

ies suggested that altered function of taste neural circuitry may contribute to restricted eating in AN.

**Aims** The aim of this study was to evaluate, in patients suffering from AN, the activation of brain areas involved in taste perception and in central reward mechanisms to both pleasant and aversive taste stimuli and to correlate gustatory neurocircuitry activity with eating behaviours, temperament measures and/or sensitivity to reward and to punishment.

**Methods** Fifteen underweight female AN patients and sixteen normal-weight healthy women underwent a functional MRI to measure brain areas activation to repeated stimuli of a pleasant taste (sucrose solution), alternated with an aversive taste (bitter solution), and water taste.

**Results** Compared to healthy controls, patients with AN showed a significantly reduced activation of left insula and left dorsolateral prefrontal cortex to sweet stimulus and reduced activation of right parietal cortex to bitter stimulus.

**Conclusions** These results, if confirmed in future studies, may improve our knowledge about the pathophysiological mechanisms of AN.

**Disclosure of interest** The authors have not supplied their declaration of competing interest.

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### EW350

#### Implication of altered $\alpha 7$ nicotinic receptors and amyloid deposition in the Alzheimer's brain

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**Introduction** Brain amyloid- $\beta$  protein (A $\beta$ ) deposition is a key pathology of Alzheimer's disease (AD). Cholinergic degeneration, including reductions in  $\alpha 7$  nicotinic acetylcholine receptors ( $\alpha 7$ -nAChR), is also known as a pathophysiology of AD. Recent imaging studies have shown cognitively normal subjects with A $\beta$  depositions, indicating a missing link between A $\beta$  deposition and cognitive decline.

**Objectives** To clarify relationships among the A $\beta$  burden,  $\alpha 7$ -nAChR availability, and cognitive declines in AD.

**Aims** To measure brain A $\beta$  deposition and  $\alpha 7$ -nAChR availability in the same patients with AD using positron emission tomography (PET).

**Methods** Twenty AD patients and age-matched 20 healthy adults were studied. The  $\alpha 7$ -nAChR availability and A $\beta$  deposition were evaluated using PET with [<sup>11</sup>C]MeQAA and [<sup>11</sup>C]PIB, respectively. Levels of specific binding were estimated by a simplified reference tissue method (BP<sub>ND</sub>) for [<sup>11</sup>C]MeQAA and a tissue ratio method (SUVR) for using [<sup>11</sup>C]PIB. The values were compared with clinical measures of various cognitive functions using regions of interest (ROIs)-based and statistical parametric mapping (SPM) analyses.

**Results** [<sup>11</sup>C]MeQAA BP<sub>ND</sub> levels were extensively lower in the cholinergic projection regions of AD. There was a significant negative correlation between [<sup>11</sup>C]PIB SUVR and [<sup>11</sup>C]MeQAA BP<sub>ND</sub> in the nucleus basalis of Meynert (NBM). The NBM [<sup>11</sup>C]PIB SUVR was negatively correlated with the [<sup>11</sup>C]MeQAA BP<sub>ND</sub> level in the anterior and posterior cingulate cortices, whereas the relation within the same region showed weak correlation. Also we found signifi-

cant correlation between cognitive decline and [<sup>11</sup>C]MeQAA BP<sub>ND</sub> levels in the NBM.

**Conclusions** A $\beta$  deposition-linked  $\alpha 7$ -nAChR dysfunction may account for cognitive decline in AD.

**Disclosure of interest** The authors have not supplied their declaration of competing interest.

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### EW352

#### 3-dimensional evaluation of lateral ventricle volumes of schizophrenia patients and investigation of the subgroups

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**Introduction** The thought of greater loss of brain tissue in Deficit Syndrome (DS) i.e. subgroup of schizophrenia with enduring primary negative symptoms defined by Carpenter et al.; this has not been verified by recent studies.

**Objective** Accumulated researches suggest that enlargement in Lateral Ventricles (LV) is related with current negative symptoms and poor prognosis. However, this has not been validated in DS.

**Aims** Our aim is to study the association between the enduring negative symptoms and LV changes schizophrenia. We included both deficit and non-deficit patients for comparison with controls.

**Methods** Forty-five patients (18 DS, 27 non-DS) and 37 healthy controls were recruited, evaluated for positive and negative symptoms, depression and extrapyramidal symptoms. Structural magnetic resonance imaging was performed. LV was assessed by MANCOVA (gender, age total brain volume as confounding factors) in 3-dimensional (3D) shape analyses. Correlations between clinical and imaging data were analyzed by Pearson correlation coefficient;  $P > 0.05$  being significant.

**Results** LV of patients was found to be greater than controls, especially in regions adjacent to parietal and temporal regions but no significant difference between subgroups was detected. Enlargement in right LV by corpus callosum adjacency was found in DS. There was no correlation between negative symptoms and LV volume.

**Conclusions** The idea of greater amount of LV enlargement in patients with predominant negative symptoms could not be observed in 3D analyses. New pathophysiological theories are needed for the explanation of negative symptoms, loss of functioning and poor prognosis rather than only commenting about tissue decrease/loss.

**Disclosure of interest** The authors have not supplied their declaration of competing interest.

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### EW353

#### Schizophrenia and dementia. Morphological and spectroscopic findings. Baseline data

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