of Living Scale, which measures Gf; the abstraction score was used to categorize premorbid Gf as relatively high (≥34; WRC n=14, TSD n=14) or low (<34; WRC n=9, TSD n=10). Task performance was analyzed using mixed-effects ANOVAs with fixed effects of session, condition, and Gf-group and their interactions, and a random effect over subjects on the intercept; the GNGr analysis also included fixed effects of post-reversal test block and its interactions.

Results: TSD degraded session 2 performance on the SM, as evidenced by a condition by session interaction ($p< 0.001$). We also observed an effect of Gf-group ($p<0.001$). The expected benefit of Gf to speed of processing on the SM was observed regardless of session or condition.

TSD also degraded session 2 performance on the GNGr, whereas subjects in the WRC showed performance improvement in session 2 (condition by session interaction, $p=0.006$). Furthermore, there was a session by Gf-group by block interaction ($p=0.048$). Those with relatively high Gf improved across sessions on the second block ($p=0.037$), but this was evident only in rested subjects ($p<0.001$).

Conclusions: Premorbid Gf predicted sleep-deprived performance on a task conceptually related to Gf, such that the benefit of Gf to speed of semantic decision processing persisted across sessions. Regarding cognitive flexibility, higher Gf did not confer resilience to TSD; only higher Gf subjects who were rested showed a benefit in session 2. This work suggests that investigating premorbid individual differences may clarify our understanding of task-specific resilience during sleep deprivation.

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EXPLORING THE ASSOCIATION BETWEEN SLEEP AND COGNITIVE PERFORMANCE IN A HEALTHY AND REAL-WORLD COGNITIVELY IMPAIRED POPULATION

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Introduction: Poor sleep is a risk factor for cognitive decline. Multiple mechanisms link sleep and brain health, including pathological accumulation of amyloid and changes in synaptic plasticity during acute sleep deprivation. However, there has been conflicting evidence as to which aspects of sleep are associated with different stages of dementia progression and healthy ageing. We aim to explore this association in both a healthy and real-world cognitively impaired population.

Materials and Methods: This cross-sectional study involves two cohorts: a healthy volunteer and a clinical one. Healthy participants completed a web-based study (SleepQuest) comprising validated questionnaires: Pittsburgh Sleep Quality Index (PSQI), General Anxiety Disorder (GAD7), Patient Health Questionnaire (PHQ8), Dysfunctional Beliefs and Attitudes about Sleep (DBAS-16) and an online cognitive assessment- Cambridge Cognition Paired Associates Learning (PAL) task through the Great Minds research registry (www.greatmindsfordementia.uk). The clinical cohort, comprising attendees to the Cognitive Disorders Clinic at Southmead Hospital, completed PSQI (to measure sleep quality) and STOP-BANG (to gauge sleep apnoea risk) questionnaires to assess sleep, with the Montreal Cognitive Assessment (MoCA) used to assess cognition.

Results: 446 healthy participants completed the SleepQuest survey and PAL task and were categorised into two groups: good sleepers (PSQI≤5) and poor sleepers (PSQI>5). Anxiety and depression scores were higher in poor sleepers compared to good sleepers [(GAD7:3.67 vs 1.60, p<0.0001); (PHQ8:5.78 vs 2.18, p<0.0001)]. Age (β=0.184,p<0.001), gender (female vs male; β=0.236,p<0.001) and quality of life (β=-0.363,p=0.048) significantly predicted cognitive performance, but sleep quality and attitudes towards sleep did not.

109 clinic participants completed the PSQI, STOP-BANG and MoCA. Overall sleep quality was poor (PSQI>5, n=67 (65.1%), mean PSQI 7.54) with 63 participants (61.1%) scoring ≥3 on STOP-BANG. STOP-BANG significantly correlated with cognitive performance (r_s=-0.233, p<0.01). Overall sleep quality (PSQI) did not significantly correlate with cognitive performance (r_s=-0.058, p>0.05).

Conclusions: Poor sleep quality was associated with increased symptoms of depression and anxiety in healthy participants. Meanwhile, in the memory clinic population, risk of sleep apnoea was negatively associated with cognitive performance, suggesting a potential role for sleep apnoea treatment in improving cognitive outcomes in a clinic population; future clinical trials should evaluate this possibility. Although average sleep quality in the memory clinic group was poor, we did not find a clear link between subjective sleep quality ratings and cognition. This mirrored the lack of an association between self-rated sleep and cognition in the healthy group. Future longitudinal studies with molecular classification of neurodegenerative disease and objective sleep markers of micro- and macro-architecture would aid our understanding of the role that sleep may have as a modifiable risk factor for dementia.

INCREASED VARIABILITY IN TOTAL SLEEP TIME IS ASSOCIATED WITH REDUCED PSYCHOMOTOR VIGILANCE IMPAIRMENT FOLLOWING EXPERIMENTAL SLEEP CONTINUITY DISRUPTION

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Introduction: Sleep deprivation, restriction, and disturbance typically result in impairments of psychomotor vigilance and sustained attention. However, there is a large degree of trait interindividual variability in these impairments. Previous evidence suggests that increased variability in habitual Total Sleep Time (TST) is associated with adverse health outcomes including reduced subjective sleep quality, subjective wellbeing, and increased depressive symptoms. Therefore, we sought to test the hypothesis that variability in TST might represent a trait phenotype of individual response to sleep disruption, using a novel sleep continuity disruption paradigm (Forced Awakenings) which aims to mimic the kind of sleep loss experienced in those with insomnia.

Materials and Methods: We performed secondary data analysis from a previously published clinical trial, where 100 healthy sleepers were randomized (stratified by age, sex, BMI) to receive two nights of sleep continuity disruption (Forced Awakenings/ FA: consisting of eight random awakenings lasting 20-60mins) and two nights of undisturbed sleep (US: 8-hour sleep opportunity) in a within-subjects crossover design, separated by a minimum two-week washout period. Participants were rigorously screened to ensure they were healthy sleepers, free of psychiatric, medical or occult sleep disorders. Prior to undergoing the inpatient sleep protocols, participants underwent seven days of at-home actigraphy, from which we derived actigraphic measures of habitual sleep variability. Variability in TST was calculated from actigraphy using two standardized measures of variability: Intra-individual Standard Deviations (iSD) and Mean Square of Successive Differences (MSSD). In accordance with previously published analyses, we assessed the degree to which variability in TST was associated with change in psychomotor vigilance (Both lapses and reciprocal reaction times), from US to FA conditions. The PVT was performed at days one and two of the respective conditions. Analyses were performed using linear regression models and controlled for age and average TST across the seven days of actigraphy.

Results: Following one day of forced awakenings, variability in TST was not significantly associated with change in PVT lapses or reciprocal reaction times between FA and US conditions. Following two days, contrary to our hypotheses, increased variability (iSD) in total sleep time was associated with a smaller increase in lapses between undisturbed sleep and sleep disruption (unstandardized beta = -0.027, SE = 0.12, $p = 0.03$), signifying reduced psychomotor impairment. MSSD of TST was not significantly associated with PVT lapses, despite trending close to significance (unstandardized beta = -0.0001, SE = 0.0003, $p = 0.05$). There was no association between TST variability and reciprocal reaction time for iSD or MSSD.

Conclusions: Contrary to the literature linking increased TST variability to adverse health outcomes, increased TST variability may represent a phenotype which is protective against the effects of sleep disruption on psychomotor vigilance. Future studies should continue to probe the association between this phenotype and other measures of cognitive-emotional impairment following sleep disruption.