SLOW were each significantly associated with depression, such that worse neurobehavioral alertness was associated with higher odds of depressive symptomatology. Similar, though attenuated, findings were observed in fully-adjusted conditional longitudinal models that examined within-subject changes in depression status in the subset of participants with repeated PVT assessments. FALSE and SLOPE were not associated with depression in either cross-sectional or conditional longitudinal models.

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DISCLOSURES

Dr. Plante has served as consultant and medical advisory board for Jazz Pharmaceuticals and Teva Australia. All other authors declare no conflicts of interest.

Conclusions: These findings suggest components of the PVT are associated with depressive symptomatology. Further research is indicated to clarify the role of the PVT in the assessment of hypersomnolence in mood disorders.

Keywords

depression; psychomotor vigilance task; cohort; sleepiness; hypersomnolence

1. INTRODUCTION

Excessive daytime sleepiness is a common and impairing symptom in persons with psychiatric disorders, particularly mood disorders[1–3]. Daytime sleepiness has been associated with treatment resistance, symptomatic relapse, increased risk of suicide, and functional impairment, highlighting the importance of somnolence in the course of affective illness[1,4–10]. In addition, the presence or absence of excessive sleepiness impacts diagnostic evaluation and choice of therapeutic strategy in depression[11–13]. Thus, daytime somnolence is a critical area in mood disorders research.

Despite the important cross-sectional and longitudinal role of daytime sleepiness in depressive illness[10,14–17], objective measures of daytime sleepiness in affective illness are lacking. The multiple sleep latency test (MSLT), the gold standard measure of daytime sleep propensity, has proven to be of limited value in quantifying hypersomnolence occurring in the context of depressive illness[18]. In fact, divergent associations between MSLT-derived sleep latency and subjective measures of somnolence have been previously described in depression[10,14]. Thus, there is a vital need to identify objective measures of daytime somnolence that can be applied in the research and clinical care of persons with mood disorders.

Complicating research in this area is the fact that hypersomnolence is a multifaceted problem for which no singular objective measure fully quantifies the subjective complaint[19]. For example, the two primary clinical electroencephalographic measures commonly employed to quantify excessive daytime sleepiness, the MSLT and maintenance of wakefulness test (MWT), capture the ability to fall asleep and stay awake under soporific conditions, respectively. Although both statistically correlate with excessive sleepiness complaints in large data sets, it is noteworthy that MSLT and MWT results only marginally explain the variance of one another, and MSLT and MWT results are frequently discordant within an individual patient, including persons with depression[20,21]. In addition, other objective measures that significantly correlate with subjective complaints of excessive daytime sleepiness, such as the psychomotor vigilance task (PVT), a measure of neurobehavioral alertness, do not or only marginally correlate with the MSLT[22–24]. Thus, it is highly plausible that specific aspects of daytime sleepiness, such as impaired psychomotor vigilance, may be more relevant to depressive illness than EEG-based measures of sleep propensity.

There are limited data that have examined relationships between PVT performance and depression. Yun and colleagues[25] recently demonstrated a significant cross-sectional association between PVT performance (number of lapses and reaction time) and depressive

symptoms in the Korean Genome and Epidemiology Study. However, this study was not able to precisely control for presence or severity of sleep disordered breathing, which can affect both PVT performance and depressive symptoms[24,26], and participants with psychiatric illness were excluded from analyses[25]. Thus, the current study sought to assess associations between depression and PVT performance in the Wisconsin Sleep Cohort (WSC) study to rigorously examine associations between impaired neurobehavioral vigilance and depression scores. We hypothesized that poorer performance on the PVT would be associated with depressive symptomatology both cross-sectionally and longitudinally.

2. MATERIALS AND METHODS

2.1. Participants

Data were derived from individuals participating Wisconsin Sleep Cohort (WSC) Study, the methodology of which is detailed elsewhere [26–28]. Briefly, beginning in 1988, state workers from Wisconsin underwent baseline overnight polysomnography and were invited for longitudinal follow-up every 4 years. All participants provided written informed consent. The University of Wisconsin-Madison Health Sciences Institutional Review Board has approved all WSC procedures. During the course of the study, the psychomotor vigilance task (PVT; described below) was administered during select follow-up waves. For these analyses, a total of 1,569 PVT measures were available from 942 unique WSC study participants.

2.2. Psychomotor Vigilance Task

The psychomotor vigilance task (PVT) is a well-validated measure of neurobehavioral alertness used in sleep research. The PVT was originally developed as a neurocognitive assay to quantify the response to sleep loss, measuring the ability to sustain attention and promptly respond to salient signals[29]. The PVT is a task that requires responses to a stimulus (digital counter) by pressing a button as soon as the stimulus appears, which stops the stimulus counter and displays the reaction time in milliseconds for a 1-second period. The subject is instructed to press the button as soon as the stimulus appears, to keep reaction time as minimal as possible, and not to press the button too soon. The PVT meets the criteria for an accurate measure of neurobehavioral performance as it is indicative of a fundamental aspect of waking cognitive function, easily performed and administered, minimally affected by learning/aptitude, brief, valid and reliable, sensitive, and able to provide meaningful outcome variables for interpretation (reviewed in[30]). In the WSC study, PVT trials were conducted in two separate (10 minute) trials, occurring at approximately 09:00–10:00 and 13:00–14:00. Data from both time points were averaged for analysis.

Six separate PVT variables were utilized: mean reciprocal reaction time (1/RT); total lapses (RTs≥500msec; LAPSE); total false responses (FALSE); reciprocal of the mean of the 10% fastest (FAST) and 10% slowest (SLOW) RTs; and slope of the linear regression line for all transformed 1/RTs across the 10 minute task (SLOPE).

2. 3. Outcome: Depression

Participants completed the Zung Self-Rating Depression scale [31,32]. Scores on this instrument range from 25 to 100, with scores 50 to 59 categorized as mild depressive symptoms, and scores ≥60 categorized as moderate or worse depressive symptoms. For these analyses, two items related to sleep ("I have trouble sleeping through the night" and "I get tired for no reason") were removed to avoid the possibility of a built-in association between depressive and sleep-related symptoms. The modified Zung score was rescaled to have a range that is consistent with the original scale (25–100). A score of 50 or greater on the modified Zung scale defined depression for these analyses.

2. 4. Statistics

Two categories of statistical modeling were used. First, cross-sectional associations between PVT variables and depression were evaluated using repeated measures logistic regression. Repeated measures logistic regression allows for the efficient use of multiple studies per person by adjusting for within-subject correlation of observations and computing robust standard errors for hypothesis testing. Second, conditional (intrasubject) logistic regression was employed to estimate the increased likelihood of development of depression associated with PVT variables of interest. Unlike cross-sectional analyses, these conditional models analyzed data from individuals that transitioned from depressed to not depressed status or vice versa. These "self-as-control" models implicitly account for fixed within-person characteristics such as genetics and sex. Additionally, the conditional models accommodate participants who meet criteria for depression at baseline but later change depression status, who would otherwise be excluded from standard longitudinal modeling approaches [33].

All models included age, sex, body mass index (BMI; kg/m^2), self-reported habitual sleep duration, chronic medical disorders (coronary artery disease, congestive heart failure, angina, hypertension, cerebrovascular accident, diabetes, asthma, emphysema, thyroid disease, epilepsy, arthritis, or back pain), tobacco use (current, past, or never), alcohol consumption (≥2 standard drinks of beer, wine, or hard liquor per day versus less), caffeine consumption (0, 1–2, 3–4, or >5 caffeinated beverages per day), sedative hypnotic medication use, antidepressant medication use (selective serotonin reuptake inhibitors, tricyclic antidepressants, and monoamine oxidase inhibitors), and apnea-hypopnea index [AHI, derived from in-laboratory polysomnography; absent (AHI < 5), mild (AHI 5 ≤ 15), or moderate or worse (AHI ≥ 15)], as potential confounders.

Self-report measures of insomnia were completed by WSC participants beginning partway through collection of PVT data. Thus, insomnia was not included as a covariate in primary analyses. However, given established relationships between insomnia and depression[34], effects of insomnia (reporting at least one of four insomnia complaints often or very often: difficulty falling asleep, waking repeatedly, waking too early in the morning, waking at night and inability to go back to sleep) on statistical models were also examined on an exploratory basis.

Additional sensitivity analyses included 1) analysis limited to one PVT observation per individual participant and 2) analysis that considered morning and afternoon PVT measurements separately, rather than using the average of these two time points.

Data were analyzed using SAS software (SAS Institute Inc., Cary, NC), with α=0.05 (two sided) for statistical significance.

3. RESULTS

Descriptive characteristics of the sample are found in Table 1. The full dataset consisted of 1569 separate PVT assessments (average of morning and afternoon time points) conducted in 942 unique individuals. On average PVTs were conducted in participants who were middle aged, had body mass indices in the obese range, and had relatively high proportion of moderate or worse obstructive sleep apnea.

Cross-sectional results (repeated measures logistic regression) are presented in Table 2. In fully adjusted models, 1/RT, LAPSE, FAST, and SLOW were each significantly associated with depression status. For each of these variables, worse performance on the PVT measure (e.g. longer reaction time and more lapses) was associated with higher odds of depression. FALSE and SLOPE were not associated with depression status in cross-sectional models. Sensitivity analyses that were conducted using only data available from 1,321 PVT observations with concordant insomnia data demonstrated very similar results to the full dataset (Table 3). Additionally, when analyses were conducted limited to only one PVT observation per participant (N=942), findings were very similar to repeated measures analyses that utilized all available data (Tables S1 and S2; Supplementary Material). Finally, findings were also very similar when the morning and afternoon PVT assessments were considered as separate PVT observations and analyzed discretely rather than averaged (Tables S3 and S4; Supplementary Material).

Results of conditional (intrasubject) logistic regression models are presented in Table 4. There were 78 participants who changed depression status and were available for these longitudinal analyses. In fully adjusted models, 1/RT, FAST, and SLOW were each significantly associated with a change in depression status, such that worse neurobehavioral performance (e.g. longer reaction times) were associated with higher odds of changing from not depressed to depressed, or vice versa (i.e., improved neurobehavioral performance was also associated with increased odds of changing from depressed to not depressed). A non-significant trend for LAPSE was similarly observed, with a tendency for increased lapses to be associated with change from not depressed to depressed, and vice versa.

4. DISCUSSION

This investigation demonstrates that impaired neurobehavioral performance on the psychomotor vigilance task is associated with depressive symptomatology in the Wisconsin Sleep Cohort study. Specifically, in cross-sectional regression models, slower reaction times and lapses on the PVT were associated with higher odds of depression (defined as Zung scale score $\geq$ 50). Similar, though attenuated, results were observed in conditional longitudinal models, such that the development of depression in those previously not

depressed was associated with worse performance on the PVT (and vice versa). These findings have important implications for the study of hypersomnolence in psychiatric disorders.

As previously described, the multiple sleep latency test has proven to be of little value in identifying hypersomnolence occurring in the course of psychiatric disorders[10,14,18]. Other investigative teams have similarly described limitations of the MSLT in identifying sleepiness in other non-cataplectic disorders of central hypersomnolence[35–37]. In the case of hypersomnolence associated with mood disorders, a non-pathologic mean sleep latency on the MSLT in an individual with co-occurring complaints of daytime sleepiness and depression is typically interpreted as an absence of 'actual' sleepiness. The results of this investigation suggest that other aspects of somnolence, such as impaired neurobehavioral vigilance, may be more relevant to the study of mood disorders than the MSLT.

While our results suggest that significant depressive symptomatology is associated with worse performance on the psychomotor vigilance task across participants in the WSC sample, these results cannot be interpreted to mean that all persons with psychiatric hypersomnolence will demonstrate impairment on the PVT. Given the heterogenous nature of depressive illness[38], it is highly likely that there are cases in which depression occurs without concurrent impairment on the PVT, both with and without complaints of excessive daytime sleepiness. However, our findings do offer a possible explanation of some potential changes in brain function that may occur in persons with symptoms of hypersomnolence and depression.

Various response elements on the PVT have been previously mapped to specific brain areas/circuits, many of which may be relevant to depressive symptomatology[39]. Greater changes in blood-oxygen level dependent (BOLD) responses have been described during fast relative to slow reaction times in cortical sustained attention networks as well as cortical and subcortical motor systems, while increases in BOLD responses map to frontal and posterior midline structures that are constituents of the default mode network[39]. Notably, all of these brain networks/regions have been associated with major depression[40,41], and may reflect specific areas of impairment that are observed in the disorder. Because the PVT is a readily employed measure of neurobehavioral performance for which different response elements map to different brain circuits/structures, its future application to the study of excessive sleepiness in mood disorders may help clarify the specific brain structures/circuits that are related to complaints of hypersomnolence in these patients. Moreover, the results of our conditional longitudinal models, in which changes in PVT variables were associated with change in depression status, suggest the possibility that PVT variables could also serve as an objective measure of improvement, which would be a significant advance in mood disorders research given the relative dearth of objective measures used in depression research. Thus, future investigations that more fully clarify the role of the PVT in psychiatric hypersomnolence are clearly indicated.

There are limitations of this investigation that merit consideration. First, consistent with the epidemiologic sample utilized in analyses, the outcome of depression was determined using a validated scale rather than clinical interview, which is generally considered the gold

standard for psychiatric diagnosis. Second, while findings of the cross-sectional and longitudinal models demonstrate similar patterns of association between PVT variables and the outcome of depression (defined as Zung score ≥ 50), longitudinal models used a smaller sample size that likely resulted in both wider confidence intervals for estimated odds ratios for all PVT measures and only a trend level association between PVT lapses and depression. Third, because the study population primarily consisted of adults in middle age, results cannot be extended to other age groups such children/adolescents or the elderly. Third, although the PVT is a measure of neurobehavioral alertness that is associated with subjective complaints of sleepiness, the PVT may also index other dimensions such as psychomotor slowing, attention, focus, and engagement. Therefore, associations between PVT reaction time and lapses with depression may be related, at least in part, to these other factors. Finally, despite controlling for various factors that could influence results, it is possible that unmeasured and/or unadjusted covariates may have affected findings. However, the ability to control for both the presence and severity of sleep apnea using gold-standard polysomnography, as well as several other potentially impactful covariates, limits the likelihood that inclusion of other covariates would meaningfully alter our results.

5. CONCLUSIONS

In summary, this investigation demonstrates both cross-sectional and longitudinal associations between worse performance on the psychomotor vigilance task and depression. These findings suggest that the PVT, a measure of neurobehavioral alertness, may be a more useful objective tool to apply to the study of psychiatric hypersomnolence than other standard measures. Future research that more fully dissects the relationships between PVT response variables, subjective complaints of sleepiness, and brain function in psychiatric hypersomnolence are indicated to advance this area of inquiry.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGEMENTS

This work was supported by the National Heart, Lung, and Blood Institute (R01HL62252) and the National Center for Research Resources (1UL1RR025011) at the National Institutes of Health. Dr. Plante has also been supported by grants from the American Sleep Medicine Foundation, the Brain and Behavior Research Foundation, NIMH, NIA, NINR, the Madison Educational Partnership, and the University of Illinois at Chicago Occupational and Environmental Health and Safety Education and Research Center (funded by the National Institute for Occupational Safety and Health).

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HIGHLIGHTS

- Excessive daytime sleepiness plays a key role in the course of depression.
- No objective hypersomnolence measure is established as useful in mood disorders.
- The psychomotor vigilance task (PVT) is a measure of neurobehavioral alertness.
- Findings demonstrate PVT components are significantly associated with depression.
- The PVT may be a salient objective measure of hypersomnolence in depression.

Table 1.

Descriptive characteristics of the sample, n=1569 studies on 942 individuals.

Characteristic	Total
N (%)	1569
Male Sex, n (%)	869 (55)
Age in years, mean (SD)	58 (9)
BMI in kg/m^2, mean (SD)	32 (7)
Chronic conditions, n (%)	982 (63)
Current Smoker, n (%)	167 (10)
14+ Alcoholic drinks per week, n (%)	85 (5)
Sleep-disordered breathing as AHI ≥15, n (%)	543 (35)
5 + Caffeinated drinks, n (%)	286 (18)
Insomnia**, %	601 (46)
Habitual Sleep Time, mean hours (SD)	7.23 (0.98)
Depression*, %	191 (12)
Antidepressant use, n (%)	285 (18)
Fast, mean (SD)	4.93 (0.49)
Slope, mean (SD)	−0.009 (0.037)
Slow, mean (SD)	2.65 (0.49)
1/RT, mean (SD)	3.95 (0.47)
Lapse, mean (SD)	1.58 (2.85)
False, mean (SD)	1.72 (3.20)

*Zung score≥50

**Waking repeatedly, Difficulty falling asleep, Waking too early, and Can't fall back to sleep often or very often (not available for 248 PVT observations).

Table 2.

Cross-sectional associations between PVT variables of interest and outcome of depression. Adjusted for age, sex, BMI, sedative-hypnotic medications, chronic conditions, alcohol, caffeine, AHI, smoking, and antidepressant medications.

PVT Variable	Beta	SE	p-value	OR	Lower CI	Upper CI
1/RT	−1.08	0.19	**<0.0001**	**0.34**	**0.23**	**0.50**
LAPSE	0.10	0.02	**<0.0001**	**1.11**	**1.07**	**1.15**
FALSE	−0.02	0.03	0.406	0.98	0.92	1.03
FAST	−0.84	0.20	**<0.0001**	**0.43**	**0.29**	**0.64**
SLOW	−0.89	0.16	**<0.0001**	**0.41**	**0.30**	**0.57**
SLOPE	−0.84	2.20	0.704	0.43	0.01	32.52

SE=standard error; OR=odds ratio; CI=95% confidence interval. Significant associations marked in bold.

Table 3.

Cross-sectional associations between PVT variables of interest and outcome of depression from 1,321 PVT observations with available insomnia data. Adjusted for age, sex, BMI, sedative-hypnotic medications, chronic conditions, alcohol, caffeine, AHI, smoking, antidepressant medications, and insomnia.

PVT Variable	Beta	SE	p-value	OR	Lower CI	Upper CI
1/RT	−1.36	0.22	**<0.0001**	**0.26**	**0.17**	**0.39**
LAPSE	0.13	0.03	**<0.0001**	**1.14**	**1.08**	**1.20**
FALSE	−0.04	0.04	0.328	0.97	0.90	1.04
FAST	−1.04	0.21	**<0.0001**	**0.35**	**0.23**	**0.54**
SLOW	−1.23	0.19	**<0.0001**	**0.29**	**0.20**	**0.43**
SLOPE	0.33	2.48	0.894	1.39	0.01	179.43

SE=standard error; OR=odds ratio; CI=95% confidence interval. Significant associations marked in bold.

Table 4.

Conditional (intrasubject) longitudinal associations between PVT variables of interest and outcome of depression. Adjusted for age, BMI, sedative-hypnotic medications, alcohol, caffeine, AHI, smoking, and antidepressant medications. Non-changing personal variables (i.e. sex) are inherently controlled for in the model.

PVT Variable	Beta	SE	p-value	OR	Lower CI	Upper CI
1/RT	−1.85	0.78	**0.017**	**0.16**	**0.03**	**0.72**
LAPSE	0.16	0.08	0.057	1.17	1.00	1.38
FALSE	−0.09	0.09	0.336	0.91	0.76	1.10
FAST	−1.74	0.78	**0.027**	**0.18**	**0.04**	**0.81**
SLOW	−1.32	0.61	**0.029**	**0.27**	**0.08**	**0.88**
SLOPE	−4.75	5.52	0.389	0.01	0.00	432.33