sive disorder NOS) during the follow-up period, 36 before the age of 79 and another 24 between the ages of 79 and 85. Logistic regression analyses consistently showed that the EPQ Neuroticism score (but not the EPQ Extroversion score) was associated with first-onset depression in this birth cohort (OR per one point increase on the EPQ = 1.07; 95% CI = 1.03, 1.10). Neuroticism was also associated with incident depression when individuals with incident dementia (n = 61) were excluded from the analyses (OR = 1.08; 95% CI = 1.04, 1.12) and also when incident cases of depression between 70 and 79 years (OR = 1.08; 95% CI = 1.04, 1.13) and between 79 and 85 (OR = 1.06; 95% CI = 1.00, 1.11) were analyzed separately.

Conclusion: We believe this is the first prospective study on a community sample of older adults to show that personality traits amplify risk for a first lifetime episode of depression. Findings have implications for theories on the etiology of later life depression and potentially for the identification and treatment of people at risk.

S095-003 Diagnosing Major Depression in Elderly Primary Care Patients: Nuances and Determinants

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Objective: We studied how general practitioners (GPs) diagnosed elderly patients with depressive symptoms. Sociodemographic factors such as younger age, female sex and more education, clinical characteristics such as severe depression and comorbidity of anxiety disorders are suggested to improve the diagnostic rate of depression. It may be decreased by comorbidity of somatic diseases. The role of these determinants was investigated in old age.

Design: A random sample of patients >55 year was screened for psychopathology. This was followed by the assessment of psychiatric disorders by a standardized psychiatric interview. Finally, 55 patients turned out to suffer from a major depressive disorder. GPs registered during one year all contact diagnoses and prescriptions for medication.

Materials and Methods: The General Health Questionnaire (GHQ-12) was used as screening instrument for psychopathology, and the Composite International Diagnostic Interview (12-month version) to assess (severity of) major depression and anxiety disorders in the last year. Contact diagnoses were defined according to the International Classification of Primary Care (ICPC). The ICPC codes P03 and P76 refer to a down/depressive feeling and depression respectively. Somatic comorbidity was determined based on contact diagnoses, and antidepressant use was assessed based on prescription data. T-tests, M-W U-tests and Chi-square tests were applied to study differences in sociodemographic and clinical factors between patients with and without a diagnosis coded as P03 or P76 by the GP.

Results: 20.8% were classified by their GP as having a depressive feeling and/or depression, 32.1% as having psychological problems other than depression and 13.2% has having no psychological problems, but social problems (closely related to psychological problems). In 11.3% of the depressed patients, antidepressants were prescribed without the diagnostic classification depressive feeling/depression. Patients who were accurately diagnosed by their GP were significantly older than patients who were not. We found no substantial differences in severity of depression and comorbidity of somatic diseases or anxiety disorders between the patient groups. However, the prescription rate of antidepressants was significantly higher in the accurately diagnosed patients.

Conclusion: In 77.4 % of elderly depressed patients GPs seem to be aware of psychological problems, but in only 20.8% of the patients the problems were specified as depressive feeling or depression. The one year registration period offered GPs the possibility to follow depressive episodes in their entire course from onset of symptoms, full manifestation of symptoms, till recovering state. Nevertheless, the diagnostic rate remained low. Training programs for improving the diagnostic rate in elderly patients should focus on a broad spectrum of psychological and social problems, while at the same time GPs should practice how to distinct these problems from each other.

S095-004 A Prospective Study into the Relationship between Premorbid Neuroticism and Mood Disorders in Dementia

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Objective: Mood disturbances are highly prevalent in patients with dementia. The pathogenesis is still
unclear. In this respect, psychological risk factors have received relatively little attention. The aim of this study was to determine if premorbid neuroticism is a predictor of mood disturbances in dementia patients.

**Design:** The Maastricht Study of Behavior in Dementia (MAASBED) focuses on the course and risk factors of BPSD. The project is a 2-year follow-up study including 199 patients with dementia. Patients are seen at 6-month intervals.

**Materials and Methods:** A reliable informant to provide information about patient personality was present for 181 patients. Analyses were performed on the 1-year data, and complete follow-up data were available for 124 patients. The NEO-FFI was completed by the primary caregivers and provided information about the patient’s premorbid neuroticism. Mood disturbances were assessed with a factor mood/ apathy from the Neuropsychiatric Inventory (NPI) and with the Cornell Scale for Depression in Dementia (CSDD).

**Results:** Female patients with high premorbid neuroticism had more mood disturbances than females with low neuroticism at baseline (p=0.03) and after 6 months (p=0.001). These results were independent of caregiver neuroticism, caregiver depression, and duration of illness. When the CSDD was used, female patients with high premorbid neuroticism had more mood disturbances at baseline (p=0.001) and after 6 months (p<0.001), but also after 1 year (p=0.05). Premorbid neuroticism was also a significant predictor of hyperactive behavior.

**Conclusion:** Premorbid neuroticism is a risk factor for mood disturbances in female patients with dementia, independent of caregiver characteristics. This effect declines after 1 year and, therefore, probably does not hold for later stages of dementia. Identification of premorbid neuroticism must alert the clinician to the development of mood disturbances.

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S095-005 Serotonin Modulation of Cerebral Glucose Metabolism in Geriatric Depression

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**Objective:** The ability to conduct in vivo studies of serotoninergic function in neuropsychiatric disorders has been limited by the lack of safe and selective pharmacologic agents for the serotonin system and by the difficulties in the development of serotonin radiotracers. An approach has been developed to measure serotonin function by evaluating the cerebral metabolic effects of a single dose of the selective serotonin reuptake inhibitor (SSRI) citalopram administered intravenously (Smith et al., 2002). The goal of the present study was to evaluate the cerebral metabolic response to acute citalopram administration in patients with geriatric depression compared to age-matched controls.

**Design:** Two resting Positron Emission Tomography (PET) studies of cerebral glucose metabolism were performed after administration of a saline placebo infusion (Day 1) and after administration of citalopram (40mg, IV, Day 2). The patients were rescanned after eight weeks of treatment with the oral medication.

**Materials and Methods:** Six patients with geriatric depression (one male/five females, mean age of 68.8, SD=5.7 years) who met DSM-IV criteria for current major depressive episode (non-bipolar, non-psychotic) and five comparison subjects (two males/three females, mean age of 64.8, SD=4.3 years) were enrolled in the study. PET data acquisition was performed on the GE Advance PET tomograph 35 minutes after injection of [18F]-2-deoxy-2-fluoro-D-glucose. The PET data were analyzed using the data driven voxel-wise analysis method, statistical parametric mapping (SPM99, Friston et al., 1995).

**Results:** The six depressed patients all met criteria for treatment response by week eight of treatment (defined as a Hamilton Depression Rating Scale Score below ten). The elderly control subjects demonstrated greater right cortical decreases relative to the patients, whereas the depressed patients demonstrated greater left cortical decreases relative to the controls. The depressed patients demonstrated greater increases relative to the controls in the right putamen and left occipital cortex. Chronic citalopram treatment resulted in progressive alterations in cerebral glucose metabolism in the patients.

**Conclusion:** These preliminary data provide evidence of a blunted right hemisphere cortical response and a less lateralized response to citalopram in the patients. Future studies in a larger sample of patients will evaluate whether the cerebral metabolic response to citalopram represents a biological marker of treatment outcome in geriatric depression. Supported in part by MH 64823, MH49936, MH57078, MO1621, MH 01509, MH 60575 and the General Clinical Research Center of the North Shore-LIJ Research Institute.