Original Paper

Exploring Remote Monitoring of Poststroke Mood With Digital Sensors by Assessment of Depression Phenotypes and Accelerometer Data in UK Biobank: Cross-Sectional Analysis

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Abstract

Background: Interest in using digital sensors to monitor patients with prior stroke for depression, a risk factor for poor outcomes, has grown rapidly; however, little is known about behavioral phenotypes related to future mood symptoms and if patients with and without previously diagnosed depression experience similar phenotypes.

Objective: This study aimed to assess the feasibility of using digital sensors to monitor mood in patients with prior stroke with a prestroke depression diagnosis (DD) and controls. We examined relationships between physical activity behaviors and self-reported depression frequency.

Methods: In the UK Biobank wearable accelerometer cohort, we retrospectively identified patients who had previously suffered a stroke (N=1603) and conducted cross-sectional analyses with those who completed a subsequent depression survey follow-up. Sensitivity analyses assessed a general population cohort excluding previous stroke participants and 2 incident cohorts: incident stroke (IS) and incident cerebrovascular disease (IC).

Results: In controls, the odds of being in a higher depressed mood frequency category decreased by 23% for each minute spent in moderate-to-vigorous physical activity (odds ratio 0.77, 95% CI 0.69-0.87; *P*<.001). This association persisted in both general cohorts and in the IC control cohort.

Conclusions: Although moderate-to-vigorous physical activity was linked with less frequent depressed mood in patients with prior stroke without DD, this finding did not persist in DDs. Thus, accelerometer-mood monitoring may provide clinically useful insights about future mood in patients with prior stroke without DDs. Considering the finding in the IC cohort and the lack of findings in the IS cohorts, accelerometer-mood monitoring may also be appropriately applied to observing broader cerebrovascular disease pathogenesis.

JMIR Neurotech 2025;4:e56679; doi: 10.2196/56679

Keywords: depression; cerebrovascular disease; remote monitoring; stroke; accelerometers; mobile phone

Introduction

Overview

Depression is an established risk factor for poor outcomes after a stroke and transient ischemic attack (TIA), including subsequent stroke and other cerebrovascular diseases (CeVDs) [1,2]. Although poststroke depression (PSD) affects roughly one-third of patients with stroke, screening for depression in patients after a stroke is not routine, with less than 10% of patients with stroke screened [3]. Furthermore, it remains unclear when follow-up PSD screening should occur, as current research suggests that not all patients will experience PSD symptoms immediately after a stroke and, for those who do, the majority will experience recurrent depression episodes in the years after a stroke [4]. A reason for this gap in screening is the shortage of neurologists, particularly those with diagnostic training in identifying PSD [5]. Accelerated by the widespread adoption of personal mobile devices, from computers to smartwatches, it is critical to investigate the potential of such devices to collect meaningful data outside of clinical settings, aiding clinicians in identifying depressed mood in patients with stroke—and, potentially, those most at risk for subsequent stroke and CeVDs [6].

Background

The prevalence of PSD remains unknown, partly due to its heterogeneous nature, spanning unique somatic, behavioral, cognitive, motivational, and emotional components [7]. The severity of its manifestation ranges from mild symptoms to clinical-grade depression, the former of which relies on self-reported scoring methods inherently subject to bias, especially in patients with cognitive impairment for whom self-reported surveys may not be reliable [8]. Although clinician-administered assessments, like the Montgomery–Åsberg Depression Rating Survey (MADRS), offer gold-standard assessments of symptoms, nurse and physician shortages complicate the routine administration of such instruments [9].

In some survivors, depression may emerge alongside the incipient pathogenesis of cerebrovascular dysfunction, while for others, depression may be a reaction to being conscious of cognitive impairment or the putative manifestation of silent cerebral infarcts [10,11]. As such, individual depression phenotypes may vary greatly across survivors with identical survey summary scores. Although investigations into the associations between stroke location within the brain and self-reported depression survey scores have yielded inconclusive results, a recent cross-sectional study of patients with prior stroke (n=200) found that symptoms assessed by MADRS correlate with specific macrostructural characteristics [12]. Considering that clinician-administered assessments, like MADRS, are more accurate than self-reported survey scores in patients with prior stroke, the need for a modified approach to monitoring patients with stroke for depression emerges.

In recent years, objective data from portable and wearable sensors have demonstrated the feasibility of augmenting self-reported mood surveys outside of clinics, a promising approach for monitoring patients with symptomatic and asymptomatic deteriorating brain health outside of standardized, clinical environments [13-20]. In addition, accelerometer measures of behavior have established a difference in PA engagement stratified by depression severity, highlighting the need for a thoughtful approach to PSD screening and monitoring that ensures patients with emerging or mild depression symptoms, unlike those with previous documented depressive episodes, are not neglected [21].

While triaging patients with PSD for preventative intervention could yield clinically meaningful functional recovery outcomes, the potential of such an approach for preventing future CeVD diagnoses remains to be seen. Numerous studies have found that depressive symptoms are associated with an increased risk of subsequent CeVD, from acute CeVDs, like stroke and TIA, to more chronic conditions, like cerebral arterial stenosis and vascular dementia [11,22-24]. Furthermore, recent research suggests daily functioning and cognitive changes may be observable up to 10 years before some types of CeVD [25]. Thus, particular attention should be paid to behavioral patterns in patients with PSD to elucidate phenotypes with predictive potential for functional outcomes and neurologic disorders.

Previous Work

Blending self-reported assessments of phenomena, like mood, recorded through web browsers and smartphone apps, with passive sensor data, like that from wearable accelerometers, is gaining popularity in real-world settings [26,27]. Numerous pilot studies have demonstrated the potential for wearable and minimally invasive sensors to detect neurologic conditions; however, these tools have neither been validated in population cohorts nor combined with survey sampling of mood [28].

Early-stage evidence suggests that monitoring lifestyle behavior and mood in PSD is feasible [29-31]. The results of a small longitudinal study (n=40) suggest that self-reported moderate-to-vigorous physical activity (MVPA) before stroke is associated with improved mobility and self-care as well as decreased discomfort after stroke [32]. While the study did not sample mood outside of clinical environments, Reinholdsson et al [33] used self-report surveys to expand on the above findings, demonstrating that patients who engage in higher levels of prestroke physical activity (PA) experienced less severe PSD compared with patients who were physically inactive .

In addition, current literature on accelerometers in PSD suggests that distinct behavioral patterns may identify patients with depression within the first year after a stroke. In a 2022 prospective observational study of recently discharged patients with minor ischemic stroke (n=76), participants wore accelerometers in-hospital for 1 week. Analyses revealed that only increased sedentary behavior (SB) and reduced light physical activity (LPA) were linked with more intense depression, assessed through a written Geriatric Depression

Scale survey, 3 months after hospitalization in this older adult cohort [34]. In a small pilot study (n=40) of stroke survivors, MVPA was linked with positive mood [35]. Although extensive research has confirmed links between sleep disorders and both depression and incident CeVD (IC), no research has observed both depressive symptoms and objectively measured sleep after stroke [36,37]. Furthermore, no previous accelerometer research into PSD beyond the first year of stroke recovery has been published.

Goal of This Study

The goal of this study is twofold: first, to investigate potential associations between objectively measured behavior and future depression frequency in patients with prior stroke assessed by a remote approach and second, to explore whether that association varies between patients with prior stroke with a prestroke depression diagnosis (DDs) and those without (controls).

We conducted a cross-sectional analysis with the UK Biobank (UKBB), the most extensive lifestyle and mood cohort to date, assessing the relationships between accelerometer-measured sleep, SB, LPA, and MVPA and a subsequent depression descriptor (depressed mood frequency). Given that depression before stroke may yield behavioral phenotypes distinct from those emergent in participants without a prestroke depression diagnosis, we created 2 cohorts of patients with prior stroke: those with a clinical depression diagnosis before stroke and those without. As this analysis focuses on participants who may develop or have undiagnosed PSD, participants whose PSD diagnosis was recorded were excluded. Adjusting for age, sex, ethnicity, multiple relevant comorbidities, and time elapsed between accelerometer monitoring and depression survey submission, we hypothesized that increased LPA and MVPA time would be associated with a reduction in the odds of being in a more frequent depressed mood category while increased SB time would be associated with a rise in the odds of being in a more frequent depressed mood category. Considering the established relationship between sleep and depressed mood, we created a binary variable (yes or no) for guideline-recommended sleep (7-9 h/d). We hypothesized that guideline-recommended sleep would be associated with a reduction in the odds of being in a more frequent depressed mood category.

Methods

Recruitment

The UKBB enrolled middle-aged (40-69 y) participants (N=502,364) at 22 assessment centers across the United Kingdom at a baseline assessment (2006-2010), which included in-person interviews, touchscreen surveys, and physical examinations to extract lifestyle and environmental data used in this study. Although all baseline participants (n=502,151) were invited, only 72,652 enrolled in the 1-week accelerometer study (2013-2015) and completed the depression frequency survey (2016-2017). Hospital and other diagnostic registries were linked to enrolled participants.

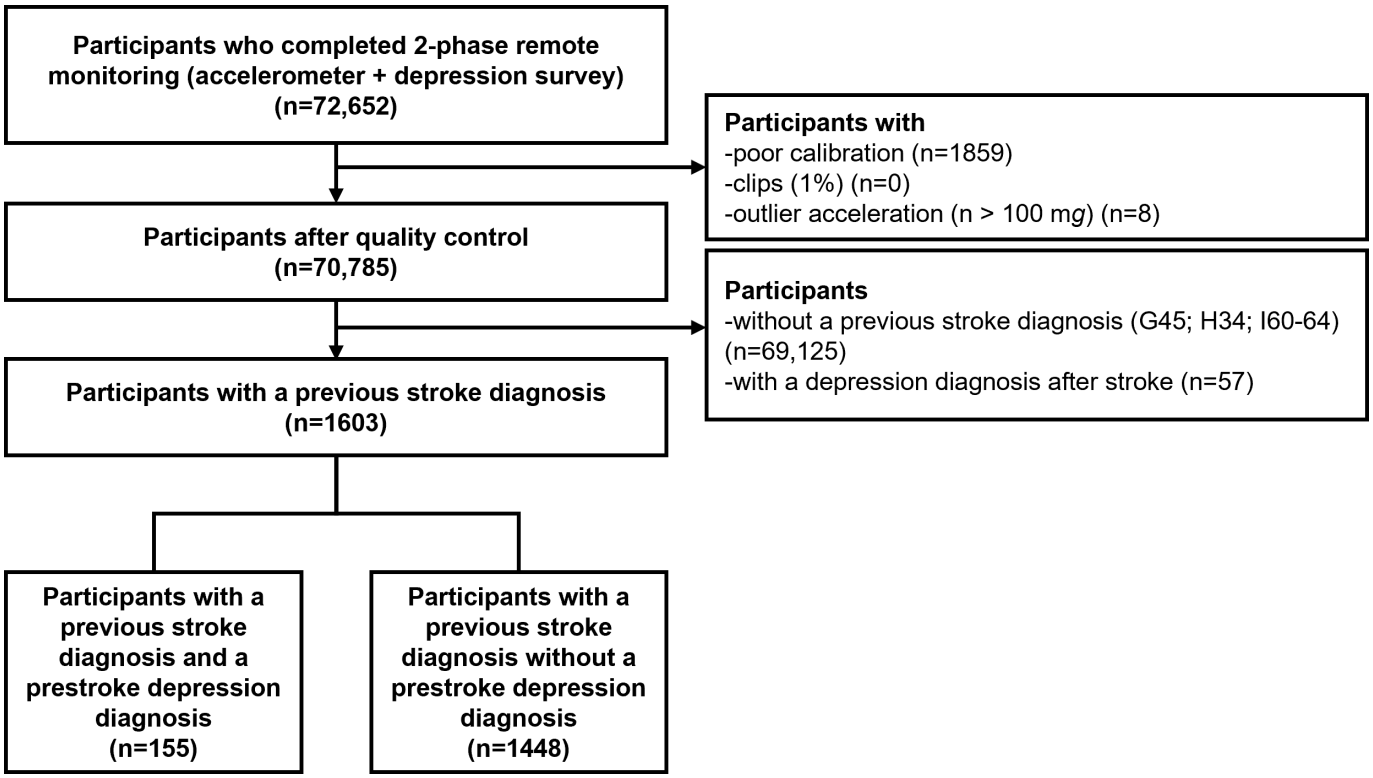
Participant Cohorts

Among participants who completed both remote monitoring components, those with dementia (n=23) were excluded. Quality control filtering demonstrated by Madjedi et al [38] was applied (n=70,785), which excluded those with outlier acceleration (>100 mg), more than 1% of readings exceeding ±8 *g* (clips), accelerometer wear time less than 3 days, and missing data for at least one 60-minute interval throughout 24-hour periods. Only participants with a previous stroke, including ischemic stroke, hemorrhagic stroke, and TIA (G45), were included (n=1660). Retinal artery occlusion (H34) was included as a stroke, as it is now considered a type of acute ischemic stroke [39]. Participants who were diagnosed with depression after stroke but before the accelerometer study (n=57) were excluded.

Among those meeting the inclusion criteria (n=1603), participants were divided into two cohorts: (1) those with a prestroke depression diagnosis at accelerometer study commencement (n=155) and (2) controls, that is, those without a prestroke depression diagnosis (n=1448) (Figure 1). No participants were diagnosed with depression between the accelerometer study and the follow-up depression survey.

Participants with a history of depression (*International Classification of Diseases, Tenth Revision* [*ICD-10*] codes F32-39) comprised the depression diagnosis (DDs) cohort. Definitions (*ICD-10* codes) used for inclusion and exclusion criteria as well as diagnostic classification are available in Multimedia Appendix 1.

Figure 1. Classification algorithm for participant cohorts.



Data Collection

Accelerometer study participants were instructed to wear the Axivity AX3 commercial accelerometer wristwatch continuously on their dominant arm for 1 week. The depressed mood frequency question was administered through a link accessible on smartphone, tablet or PC browsers as part of the standardized Patient Health Questionnaire-2 (PHQ-2) survey: "Over the past two weeks, how often have you felt down, depressed, or hopeless?" Responses were ordinal scores indicating the frequency of depressed mood, with 1="Not at all"; 2="Several days"; 3="More than half of days"; and 4="Nearly every day."

Permanent covariables were obtained at baseline visit, including sex and ethnicity. For each participant, age at the time of accelerometer study was calculated. Time-to-assessment was individually calculated by subtracting the accelerometer start date from the date of submitting the depressed mood survey. Comorbidity diagnoses before the accelerometer study were obtained from linked patient and hospital databases.

Statistical Analysis

To compare continuous and categorical covariables, the Mann-Whitney U test and χ^2 test, respectively, were used. A cross-sectional analysis using ordinal logistic regression to investigate the association between objective behavior predictors and the ordinal outcome variable, depressed mood frequency over the past 2 weeks, was conducted on data obtained at the accelerometer study and remote follow-up survey.

For both DD and control cohorts, separate models were fitted to evaluate whether the role of objective behavior predictors in depressed mood frequency differed between cohorts.

Analyses were performed in R (R Foundation for Statistical Computing), using *polr* from the library MASS. The effect sizes of objective behavior predictors, adjusted for confounders, on depressed mood frequency were plotted as odds ratios with 95% CIs. The Likelihood Ratio Test was used to obtain all *P* values and associated CIs. $P<.05$ was statistically significant.

Sensitivity Analysis

Three sensitivity analyses (also using ordinal logistic regression models), each considering DDs and controls, were performed using UKBB data. First, a general population dataset wasf generated. This included all participants eligible for inclusion in the accelerometer study and follow-up depression frequency survey who did not have a previous stroke diagnosis.

Next, participants with an initial IC diagnosis (after the depression frequency survey) were filtered into a separate dataset. Ordinal logistic regression models were fitted to assess the relationships between objective behavior predictors and depressed mood frequency. Finally, participants in the IC cohort who had an IS diagnosis were filtered into a separate dataset, and ordinal logistic regression models were fitted to assess the target relationship. The investigation of IC as a composite end point reflects updated understanding of stroke as sharing etiology with other neurologic rather than circulatory system disorders, as defined in the most recent

International Classification of Diseases, Eleventh Revision (ICD-11) [37].

For each filtered cohort, sample characteristics were obtained for review.

Ethical Considerations

National Health Service Research Ethics Committee (11/NW/0382) granted ethical approval for the UKBB population cohort study. Informed consent was obtained from all UK Biobank participants under National Health Service National Research Ethics Service (Ref 11/NW/0382). All UKBB data are deidentified.

Results

Study Characteristics

For participants in the 2-stage remote monitoring study (Table 1), the DDs had a higher proportion of women compared with controls (58.7% vs 40.7%). On average, DDs were younger (64 vs 66 y), slept slightly longer (9.2 vs 9.0 h/d), spent slightly less time in MVPA (29.3 vs 37.3 min/d) and SB (580.1 vs 583.6 min/d), and spent slightly more time in LPA (281.4 vs 278.2 min/d).

Table 1. Baseline characteristics of patients with previous stroke.

	Prestroke depression	Controls	*P* value
Number of participants, n	155	1448	
Age, mean (SD)	64 (7)	66 (6.5)	<.001
Gender, n (%)			
Men	64 (41.3)	859 (59.3)	<.001
Race, n (%)			
White	153 (98.7)	1418 (97.9)	.68
Sleep, mean (SD)	9.2 (1.8)	9.0 (1.8)	<.001
Sleep (7-9 h/d), n (%)	71 (45.8)	736 (50.8)	.27
SB[a], mean (SD)	580.1 (114.4)	583.6 (112.8)	<.001
LPA[b], mean (SD)	281.4 (106.9)	278.2 (102.4)	<.001
MVPA[c], mean (SD)	29.3 (31.2)	37.3 (33.0)	<.001
Time-to-assessment, mean (SD)	1.8 (0.7)	1.8 (0.6)	<.001
Diabetes, n (%)	21 (13.5)	130 (9.0)	.09
Hyperlipidemia, n (%)	76 (49)	615 (42.5)	.14
Hypertension, n (%)	155 (100)	1448 (100)	1
Multiple strokes, n (%)	40 (25.8)	348 (24.0)	.70
Time since most recent stroke, mean (SD)	7.8 (6.4)	9.8 (8.8)	<.001

[a]SB: sedentary behavior.
[b]LPA: light physical activity.
[c]MVPA: moderate-to-vigorous physical activity.

All participants had a hypertension diagnosis. The average time between accelerometer study start and depressed mood survey submission (time-to-assessment) was 1.8 years for both cohorts.

The average time from the initial stroke to the accelerometer study commencement was less for DDs than controls (7.8 vs 9.8 y).

Among DDs, 9 participants slept less than 7 hours while 75 slept more than 9 hours. In the control group, 79 participants slept less than 7 hours while 633 slept more than 9 hours.

Cross-Sectional Analysis

No significant association persisted in both the DD and control cohorts (Table 2). In controls, for each minute spent in MVPA per day, the odds of being in a higher depressed mood frequency category decreased by 23% (*P*<.001).

Table 2. Ordinal logistic regression assessing objective behavior predictors and depressed mood frequency.

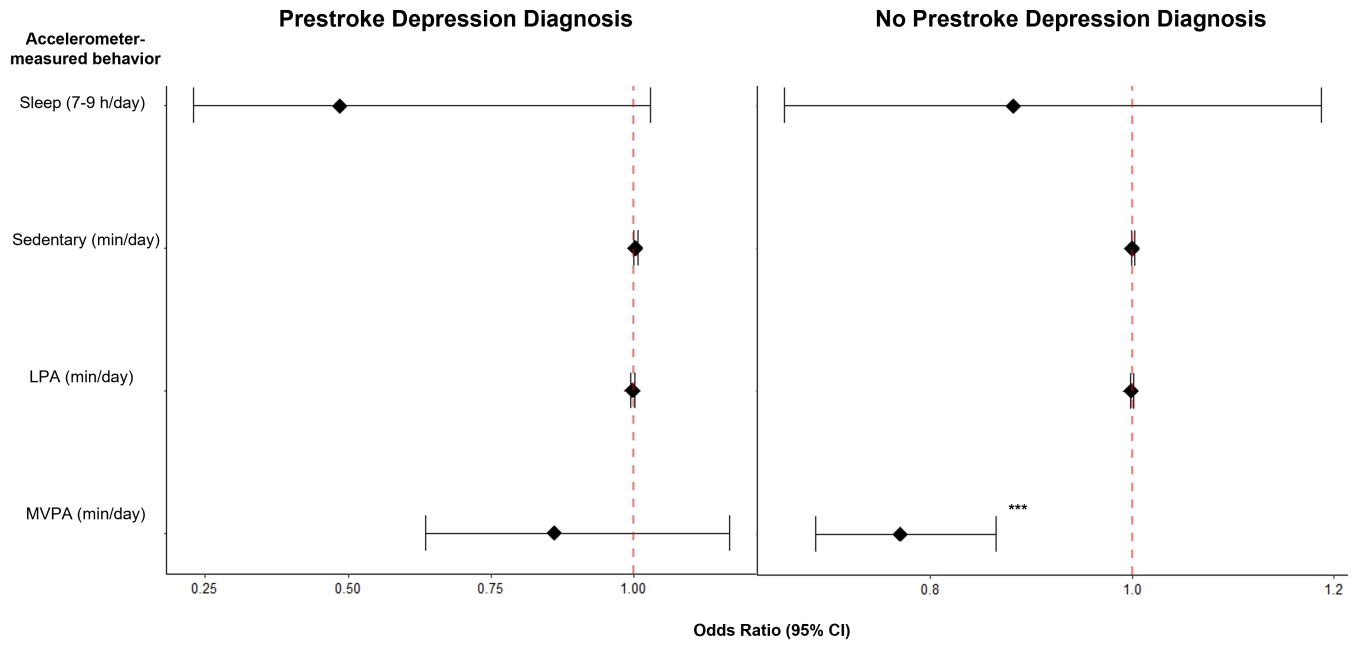
Previous stroke participants	Prestroke depression		Controls	
	OR[a] (95% CI)	*P* value	OR (95% CI)	*P* value
Sleep (7-9 hr/d)	0.49 (0.23-1.03)	.06	0.88 (0.66-1.19)	.41
SB[b] (min/d)	1.00 (1.00-1.01)	.10	1.00 (1.00-1.00)	.63
LPA[c] (min/d)	1.00 (0.99-1.00)	.20	1.00 (1.00-1.00)	.35
MVPA[d] (min/d)	0.86 (0.64-1.17)	.33	0.77 (0.69-0.87)	<.001

Previous stroke participants	Prestroke depression		Controls	
	OR[a] (95% CI)	*P* value	OR (95% CI)	*P* value

[a]OR: odds ratio.
[b]SB: sedentary behavior.
[c]LPA: light physical activity.
[d]MVPA: moderate-to-vigorous physical activity.

Models were adjusted for age, sex, ethnicity, time-to-assessment, hyperlipidemia diagnosis, and diabetes diagnosis. Odds ratios (ORs) with 95% CIs for frequency of depressed mood are reported (Figure 2). ORs above 1 correspond to an increase in the accelerometer-measured behavior associated with increased depressed mood frequency.

Figure 2. Forest plot of odds ratios for depressed mood frequency by accelerometer-measured behavior comparing participants with prestroke depression diagnosis (DDs) and control cohorts. LPA: light physical activity; MVPA: moderate-to-vigorous physical activity. *** denotes statistical significance.



Sensitivity Analysis

Study Characteristics

In each filtered cohort (Multimedia Appendix 2), DDs were younger than controls (general cohort: 60 vs 62 y; IS: 64 vs 66 y; IC: 65 vs 67 y) and had a greater proportion of females (69.4% vs 56.8%; 60.0% vs 45.6%; 61.8% vs 45.1%). In the general population cohort, DDs had a greater proportion of White participants (97.7% vs 97%). On average, DDs also spent less time across cohorts in MVPA (35.0 vs 42.9 min/d; 31.0 vs 39.5 min/d; 32.0 vs 38.4 min/d), less time in LPA (295.0 vs 300.3 min/d; 286.7 vs 291.0 min/d; 287.5 vs 287.7 min/d), and more time asleep (9.1 vs 8.9 h/d; 9.1 vs 9.0 h/d; 9.04 vs 8.98 h/d).

While DDs in the general cohort spent slightly less time, on average, in SB than controls (564.3 vs 564.4 min/d), DDs in the IS and IC cohorts spent more time sedentary on average (577.1 vs 569.9 min/d; 578.2 vs 575.3 min/d).

In the general cohort, DDs had a higher proportion of diabetes (4.4% vs 2.9%) and hyperlipidemia (17.9% vs 14.9%) diagnoses and a lower proportion of participants with optimal sleep duration per day (49.5% vs 55.4%).

For the IS cohort, the average time from the completion of the depression survey to first stroke diagnosis was slightly more for DDs (1.9, SD 0.7 y) than controls (1.8, SD 0.6 y). In the IC cohort, the average time from the completion of the depression survey to first CeVD diagnosis was similarly more for DDs (1.9, SD 0.7 y) than controls (1.8, SD 0.7 y).

Cross-Sectional Analysis

In the general model (Multimedia Appendix 3), for each minute spent in MVPA, the odds of being in a higher depressed mood frequency category decreased by 18.4% (*P*<.001) and 13.5% (*P*<.001) for DDs (n=6096) and controls (n=62,589), respectively.

Also in the general model, specific only to controls, getting guideline-recommended sleep hours (7-9 h) each day was associated with a decreased odds of being in a higher depressed mood frequency category (5.3%; *P*=.02).

No significant associations were identified for those in the IS-only cohort (Multimedia Appendix 4).

For the final sensitivity analysis (Multimedia Appendix 5), assessing only those participants with an IC diagnosis, including stroke, the odds of being in a higher depressed mood frequency category decreased by 12.2% for each minute increase in MVPA (P=.03), only in controls (n=1526).

Discussion

Principal Findings

This investigation partially supports the hypothesis that objective behavior predictors would be associated with future depressed mood frequency. Although we found no significant associations between depressed mood frequency and SB, LPA or sleep for patients with prior stroke, regardless of prestroke depression diagnosis, we did find that the odds of being in a higher depressed mood frequency category decreased for each minute spent in MVPA; however, this association was only observed in participants without a prestroke depression diagnosis. This finding supports the exploratory aim of this manuscript, suggesting that participants with prestroke depression may experience different behavioral patterns compared to those without a prestroke depression diagnosis. Such a finding can potentially help clinicians tailor programs monitoring patients at risk of PSD.

The sensitivity analysis in the general cohort corroborates established findings that MVPA confers a protective effect on mood, regardless of previous depression diagnosis. The lack of findings for the sensitivity analysis including only IS cases may be driven by the small sample sizes; however, the lack of findings also brings into question the potential for accelerometers to capture clinically actionable aberrations in patients before a stroke. Given that the protective effect of MVPA on depressed mood frequency was observed in the control cohort of patients with IC, accelerometer monitoring may be more appropriately directed to assess a broader range of neurologic changes, not just those linked with strokes.

Overall, the results suggest that accelerometer-based monitoring of behavior linked to depressed mood frequency may help clinicians identify patients who would benefit from resource-intensive screening, like the MADRS assessment. The sensitivity analyses support a separate approach for monitoring patients with a previous depression diagnosis, or more severe depression, compared to those with no documented depression or mild undiagnosed depression. When applied to predictive monitoring, a remote accelerometer-mood survey approach may be useful in cohorts of patients without a previous depression diagnosis, considering that patients with IC without clinical depression may experience observable behavior and mood changes before a CeVD diagnosis while their clinically depressed counterparts may not.

Limitations

A chief limitation of this study is that self-report data, like the depressed mood frequency survey, are subject to inaccuracies. Self-reported bias in survey responses may lead to misclassification of depressive symptom frequency and could influence different time-dependent results in our cohorts. Furthermore, the frequency of depression measures was not obtained by a clinician-graded protocol but, rather, by a survey questionnaire. Also, as the accelerometer study was only administered for one week and, on average, over a year before the follow-up mood survey, the impact of time between the objective measures and follow-up could have introduced substantial changes. The lack of associations observed for DDs may be due to the small sample size of participants with a previous depression diagnosis across cohorts. Moreover, the accelerometer study was only 1-week long and, therefore, may not generalize well to accurately represent busier or less busy weeks for patients. Accelerometer data collected on weekends versus weekdays may be distinct; however, this was not considered in this study.

The dichotomous investigation of clinically depressed and control patients are study strengths. In addition, UKBB participants were primarily White, limiting the generalizability of our findings outside of European populations. This UKBB study also primarily included participants aged 60 years and older and, as such, may not generalize well to young or middle-age adult populations. The majority of DDs were female across cohorts, a frequent finding in studies; however, male patients are less likely to seek out mental health resources, and the cohort stratification may be impacted by this.

Also, in the main analysis of previous stroke patients, participants diagnosed with clinical-grade depression after first stroke were excluded from this analysis. Considering the long gap in time from initial stroke to accelerometer study commencement, participants with a more immediate PSD diagnosis may either exhibit more intense symptoms or experience an underlying pathogenesis distinct from participants whose PSD symptoms are mild or emerge in the years after stroke.

Combining stroke types together as a single end point, as was done in the main analysis as well as the IS sensitivity analysis, may not consider unique characteristics of each stroke type and, as such, generated no significant results. Sleep was also assessed as a daily composite value, without consideration for time spent in a nap or broken sleep throughout the day. Together, these 2 limitations may have introduced confounding effects when considering sleep and depressed mood frequency, as previous research has shown short and long sleep to be associated with increased risk of intracerebral hemorrhage and ischemic stroke, respectively [40]. Furthermore, considering that all participants in our cohorts were hypertensive, MVPA's protective effect on depressive mood frequency may occur through improved cardiovascular health, rather than by conferring direct cerebral effects.

Comparison With Previous Work

No previous study assessed objective behavior measures and self-reported depressed mood frequency in patients with prior

stroke years after their initial diagnosis. A key problem inherent in accelerometer research is that adherence to study designs is less-than-satisfactory for most studies [41]. This study also excluded participants with a more immediate PSD diagnosis, considering only those with prestroke depression diagnoses or those with no or mild depression after stroke. A self-report survey study of recent patients with prior stroke found that patients with high levels of PA before a stroke experienced less severe PSD [33]. Although our study could not confirm this analysis due to the design of the UKBB study, we extended those results by confirming that MVPA confers a protective effect on mood before a CeVD diagnosis in patients without a previous depression diagnosis, but not before a stroke-only diagnosis.

One plausible explanation for the lack of association between MVPA and depressed mood frequency in DDs may be that stroke survivors with a previous depression diagnosis have persistently deficient levels of brain-derived neurotropic factor (BDNF), a trophic factor released after exercise that is linked with improved mood benefits. It is well established that stroke patients in general have lower levels of BDNF, a marker of poor functional recovery [42]. The lack of a link between improved mood and MVPA in DDs may be driven by a less intense "exercise high" due to reduced or impaired BDNF function. In addition, other contributing factors, such as time spent in MVPA or neuroinflammation, may play a role in modulating BDNF expression in DDs. Of note, the lack of a significant association between MVPA and depressed mood frequency in participants with a previous depression diagnosis may be attributed to less time spent in MVPA compared with controls across all cohorts (patients with prior stroke, general population, IS, and IC). Time spent in MVPA may need to exceed a time threshold in participants with previous depression diagnoses to improve mood.

The significant findings for IC cases, compared with the lack of findings for IS-only cases, are consistent with the updated *ICD-11* classification of CeVDs as a type of brain disease with shared etiology, rather than circulatory system disorders [37]. The protective effect of guideline-recommendation sleep (7-9 h/d) only observable in controls in the general cohort corroborates established work; however, the lack of associations across other cohorts may be explained by high levels of individual variability in sleep patterns, that is, nighttime disturbances, insomnia, and so on, previously identified in patients with depression as well as those at high risk of stroke [43,44].

A small pilot study of patients with minor ischemic stroke that found SB was positively associated with depression intensity and LPA was inversely associated with depression intensity [34]. Considering that this accelerometer study was conducted within the first 3 months after hospital discharge, our results extend these findings to look at mood in the years after a stroke. For instance, SB and LPA may be significant to monitor in the months after a stroke, while MVPA may be appropriate to monitor in the years after a stroke. Alternatively, MVPA may be less useful to monitor in minor ischemic stroke cases.

Using a larger dataset, our study builds on the feasibility demonstration of a small real-world study with patients with prior stroke, years after diagnosis, collecting one week of accelerometer data and ecological momentary assessments [45]. The results of our general cohort analysis considering participants without a previous depression diagnosis align with those from Sarris et al [46], who found that self-reported optimal sleep and PA were linked with decreased frequency of depressed mood in UKBB participants.

Conclusions

Our results highlight the importance of encouraging MVPA in patients with prior stroke without a depression diagnosis. Patients with prior strokes may be able to minimize short- and long-term disability and improve outcomes by proactively managing depressive symptoms. Applying MVPA to improve mood provides the added benefits of exercise-induced inflammation reduction and enhanced vascular elasticity while simultaneously reducing the risk of developing comorbidities and arterial stenosis or occlusion [47].

Considering that the only significant associations in the main analysis and incident sensitivity analyses were those that involved MVPA, it calls into question whether using accelerometer and depressed mood frequency survey data together can help clinicians identify patients who would benefit from remote monitoring, that is, this approach may generate more noise than signal over time. This study only considered a brief (1-week) accelerometer study, and over a year, on average, eclipsed between the *in situ* accelerometer study and the remote mood follow-up survey. Since neither the main analysis (previous stroke cohort) nor the incident sensitivity analyses resulted in significant associations for participants with a previous depression diagnosis, this underscores the need for additional research to determine whether this type of monitoring strategy can generate clinically actionable insights in participants with a previous depression diagnosis. Behavioral monitoring with accelerometer data and self-report surveys may not be helpful in patients with severe, or clinical-grade, depression. Future research should consider large sample sizes, longitudinal study designs, and analyze results stratified by time-to-diagnosis. Relevant to remote monitoring researchers, our findings highlight behavioral differences for those developing exploratory programs and clinically meaningful digital endpoints.

Overall, the cross-sectional analyses offer a robust perspective into the appropriateness of depression monitoring by digital sensors, using accelerometer wristwatches and smartphone, tablet, or PC-linked sensors. These insights offer clinical teams a strategy for translating digital health data, in this case, objective and subjective behavior measures, into scientifically valid frameworks for investigation. Future monitoring of patients at risk of different CeVD types, including those with a previous stroke diagnosis, should expand on our strategy and use both active and passive data to investigate relationships between objective digital sensor data and subsequent mood reports in patients diagnosed with and screened for depression. Based on our exploratory analysis,

the potential for longitudinal data from objective sensors to predict mood appears feasible. In addition, PSD researchers should aim to characterize behavior measures linked with depressed mood across defined and clinically meaningful time periods, such as in the 3-month routine monitoring period after a stroke or TIA, considering that observable behaviors may evolve as CeVD or other neurologic disorder pathogenesis progresses.

Acknowledgments

This research has been conducted using the UK Biobank Resource under Application Number 91159. This research was funded by Mayo Clinic. SJZ was supported by a predoctoral fellowship in value assessment from the PhRMA Foundation and a National Center for Advancing Translational Sciences TL1 training grant (5TL1TR002380-05).

Disclaimer

The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript; or in the decision to publish the results.

Data Availability

All data are publicly available, upon research approval access, from UK Biobank [48]. The datasets generated during and analyzed during this study are available from the corresponding author on reasonable request. Analysis code is available [49].

Authors' Contributions

SJZ completed the study design and manuscript drafting. BJE, BMD, and AG provided clinical expertise and contributed to manuscript editing. GMC provided expertise for obtaining data access and designing the study. AG conducted statistical review.

Conflicts of Interest

None declared.

Multimedia Appendix 1

Definitions for classifying patients.
[DOCX File (Microsoft Word File), 17 KB-Multimedia Appendix 1]

Multimedia Appendix 2

Sample characteristics across sensitivity cohorts.
[DOCX File (Microsoft Word File), 19 KB-Multimedia Appendix 2]

Multimedia Appendix 3

Ordinal logistic regression assessing objective behavior predictors and depressed mood frequency in the general cohort.
[DOCX File (Microsoft Word File), 16 KB-Multimedia Appendix 3]

Multimedia Appendix 4

Ordinal logistic regression assessing objective behavior predictors and depressed mood frequency in incident stroke cohorts.
[DOCX File (Microsoft Word File), 16 KB-Multimedia Appendix 4]

Multimedia Appendix 5

Ordinal logistic regression assessing objective behavior predictors and depressed mood frequency in incident cerebrovascular disease cohorts.
[DOCX File (Microsoft Word File), 16 KB-Multimedia Appendix 5]

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Abbreviations

BDNF: brain-derived neurotropic factor
CeVD: cerebrovascular disease
DD: depression diagnosis
IC: incident cerebrovascular disease
***ICD-10*:** *International Classification of Diseases, Tenth Revision*
***ICD-11*:** *International Classification of Diseases, Eleventh Revision*
IS: incident stroke
LPA: light physical activity
MADRS: Montgomery–Åsberg Depression Rating Survey
MVPA : moderate-to-vigorous physical activity
OR: odds ratio
PA: physical activity
PHQ-2: Patient Health Questionnaire-2
PSD: poststroke depression
SB: sedentary behavior
TIA: transient ischemic attack
UKBB: UK Biobank

Edited by Pieter Kubben; peer-reviewed by Ehab Harahsheh, Nathan Allen; submitted 23.01.2024; final revised version received 23.10.2024; accepted 14.11.2024; published 10.01.2025

Please cite as:
Zawada SJ, Ganjizadeh A, Conte GM, Demaerschalk BM, Erickson BJ
Exploring Remote Monitoring of Poststroke Mood With Digital Sensors by Assessment of Depression Phenotypes and Accelerometer Data in UK Biobank: Cross-Sectional Analysis
JMIR Neurotech 2025;4:e56679
URL: https://neuro.jmir.org/2025/1/e56679
doi: 10.2196/56679