

analysis of study group was conducted and subgroups were compared with control groups after adjusting age, gender and BMI. Regarding presence of OSAS, there was no significant difference between e-GFR>60 ml/min ADPKD patients and control group ($p=0,759$). However, there was significant difference between e-GFR<60 ml/min ADPKD patients and control group ($p=0,018$). Regarding effect of RAS blockage on frequency of OSAS in hypertensive ADPKD patients, there was no significant difference in terms of OSAS between patients using ACE-I/ARB compare to patients not using RAS blockers (18/27(66,6%), 3/5(60%), respectively; $p=0,77$)

Conclusions: It is well known that ESRD (e-GFR<15 ml/min) is associated with sleep disorders. In our study, we showed that ADPKD patients with CKF(e-GFR 15-60 ml/min) had higher rate of OSAS compare to non-CKF patients and healthy control group. As conclusion, uremia progression of rather than RAS activation seems to play a role for OSAS in ADPKD patients.

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HEARTBEAT-RELATED RESPONSES OF FRONTAL CORTICAL NEURONS IN THE SLEEP-WAKE CYCLE IN CATS

V. Lavrova¹. ¹ Institute for Information Transmission Problems (Kharkevich Institute) RAS, Moscow, Russian Federation

Introduction: The visceral theory of sleep (Pigarev, 2013) assumes that the cerebral cortex switch to the analysis of interoceptive information coming from visceral organs during sleep. This was first confirmed in gastrointestinal tract researches, when cortical responses related to its activity were actually detected in visual cortical areas during sleep. Moreover, we found some sleep-related responses for cardiac activity on iEEG and local field potentials (LFPs), which appeared in normal sleep in frontal and insular cortical region. This study aimed to explore heartbeat-related activation of single neurons in frontal cortex regions during sleep-wake cycle.

Materials and Methods: In two adult cats, LFP and neuronal firing were recorded with transcranial intracerebral bipolar microelectrodes from frontal cortex. Electrodes' placement was selected according to pre-existing assumptions about the possible whereabouts of cortical areas related to heart activity. ECG was recorded with two electrodes located in the stomach and on the cats head. We recorded iEEG, breath rhythm and eye movements as well, to identify the sleep phases. Our analysis included 2-5 hours records, with periods of wake, normal NREM and REM sleep. The processing and statistical analysis were made with Spike2 CED, including special self-made scripts.

Results: In 20 records, we marked out over 120 single neurons. Heartbeat-related responses as changes of neuronal firing were found in 32,4%, in frontal cortex of both hemispheres. This connection between neuronal firing and cardiac activity appeared during slow-wave sleep but was not observed in wakefulness.

Conclusions: Now we see that information related to cardiac activity reaches cerebral cortex during sleep indeed. Our results confirm that cerebral cortex becomes visceral-analyzing during sleep, and this special brain-heart axis develops information in sleep in order to restore the somatic functionality of all the body organ systems.

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HUMAN SERUM PROTEIN CHANGES AFTER 6 H OF SLEEP DEPRIVATION INVESTIGATED WITH NEWER PROTEOMIC METHODS

A.A. Bjørkum¹, J.T. Olsen¹, H. Hvesser¹, N. Omar¹, F. Berven^{2,3}, E. Birkeland^{2,3}. ¹ Western Norway University of Applied Sciences, Department of Safety, Chemistry and Biomedical Laboratory Sciences, Bergen, Norway; ² University of Bergen, The Department of Biomedicine, Bergen, Norway; ³ The Proteomics Unit at the University of Bergen (PROBE), Bergen, Norway

Introduction: Sleep-wake associated studies using omic-methodology are increasing (O'Callaghan et al. 2019). Studying effects of partial sleep deprivation (SD) at night using proteomics- and systems biological approach has been sparse (Mauvoisin, 2019 and Noya et al. 2019). Earlier finding revealed changes in 34 proteins in human blood serum after 6 hour

of sleep deprivation at night (Bjørkum et al. 2021). The aim of this study was to further identify differentially expressed proteins in human blood serum after loss of 6 h sleep at night using newer proteomic methods and exploring systems biological databases.

Materials and Methods: In a within subject-design-study a control night were the participants (n=6 females) slept from 10:00 pm to 07:00 am and the following night sleep deprivation (SD) was performed from 10.00 pm to 04:00 am. Sleep/wake data can be found in Bjørkum et al. 2021. Venous blood was sampled at 4:00 am. Proteins from blood serum was heat denatured at 95°C for 5min, prior to reduction (DiThioThreitol) and alkylation (Iodoacetamide). Denatured proteins were digested overnight (16h) at 37°C and desalted using Oasis (waters) spin columns. Desalted proteins were lyophilized and dissolved in HEPES buffer (pH 8.5). TMT-labels were added to each sample (16plex, ThermoFisher), and desalted and lyophilized prior to high-pH fraction using an offline HPLC (Waters, HPLC). The samples were run a Orbitrap Exploris massspectrometer (ThermoFisher) coupled to an Ultimate 3000 HPLC. Raw-files were search against the Swissprot database using Proteome Discoverer 2.5. Further analysis of the data was performed in Perseus. Gene ontology analysis were performed using Gene Set Enrichment Analysis, Omim, Webgestalt.

Results: We identified 590 proteins, 63 proteins were differentially expressed, 25 upregulated and 38 downregulated. The 63 proteins took part in 229 biological processes and 31 molecular functions.

The differentially expressed proteins after 6 hours of sleep deprivation at night could be linked to affected biological processes such as e.g., immune-, coagulation- and metabolic related cellular processes. Also, proteins associated with pathological conditions such as cardiovascular- and dementia related diseases and various types of cancer were affected.

Earlier published omic-studies after lack of sleep indicate cellular stress reflected in a distinctly changed serum proteome by identifying specific protein markers to reveal distinctly affected biological processes, molecular functions, cellular pathways, DNA damage and repair and disease related proteins after sleep deprivation (see refs. In Bjørkum et al. 2021). Impaired immune system and diseases associated with sleep deprivation have been reported (Bjørkum et al. 2021, Ma et al, 2018; O'Callaghan et al. 2019, Pellegrino et al., 2012 and Pinotti et al., 2010).

Conclusions: Acute sleep deprivation as little as 6h at night, at least in females, affects several differential expressed proteins taking part in several distinct biological processes- and molecular function categories. Also, the differentially expressed proteins are related to pathological associated conditions like impaired coagulation, oxidative stress, inflammation and immune suppression, neurodegenerative related disorders, and cancer. This is in line with earlier studies from our group (Bjørkum et al. 2021).

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HYPOCRETIN RELEASE AND PLASTICITY OF HYPOCRETINERGIC RECEPTORS IN A PHARMACOLOGICAL MODEL WITH NARCOLEPSY-LIKE FEATURES INDUCED BY SUVOREXANT IN RATS

C. Carrera-Cañas^{1,2}, M. Garzón¹, M. Callejo¹, I. De Andrés¹. ¹ Universidad Autónoma de Madrid, Anatomy, Histology and Neuroscience, Madrid, Spain; ² Tatiana Pérez de Guzmán el Bueno Foundation, Predoctoral Fellow, Madrid, Spain

Introduction: The hypocretinergic (Hrct) system is a neuromodulatory network involved in many physiological processes among which is the control of the sleep-wake cycle. This system comprises two excitatory hypothalamic neuropeptides -Hrct1 and Hrct2 (or orexins A/B)- and two G-protein-coupled receptors -HrctR1 and HrctR2- widely distributed throughout the central nervous system. Malfunction of this system is related to narcolepsy. Low or undetectable levels of Hrct1 in cerebrospinal fluid (CSF) constitutes a diagnostic criterion for Narcolepsy Type I. In the present study we have used Suvorexant, a dual Hrct receptor antagonist, to obtain a pharmacological experimental model with Narcolepsy-like features in rats by blocking the two Hrct receptors. In this model we have explored CSF Hrct1 levels and HrctR1 and HrctR2 expression within the hypothalamus.

Materials and Methods: In three groups of 8 rats daily i.p. injections of Suvorexant (10 or 30 mg/kg doses) or vehicle (DMSO) were done in the