as well as defense response (DR) paradigms. In order to determine the contributory role of noradrenergic (NA) and dopaminergic (DA) systems to these parameters, a pharmacological challenge approach was applied.

In this study, 14 normal subjects received placebo, the DA agonist bromocriptine (2.5 mg p. m.) and the NA agonist clonidine ($150 \mu g i. v.$). Only one compound was given at a particular lab session, the 3 lab tests being separated by 1-week intervals. On each test day, a series of 3 experimental conditions was presented before and again 60min after application of the challenge compound: a 5-min baseline, an OR paradigm (10 tones: 1000 Hz, 70 dB, 1 s), and a DR paradigm (10 tones: noise, 102 dB, 1 s). The following psychophysiological channels were monitored: EDA, ECG, fingerpulse volume, respiration, EOG, and EEG.

Three major findings emerged from pre-/postchallenge comparisons: 1) As expected, tonic heart rate showed a significant decrease (P < 0.01) under clonidine, attesting to NA sources in the control of this autonomic measure. 2) Blink rates displayed a trend (P < 0.10) towards increase under bromocriptine, pointing to a status as marker for DA activity. 3) With regard to ED measures, both skin conductance level (P < 0.07) and number of ORs/DRs (P < 0.01) showed decreases under clonidine challenge. These findings indicate that electrodermal parameters primarily reflect changes in NA metabolism and/or at NA receptor sites.

Finally, it should be mentioned that on placebo days the psychophysiological measures examined showed a rather inconsistent pattern.

Assessment of P300 with varying stimulus intensities of pure olfactory odorants

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The occurrence of the late cognitive component P300 is not only dependent on stimulus qualities but also on stimulus probability and the subjective task relevance. Moreover, using olfactory stimuli, the recording of P300 could reveal useful information about the varying capability of human olfaction in and between subjects. Olfactory stimuli presented within a constant flow airstream (Kobal, 1981, *Elektrophysiologische Untersuchungen des menschlichen Geruchssinns*.

Stuttgart: Thieme) were used in a discriminative task.

Five female subjects (non-smokers who did not take any drugs or oral contraceptives with a regular menstrual cycle) ranging in age from 20 to 35 years participated twice in this experiment: Before and after their ovulation, taking into account the individual length of their menstrual cycle. To assess the general ability to perceive odors we determined the olfactory thresholds of 5 different odorants (Linalool, Menthol, Citral, Isoamylacetate, and Androstenone) for each subject a few days before the EEG sessions. Probes were presented in random order using the staircase method of limits. Before recording EEG data the threshold for Citral was again determined separately for each nostril. For EEG recording and later ERP analysis stimuli were presented to the nostril with the lower threshold. Each session consisted of two sets of 100 trials. ERP data were collected from Fz. Cz. and Pz electrodes referred to linked mastoids. The duration of stimulus presentation was 200 ms, mean ISI 25 s, as short ISIs of about 10 s drastically augmented the habituation effect. Olfactory stimuli were presented in an oddball paradigm using low concentrated Citral as a frequent stimulus and high concentrated Citral as a target stimulus (P = 0.16). The subject's task consisted of responding to the target stimuli by lifting a finger, 3.5 s after stimulus onset indicated by a 500 Hz tone. Correctness of response and reaction time were measured. Moreover, target stimuli had to be counted.

Results show that rare olfactory stimuli differing only in concentration can evoke a P300 component its maximum appearing approximately 600 ms after the N1 component of the olfactory evoked potential. Differences in latencies and amplitudes can provide valuable information when looking for variations in olfactory sensitivity, e.g., due to the hormonal status of subjects.

Sleep-related variations of immune-functions

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Sleep may influence immune functions. Plasma levels of cytokines vary with sleep or sleep deprivation. Levels of interleukin-1alpha (IL-1–) rise