

## 0513

**CURRENT MAJOR DEPRESSION IN OBSTRUCTIVE SLEEP APNEA SYNDROME (OSAS) RATIONALE, DESIGN AND PRELIMINARY RESULTS OF A NORWEGIAN, POPULATION-BASED STUDY***Hrubos-Strøm H<sup>1,2</sup>, Dammen T<sup>3</sup>, Nordhus P<sup>4</sup>*

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**Introduction:** OSAS is a prevalent disorder in the community associated with depression. The latest review of research concerning this association recommend further emphasis to subject selection and quality of diagnostic tools in future research. The aims of the study are: 1: To estimate the prevalence of current major depression in subjects derived from the general population with high risk of having OSAS. 2: To assess how current major depression is associated to severity of OSAS measured by the Apnea Hypopnea Index (AHI).

**Methods:** Phase-I: The Berlin Questionnaire (BQ) is designed to identify persons with high risk of OSAS based on self-reported snoring, daytime sleepiness and hypertension or obesity. A questionnaire consisting of the Norwegian version of the BQ and additional questions was mailed to 30 000 subjects. The draw was stratified in gender and age from 30 to 65 years in subjects residing in Akershus, Hedmark or Oppland counties, registered in the Norwegian National Register. Phase-II: A random sample of 292 subjects with high risk of having OSAS were interviewed with the Structured Clinical Interview for DSM-IV, axis-I disorders (SCID-I) and completed a full night polysomnography after the interview. OSAS diagnosis was based on AHI > 5 plus self-reported excessive daytime sleepiness or AHI >15 alone.

**Results:** The phase-I response-rate was 54%. Among subjects eligible for further investigations, 23% had high risk and 77% low risk for OSAS. Preliminary results indicated a point prevalence of current major depression of 12%. This estimate is 3-4 times higher than estimates for published for current major depression in the general population. Current major depression was not associated with severity of OSAS measured by AHI.

**Conclusion:** In a population based sample of subjects with high risk of having OSAS, current major depression was highly prevalent, but not associated with severity of AHI.

## 0514

**THE SLEEP SYMPTOMS CHECKLIST: USEFUL FOR SCREENING BUT NOT DIAGNOSIS***Bailes S<sup>1</sup>, Baltzan M<sup>2,4</sup>, Dorrie R<sup>1</sup>, Fichten C<sup>1,5</sup>, Grad R<sup>2,4</sup>, Libman E<sup>1,4</sup>*

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**Introduction:** We have demonstrated that the Sleep Symptoms Checklist (SSC) can identify some primary care patients at risk for sleep apnea. The present study seeks to determine if the SSC can distinguish patients with and without apnea in (a) a sample of physician-referred sleep clinic patients, and (b) using a conceptually similar questionnaire format, an older community sample of sleepy/tired individuals with sleep problems.

**Methods:** Participants included the Sleep Clinic (N=95, Mean Age=52), Sleepy/Tired Older Community (N=107, Mean Age=62), and Control (N=21, Mean Age=42) samples. The Sleep Clinic and Control samples were administered the SSC, a 21-item screening instrument including signs and symptoms of sleep disorders reduced to 4 factors: SSCSleep-Disorder, SSCDaytimeDistress, SSCInsomnia, SSCPsychological. The

Older Community and Control samples completed an extensive questionnaire battery (Q). A subset of 21 Q items, selected to match items of the SSC, have a similar, 5-factor structure: QSleepDisorder, QFatigue, QSleepiness, QInsomnia, QPsychological. All participants underwent nocturnal screening for sleep apnea.

**Results:** In the Sleep Clinic sample, all referred patients (with and without diagnosed apnea) had significantly higher scores on the SSCSleep-Disorder, SSCDaytime and SSCInsomnia subscales than healthy Controls. No differences were found between those with and without apnea. Similarly, in the Older Community sample, no significant differences were obtained between those with and without apnea, but both groups were significantly more symptomatic on 4 of the 5 Q factors than the healthy controls.

**Conclusion:** The SSC can identify those individuals at risk for sleep apnea, who should be evaluated with polysomnography. It cannot be used to definitively diagnose sleep apnea. A similar pattern of results was obtained in the physician-referred Sleep Clinic and the Community samples using a different questionnaire format. This indicates that it is the symptom profiles themselves, independent of questionnaire format, that identify individuals at high risk for sleep apnea.

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## 0515

**URINE PH DOES NOT DECREASE IN RESPONSE TO OBSTRUCTIVE SLEEP APNEA***Watenpaugh DE<sup>1,2</sup>, Dao D<sup>1</sup>, Burk JR<sup>1</sup>*

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**Introduction:** Nightlong compromised ventilation associated with obstructive sleep apnea (OSA) leads to sustained nocturnal hypercapnia and possibly respiratory acidosis. Such acidosis should in turn elicit renal compensation with reduced morning urine pH relative to pH at bedtime. Because positive airway pressure (PAP) normalizes nocturnal respiration in OSA, one would expect PAP treatment to counteract any OSA-induced nocturnal urine pH reduction. We hypothesized that: 1) morning urine pH would decrease relative to urine pH measured prior to bedtime in untreated OSA patients, and 2) treatment with PAP prevents this nocturnal urine pH reduction.

**Methods:** Patients (137 total) had mild to severe OSA. Urine samples were collected before and after full-night diagnostic nocturnal polysomnography (NPSG) and full-night PAP titration. Patients provided a urine sample prior to bedtime and from the first morning void. NPSG always occurred before PAP titration, but studies did not occur on consecutive nights. Color-indicator dipsticks quantified urine pH to +/- 0.5 units. There was no control or monitoring of diet, electrolyte metabolism, or fluid balance.

**Results:** No evening-to-morning change in urine pH occurred during NPSG or PAP titration. Urine pH averaged (SD): 5.8 (0.8) before NPSG, 5.9 (0.8) after NPSG, 5.6 (0.7) before PAP, and 5.7 (0.7) after PAP.

**Conclusion:** The data refuted our hypotheses: urine pH failed to decrease after a night with untreated OSA, and PAP treatment had no affect on this finding. These results suggest that no significant respiratory acidosis occurs during a night with untreated OSA, or that no renal compensation occurs in response to such acidosis. Furthermore, the findings do not support use of urine pH as an indirect indicator of the hypercapnia associated with OSA.