elevated cortisol on cognition, assuming a likely role of stressful events. Yet, very few studies actually examined these assumed links between life events, cortisol and cognition.
Objective To examine associations between salivary cortisol, cognition and life events in a population of non-demented old individuals.
Methods A cross-sectional analysis was conducted using data from Colaus/PsyColaus, a longitudinal population-based study involving 6733 Lausanne residents. Salivary cortisol samples (upon waking, 30 minutes after waking, at 11 am and at 8 pm) were obtained from 799 non-demented participants aged at least 60.
Life events, activities of daily life along with depressive symptoms were assessed using a standardized questionnaire. A comprehensive neuropsychological test battery was used to determine the Clinical Dementia Rating (CDR).
For multiple comparisons, P values were adjusted (P') according to Holm-Bonferroni's method.
Results Cortisol at 11 am and cortisol area under the curve (AUC) were positively correlated with CDR sum of boxes (CDRSOB) scores ($P' = 0.035$; Rho = 0.097 and $P' = 0.024$; Rho = 0.110, respectively). The association between cortisol AUC and CDRSOB remained significant after controlling for age, sex, body mass index, education, smoking and depression ($P = 0.001$; $\beta = 0.001$; R^2 change = 0.016). The number and the total impact of life events were associated neither with cortisol nor with CDRSOB.
Conclusions Elevated cortisol was associated with poorer cognitive functioning yet independently of life events. This suggests that the increased cortisol associated with poorer cognition might be not a mere reflection of stressful events but rather explained by other factors, yet to be elucidated.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.021

FC18

The EVACO Project: A new battery for assessing social cognition disorders and related psychiatric disability in schizophrenia

P. Roux[1,*], M. Urbach[1], S. Fonteneau[1], B. Aouizerate[1,3,4,5,6,7,8], F. Berna[1,6,7,8], L. Brunel[1,9], D. Capdevielle[1,10], I. Chereau[1,11], J. Danion[1,7], J. Dorey[1,12], C. Dubertret[1,13], J. Dubreucq[1,14], C. Faget[1,15], F. Gabayet[1,14], P.M. Llorca[1,11], J. Mallet[1,13], D. Misdrahi[1,3,4,5,6,7,8,16], R. Rey[1,12], R. Richieri[1,15], F. Schürhoff[1,9], H. Yazbek[1,10], C. Passerieux[1,2], E. Brunet-Gouet[1,2], FACE-SCZ-Group[16]
[1] *Fondation FondaMental, Créteil, France*
[2] *Service de psychiatrie d'adulte, Centre Hospitalier de Versailles, UFR des Sciences de la Santé Simone Veil, Université Versailles Saint-Quentin-en-Yvelines, Versailles, France*
[3] *Centre Hospitalier Charles-Perrens, 33076 Bordeaux, France*
[4] *Université de Bordeaux, Bordeaux, France*
[5] *Inserm, Neurocentre Magendie, Physiopathologie de la Plasticité Neuronale, U862, 33000 Bordeaux, France*
[6] *Hôpitaux Universitaires de Strasbourg, Université de Strasbourg, Inserm U1114, Fédération de Médecine Translationnelle de Strasbourg, Strasbourg, France*
[7] *Inserm U955, Translational Psychiatry team, Créteil, France*
[8] *Paris Est University, DHU Pe-PSY, Pôle de Psychiatrie des Hôpitaux Universitaires H.-Mondor, Créteil, France*
[9] *Service Universitaire de Psychiatrie Adulte, Hôpital la Colombière, CHRU de Montpellier, Université Montpellier 1, Inserm 1061, Montpellier, France*
[10] *CMP B, CHU, EA 7280 Faculté de Médecine, Université d'Auvergne, BP 69, 63003 Clermont-Ferrand cedex 1, France*
[11] *Université Claude-Bernard Lyon 1, Centre Hospitalier Le Vinatier Pole Est, BP 300, 39–95, boulevard Pinel, 69678 Bron cedex, France*
[12] *AP–HP, Department of Psychiatry, Louis-Mourier Hospital, Inserm U894, Université Paris Diderot, Sorbonne Paris Cité, Faculté de médecine, Colombes, France*
[13] *Centre Référent de Réhabilitation Psychosociale, Centre Hospitalier Alpes Isère, Grenoble, France*
[14] *Assistance publique des Hôpitaux de Marseille (AP–HM), pôle universitaire de psychiatrie, Marseille, France*
[15] *CNRS UMR 5287-INCIA, France*
[16] *FondaMental Academic Centers of Expertise for Schizophrenia (FACE-SZ) group, France*
[*] *Corresponding author.*

The relation of social cognitive disorders and schizophrenic symptoms are well-established. Yet, assessment methods have not reached a consensus. In addition, causal paths between neurocognition, social cognition, symptoms and functional expression are not clearly understood. During the past few years, some authoritative accounts proposed specialized batteries of tests and emphasized theory of mind, emotion recognition, and interpretation bias constructs:
– NIMH's "Social cognition psychometric evaluation" battery (Pinkham AE, Penn DL, Green MF, Harvey PD. *Schizophrenia Bulletin*, 2015);
– "Social cognition and functioning in schizophrenia" (Green MF, Lee J, Ochsner KN. *Schizophrenia Bulletin*, 2013).
Interestingly, these accounts stemming either from expert consensus and psychometric considerations or from neuroscience knowledge recognized some difficulties in providing a fully usable set of instruments. The project described here (EVACO protocol, funded by the Programme Hospitalier de Recherche Clinique national) follows an alternative approach and aims at providing a psychometrically validated battery. Based on a cognitive neuropsychology view on schizophrenic functional disability, several tests were gathered and are assessed in a 12-months multi-center follow-up of 160 individuals with schizophrenia. The FondaMental foundation network of Expert Centers is involved in recruiting patients from eight centers (Clermont-Ferrand, Colombes, Créteil, Grenoble, Marseille, Montpellier, Strasbourg, Versailles). To-date, the first evaluation of the population has been achieved. Experience reports and inclusions follow-up demonstrate the good acceptability of this battery both on the patients and the evaluator's side. We emphasize the usefulness of this project to meet the clinicians' needs of validated social cognition tools, by describing different scenarios of use.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.022

FC19

The relationship between premorbid adjustment and cognitive dysfunction in schizophrenia

P. Stefanatou[1,*], C.S. Karatosidi (MSc)[2], E. Kattoulas[1], N. Stefanis[1], N. Smyrnis[1]
[1] *Eginition Hospital, Medical School, University of Athens, 1st Department of Psychiatry, Athens, Greece*
[2] *Eginition Hospital, Medical School, University of Athens, Clinical Neuropsychology, Athens, Greece*
[*] *Corresponding author.*

Introduction Premorbid adjustment (PA) is one of the main prognostic indicators of schizophrenia. Both social and cognitive deficits observed during the premorbid period hold a predictive value for the onset of schizophrenia.
Objectives To investigate how cognitive functions are related to aspects of PA.
Aims To examine the relationship of each PA domain (academic and social) at each of the three developmental stages (childhood,

https://doi.org/10.1016/j.eurpsy.2016.01.023 Published online by Cambridge University Press

early adolescence and late adolescence), as well as their course with the cognitive functions in schizophrenia patients.
Methods PA, intellectual quotient (IQ), verbal learning, memory, processing speed, executive functions and verbal fluency were assessed using PAS, WAIS, RAVLT, TMT, WCST and COWAT measures respectively in a sample of 85 clinically stabilized male schizophrenia inpatients.
Results Negative correlations emerged between academic PA during adolescence and both verbal IQ and processing speed, while positive correlations were found with working memory. Negative correlations emerged between deterioration in academic PA during adolescence and both processing speed and immediate auditory verbal recall, while correlations with verbal learning were positive. There was no relationship between cognitive functions and either social PA or its deterioration.
Conclusion Our findings revealed significant associations between both academic PA and its course with cognitive functions in schizophrenia patients. In summary, deficits in several fields of cognitive functions seem to follow a different path long before and after the onset of the disease, but further investigation is necessary.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.023

Comorbidity/dual pathologies

FC20

Role of metabolic, atherogenetic and psychological factors in patients with colorectal adenomas: Preliminary results of the psycho-Neuro-Endocrino-Immunology Modena (PNEI-MO) Research Group

S. Ferrari [1], S. Mancini [1], S. Alboni [1], C. Artoni [1], A. Fabbrizzi [1], L. Feltri [1], G. Galli [1], A. Guicciardi [1], M. Marchi [1], F. Mariani [1], G. Mattei [1,*], G. Pollutri [1], S. Saraceni [1], P. Sena [1], G. Spiga [1], S. Tassi [1], C. Visentini [1], L. Roncucci [1], A. Merighi [2], M. Marsico [2]
[1] *University of Modena and Reggio Emilia, Department of Clinical-Diagnostic Medicine and Public Health, Modena, Italy*
[2] *Policlinico Hospital, Endoscopy Unit, Modena, Italy*
[*] *Corresponding author.*

Introduction Pro-inflammatory states of the large bowel have a multifactorial aetiology, including metabolism, atherogenesis, and psychological determinants. Inflammation plays a role in depressive and anxiety disorders, is tightly associated with early pro-atherogenetic alterations and metabolic dysregulation, and is also a key factor for the development of colorectal cancer.
Aim To investigate the association between pro-atherogenetic factors, metabolic status, psychological assessment and presence of colorectal adenomas.
Methods Case-control study, approved by the local Ethic Committee. Patients aged 40 or more and undergoing colonoscopy for positive faecal blood test and/or abdominal symptoms, with a negative history for neoplasia or inflammatory bowel diseases, were enrolled. For each patient the following data were collected: waist and hip circumferences, BMI, arterial pressure, fasten serum glycemia, current medications. Beside colonoscopy, carotid intima-media thickness (IMT) was assessed by means of echographic evaluation. Psychometric assessment included HADS, TCI, IMSA, SF-36. Statistics performed with SigmaPlot v.12 Platform.
Results Preliminary results are available for 18 patients (male/female 8/10) Mean age 62.6 ± 8.4. Ten patients had at least one adenoma, 8 patients had no lesions. The following differences were noticeable: HADS-depression (mean ± SD) adenoma vs. no-adenoma: 4.9 ± 3.2 vs. 1.7 ± 1.8 ($P < .01$); IMT median value adenoma vs. no-adenoma: 793 vs. 638 micrometers ($P = .04$); Body weight (mean ± SD) adenoma vs. no-adenoma: 66.4 ± 8.7 kg vs. 80.9 ± 15.3 kg ($P = .03$); waist circumference (mean ± SD) adenoma vs. no-adenoma: 105.2 ± 13.4 cm vs. 89.5 ± 4.7 cm ($P < .01$).
Conclusions Preliminary data from PNEI-MO Research Group support the relation between systemic inflammation, psychological status and development of precancerous colorectal cancer lesions. Depression seems associated with the presence of colorectal adenomas.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.024

Depression

FC21

Epigenetic signature of glucocorticoid receptor is associated with the familial component of depression: A twin-based study

H. Palma-Gudiel, A. Cordova-Palomera, L. Marquès Feixa, F. Cirera Miquel, L. Fañanás [*]
University of Barcelona, Animal Biology, Barcelona, Spain
[*] *Corresponding author.*

Introduction Despite the fact that depression has an estimated heritability around 40%, there is no definitive candidate gene contributing to its etiology. The lack of an identified genetic component for high-heritability disorders, like depression, gave rise to the concept of missing heritability. The epigenetics' field has pushed forward new hypotheses to fill this gap since transgenerational inheritance of epigenetic patterns has been described both in animal models and, more recently, in humans. Depression is usually associated with an abnormal stress response and an altered hypothalamic-pituitary-adrenal axis, regulated by the glucocorticoid receptor (coded by *NR3C1* gene). Therefore, NR3C1 has been widely investigated as a functional candidate gene involved in anxious-depressive spectrum disorders (ADSD) although a more comprehensive study of its methylation is further required (Palma-Gudiel et al., 2015).
Aims To analyze NR3C1 promoter's methylation and to study its association with anxious-depressive spectrum disorders.
Methods The sample consisted of 48 pairs of monozygotic twins, from the UB twin register, grouped as concordant, discordant and healthy pairs depending on whether both, one or none of the co-twins of each pair were affected by a lifetime ADSD, according to DSM-IV criteria (SCID). DNA methylation was assessed by bisulfite conversion and subsequent pyrosequencing.
Results Hypermethylation at specific CpG sites, not previously reported, was detected in concordant twin pairs as compared with discordant and healthy groups ($P = 0.03$).
Conclusions The epigenetic pattern newly described in *NR3C1* gene may be contributing to the familial component of depression and thus could be putatively explained by transgenerational inheritance of epigenetic phenomena.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.025