33 ASSOCIATIONS BETWEEN DAILY SLEEP AND AFFECTIVE EXPERIENCES: A SYSTEMATIC REVIEW

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10.1136/bmjresp-2021-bssconf.30

Introduction This work reviews empirical research investigating the bidirectional relationship between daily sleep and affective experiences. In particular, the review focuses on ambulatory assessments such as experience sampling (ESM) and daily diaries. A secondary objective explored the differential impact of affective disorder diagnosis and shift work on daily sleep-emotion dyads.

Methods EMBASE (Ovid), Ovid MEDLINE(R), PsycINFO (Ovid), and Scopus (Elsevier) were searched to January 2021. Additional studies were identified through reference checking and hand searching. Records were deduplicated on EndNote and uploaded to Rayyan.

Results 1526 studies were identified and 51 met the full inclusion criteria. Studies predominantly included healthy populations (N=42), of which four involved shift workers; remaining studies investigated mood disorders (N=9). Studies with only self-report sleep measures were most common (N=31) but a high number incorporated actigraphy (N=20). Overall, 13 studies used both actigraphy (objective) and self-report (subjective) sleep markers. Sleep diaries (N=13), the Pittsburgh Sleep Quality Index (PSQI; N=10), and Positive and Negative Affect Schedule (PANAS; N=20) were the most widely used measures. In general, findings support a mutual relationship between sleep and next-day affective experiences among healthy populations and individuals diagnosed with a mood disorder.

Discussion This work expands on prior reviews by Konjarski, Murray, Lee, and Jackson (2018) and Ong, Kim, Young, and Steptoe (2017) across four areas: to include affective disorders and shift workers; to focus on the situational context of daily assessments; to account for interchangeable affective definitions; and to include studies published after 2017.

REFERENCES

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35 MANAGEMENT OF CENTRAL APNOEAS – ANALYSIS OF A PAEDIATRIC COHORT REFERRED TO A TERTIARY CARE SLEEP SERVICE

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10.1136/bmjresp-2021-bssconf.31

Introduction Central sleep apnoea (CSA) is common in childhood and is usually clinically associated with developmental delay, syndromes, brain and/or brainstem involvement. The threshold for significant CSA remains controversial. Only one study so far has described CSA in a large paediatric cohort.[1]

The aim of this study was to review the prevalence, clinical correlation and management of CSA in a cohort of paediatric patients referred to a tertiary UK sleep service.

Methods Retrospective study of children <18 years referred to the paediatric sleep service for a sleep study between April 2018-July 2020. We included patients with a cAHI of ≥1. Patients with previous sleep studies, diagnosis of CSA and on ventilatory support or oxygen therapy were excluded.

Results 162 patients were included with a median age of 2.7 years (range 9 days to 9.7 years). 129 patients had isolated cAHI of 1-5 without association with obstructive sleep apnoea (OSA), defined as oAHI>5. 14 had isolated cAHI ≥5 and 19 had CSA with OSA.

Abstract 35 Table 1 Background and severity of cAHI

Diagnoses	Isolated CSA with cAHI 1-5	Isolated CSA with cAHI>5	CSA with OSA>5
Congenital heart disease	8 (6.0%)		3 (15.8%)
PWS Pre-GH	6 (4.5%)	1 (10%)	
PWS Post-GH	1 (0.8%)		
Trisomy 21	6 (4.5%)	1 (10%)	1 (5.3%)
Ex-preterm	13 (9.8%)	2 (20%)	2 (10.5%)
Chronic lung disease	2 (1.5%)		
Unsafe swallow	3 (2.3%)		1 (5.3%)
Obesity/overweight	4 (3.0%)		3 (15.8%)
Neurodisability/neuromuscular including seizures	9 (6.8%)	2 (20%)	3 (15.8%)
Congenital syndromes	3 (2.3%)	2 (20%)	
Recurrent lower respiratory tract infections	4 (3.0%)		1 (5.3%)
Adenotonsillar hypertrophy/symptoms osa	59 (44.4%)	2 (20%)	9 (47.4%)
Asthma/VIW/bronchiolitis	24 (18.0%)	1 (10%)	2 (10.5%)
Gastro-oesophageal reflux disease	3 (2.3%)	1 (10%)	1 (5.3%)
Miscellaneous	11 (8.3%)		3 (15.8%)

Abstract 35 Table 2 Investigation and management cAHI - 1-5 vs ≥5

cAHI	No of patients	Investigations				Management		
		ENT referral	EEG	Neuroimaging	Genetics	O2	CPAP/NIV	Follow-up study
1-5.0	148	30	0	0	0	0	4	51
≥5.0	14	4	2	3	2	4	2	7

Patients with isolated cAHI 1-5 had no specific clinical features except adenotonsillar hypertrophy. Isolated severe CSA was identified in ex-preterm or complex neurodisability (table 1). Only severe CSA patients had further investigations to exclude a central/genetic cause (table 2).

45 patients with a cAHI of 1-5 had a follow-up study. of these, 41/45 had a comparable CSA severity with cAHI <5. 4 patients had cAHI 1-5 but newly identified OSA on follow-up.

Discussion Our analysis shows that children with an isolated cAHI of 1-5 had reproducible results on follow-up study. There was an association between CSA≥5 and OSA which has not been reported in the literature suggesting the central component may resolve after treating OSA.

REFERENCE

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36 LITERATURE REVIEW ON THE EFFECTS OF ACUTE AND CHRONIC ALCOHOL USE ON THE GLYMPHATIC TRANSPORT SYSTEM

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10.1136/bmjresp-2021-bssconf.32

Introduction Alcohol is known to disrupt various brain processes, including accumulation of βamyloid involved in the pathology of Alzheimer's disease. The glymphatic system clears toxins including βamyloid. It is mainly active during Non-REM slow wave sleep [1]. We wanted to research effects of acute and chronic use of alcohol on glymphatic clearance.

Method Peer reviewed articles identified in PubMed from inception until 5 September 2021. Search terms included: 'glymphatic', 'glial-lymphatic', 'alcohol' and 'ethanol'. This resulted in thirteen publications of which four were relevant and included.

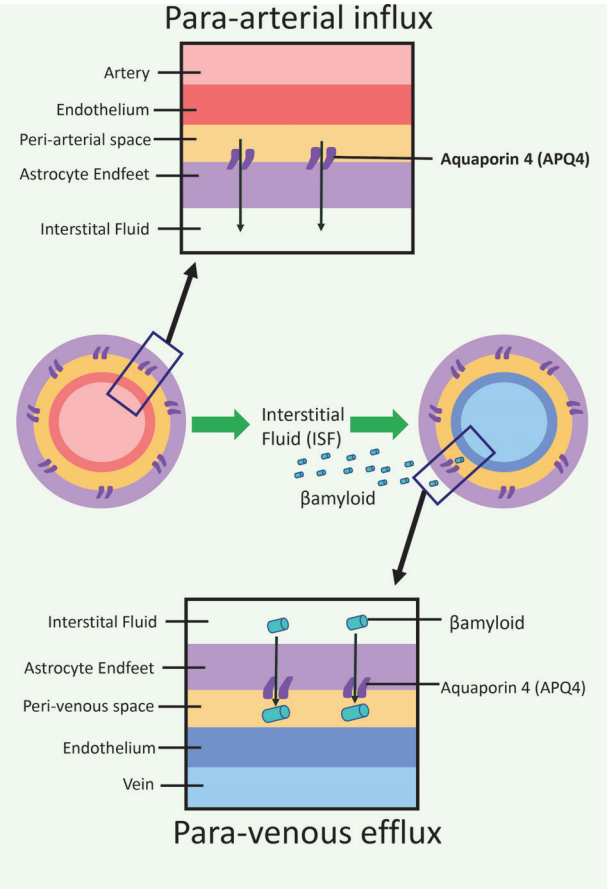
Results The glymphatic system is a perivascular transport system (figure 1).

Low dose alcohol improved glymphatic clearance, increasing influx and efflux of CSF also influenced by increased cerebral blood flow [2]. This promoted clearance of βamyloid [7]. There was a decline in GFAP with reduced AQP4 loss in chronic low use [2] as well as improving endothelial tone [7]. After stopping chronic low alcohol an increase in CSF influx and glymphatic function was observed [2].

Acute medium-high alcohol causes reversible decline in glymphatic transport, worse with higher dose [2,4]. The decreased influx may be mediated by release of endogenous opioids. Alcohol decreases cardiac output [6], with decline in heart rate [1] decreasing pulsatility of vessels and CSF influx [4]. This process may also be influenced by reduced glucose metabolism [2].

Chronic high alcohol caused decreased glymphatic clearance [1,2,6]. This process is partially irreversible due to astrogliosis causing increased GFAP with depolarization of APQ4 [6] and compromise of the blood brain barrier [3]. Although there is evidence of some reversible changes following abstinence [2].

Discussion Low dose alcohol appears to promotes glymphatic function. Medium-high alcohol disruption is initially reversible, becoming at least partially irreversible with chronic use. These changes impact the role of the glymphatic system, inhibiting its function during sleep and increasing risk of Alzheimer disease.



Abstract 36 Figure 1 Representation of CSF flow from subarachnoid space into interstitial fluid and parenchyma. Aquaporin 4 (AQP4) water channels are polarised toward the perivascular space allowing flow of soluble molecules, including efflux of βamyloid from the interstitial fluid