Methods: Female participants (n=149) with sleep disturbance and temporomandibular joint (TMJD) pain were recruited as part of a treatment trial. Data were used to identify a cohort of participants with clinically significant depressive symptoms (CES-D >16), with and without tinnitus. We examined the pre-randomization polysomnography (PSG) dataset, and calculated sleep architecture, and relative spectral power (Alpha, beta, theta, delta). We compared cohorts using independent t-tests, testing for differences in architectural and spectral sleep parameters, controlling for anxiety symptoms.
Results: 14 females (mean age = 41.76) reporting current tinnitus were age and depression severity matched with 14 females reporting no tinnitus (mean age = 41.27). Groups did not differ significantly in age (p = 0.91), BMI (p = 0.868), race (p = 0.328) or severity of depressive symptoms (CES-D: 23.93 No tinnitus vs 25.07 Tinnitus, p = 0.540), but the tinnitus group reported significantly higher anxiety (GAD-7: 9.43 no tinnitus vs 13.36 Tinnitus, p = 0.016). Data indicated TMJD patients with tinnitus had greater N2 sleep percentage (24.243% no tinnitus vs 57.200% Tinnitus, p = 0.033) compared with controls. There were no significant differences in N1% (4.2% No tinnitus vs 3.7% Tinnitus, p = 0.874), SWS% (23.164% No tinnitus vs 17.236% Tinnitus, p = 0.217) or REM% (26.386% No Tinnitus vs 21.88% Tinnitus, p = 0.238) between groups. Analysis of spectral data showed no significant differences in relative alpha (0.129 No tinnitus, 0.143 Tinnitus, p = 0.099), beta (0.186 No tinnitus vs 0.188 Tinnitus, p = 0.814), theta (0.123 No Tinnitus vs 0.125 Tinnitus, p = 0.069), or delta power (0.360 Tinnitus vs 0.346 Tinnitus, p = 0.399).
Conclusion: Our results indicate an association between tinnitus and increased N2% in TMJD participants reporting sleep disturbance and depressive symptoms. The effect of tinnitus on objective sleep parameters, in the context of depressive symptoms warrants further study.
Support (If Any): This research study was supported financially by the NIH Grant R01 DE019731 (Haythornthwaite, JA and Smith, MT).[MR1] [MR1]Don't include this in the main body, it'll use too many words. If there's a section on the submission portal to include this, add it. If not can just put the grant reference (as one word) in brackets after "treatment trial"

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THE EFFECT OF ANTIDEPRESSANT MEDICATIONS ON SLEEP ARCHITECTURE IN A PRIMARILY MIDDLE-AGED SAMPLE OF WOMEN WITH MULTI-MORBIDITIES: CHRONIC INSOMNIA, CHRONIC PAIN, AND DEPRESSION

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Introduction: Depression commonly impacts sleep architecture by increasing REM and decreasing stage 3 sleep. Growing evidence suggests antidepressants may reduce or reverse those effects in depressed individuals but has largely ignored their impact in the context of multi-morbidities. Depression, chronic insomnia, and chronic pain are common in middle-aged women and are thought to share a common neurobiological basis. They often co-occur, and thus, represent a common multi-morbidity triad. Here we examine whether %REM and %stage 3 sleep differ as a function of antidepressant use (yes/no) in a primarily middle-aged sample of women with all three morbidities.
Methods: Female adults (18+ years, n=91, Mage=51, SD=10.16) with comorbid insomnia, chronic pain, and depression (≥17 BDI-II score) completed one night of ambulatory polysomnography and reported antidepressant medication use (yes/no) and type (SSRI/SNRI/SARI/TCA/NDRI) as part of baseline data collection for a RCT (SPIN, NCT02688569). ANCOVA (R v4.1.1) examined group differences (antidepressant use: yes/no) in %REM and %stage3 sleep, controlling for age and sleep medication use.
Results: Only %REM was significantly lower (F=6.213, p=.015) in antidepressant users (n=48, M=14.85, SD=9.38) versus non-users (n=43, M=19.23, SD=6.70). An exploratory follow-up ANCOVA examined whether antidepressant type was important. Two groups were formed based on antidepressant mechanism of action (medications affecting serotonin, i.e., SSRI/SNRI/SARI vs medications affecting other neurotransmitters, i.e., TCA/NDRI), and their means were compared. Only %stage3 sleep was significantly lower (F=7.937, p=.007) in SSRI/SNRI/SARI users (n=37, M=11.18, SD=9.28) versus TCA/NDRI users (n=11, M=19.36, SD=13.04).
Conclusion: Thus, general antidepressant medication use may help decrease REM, but the increase of stage 3 sleep depended on which medications were used (i.e., TCA/NDRI). These findings suggest a neurophysiological functioning difference between types of antidepressants, particularly for this population of women with chronic pain, insomnia, and depression. This difference may be due to SSRI/SNRI/SARIs impacting serotonin, which may have an impact on REM, but not stage 3 sleep. However, the current study only had a small group of people using TCA/NDRIs, limiting generalizability and inferences. Future longitudinal research with a larger sample size is needed to parse out the effects of each antidepressant medication type individually and over time.
Support (If Any): ClinicalTrials-NCT02688569, PI McCrae.

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THE ROLE OF HYPERSOMNOLENCE IN DEPRESSION: RESULTS FROM A LONGITUDINAL STUDY OF THE AMERICAN GENERAL POPULATION

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Introduction: Insomnia symptoms have often been pointed out as an important factor for the relapse and/or the maintenance of depression. However, the contribution of hypersomnolence in depression is less studied and the conclusions are unclear. Our aim is to examine the association between hypersomnolence and depression in the US general population.
Methods: This longitudinal study was carried out in eight states in the U.S. A total of 12,218 subjects were interviewed by phone during the first wave (W1) and 10,931 at the second wave (W2) three years apart. The analyses included only the subjects who participated in the 2 waves (N=10,931). Hypersomnolence symptoms and Major Depressive Disorder (MDD) were assessed according to DSM-5 criteria.
Results: At W1, 27.2% (95% CI 26.4%-28.0%) of the sample had at least one hypersomnolence symptoms listed in the DSM5 occurring at least 3 days per week. This prevalence at W2 was 26.4% (95% CI 25.6%-27.2%). The incidence of DSM5 hypersomnolence symptoms was 20.5% which represents a yearly incidence of 5.8%. For MDD, the prevalence was 5.1% (95% CI 4.7%-5.5%) and 4.2% (95% CI 3.8%-4.6%) in W1 and W2, respectively. At W1, 54.2% of participants with a MDD also had hypersomnolence. At W2, 50.7% of MDD subjects also reported hypersomnolence. After adjusting for age, gender and medical conditions, persistent hypersomnolence (i.e., reported at both interviews) predicted incident MDD (i.e., absent at the first interview but present at the follow-up) with a relative risk