follow-up 68%). Mental health was screened for symptoms of anxiety and depression at baseline by self-report on 12 items (the Anxiety Depression Index-12). Self reported whiplash trauma was registered as a dichotomy at follow-up, and followed up with age at whiplash trauma in positive cases.

Results: Whiplash trauma was reported by 956 individuals at follow-up, whereof 277 were reported to have occurred between baseline and follow-up. Symptoms of anxiety and depression increased the likelihood of self-report of whiplash trauma at follow-up (OR=1.24 per SD increase in mental symptom load, 95% confidence interval 1.10 − 1.40, p<.001), adjusted for age and gender. Whiplash was associated with increased disability pension award.

Discussion: Our finding suggests that the increased level of psychopathology found in individuals with a history of whiplash trauma might partly be present already prior to the whiplash injury. This finding is contrary to the common conception of causality in the whiplash-mental health association.

Symposium: Recent findings in alexithymia research

S25.01

Alexithymia among finnish male prisoners

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Some earlier studies have reported a positive association between alexithymia and delinquency. We studied this association in a sample of Finnish prison inmates. A questionnaire including the 20-item Toronto Alexithymia Scale (TAS-20), the 13-item Beck Depression Inventory and questions on socio-demographic variables as well as current and previous convictions, was delivered to 209 male prisoners. Of these, 113 individuals (54.1%) aged 17-65 years (mean 33.5) returned the questionnaire acceptably filled in. From a general population study, 1300 men aged 30-50 years (mean 40.3) were drawn as a control group.

The prevalence of alexithymia (TAS-20 cut-off point 60/61) was 7.5% in the population sample and 26.5% in the prisoner sample (p<.001). In a logistic regression analysis, controlling for age, marital status, basic education and depression, being a prisoner was still highly significantly associated with dichotomous alexithymia (OR 2.60, p=.003). Moreover, the mean TAS-20 score differed significantly between the samples (45.9 vs. 50.6 points, p<.001).

Of the prisoners, 18 (15.9%) reported having committed homicide. When they alone were compared with the population sample, no significant difference in the prevalence (7.5% vs. 11.1%) or level (mean TAS-20 score 45.9 vs. 46.8) of alexithymia was found. In a logistic regression analysis with confounders, being a convict confessing to homicide was not associated with alexithymia.

Male prisoners are more alexithymic than men in general population. There are, however, differences between different types of crimes. Those who confessed to homicide were, surprisingly, not more alexithymic than controls. Studies with larger samples are needed.

S25.02

Familial transmission of alexithymia

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Alexithymia represents a risk factor for psychiatric and psychosomatic disorders and is associated with a less favourable outcome in various treatments modalities. With prevalence rates up to 30% in subjects seeking psychiatric or psychotherapeutic treatment, there is an urgent need for a better understanding of the psychobiology of alexithymia. Previous studies have described an association between alexithymic traits of mothers and their offspring but did not investigate the fatherś contribution. Therefore, psychological mechanisms like the mother-child bonding may exclusively account for the observed association. The aim of the present study was to extent this research strategy to fathers, too.

The familial transmission of alexithymia was assessed in 86 child-parents trios. Significant associations between the TAS-20 scores of the children and mothers and children and fathers were found. The results were adjusted for age, gender and education. Factor 1 (difficulties identifying feelings) showed the largest intrafamilial association.

The significant association of both fatherś and motherś TAS-20 scores with the TAS-20 scores in the offspring strongly support a familial transmission of alexithymia. As both parents contributed to the TAS-20 score of their offspring, psychological and genetic factors may be responsible for the observed association. Thus, in addition to psychological research of affect development and differentiation the search for genetic mechanism for alexithymia should be started.

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S25.03

Does alexithymia predict non-response to psychotherapy?

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Background and Aims: Some studies have shown that alexithymic patients respond poorly to pharmacotherapy and that alexithymia may have a negative impact on the naturalistic course of psychiatric illnesses. The view that alexithymic patients are also less responsive to psychotherapy is often described in the literature, but few empirical studies have examined this issue, with inconsistent results.

Methods: We conducted two prospective studies (pre/post/follow-up) with patients with panic disorder and obsessive-compulsive disorder, to evaluate alexithymia as a potential predictor of the outcome of cognitive-behavioral therapy (CBT) including exposure response management. A further aim was to examine the absolute and relative stability of alexithymia.

Results: Regression analyses revealed that alexithymia, as measured with the 20-item Toronto Alexithymia Scale, was related neither to the post-treatment nor to the follow-up outcome. The repeated measures ANOVA showed a significant decrease of alexithymia over time,

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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.

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