ing, particularly; it has been shown that amyloid can cause reduction of glutamatergic transmission and inhibition of synaptic plasticity via increased endocytosis of NMDA receptors. Trace Amines (TAs) are a family of endogenous compounds with strong structural similarity to the classical monoamine neurotransmitters. The molecular mechanism of the TAs involves binding to a novel G protein-coupled receptor, called TAAR (trace amine-associated receptor). TAAR1 is distributed in the CNS. Recently, it has been shown that selective activation of TAAR1 are able to reverse glutamatergic hypofunction induced by selective NMDA receptor antagonists suggesting that TAAR1 activation may enhance also glutamatergic function. There are several lines of evidence suggesting pro-cognitive action of TAAR1 agonists in various behavioral experimental protocols and there is evidence indicating that TAAR1 can modulate frontal cortex glutamate NMDA receptor- related functions [2,3,4,5].

**Objectives:** 1. To study in vitro the role of TAAR1 agonists on basal cortical glutamatergic transmission and their beneficial effect on Ab-induced dysfunction.

2. To study, in vivo, the role of TAAR1 in cognitive dysfunction induced by Ab and the beneficial role of TAAR1 agonists on cognition in Alzheimer's mouse models.

**Methods:** In vitro experiments were conducted on primary cortical cultures. Cortices of E17 embryo from TAAR1 and control mice were isolated and incubated for 14 days at 37°C and 5% CO2. Cells were then stimulated with Ab 1-42 (1 $\mu$M, AnaSpec, USA), TAAR1 agonist (RO5256390, Sigma Aldrich, Belgium, 1 $\mu$M) or both 1hr at 37°C and NMDA surface expression was assessed using biotinylation assay and Western blots. In vivo studies were performed using 10-weeks mice ICV injected with: Ab 1-42 (3 $\mu$l), TAAR1 agonist (3$\mu$l) or both and vehicle treated controls. 7 days later, a series of behavioral tests were performed to evaluate the effects of Ab 1-42 and TAAR1 agonist, including Morris Water Maze (MWM), novel object recognition (NOR) and open field.

**Results:** In vitro data showed that, as expected in WT mice, Ab 1-42 significantly decreased NMDA surface (NR1: -35$\pm$ 2.6%; NR2A: -38$\pm$ 1.8%; NR2B: -47$\pm$ 4.2%) expression while TAAR1 agonist promotes their membrane localization (NR1: +48$\pm$4.8%; NR2A: +67$\pm$3.5%; NR2B: +52$\pm$3.8% $p<0.05$, Student t test) on cortical cells.

**Conclusion:** Altogether, our results showed that in vitro, TAAR1 agonist displayed the ability of increasing NMDA receptors surface expression, suggesting the possibility of displaying therapeutic effect on cognitive Ab induced impairments. Whether these effects are reproducible in vivo, are currently addressed.

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## P.480 The effects of propolis extract on age-associated cognitive deficits in rats

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**Background:** Propolis is widely used as alternative medicinal product due to their antimicrobial, antiinflammatory and antioxidant properties. Previous studies have shown that propolis has a neuroprotective effect and alleviates the cognitive impairments in scopolamine or beta-amyloid induced learning and memory impairment animal models [1,2]. The incidence of the physiological aging associated neurodegenerative diseases characterized by memory loss and dementia is increasing. The aim of this study is to evaluate the effect of chronic propolis administration on cognitive dysfunctions following physiological aging processes.

**Methods:** In this study, male Wistar rats were divided into 4 groups (n=10 for each group): young-control (YC-6 months), young-propolis (YP-6 months), old-control (OC-24 months), old-propolis(OP-24 months). The water-soluble form of propolis will be prepared from fresh Turkish propolis (Aksuvital Natural Products Company). The main components in this extract will be identified by Gas Cromathography-Mass Spectrometry (GC-MS) analysis. The extract of propolis (100 mg/kg) was administered orally for 28 consecutive days to YP and OP groups. At the end of 28 days period, locomotor activities, passive avoidance and elevated plus maze tests were performed respectively. In passive avoidance apparatus, which measures emotional memory, acquisition (on day 1), and retention (on day 2) trials were carried out. In acquisition trial, an electric foot-shock was delivered to the animal via grid floor. The time taken for animals to enter the dark compartment was recorded as the training latency. Retention latency was evaluated 24-h after acquisition trial. In EPM test, which measures spatial memory, acquisition (on day 1) and retention (on day 2) sessions were performed. Transfer latency (the time in which the animal moves from the open arm to the enclosed arm) was utilized as an index of learning and memory processes. The rats were placed into the open arm and the transfer latency was recorded for both days. The results of the study were evaluated by one way ANOVA post hoc Tukey test. The data were considered to be significant statistically if the probability had a value of 0.05 or less.

**Results:** There is no statistically significant differences between the first day transfer latencies in all groups in passive avoidance and elevated plus maze tests. The retention latencies in passive avoidance test significantly reduced in physiologically aged, OC group compared to the YC group ($p<0.05$). Also, the second day transfer latencies in elevated plus maze test increased in OC group compared to the young controls ($p<0.05$). The results of both behavioral tests shown the development of cognitive dysfunction because of the physiological aging. After chronic propolis administration, both latencies in OP group reversed to the young control levels.
**Conclusion:** The results of the study have shown that the chronic propolis extract administration may prevent the emotional and spatial memory impairment during physiological aging. Hence the propolis extract could be considered as a new strategy to prevent or slow down the development of cognitive dysfunctions following physiological aging processes. However, further studies are needed to explore the biological mechanisms of propolis and to support these findings.

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### P.481 Increased risk-taking behaviour following a loss by women with obesity is associated with ventromedial prefrontal cortex and insula alterations

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**Background:** Obesity is linked with increased risk-taking behavior in terms of food choices. Multiple studies have suggested that altered activity in interoceptive and decision-making circuitry may underlie the heightened risk-taking found in obesity. Whether this risky behavior is driven by dysfunctional salience processing or a lack of appreciation of danger, or both, remains unclear.
**Aim:** In the study at hand we aimed to describe the brain regions purportedly underpinning the increase risk-taking behavior in obesity by assessing a group of adult women with obesity in comparison with healthy weight controls during the performance of a task assessing risk behavior in a food-independent context.
**Methods:** Twenty-three adult women with obesity and twenty-three age-matched, healthy weight controls completed the Risky Gains Task during a 3T functional magnetic resonance imaging scan. The Risky Gains Task consists of participants choosing between a safe option for a small, guaranteed monetary reward and risky options with larger rewards but also a higher chance of losing money. The frequency of risky choice overall and following a winning trial versus a losing trial were compared. Likewise, brain responses during winning trials vs. loses, and during risk vs. safe decisions (overall and following winning and losing trials) were also compared between both groups. The UPPS-P questionnaire was completed by all participants to assess impulsivity trait levels.
**Results:** As expected, the body mass index (BMI) of the obesity group (mean=$43.59\pm6.98$) was higher than the control group (mean=$20.93\pm6.98$; $p<0.001$). Regarding psychometric assessments, there were no significant between-group differences in UPPS-P scores, although we observed trending-level differences in particular UPPS-P subscales. Specifically, scores in lack of perseverance tended to be reduced in the obesity group ($p=0.07$), while scores in sensation seeking tended to be higher in obese subjects ($p=0.08$). Neuroimaging analyses demonstrated that participants with obesity showed decreased activity in the right anterior insula during losing trials in comparison to the control group ($p<0.05$, AlphaSim corrected), suggesting that the insula plays a role in giving more weight to positive response to reward over the impact of a loss. In addition, right insula activation during losses was negatively correlated with UPPS-P sensation seeking scores ($p=0.30$, $r=-0.320$). During safe trials following a loss, participants in the obese group presented decreased activation in the ventromedial prefrontal cortex (vmPFC) ($p<0.05$, AlphaSim corrected). vmPFC response during post-loss trials was positively correlated with safer choices on the task overall ($p=0.14$, $r=-0.371$), indicating the vmPFC may underpin the assessment of risk-taking behaviors.
**Conclusions:** The brains of individuals with obesity may be hyposensitive to interoceptive cues stemming from losses, thereby bringing about increased risk-taking behaviors. In addition, disrupted tuning of the insula towards interoceptive signals may lead to a lack of input to the vmPFC when weighing the costs and benefits of risky choices. This could partly explain why individuals with obesity overeat despite harmful outcomes.

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### P.482 Olfactory neurons as surrogate tissue to investigate disorders of the brain

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