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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It is therefore likely that a multi-modal biomarker approach stratified for ease of use, sensitivity and specificity will be needed in AD. This paper will focus on advances in the development of an immunotherapy for AD. In summary, clinically useful biochemical and imaging derived markers are clearly required in AD to inform regulatory and therapeutic decision making regarding candidate drugs and their indications in order to help bring new medicines to the right patients faster than they are today. Clearly, for AD a good biomarker could significantly reduce drug development timelines and optimize resources, thereby facilitating the evaluation of multiple molecules and therapeutic approaches. Currently, biomarkers, as well as novel therapeutic strategies need to be simultaneously developed and synergistically implemented in clinical AD research.

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Symposium: Preventing depression

S26.01

Prevention of depression: Possibilities and challenges

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Depressive disorders are highly prevalent, have a high incidence, and are associated with huge losses in quality of life in patients and their relatives, with increased mortality rates, with high levels of service use, and with huge economic costs. Prevention is an alternative to treatment that has not been studied elaborately until now. In this presentation, the current state-of-the-art on prevention of depression will be presented. Some recent methods will be shown to identify high risk groups that can be target populations for these interventions. These studies have made it clear that the majority of the first-ever incident cases of depression can be predicted with some simple risk-indicators. In the next part of the presentation, the research that has been conducted on the effects of preventive interventions will be summarized. In the past decade, several randomised studies have examined the effects of preventive interventions on the incidence of depressive disorders. These studies show that indicated prevention (with subjects who already have some symptoms but no DSM-disorder) can reduce the incidence of major depression with about 30%. Finally, an overview of interventions that are used as prevention in routine practice will be presented. Most of these interventions are based on cognitive-behavior therapy and are aimed at subjects who have some symptoms of depression but do not meet diagnostic criteria for a mood disorder. New developments in the prevention field and innovative interventions that are now being tested, will be presented.

S26.02

Temperament, personality and depressions: The case of the melancholic type

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The melancholic type is a phenomenological construct useful to recognize and assess persons vulnerable to develop major depression. Its main features are consciousness, orderliness, hyper-heteronomia and intolerance of ambiguity. These features, which mainly describe the social behaviour and the value system of these persons, were first described in qualitative studies mainly developed in Continental and Japanese psychopathology, and later established through quantitative research. The evolution of this construct nicely illustrates how qualitative and quantitative methods may be integrated in an epistemologically sound research agenda.

S26.03

Preventive strategies for depression: Conceptual, methodological, and practical issues

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While there is growing evidence for the efficacy of programs aimed at preventing depression, extensive variability exists in related research designs and methodology. The present contribution focuses on conceptual, methodological and practical considerations relevant to prevention research and practice in depression. First, it will outline characteristics of particular types of prevention (universal, selected, and indicated prevention) along with their strengths and weaknesses. It will also address the problem of reliable case identification, the adequate assessment of key symptomatology and relevant risk, protective, and vulnerability factors, the significance of preceding comorbidity which may affect the course of depressive symptoms, and, finally, the necessity for instruments allowing the longitudinal examination of subjects over long time periods or over developmental transitions. Furthermore, it will discuss the use of different outcome criteria (e.g., case status, symptoms, risk factors) and related implications, as well as effects of base rates and length of follow-up intervals on the efficacy of interventions. Finally, it will address the evidence of targeted intervention components in relation to particular risk groups, and will point to adequate implementation and related adherence measures. Recommendations for future prevention research and clinical work will also be provided.

Core Symposium: Alcoholism, from neurobiology to new treatment approaches

CS05.01

Neurobiology of alcoholism, an update

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