Symposium: The role of the telomere-telomerase system in psychiatric disorders and treatments: **Underlying mechanisms linking mental illness** with cellular aging

S034

Telomere length and depressive and anxiety disorders: Longitudinal associations and underlying mechanisms

B. Penninx*, J. Verhoeven VU University Medical Center, Psychiatry, Amsterdam, The Netherlands

* Corresponding author.

Many psychiatric disorders have been associated with increased risk of mortality and various aging-related somatic diseases. In addition to unhealthy lifestyles, also various stress-related physiological processes likely play a role in explaining these detrimental health consequences of psychiatric disorders. The impact could be visible at the cellular level, with psychiatric patients presenting more signals of physiological aging for instance as determined by measuring telomere length. In this talk we will first highlight the current state-of-the art evidence that various psychiatric conditions, including e.g. depression, anxiety and PTSD, are associated with shorter telomere length. Second, we will provide results from the Netherlands Study of depression and anxiety (n=2981) that tested longitudinal associations using 6 year data on psychiatric status and telomere length. These results indicate that the association between depressive and anxiety disorders with telomere length is stable over time, and doesn't show many dynamic associations. Finally, in the same study we have also tested to what extent lifestyle and dysregulations of physiological stress systems such as the immune, HPA-axis and autonomic nervous systems are partly responsible for the observed shorter telomere length in depressed or anxious patients. Results indicate that especially smoking behavior and systemic inflammation partly contribute to the shorter telomere length, but can't completely explain found associations.

In sum, this talk will highlight the current state-of-evidence for an association between various psychiatric conditions with shorter telomere length, and will provide insights into its dynamics and its contributing mechanisms.

Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.108

S035

The role of telomeres and telomerase in the clinical effect and mechanism of action of psychopharmacological interventions

F.S. Bersani

Sapienza University of Rome. Department of Neurology and Psychiatry, Roma, Italy

Originally studied in relation to aging and cancer research, telomeres and telomerase are now also investigated in relation to psychiatric disorders and treatments. Based on findings emerging from clinical and preclinical data, we hypothesize that the telomere-telomerase system represents a novel element mediating the mechanism of action of certain psychopharmacological interventions.

In this symposium I'll present the preliminary evidence on the complex translational relationships between specific psychiatric

medications (i.e. antidepressants, lithium and antipsychotics), the telomere-telomerase system and clinical outcomes. The modulation of intracellular Wnt/b-catenin or PI3 K/Akt signaling pathways, the interaction with BDNF and 5-HT, and the antioxidant properties could represent possible mechanisms by which the different types of psychiatric medications could modulate telomere length and telomerase activity. The potential of the telomere-telomerase system in promoting cellular survival and/or function in the brain and in the periphery could, in turn, represent a neurobiological substrate through which these molecules can mediate the therapeutic effect of such interventions.

Further, in the present symposium I'll show data from our research team on telomere length and telomerase activity in leukocytes predicting clinical response to serotonin-specific reuptake inhibitors (SSRIs) in subjects with major depressive disorder.

Disclosure of interest The author has not supplied his declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.109

S036

Can reducing psychological distress slow down the rate of telomere attrition?

E. Epel 1,*, J. Verhoeven 2

1 USA

² VU university Amsterdam, VU university medical center, Amsterdam, The Netherlands

* Corresponding author.

Specific types of cognitions and mental processes may lead to greater stress arousal and may subsequently impact cell longevity. The study of telomeres and telomere-related molecular systems may provide a pathway for exploring the link between psychological domains and cell physiology. Based on findings emerging from clinical and preclinical data, we hypothesize that the telomere-telomerase system contributes to explain certain biological underpinnings of psychological interventions.

In this symposium we'll present the preliminary evidence on the complex translational relationships between specific psychological domains (i.e. childhood adversities, stressful life events, mindfulness-based interventions and perceived distress), the telomere-telomerase system and clinical outcomes. Further, we'll discuss preliminary data on the effect of mindfulness- and meditation-based interventions on cellular ageing and diseaseassociated molecular phenotypes.

Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.110

Evidence of accelerated biological ageing in post-traumatic stress disorder

D. Lindqvist Lund, Sweden

Post-traumatic stress disorder (PTSD) is a common and debilitating condition, affecting between 10-20% of soldiers returning from combat zones, and with even higher prevalence rates in Veterans Affairs healthcare settings. PTSD is associated with an increased risk for various medical illnesses, many of which are commonly seen with older age. This raises the possibility that PTSD is associated with accelerated biological aging at the cellular level. Accelerated biological aging occurs when biological age outpaces chronological age, and this process is driven by a number of biological mechanisms including immune activation, oxidative stress, and mitochondrial dysfunction.