*Introduction* Among patients with major depression, increased inflammatory markers at baseline may predict an anti-depressant response. Reducing inflammation may augment response to psychotropic medications. Few studies have investigated an association between Leukocyte Telomere Length (LTL) and therapeutic response in depression, reporting mixed results. No studies assessed LTL and treatment response with PPAR-γ agonists.
*Objectives* (1) LTL as a predictor of anti-depressant response to PPAR-γ agonist in patients with unremitted depression.
(2) the correlation between LTL and insulin resistance (IR) status.
*Aims* We aimed to assess LTL as a predictor of antidepressant response to Pioglitazone in groups of insulin resistant and insulin-sensitive subjects using surrogate markers of IR.
*Methods* Medically stable men and women ($n = 42$) ages 23–71 with non-remitted depression participated in double-blind placebo controlled add-on of Pioglitazone to treatment-as-usual. Oral glucose tolerance tests were administered at baseline and at 12 weeks.
*Results* At baseline, no differences in LTL were detected by depression severity, duration or chronicity. LTL was also not significantly different between insulin-resistant and insulin-sensitive subjects. Subjects with longer telomeres exhibited greater declines in depression severity in the active arm, but not in a placebo arm. LTL also predicted improvement in insulin sensitivity in the group overall and did not differ between the active and placebo arm.
*Conclusions* LTL may emerge as a viable predictor of antidepressant response. An association between insulin sensitization and LTL regardless of the baseline IR status points to potential role of LTL as a non-specific moderator of metabolic improvement in these patients.
*Disclosure of interest* I, Dr. Natalie Rasgon, am a consultant for Shire Pharmaceuticals and Sunovion Pharmaceuticals.

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**W030**

## Effect of BMI on resting-state functional architecture of the brain in healthy individuals and patients with psychosis

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Elevated body mass index (BMI) is associated with increased multimorbidity and mortality. The investigation of the relationship between BMI and brain organization has the potential to provide new insights relevant to clinical and policy strategies for weight control. Here, we quantified the effect of BMI on the functional connectivity of the Default-Mode (DMN), Central Executive (CEN), Sensorimotor (SMN) and Visual (VN) networks in 496 healthy individuals that were studied as part of the Human Connectome Project. We found that elevated BMI was associated with disrupted functional integration of sensory-guided (SMN, VN) with internally controlled (DMN, CEN) networks, implicating increased attention to sensory stimuli as a possible mechanism underpinning overeating and weight gain. Our results suggest that weight control strategies should expand to include wider societal policies that incorporate modifications to eating environments and to the visual presentation and branding of food products.
*Disclosure of interest* The authors have not supplied their declaration of competing interest.

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**W031**

## Comorbidity of depression and diabetes: Questions recently answered and raised

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This paper will present information about a multicentric international collaborative study, which explored the frequency of depressive disorders in people with diabetes Type 2. The study was carried out in 14 countries–5 in Asia (Bangladesh, China, India, Pakistan and Thailand), two in Africa (Kenya and Uganda), two in Latin America (Argentina and Mexico) and five in Europe (Germany, Poland, Russia, Serbia and Ukraine). The study found that depressive disorders and sub threshold depression are frequent in people with diabetes: one tenth of all the nearly 3000 patients examined had major depression and another 15% sub threshold depressive disorders. Depression was only rarely recognized by the physicians dealing with diabetes and those few who were recognized were not provided treatment of their depressive disorders. The presentation will draw attention to the need to improve skills of diagnosis and treatment of psychiatric disorders of physicians who are not psychiatrists.
*Disclosure of interest* The author has not supplied his declaration of competing interest.

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**W032**

## Depression and its somatic consequences: Allostatic load as the connecting link

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Stress-related psychiatric disorders, such as depressive and anxiety disorders, have been associated with increased risk of overall mortality as well as with the onset of various aging-related somatic diseases. In addition to unhealthy lifestyles and poorer (self) care, various stress-related physiological processes likely contribute to these detrimental health consequences of psychiatric disorders. Considering the fact that the impact of stress-related disorders is visible on many different somatic health outcomes, it is unlikely that contributing biological systems are very specific. In fact, it is likely that multiple dysregulations of stress systems, including the immune, HPA-axis and autonomic nervous systems, but also various general proteomic or metabolomic pathways are involved. The concept of Allostatic Load (AL) emphasizes the presence of a multi-system physiological dysregulation.
In this talk I will summarize what the evidence is for somatic health consequences of psychiatric conditions, with depression as an important example. Subsequently, I will provide an overview of the various stress systems that are dysregulated in depressed patients. In addition, I will provide empirical data from the Netherlands Study of Depression and Anxiety ($n = 2981$) that illustrate that there is evidence that depressed patients are especially at risk for a dysregulation in multiple physiological stress systems. I will also illustrate how such a state of AL can impact on basis cellular aging indicators like telomere length and epigenetic age.
In sum, this talk will highlight the current state-of-evidence for an association between depressions with the onset of many adverse somatic health outcomes, and will provide insight into the contributing role of a multisystem physiological dysregulation.
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