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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Implication of altered α7 nicotinic receptors and amyloid deposition in the Alzheimer's brain

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Introduction Brain amyloid-β protein (Aβ) deposition is a key pathology of Alzheimer's disease (AD). Cholinergic degeneration, including reductions in α7 nicotinic acetylcholine receptors (α7-nAChR), is also known as a pathophysiology of AD. Recent imaging studies have shown cognitively normal subjects with Aβ depositions, indicating a missing link between Aβ deposition and cognitive decline.
Objectives To clarify relationships among the Aβ burden, α7-nAChR availability, and cognitive declines in AD.
Aims To measure brain Aβ deposition and α7-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 α7-nAChR availability and Aβ deposition were evaluated using PET with [^{11}C]MeQAA and [^{11}C]PIB, respectively. Levels of specific binding were estimated by a simplified reference tissue method (BP_{ND}) for [^{11}C]MeQAA and a tissue ratio method (SUVR) for using [^{11}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 [^{11}C]MeQAA BP_{ND} levels were extensively lower in the cholinergic projection regions of AD. There was a significant negative correlation between [^{11}C]PIB SUVR and [^{11}C]MeQAA BP_{ND} in the nucleus basalis of Mynert (NBM). The NBM [^{11}C]PIB SUVR was negatively correlated with the [^{11}C]MeQAA BP_{ND} level in the anterior and posterior cingulate cortices, whereas the relation within the same region showed weak correlation. Also we found significant correlation between cognitive decline and [^{11}C]MeQAA BP_{ND} levels in the NBM.
Conclusions Aβ deposition-linked α7-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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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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