even after controlling for depression. The high test-retest correlations of alexithymia total and factor scores indicated relative stability of this construct, suggesting that it is a stable personality trait rather than a state-dependent phenomenon in these patients.

Conclusions: The results are encouraging for cognitive-behavior therapists working with alexithymic patients with panic disorder and obsessive-compulsive disorder, since the CBT outcome of these patients does not appear to be negatively affected by alexithymia. Furthermore, some alexithymic characteristics may decrease during CBT, even when the therapy program is not specifically directed to alexithymia. Future controlled studies should examine whether these improvements of alexithymia are due to psychotherapeutic interventions, in particular exposure therapy.

S25.04

Cellular phone communication and alexithymia - results of the Radep study

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No studies exist dealing with alexithymia and cellular phone (=CP) use. We hypothesised that there is an association between alexithymia and 1.not owing a CP and 2. with sparse use of CP.

The material consisted of 696 primary health care patients in Finland. Data was gathered with a questionnaire. Alexithymia was measured with the Toronto Alexithymia Scale-20. In addition to owing and using of CP several other factors were measured. The sociodemographic background factors consisted of gender, age, marital status, working status, living situation, and interpersonal relationships. The health status was measured with two subjective assessments (self perceived general health and functional ability) and with three standardized scales (the Depression Scale, Mood Disorder Questionnaire, and 22 questions from the core psychosis section of the Composite International Diagnostic Interview). In addition the childhood emotional, sexual and physical abuse was measured with the Traumatic and Distress Scale.

Only 9 % of the participants did not own a CP. Among them the means of TAS total score and TAS-factor3 (externally oriented thinking) were significantly higher than among other participants. Among those who used CP at least daily the means of all alexithymia measures: TAS total score, and the three factors (difficulty in identifying feelings, difficulty in describing feelings, externally-oriented thinking) were significantly lower than among other participants. In case of TAS total score and difficulty in describing feelings these associations still remained after controlling for all the above mentioned other factors. These findings fit well with the alexithymia construct.

Symposium: Immunotherapy of neurodegenerative disorders

S33.01

Human anti-prion protein antibodies block A117V PrP peptide fibril formation and prevent A117V PrP peptide-induced neurotoxicity

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Prion diseases, or Transmissible Spongiform Encephalopathies (TSEs), are a group of fatal neurodegenerative disorders associated with a conformational transformation of the cellular prion protein (PrPC) into a self-feplicating and proteinase K (PK)-resistant conformer, scrapie PrP (PrPSc). Aggregates of PrPSc around neurons lead to neuropathologyical change including neuronal loss, astrogliosis, spongiform degeneration and deposition of amyloid plaques. Currently no effective treatment for prion disease exists. The development of novel therapeutic strategies against prion diseases has become a priority. Several reports have demonstrated that passive and active immune-based therapy can significantly prolong the incubation period of prionoses in vivo, and also some anti-PrP monoclonal can prevent PrP peptide toxicity in vitro. In this study, we have first time identified and purified anti-PrP antibodies from human intravenous immunoglobulin (IVIG) by using PrP peptide affinity chromatography column. The ratio of anti-PrP antibody and IVIG is about 1:1200. In vitro study indicates these anti-PrP antibodies strongly block PrP A117V peptide fibril formation and disrupt formation of fibrillar structures. Furthermore, these antibodies almost completely prevented neurotoxicity of PrP A117V peptide in cultured rat cerebellar granule neuron cultures (CGN). In contrast, immunoglobulins depleted of anti-PrP antibodies had little effect on PrP fibril formation or protection of neuronal cells. Our study suggests that human anti-PrP antibodies may interfere with the pathogenesis of prion disease and these purified antibodies may be a potential therapeutic agent to prevent or slow prion disease progression.

S33.02

Biological and Imaging markers as outcome measures for secondary prevention trials in Alzheimer's disease

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With the evolving pharmacological era for the treatment of Alzheimer's disease (AD), there is a growing urgency to develop biochemical markers, as well as biomarkers based on imaging techniques to aid early accurate diagnosis, characterize patient populations and quantify the extent to which new drugs reach intended targets, alter proposed pathophysiological mechanisms and achieve clinical outcomes. Biomarkers support stratification of patient populations or quantification of drug benefit in primary prevention or disease-modification studies. Enrichment of trials with patients with similar prognosis according to a particular biomarker or combination of biomarkers could speed up proof-of-concept and dose-ranging studies. A wide range of imaging-based biomarkers are presently being studied for AD. These include an ever growing array of manual or fully automated MRI post-processing techniques of whole brain, grey or white matter, fiber tracts or specific brain regions, as well as metabolic, functional MRI and PET investigations. Multiple biochemical analytes in blood, urine or cerebrospinal fluid (CSF) have been proposed and studied, the most obvious of which are CSF β-amyloid related proteins, including abeta-antibodies and BACE 1, or tau proteins (total and phosphorylated tau), as they seem intimately involved in key mechanisms of AD. However, there are many different therapeutic approaches predicated on different pathophysiological hypotheses that might require different mechanistic markers.

https://doi.org/10.1016/j.eurpsy.2008.01.142 Published online by Cambridge University Press