Table 1. Results of neuropsychological tests.

Scale	CSII group	MDII group
MMSE	30 (30; 30)	29 (27; 30)
MoCA	29 (28; 30)*	26 (24; 28)*
TMT-A	47,5 (38; 65)*	85 (60; 96)*
TMT-B	90 (60; 120)*	145,5 (115; 180)*
Test of correction	0,67 (0,67; 0,81)*	0,68 (0,6; 0,8)*
Benton's similarity test	24 (22; 26)*	18 (16; 23)*
Words memorising test	a) 6 (4; 7)*	a) 4 (4; 5)*
a) 1 trying	b) 5 (5; 7)*	b) 9,5 (8; 12)*
b) number of tryingsc) after 30 min	c) 8 (6; 8)*	c) 5 (4; 6)*
HADSanxiety	4 (3;6)*	7 (5; 10)*
HADSdepression	3 (2; 4)*	4 (3;6)*
SF-36		
Physical component of health	52,7 (49,2; 55,3)*	44,7 (41,4; 52,4)*
Mental component of health	52,2 (41,3; 54,6)*	37,5 (35,5; 45,2)*
4DSQ distress	5 (3; 6)*	10 (5; 13)*
4DSGdepression	0 (0; 1)*	2 (0; 3)*
4DSQanxiety	1 (0; 2)*	4 (2; 8)*
4DSQsomatisation	5 (3; 9)*	9 (6; 11)*

* ($p<0.05$)

P.478 One-year change in telomere length and cognitive function in the context of HIV, childhood trauma and major depressive disorder

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Background: HIV-associated neurocognitive disorder (HAND) refers to a spectrum of motor, cognitive and behavioural difficulties that may occur consequent to HIV infection. In spite of improved access to antiretroviral therapies, the prevalence of HAND remains high. The physical, psychological and social consequences of HAND are particularly concerning in sub-Saharan Africa, where the burden of HIV infection is highest. The risk of developing HAND is likely influenced by gene-environment interactions. Previous research from our group suggested that the interaction of childhood trauma (CT) and depression may increase the risk of neurocognitive decline, and that telomere length (TL) attrition, a marker of biological aging, may mediate this relationship. We expanded our investigation of whether change in TL is predictive of declining neurocognitive function.
Methods: Sixty-one HIV-positive and 49 HIV-negative women were recruited from community health care facilities in and around Cape Town, South Africa. Consenting participants underwent psychiatric diagnostic and neurocognitive assessments, which were administered by a trained research psychologist. Study participants completed the HIV Neurobehavioral Research Center Neuropsychological battery, which uses seventeen tests to cover seven domains of cognitive function: motor skill, verbal fluency, attention and working memory, processing speed, learning, recall, and executive function, from which a global age- and education-adjusted cognitive score was calculated. The Childhood Trauma Questionnaire (CTQ) and Center for Epidemiological Studies Depression Scale (CESD) were used to assess CT experience and depressive symptoms respectively. Quantitative polymerase chain reaction using primers specific to telomeric repeats and the reference gene human β-globin was performed on DNA extracted from peripheral blood mononuclear cells. Neurocognitive tests and TL measurements were performed at baseline and at 12 months and change scores were calculated. Multiple linear regression models using the R statistical language were used to assess the relationships between HIV, CT, depression, change in TL and change in cognitive scores.
Results: Women with HIV had significantly higher CTQ and CESD scores ($p = p < 0.001$ and $p = 0.035$). Relative TL was significantly reduced in HIV-positive compared to HIV-negative women at baseline ($p = 0.022$) but did not differ at the one-year follow-up. Depressive symptoms alone, and in interaction with CT, were associated with increased TL shortening across participants ($p = 0.037$ and $p = 0.017$ respectively). HIV seropositivity was strongly associated with worsening global cognitive scores over one year ($p = 2$ x 10-4). The interaction of CT experience, HIV status and change in TL was associated with a decline in cognitive performance ($p = 0.025$).
Conclusions: HIV infection is significantly associated with CT and depressive symptom severity, as well as accelerated biological aging. Our longitudinal data support the deleterious impact of HIV on cognitive function and suggest that TL attrition is predictive of worse cognitive performance in the context of CT and HIV.

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P.479 Trace amine associate receptor 1 (TAAR1) as a new target for the treatment of cognitive dysfunction in Alzheimer disease

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Background: Alzheimer disease (AD) is the main cause of dementia with approximately 27 million people affected worldwide. Beta-Amyloid peptide (Ab) is elevated in the brains of patients with AD and is believed to be causative in the disease process. Ab can reduce long-term potentiation (LTP), a form of synaptic plasticity that is closely associated with learning and memory [1]. LTP involves postsynaptic phosphorylation and glutamate receptor traffick-

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 μM, AnaSpec, USA), TAAR1 agonist (RO5256390, Sigma Aldrich, Belgium, 1 μM) 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 μl), TAAR1 agonist (3μl) 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.