Abstract # 1844

A high-sensitivity magnetic multiplex assay detects changes in plasma concentrations of interleukin-6 induced by the Trier Social Stress Test

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Concern exists about whether or not multiplex assays are sensitive enough to detect subtle yet biologically relevant changes in circulating concentrations of inflammatory biomarkers such as interleukin (IL)-6 in healthy humans. We tested two different multiplex kits (human Magnetic Luminex Performance Assay, R&D Systems [Minneapolis, MN]; and human Cytokine/Chemokine Magnetic Bead Panel, Millipore [Billerica, MA]) to detect IL-6 concentration changes versus a high sensitivity (HS)-ELISA (R&D Systems) in plasma collected from eighteen healthy adults (mean age 35.3 [SD = 8.88]) (10 women) before and 90 and 210 min after the start of an acute laboratory psychosocial stressor, the Trier Social Stress Test (TSST). Multiplexes were completed per manufacturer instructions and analyzed using a MAGPIX (Luminex, Austin, TX). For the R&D multiplex, IL-6 was detected in all samples and values were associated with concentrations determined by HS-ELISA ($r = 0.81$, $p < 0.001$). For the Millipore multiplex, IL-6 was detected in only 83% of the samples and values were not correlated with those determined by HS-ELISA ($p = 0.46$). To examine associations between IL-6 responses to the TSST we computed areas under the curve (AUCs) using the trapezoidal method. AUCs were positively correlated between the R&D multiplex and HS-ELISA ($r = 0.83$, $p = 0.001$), but not between the Millipore multiplex and HS-ELISA. These results suggest that certain multiplex assays may be able to detect changes in plasma concentrations of IL-6 as a result of TSST challenge.

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Abstract # 1845

Age- and depression-related changes in FKBP4 and FKBP5 expression across adolescence

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FK506-binding protein 5 (FKBP5) has repeatedly been shown to be a critical determinant of depression. For example, FKBP5 polymorphisms emerged as moderators of depression risk and protein levels have been linked to disease severity. However, little is known about FKBP5 in the context of depression pathogenesis. To address this gap, we repeatedly assessed 42 female adolescents (14-19yrs) at high risk for depression over the course of 2.5 years. Every 6 months, participants underwent a diagnostic interview and venipuncture to measure FKBP5 and FKBP4 mRNA levels. To assess the role of age, four equally-sized age groups were formed (14–15, 16–17, 18, 19 yrs). Over the study period, 21 adolescents showed at least one episode of depression (DEP), while 21 stayed symptom-free (SF). FKBP5 expression increased across age groups independent of health status, such that 14–15yrs old DEP and SF adolescents showed the lowest and 19yrs old DEP and SF adolescents showed the highest FKPB5 expression across time (age: $F = 3.22$, $p = .035$). While a similar pattern emerged for FKBP4 in SF adolescents, DEP adolescents exhibited overall lower as well as less age-variable FKBP4 mRNA levels (FBKP4-by-age-by-health status: $F = 2.28$, $p = .023$). In summary, while changes in FKBP4 and FKBP5 expression across adolescence showed a similar up-regulatory pattern in healthy participants, depression was associated with a potential de-synchronization of the two co-chaperons suggesting reduced glucocorticoid receptor signaling.

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Abstract # 1847

Abstract # 1847 Cross-sectional and longitudinal associations between immune cells in blood and brain – A TSPO PET study in healthy control subjects

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The interaction between immune cells in the periphery and brain in humans is poorly understood, partly due to the lack of *in vivo* studies. Here, we examined 32 healthy individuals using positron emission tomography (PET) and [11C]PBR28, a radioligand for the 18-kDa translocator protein (TSPO) which is expressed in immune cells both in brain and blood. In 26 individuals, two measurements were performed. In a subgroup of 19 individuals, of which 12 had repeat examinations, leukocyte numbers in blood was measured on each day of PET measurements. We assessed TSPO binding expressed as total distribution volume of [11C]PBR28 in brain and in blood cells. TSPO binding in brain was strongly and positively correlated to binding in blood cells at baseline ($r = .85$, $p = 2.1 \times 10^{-9}$, corrected for TSPO genotype). A correlation was also observed when analyzing change in TSPO levels in brain and blood between two PET examinations ($r = .60$, $p = .002$). Finally, there was a significant correlation between change of leukocyte numbers and change in brain TSPO binding, and a trend-level correlation to TSPO change in blood cells ($r = .63$, $p = .038$; $r = .6$, $p = .052$). These findings indicate a functional association between immunological cells in blood and brain at physiological conditions, such as interchange of peripherally derived cells or a common regulatory mechanism.

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