

initiation of arousals of ≥ 5 minutes were noted and then the first five minutes were 'acti-blipped' for each night. These were cross referenced with each other for awakening in the 5-min a) epoch before (-1), b) same epoch (E-E), and c) epoch after (+1). Hit-rates were calculated as \sqrt{xy} , where x =proportion of child acti-blips matched to parents and y =parent acti-blips matched to child's acti-blips. Combined 5-minute blip hit-rates ($E-E \pm 1$) were calculated for 13 true and five pseudo parent-child dyads.

Results: Sleep concordance ranged from 58–82% ($M=71.42 \pm 9.37$). Mann Whitney U-test revealed significant differences in a) combined hit-rate ($E-E \pm 1$) between true ($M=.30 \pm .10$) and pseudo ($M=.10 \pm .04$) parent-child dyads ($U=.00$ $p=.001$), and b) between percentage of parent awakening within 10 minutes of child's night awakening ($M=33.50 \pm 20.75$) compared to percentage of child awakening within 10 minutes of parent's ($M=10.83 \pm 3.60$; $U=11$, $p < .001$). During the 14-nights, 29% of parent awakenings occurred within 5 mins of their child's awakening. In contrast, only 6% of child's awakenings occurring within 5-min of their parents awakening.

Conclusions: Preliminary evidence demonstrates strong sleep concordance in parent-child dyads. Parents have child related night-awakenings, which can make them vulnerable to insomnia. Future studies should address possible interventions for these parents.

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Pharmacology

IMPACT OF QUETIAPINE OVER SLEEP ARCHITECTURE- A REVIEW OF CURRENT DATA

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Introduction: Quetiapine is recommended quite frequently by clinicians for the treatment of insomnia associated to different psychiatric disorders and even for primary insomnia, although clear recommendations for its use in these cases are lacking from specific guidelines. If the anti-histaminic properties of low-dose quetiapine are confirmed by basic pharmacological studies, one should consider both beneficial effects and adverse effects that could arise from this pharmacodynamic profile.

Materials and methods: A search of main electronic databases (PubMed, Cochrane, CINAHL, Embase, Thomson Reuters/Web of Science) was performed using as paradigm „quetiapine” OR „quetiapine XR” AND „sleep architecture”, „REM sleep”, „non-REM sleep”, „insomnia” OR „sleep disorders”. All papers published between 2000 and 2018 were collected and filtered out non-significant citations based on pre-defined inclusion/exclusion criteria. No limitations regarding the language of published papers, age, primary diagnosis or specific comorbidities were formulated.

Results: Quetiapine and other atypical antipsychotics as augmentation therapy or monotherapy to unipolar and bipolar disorder patients have been shown to improve sleep continuity and sleep architecture. Quetiapine showed in polysomnographic studies in patients diagnosed with schizophrenia that it can increase the sleep latency, wake time after sleep onset, and REM sleep latency, while increasing total sleep time and sleep latency in healthy subjects. A randomized clinical trial showed that under acoustic stress quetiapine increased total sleep time by half an hour and reduced the number of awakenings by 35–40% compared to placebo and similar to mirtazapine. In the same trial, low doses of quetiapine specifically increased the duration of non-rapid eye movement sleep (N2) and induced daytime sleepiness and lessened sustained attention in patients with transient insomnia. In alcohol-dependent patients with insomnia quetiapine had no effect on sleep efficiency, but reduced awakenings after sleep onset and increased non-significantly sleep onset latency and stage 2 sleep time. Quetiapine XR improved sleep quality in elderly patients with major depressive disorder, and in midlife women with the same disorder. Quetiapine XR also improved sleep quality in patients with generalised anxiety disorder (monotherapy) and in those diagnosed with fibromyalgia (as add-on therapy).

Conclusions: Quetiapine had positive effects over insomnia associated with major depressive disorder, bipolar disorder, fibromyalgia, generalised anxiety disorder, schizophrenia, but larger, better-designed trials using polysomnography should be realised in order to confirmed the impact of this antipsychotic over sleep architecture in both primary and secondary insomnia. The risk for daytime sleepiness and metabolic adverse effects should be considered whenever this antipsychotic is recommended.

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Pharmacology

TRAZODONE AS ADD-ON FOR RESIDUAL INSOMNIA ASSOCIATED TO SEVERE MAJOR DEPRESSIVE DISORDER IN ELDERLY PATIENTS

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Introduction: Residual symptoms of severe major depressive disorder are important predictors for relapse and they are associated with poorer quality of life, lower functionality, and risk for contracting new psychiatric disorders (e.g., alcohol or benzodiazepine dependence). Insomnia is one of the most frequently reported residual symptom in major depression, and this phenomenon has attracted complex pharmacological and psychotherapeutic-oriented interventions, which have been associated with various degrees of success.

Materials and methods: Five patients, mean age 71.5 years, diagnosed with major depressive disorder have been treated during their last episode (severe, without psychotic features) with either a serotonin selective reuptake inhibitor (sertraline, fluoxetine or escitalopram), or a serotonin and norepinephrine reuptake inhibitor (venlafaxine or duloxetine). All these patients were evaluated after 4, 8, 12, 16, 20 and 24 weeks since the treatment onset and both their global clinical status and their Geriatric Depression Scale (GDS-30) improved significantly after 24 weeks. Also, their overall functionality, evaluated through Global Assessment of Functionality Scale (GAF) improved considerably (+65% to baseline). However, all these patients accused the persistence of difficulties in falling asleep, combined with multiple awakenings during the night, observed for the majority of nights within a normal week. The self-rated discomfort associated with sleep problems was evaluated as moderate to severe (6.8 on a 10-point visual analogic scale-VAS). A clinical thoroughly examination was performed and other organic or toxic causes for sleep problems were excluded. A flexible dose of trazodone (25–100 mg QD) was recommended as add-on to their current treatment, in order to manage residual insomnia.

Results: Patients reported improved quality of sleep and the VAS score declined to a mean value of 4.6 after 7 days. **This improvement persisted after 14 and 28 days**, without significant fluctuations on GAF, GDS-30, or CGI-S. Two patients discontinued treatment with trazodone after 28 days, but in the other 3 cases, trazodone was administered as add-on for the remaining of the antidepressant treatment. In one case trazodone daily dose was increased up to 200 mg. No significant adverse events were reported during the treatment.

Conclusions: Continuous monitoring of the sleep quality and quantity should be initiated by the treating physician in cases of major depression in elderly patients, even if the mood and other core symptoms of depression are considered in remission. **Adding trazodone is granted if residual insomnia is detected**, but individual responsibility may request dose adjustment during the entire duration of the antidepressant treatment.

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Sleep Breathing Disorders

SLEEP APNEA PATIENTS EXHIBIT INCREASED NOCTURNAL PLASMA LEVELS OF IL-6 AND TNF- α : EFFECTS OF CPAP, OXYGEN & ANTIOXIDANTS

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Introduction: Sleep apnea patients exhibit increased plasma levels of IL-6 and TNF- α and this response may be implicated in increased cardio-cerebrovascular morbidity.

Aims: We hypothesized that this inflammatory response is causatively related to the intermittent strenuous diaphragmatic contractions and the