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Accelerometer-derived sleep measures in idiopathic dystonia: A UK Biobank cohort study

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Abstract

Background: Sleep disturbance is an increasingly recognised non-motor trait in dystonia, with varying findings reported to date. Here, we examine sleep in a UK Biobank derived dystonia cohort using subjective self-reported sleep symptoms and objective accelerometer-derived sleep measures, with comparison to a control population.

Methods: 241 dystonia cases were compared to 964 matched controls in analysis of self-reported sleep symptoms and changes in sleep architecture using wrist-worn triaxial accelerometers.

Results: Dystonia participants had poorer self-reported sleep patterns compared to controls. Accelerometry measurements demonstrated later sleep times, reduced time in bed and shifts in circadian rhythm. No association was observed with pain, and only limited relationships with psychiatric symptoms.

Discussion: This study demonstrates the utility of accelerometers in longer-term evaluation of sleep in dystonia, for measurement of disturbance and response to treatment. Compared to controls, altered sleep and circadian rhythm were more common in dystonia patients which may contribute to the clinical phenotype.

Introduction

Dystonia is a hyperkinetic movement disorder characterised by involuntary muscle contractions causing twisting, repetitive movements, and abnormal postures. Multiple non-motor symptoms have been reported in association with dystonia, with sleep disturbance described in 40-70% of idiopathic dystonia cohorts.^{1,2} However, to date few studies have sought to examine sleep architecture through use of objective measures such as laboratory-based polysomnography (PSG). Those that have, have demonstrated changes in participants with cranio-cervical dystonia. Changes reported include a decrease in sleep efficiency (SE), total sleep time (TST) and percentage rapid eye movement (REM) sleep, and an increase in sleep latency (SL), wakefulness after sleep onset (WASO) and REM sleep latency, compared to controls.³⁻⁸ Sleep fragmentation and increased REM sleep latency have also been observed in *GCHI* mutation-positive dopa-responsive dystonia,⁹ as well as higher numbers of sleep spindles observed in primary and secondary forms of dystonia.¹⁰

Multiple factors influence sleep structure and quality, including for example, pain and psychiatric symptoms. Both these traits that have been reported at higher than population levels in dystonia cohorts. However, the direction of the causal relationship in the interaction between these symptoms remains uncertain.¹¹⁻¹⁵ Changes to sleep architecture, particularly REM sleep, are considered a distinctive neurological marker of depression, suggesting a complex bidirectional relationship between sleep and mental health disorders.¹⁶ Pain is also widely associated with impaired sleep quality, and has been linked to sleep disturbances such as insomnia and reduced sleep duration. Excess pain is well recognised in dystonia, and reported in up to 90% of those with cervical dystonia.¹⁴ It also appears to be a major contributor to impaired sleep quality amongst dystonia cohorts, with evidence of sleep impairment and pain severity being linked.¹⁵ There are no studies which have specifically investigated the relationship between self-reports of pain and psychiatric symptoms, and actigraphy-derived sleep variables in dystonia. Furthering our understandings of these relationships using a minimally intrusive monitoring system has the potential to impact the management of non-motor symptoms in a primary care setting, particularly given the detrimental impact on an individual's quality of life.

Although laboratory-based PSG remains the gold-standard objective measure of sleep, accelerometer devices validated to detect sleep/wake patterns, are increasingly being used to assess sleep parameters in the home environment and for extended periods of time.¹⁷⁻²⁰ The

UK Biobank (UKBB) is a large population-based study integrating detailed clinical data, including raw accelerometer data, self-reported symptom questionnaires including pain and psychiatric symptoms, primary care records and hospital admissions.²¹ Here, we use UKBB actigraphy data to compare sleep disturbances and circadian rhythm across dystonia and matched controls, and investigate correlations between actigraphy variables and self-reported non-motor symptoms including pain and psychiatric symptoms. We hypothesise that sleep disturbances would be common in dystonia, relative to the comparison group, and that these relationships would be dependent of confounding factors.

Methods

Study cohort

Data was analysed from the UKBB (MREC reference: 21/NW/0157) released to Cardiff University (project 13310), involving >500,000 adults (40-69 years) recruited throughout the UK (2006-2010). Individuals diagnosed with dystonia were identified using a previously validated algorithm,^{22,23} combining International Classification of Diseases version 10 (ICD-10) codes and Read Code version 2 (Supplementary Tables 1 & 2).²² The control cohort were derived from participants matched for both age and gender on a 4:1 ratio (n = 964).

Non-motor symptoms

Psychiatric, pain and sleep disturbance symptoms were determined through a combination of diagnostic codes and symptom-based questionnaires administered by the UKBB (Supplementary Table 3). These codes were used due to their high completion rate within the dystonia cohort (>93%), and in keeping with previous studies.²⁴⁻²⁷

Accelerometer data

Physical activity was extracted from 100Hz raw triaxial acceleration data obtained via Axivity AX3 wrist-worn accelerometers in a total of 103,712 participants within the UKBB (2013-2015).²¹ Data was collected continuously over seven days, and processed and analysed using R package GGIR (version 2.3-0).^{28,29} Processing of data, including calibration, resampling and epoch generation has been described elsewhere.²¹ Participants were excluded if they had fewer than three days recorded data (>16 hours per day) or if they failed calibration (> 0.01g). Derived sleep features and physical activity (Supplementary Table 4) measures were averaged across valid days.

Statistical Analysis

Categorical data was compared between groups using Chi-square test. Quantile plots were used to examine the normality of data, with parametric (Student's t-test) or non-parametric (Mann-Whitney U) tests used for case-control comparison where appropriate, and Bonferroni correction applied for multiple comparisons. Associations between self-reported sleep properties, physical activity, psychiatric diagnoses, pain symptoms and accelerometry outcomes were assessed using linear regression analysis and adjusted for age at recruitment and sex.

Results

The dystonia cohort (n=1,572) identified within the UKBB has been reported elsewhere,²³ of these, accelerometer data available for 241 (159 female, 82 male) unique individuals, and 15,768 age and sex-matched controls (Table 1). Except for higher rates of daytime sleepiness in those with unspecified forms of dystonia (p=0.007), no significant differences were observed across the self-reported sleep symptoms. However, significantly poorer overall sleep patterns and lower sleep scores were seen in the overall cohort and those diagnosed with cervical dystonia (p < 0.001), compared to controls (Table 1).

Accelerometer activity was analysed for the overall dystonia cohort owing to small cohort sizes within the individual sub-groups (blepharospasm, n =14; cervical dystonia, n=128; familial, n =2; orofacial, n =2; other/unspecified, n =18; tremor, n =75; writer's cramp, n =2) and controls. Individuals with dystonia were assigned to a single subtype of dystonia. Mann-Whitney U tests found that those with dystonia had a significantly later bedtime (p = 0.001) and spent less time in bed (TIB) (p = 0.002) compared to controls (Table 1). Compared to controls, individuals with dystonia had shifts and delays in circadian rhythm, least and most active five hours were later than controls (p <0.001), although acceleration activity did not differ between groups. Those with dystonia were also less active during waking hours than controls, with reduced daytime acceleration (p <0.001). Visual outputs reported by GGIR are shown in Figure 1.

Linear regression analysis found self-reported sleep variables associated with their respective accelerometer derived sleep measures, including self-reported chronotype and accelerometry-derived sleep and wake time (p < 0.001 and p = 0.01), and self-reported and accelerometry-derived sleep duration (p <0.001) (Supplementary Table 5). Other associated variables included insomnia and sleep onset (p =0.007), and nocturnal awakenings (p =0.02)

and number and duration of daytime naps ($p < 0.001$). Interactions between psychiatric diagnoses and the number and duration of daytime naps ($p = 0.001$ and $p < 0.001$, respectively) were also observed, alongside associations with accelerometer-measured physical activity including daytime acceleration and earlier bedtimes ($p = 0.001$) and time spent being inactive and later bedtimes ($p < 0.001$). Daytime acceleration was associated with fewer and shorter daytime naps ($p < 0.001$), while duration of periods of inactivity were associated with reduced TIB ($p < 0.001$), TST ($p < 0.001$) and higher numbers of nocturnal awakenings ($p < 0.001$). There were no associations between self-reported pain and accelerometer derived sleep variables.

Discussion

This study represents the largest analysis of accelerometer-derived sleep measures in dystonia to date. It also combines both the objective accelerometer measurements and subjective participant reported symptom measures, to determine their reliability when determining disturbances to sleep in dystonia. Both measures demonstrated an excess of sleep disturbance in the dystonia cohort compared to controls, including higher rates of self-reported poor sleep, and accelerometer determined reduced time in bed and shifts in circadian rhythm. No association was observed between pain and sleep, and elevated psychiatric symptoms were only associated with a higher number of and longer daytime naps, suggesting that sleep disturbance is independent of these other non-motor symptoms in dystonia. These findings, in contrast with previous literature, may in part be explained by the ‘healthy volunteer’ selection bias within the UK Biobank.³⁰

Several of the self-reported sleep symptoms are consistent with previous studies, including poorer sleep patterns in the overall and cervical dystonia cohorts, while higher levels of daytime sleepiness were described by those with unspecified/other dystonia.^{11,15,31,32} In contrast, previous findings have shown high proportions of excessive daytime sleepiness and abnormal sleep duration amongst those with cranio-cervical dystonia, potentially owing to use of standardised sleep questionnaires such as the Pittsburgh Sleep Quality Index and Epworth Sleepiness Scale.^{11,15} Further, the UKBB used only single questions for five aspects of sleep, potentially not allowing for the depth of symptom information needed to identify sleep disturbances.

Analysis of the accelerometer data found those with dystonia to have a significantly later bedtime and less time spent in bed compared to controls, in keeping with the findings from

previous studies involving self-reported sleep duration.¹¹ A single PSG-study to date has also reported increased sleep latency in cervical dystonia, providing a potential explanation for the observed reduced sleep time.⁶

Consistent with a single previously reported PSG-based study, subjective self-reported sleep variables were associated with their more objective, respective accelerometer measures, including sleep duration, chronotype, sleep onset, wake time, insomnia and number of nocturnal awakenings.⁶ The domains in which less consistency was observed, such as self-reported insomnia, SE and TST, may in part be due to the long interval between baseline assessment and accelerometry data capture in some cases (average 5.9 years), as well as the well-recognised differences that emerge between subjective and objective sleep measures.^{33,34}

In contrast to previous literature, we found no association between pain or psychiatric symptoms and accelerometer determined sleep disturbances. Several PSG and questionnaire-based studies have shown that pain and mood disorders are strongly correlated with sleep impairment amongst dystonia cohorts,^{7,12,31} although findings are inconsistent as to whether sleep-related measures are independent of motor severity.^{7,12} It is possible that the small sample size (n =13) amongst PSG-based studies accounts for the association, whilst accelerometer data may provide a balance between accuracy and cohort size, and an area for future work.

In line with existing evidence, several associations between physical activity and accelerometer-derived sleep measures were observed.³⁵ Bouts of inactivity were significantly associated with later sleep time, reduced TIB, reduced TST and an increased number and duration of daytime naps. These findings suggest that physical activity can promote healthy sleep in those with dystonia, although we cannot rule out reverse causality.

Whilst this study benefits from the extensive longitudinal information collected at scale, several limitations also exist. Firstly, the UKBB is a volunteer-based sample and not representative of the general population,³⁰ with participants potentially experiencing fewer symptoms that would interfere with participation. This is particularly relevant in the context of sleep disturbance as those who experience more severe dystonic symptoms may experience increased sleep impairments.⁷ Secondly, whilst the UKBB is a great resource for non-motor symptom research, the reliability and validity of the measures remains largely unknown. For example, the validity of sleep behaviours in comparison with other established sleep questionnaires such as the PSQI, has not been examined. Lastly, we were unable to

confirm clinical diagnoses and therefore it remains possible that those without dystonia were included in the cohort. However, our cohort identification involved use of a previously published algorithm with 79% sensitivity in identifying those with dystonia.²²

The present findings add to a growing body of evidence of sleep disturbances in dystonia, as well as demonstrating the capacity of accelerometry measurements in providing more detailed information about sleep patterns, beyond questionnaire-based surveys. Our findings also suggest need for future investigation of physical activity and sleep in the context of dystonia, as well as the potential for use of physical/exercise therapies as means for improved sleep quality in dystonia. Future work includes application of accelerometer-based sleep measurements in larger dystonia cohorts, in conjunction with PSG-based measurements, as well as longitudinal studies to examine whether sleep parameter variation is observed with varying motor symptom severity and therapeutic intervention.

Data Availability Statement

Anonymized data not published within this article will be made available by request by application to the UK Biobank (<https://www.ukbiobank.ac.uk/enable-your-research/apply-for-access>).

Ethical Compliance

The UKBB has approval from the North West Multi-centre Research Ethics Committee (MREC) (reference 21/NW/0157). All participants gave written informed consent. The data used in this thesis was released to Cardiff University following project application to UKBB (Project Code: 13310). We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this work is consistent with those guidelines.

Author Contributions:

GAB: Conception, acquisition, execution, analysis and interpretation of the data, drafting and revising critically for important intellectual content.

MEW: Acquisition, execution, analysis and interpretation of the data, drafting and revising critically for important intellectual content.

SK: Acquisition, analysis and interpretation of the data, drafting and revising critically for important intellectual content.

CM: Acquisition, analysis and interpretation of the data, drafting and revising critically for important intellectual content.

EHD: Acquisition, analysis and interpretation of the data, drafting and revising critically for important intellectual content.

KJP: Conception, acquisition, analysis and interpretation of the data, drafting and revising critically for important intellectual content.

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Financial disclosure/Competing Interests

The authors report no competing interests. SK and CM are employees at Aparito. EHD is employee at Aparito and holds shares.

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Legends

Figure 1. Example of visual output reported by GGIR in the A) dystonia and B) control cohort

i) Bar plots with information on key physical activity **ii)** Bar plots with key/illustrative sleep variables
iii) Visual summary of physical activity and sleep patterns

Abbreviations: PA: Physical activity, SPT: Sleep period time

Supplementary Table 1. ICD-10 Codes and Read Codes used to identify dystonia patients

Supplementary Table 2. Exclusion codes (ICD-10 and Read Codes) for UK Biobank participants

Supplementary Table 3. UK Biobank questions related to non-motor symptoms and responses

Supplementary Table 4. Accelerometer-derived sleep features and physical activity and their definitions

Supplementary Table 5. Associations between self-reported sleep, pain/psychiatric symptoms and physical activity, and accelerometer-derived sleep variables in the dystonia cohort

Tables

Table 1. Demographics, self-reported and accelerometer derived sleep variables

	Dystonia	Blepharospasm	Cervical Dystonia	Idiopathic Familial Dystonia	Idiopathic Non-familial Dystonia	Idiopathic Orofacial Dystonia	Other Dystonia	Dystonia, unspecified	Dystonic Tremor	Control	
Number	241	14	128	2	-	2	1	17	75	15768	
Age (mean, SD)	57.5 (7.9)	60 (8.18)	56.6 (7.6)	62 (0)	-	43 (1.4)	57	57.1 (9)	59 (8.1)	58.4 (7.8)	
Age (range)	40-70	42-68	41-70	-	-	42-44	-	43-69	40-70	40-69	
Gender	Male	82	4	47	0	-	0	0	4	27	4
	Female	159	10	81	2	2	1	13	48	13	
	Dystonia	Blepharospasm	Cervical Dystonia				Other/Unspecified Dystonia	Tremor	Control		
Sleep symptoms											
Insomnia	179/241 (74.3) 0.4	8/14 (57.1) 0.086	95/128 (74.2) 0.53	-	-	-	10/17 (58.8) 0.08	63/75 (84) 0.13	12071/15763 (76.7)		
Chronotype	89/218 (40.8) 0.09	6/13 (46.2) 0.41	46/115 (40) 0.3	-	-	-	6/15 (40) 0.7	29/68(42.6) 0.21	5029/14238 (35.5)		
Sleep Duration	79/240 (32.9) 0.11	3/14 (21.4) 0.57	46/128 (35.9) 0.054	-	-	-	5/16 (31.25) 0.79	25/75(33.3) 0.33	4443/15727 (28.3)		
Snoring	83/225 (36.9) 0.22	5/12 (41.7) 0.52	52/124 (41.9) 0.035	-	-	-	5/14 (35.7) 0.83	18/70 (25.7) 0.2	4857/14725 (33)		
Daytime Sleepiness	9/241 (3.7) 0.1	0/14 (0) 0.58	4/128 (3.1) 0.47	-	-	-	2/17 (11.8) 0.007	3/75 (4) 0.28	343/15740 (2.2)		
Sleep pattern											
Healthy	79/203 (38.9) 0.48	6/11 (54.5) 0.38	42/111 (37.8) 0.45	-	-	-	5/13(38.5) 0.83	4/63(6.3) 0.43	5500/13920 (41.4)		
Intermediate	109/203 (53.7) 0.62	4/11 (36.4) 0.2	59/111 (53.2) 0.63	-	-	-	8/13 (61.5) 0.66	36.63 (57.1) 0.78	7366/13290 (55.4)		
Poor	15/203 (7.4) 0.0008	1/11 (9.1) 0.27	10/111 (9) 0.0006	-	-	-	0/13 (0) 0.51	23/63 (36.5) 0.16	424/13290 (3.2)		
Sleep score	3 (2-4) 0.05	4 (2-4) 0.83	3 (2-4) 0.02	-	-	-	3 (2-4) 0.78	3 (3-4) 0.32	3 (3-4)		
Accelerometry											
<i>Sleep</i>	241	964									
Bedtime	23:55 (01:14)	23:36 (01:16)	0.001								

Wake-up time	07:11 (01:41)	07:06 (01:17)	0.42
Time in bed (hours)	7.24 (1.37)	7.45 (1.4)	0.002
Sleep duration (hours)	6.5 (1.43)	6.71 (1.36)	0.007
Sleep efficiency (%)	90.5 (5)	90.26 (5.26)	0.56
WASO	0.69 (0.37)	0.74 (0.43)	0.07
Number of nocturnal awakenings	12.8 (4.67)	13.29 (4.5)	0.39
Number of daytime naps	8.47 (6.67)	8.3 (6.17)	0.39
Duration of daytime naps	1.55 (1.35)	1.52 (1.34)	0.29
Duration of longest sleep bout	8 (1.4)	8 (1.4)	0.81
<i>Circadian rhythm</i>			
L5 time	04:51 (06:38)	00:25 (01:14)	<2.2e-16
L5 acceleration (mg)	3.57 (1.39)	3.74 (1.97)	0.011
M5 time	10:23 (02:30)	03:47 (05:53)	<2.2e-16
M5 acceleration (mg)	58.7 (22.1)	59.19 (24.31)	0.22
<i>Physical activity</i>			
	240	964	
Overall physical activity (mg)	27.5 (10.15)	28.14 (10.55)	0.1299
Daytime acceleration (mg)	37.95 (14.47)	38.59 (15.03)	<2.2e-16
Sleep time acceleration (mg)	3.98 (1.53)	3.97 (1.68)	0.8177
Inactive time (minutes)	629.6 (149)	633.81 (130.74)	0.84
Light time (minutes)	258.77 (72.08)	260.02 (75.82)	0.4
Moderate time (minutes)	94.56 (60.61)	95.68 (58.74)	0.9
Vigorous time (minutes)	2.13 (2.9)	2.31 (3.73)	0.17
Bouts of 30 minutes inactivity	322.33 (202.11)	333.89 (193.37)	0.93
Bouts of 1-minute MVPA	38.36 (36.74)	36.71 (37.81)	0.74
Least active 5 hours (mg)	3.74 (1.68)	3.53 (176)	0.23
Most active 5 hours (mg)	58.77 (21.9)	60.35 (24.35)	0.02

Abbreviations: mg: milligravity, MVPA: Moderate-vigorous physical activity, SD: Standard Deviation, WASO: Wakefulness after sleep onset

Sleep symptom values given as n (%), p-value. P-values are all vs controls

Bold p-values represent significant values post Bonferroni correction for multiple comparisons

