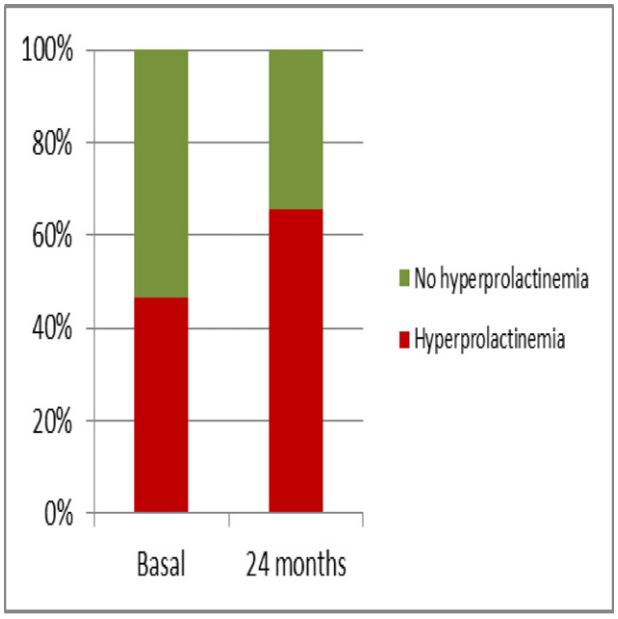

Fig. 1 Prolactine variation at 24 months.

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

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EW514

Cortical and subcortical morphology deficits in cerebral gray matter in patients with schizophrenia and not affected siblings

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Objective Explore the basis of cortical morphometry in patients with schizophrenia and non-affected siblings by Magnetic Resonance Structural analyzing cortical thickness.
Methods Twenty-nine patients with schizophrenia treated with atypical antipsychotics and clinically stable in the last 6 months were recruited. Twenty-three not affected siblings of patients with schizophrenia and 37 healthy volunteers were recruited. Magnetic Resonance Structural was performed. FreeSurfer the brain imaging software package for analysis of Cortical Thickness is used. In the analysis of group differences in cortical thickness (CT) with the general linear model (GLM), the *P*-value was established in 0003 following the Bonferroni correction to control for multiple comparisons (seven regions of interest a priori in each hemisphere).
Results Significant differences in cortical thickness between patients and healthy controls. Differences between groups were calculated by general linear model (GLM) with age and sex as covairables (Table 1).
Conclusions In applying the correction for multiple comparisons, differences in bilateral-lateral orbitofrontal, medial orbitofrontal-right and left temporal transverse frontal cortex are significant. Our study replicates previous findings and provides further evidence of abnormalities in the cerebral cortex, particularly in the frontal and temporal regions, being characteristic of schizophrenia.

Table 1 Significant differences in cortical thickness in healthy controls, not affected siblings and patients with schizophrenia.

		Controls	Siblings	Patients	F	P	
		n=37	n=23	n=29			
Frontal	L caudalmiddlefrontal	2.41	2.36	2.27	4,65	<0.05*	P<C=S
	L lateralorbitofrontal	2.66	2.57	2.5	8,5	<0.001***	P<C= S
	R lateralorbitofrontal	2.59	2.45	1.96	9,28	<0.001***	P<S<C
	L medialorbitofrontal	2.44	2.41	2.3	5,72	<0.01**	P<S<C
	R medialorbitofrontal	2.57	2.51	2.36	14,32	<0.001***	P<S<C
	L rostralmiddlefrontal	2.2	2.21	2.17	5,39	<0.01**	P<C=S
	R rostralmiddlefrontal	2.33	2.27	2.2	4,19	<0.05*	P<C=S
	L superiorfrontal	2.62	2.58	2.46	5,56	<0.01**	P<C=S
	R superiorfrontal	2.65	2.6	2.54	3,1	0.051	P<C=S
Temporal	L superiortemporal	2.78	2.71	2.65	4,01	<0.05*	P<C=S
	R superiortemporal	2.83	2.78	2.67	4,59	<0.05*	P<C=S
	L transverseltemporal	2.43	2.24	2.19	7,68	<0.001***	P<S<C
	R transverseltemporal	2.4	2.36	2.2	5,82	<0.01**	P<C=S
	R middletemporal	2.89	2.83	2.76	4,35	<0.05*	P<C=S

P: patients; S: siblings; C: controls.

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EW516

Paliperidone palmitate log-acting injection in patients with psychotic active clinic: start, change or increase of dose

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The aim is to describe the experience of treatment with Paliperidone Palmitate long acting injection (PP) in patients with psychotic active clinic, whether diagnoses with schizophrenia or in patients with the first episode psychosis, as well as to reflect the improvement in the control of the symptoms that the patients can improve increasing the dose.
Methods We have done a descriptive study of 34 patients hospitalized in psychiatry between January and July 2015 for psychotic active clinic who started treatment with PP or the previous dose was increased.
Results 91.2% of patients admitted for acute exacerbation of their usual pathology and 8.8% for a first episode psychosis. In the CGI scale, all the patients admitted scored as severe or markedly ill; going mostly mildly ill at discharge. For 55.9% of patients, the treatment was changed to PP, 29.4% of the dose was increased PP and 14.7% antipsychotic treatment was started with PP. Among patients change treatment, the main reason was non-adherence (47.4%). 70.6% of our patients were discharged with PP as only antipsychotic

and 29.4% which was discharged with another antipsychotic, the most frequent association was of PP with Quetiapine (80%).
Conclusions PP is a highly effective medicament in the treatment of the schizophrenia that improves the adherence to the treatment, so in our experience and we consider it a medicament to be considered in the early stages of the disease. According to our experience and there are patients who can benefit from better control of symptoms adjusting the dose individually.
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EW517

Inflammatory and metabolic biomarkers of psychopathological dimensions of schizophrenia

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Introduction The concept of schizophrenia as a systemic disease includes, not only psychosis, but an increase in somatic comorbidity and cardiovascular risk [1]. Furthermore, it is known the implication of inflammation in the pathogenesis of schizophrenia [2].
Objectives To determinate potential inflammatory/metabolic biomarkers of schizophrenia's dimensions.
Methods Sample: 36 outpatients with schizophrenia for less than 11 years, under stable maintenance treatment (mean age [32.25], males [63.9%]) and their 36 matched controls (age [32.53 ± 6.63]; males [72.2%]).
Evaluation PANSS, Clinical Assessment Interview for Negative Symptoms(CAINS), Calgary Scale(CDS), CGI, Personal and Social Performance Scale(PSP). Biomarkers: C-reactive protein (CRP), homocysteine, glucose, insulin, HOMA-IR (insulin resistance), cholesterol, HDL, LDL, triglycerides.
Results Biomarkers differences between groups are shown in Table 1. Table 2 shows the correlations found after controlling for Body Mass Index [patients(28.61 ± 5.69);controls(24.64 ± 3.80); p = 0.001] and Smoking [patients(52.8%-yes);controls(5.6%-yes);p = 0.000].
Conclusions 1. CRP, a potential inflammatory biomarker in schizophrenia, is related to depression severity. Homocysteine, representing an oxidative stress, is related to positive, negative, cognitive and depressive symptoms severity, and worse functioning. 2. Patients with schizophrenia have lower HDL–related to negative and cognitive symptoms severity and worse functioning–and insulin resistance – related to worse cognition –.

Table 1

	Patients(Mean±SD)	Controls(Mean±SD)	*t*
CRP(mg/dl)	0.42±0.73	0.11±0.09	*2.50**
Homocysteine(mg/dl)	12.97±3.35	12,05±3.78	0.98
Glucose(mg/dl)	85.2±11.34	80.88±9.76	1.80
Insulin(mg/dl)	17.85±14.73	7.91±3.33	*3.93***
HOMA-IR	1.91±1.30	0.99±0.42	*3.88***
Cholesterol(mg/dl)	185.89±34.65	178.09±22.99	1.12
HDL(mg/dl)	46.19±13.55	62.14±15.10	*-4.68***
LDL(mg/dl)	113.81±29.40	103.97±27.11	1.46
TG(mg/dl)	134.08±67.63	79.11±30.39	*4.44***

*p<0.05,**p<0.01

Table 2

	CRP	Homocysteine	Insulin/HOMA-IR	Chol/LDL/TG	HDL
PANSS-Positive		0.59**			
PANSS-Negative		0.46*			-0.51**
PANSS-General		0.58**			
PANSS-Total		0.60**			-0.35*
CAINS		0.49*			-0.49**
CDS	0.55**	0.41*			
CGI-Cognition		0.47*	0.42*/0.42*		-0.40*
CGI-Global		0.49*			-0.46**
PSP		-0.51**			0.49**

*p<0.05,**p<0.01

Disclosure of interest The authors have not supplied their declaration of competing interest.
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EW518

Clinical and functional response to paliperidone palmitate in early schizophrenia–A retrospective observational study in newly diagnosed patients treated over a 12-month period

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Introduction Data on clinical outcomes with long-acting antipsychotic treatment in young, newly diagnosed patients with schizophrenia is sparse.
Objectives To explore hospitalization, drug utilization and clinical outcomes from medical records of newly diagnosed schizophrenia patients during first 12 months of treatment with once-monthly paliperidone palmitate (PP).
Methods International, multicenter, retrospective, observational study. Outcomes presented: baseline (BL) characteristics and demographics, clinically relevant improvements in disease severity (ie ≥ 20% decrease in PANSS or BPRS total score or CGI-S Change ≥ −2 or CGI-C ≥ 3, with no score showing worsening) and clinically relevant functional improvement (i.e. change in PSP total score ≥ +7 points or change in GAF total score ≥ +20 points, with no score showing worsening) from BL to last-observation-carried-forward endpoint (LOCF-EP) within 12-month documentation period, mean mode PP dose and adverse drug reactions.
Results Eighty-four patients analyzed: 69% male, mean age at initiation of PP was 24.1 (SD2.7) years, mean BL weight was 78.7 (SD16.0) kg and 80.0 (SD14.7) kg at LOCF-EP, with a mean change of 1.2 (SD3.9) kg; mean time from first psychotic episode to initiation of PP was 5.5 (SD3.3) months. At LOCF-EP 86.6% achieved a clinically relevant improvement (71/84, Kaplan-Meier median time from initiation of PP: 52.4 days). 63.4% achieved a clinically relevant functional improvement (52/84, Kaplan-Meier median time from initiation of PP: 53.1 days). PP mean mode maintenance dose was