



Data in Brief

Gene expression profiling in peripheral blood mononuclear cells of early-onset schizophrenia

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ABSTRACT

Schizophrenia (SZ) is a severe chronic psychiatric disorder with wide prevalence and high morbidity. We know little about SZ's etiology and pathophysiology at present. The study of gene expression profile is useful for us to identify potential biomarkers at molecular level and explain possible pathogenesis of SZ. Therefore we recently compared gene expression profiles in PMBCs from EOS cases and healthy controls using microarrays. Here we will describe in detail the contents and quality control of the microarray experiment. The raw microarray data are accessible through GEO series accession number [GSE54913](https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE54913).

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Specifications	
Organism/cell line/tissue	<i>Homo sapiens</i> /peripheral blood mononuclear cell
Sex	Male and female
Sequencer or array type	Arraystar LncRNA Array v2.0
Data format	Raw and processed
Experimental factors	Early-onset SZ cases vs. healthy controls (<18 years)
Experimental features	Microarray gene expression profiling to identify differential expressed genes in SZ cases compared with controls
Consent	All the participants and their parents signed the informed consent
Sample source location	China

1. Direct link to deposited data

<http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE54913>.

The disease onset before age 18 is generally regarded as early-onset (EOS) when the patients confer more familial vulnerability and poor outcomes [1]. The neurodevelopmental hypothesis posits that the onset of SZ is associated with early development of the nervous system

[2]. We paid attention to this period and speculate that the altered gene expression in these patients may be associated with the disease process.

Peripheral blood mononuclear cells (PBMCs) have represented an accessible tissue source for gene expression, as it is easily collected from patients. There already have many gene expression profiling studies using PBMCs, a consistent conclusion about the expression alteration of schizophrenia is lacked [3,4].

2. Experimental design, materials and methods

We recently collected blood samples from 18 EOS cases and 12 controls. Then we generated whole-genome gene expression profiles on PBMCs from these samples by using microarray. 17,200 valid probes detected in our experiment were used to identify altered gene expressions.

2.1. Study population

A total of 18 first-onset SZ patients (8 males and 10 females, aged 14.78 ± 1.70 years) were included in our study. They were untreated and drug-naïve patients diagnosed by at least two experienced psychiatrists independently according to the Diagnosis and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV) criteria for SZ. 12 healthy controls (6 males and 6 females, aged 14.75 ± 2.14 years) were recruited into the study. Teenagers with a history of other mental health or neurological diseases were enrolled into our study. All participants

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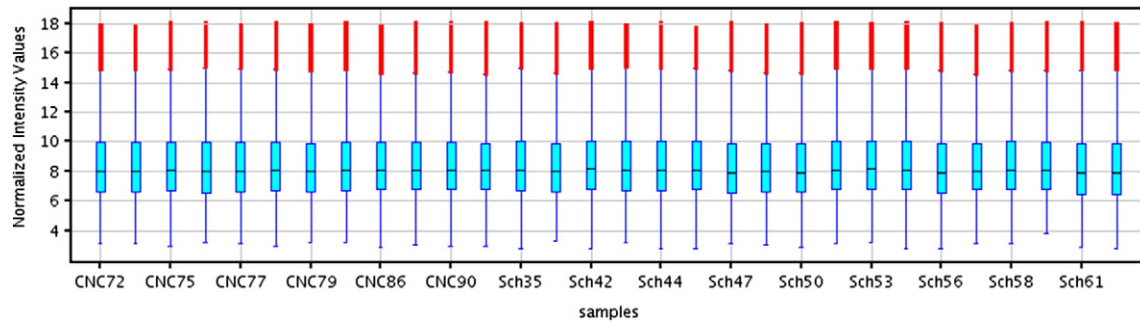


Fig. 1. Quality assessment of mRNA data after filtering. The box-plot shows the distribution of normalized signal intensity by array; the distributions of log₂-ratios among all samples are nearly the same after normalization.

were unrelated Han Chinese recruited from the north of China. And both the participants and their parents signed the informed consent before participation. The study was approved by Medical Research Ethics Committee of Shanxi Medical University.

2.2. Microarray and quality control

Peripheral blood was collected. NanoDrop ND-1000 was used to quantify total RNA after RNA extraction, and RNA integrity was assessed by standard denaturing agarose gel electrophoresis. Agilent Array platform was employed to perform the microarray analysis. Following RNA amplification, hybridization and image scanning, signal intensities were normalized in the quantile method using GeneSpring GX v11.5.1 (Agilent Technologies), and low intensity mRNAs were filtered (mRNAs that at least 20 out of 30 samples have flags in Present or Marginal were chosen for further analysis). R [5] was used to perform the data processing and analyses of mRNA data. The sample preparation and microarray hybridization were performed based upon the manufacturer's standard protocols with minor modifications.

Log₂-ration was used by quantile normalization. The distributions of the intensities after normalized among all samples were shown in Fig. 1. Identification of differentially expressed genes between SZ cases and controls was made using R package genefilter [6]. We identified 84

differentially expressed genes through fold change and P value filtering (FC ≥ 2 and P_{adjusted} < 0.05) listed in Table 1.

3. Discussion

All the participants in our study were teenagers with similar age (<18 years), and their brains were still developing. The SZ cases were neither under medication nor had a history of pharmacotherapy. We mainly described a dataset about gene expression profiles of the 30 samples measured by Arraystar.

Among the 84 DE genes listed above, *SLC18A1* has been reported to be associated with SZ [7,8]. In addition, *CTLA4* was also identified showing a high expression level in SZ [9] which is consistent with the results from our study. Through our description above, we believe that this dataset will be useful for the exploration of SZ's pathogenesis in the future.

Conflict of interest

The authors have no conflicts of interest.

Acknowledgments

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Table 1
List of differentially expressed genes.

Up-regulated genes (82)	C11orf49	NKAIN4	LCE2D	GAGE10
	SLC18A1	EYA2	UBAP2L	ATP2B3
	NAT1	OPRL1	RFPL4B	TKTL1
	MYBPC1	C21orf56	CCL26	USP9Y
	KIF23	SLC5A4	DARC	LDB1
	GTF2H1	GJB5	POU6F2	ACP2
	ALDH4A1	CCDC134	PNMA2	P4HA3
	IL28RA	MYL3	CNGB3	C11orf1
	ERVFRDE1	SCAP	DEFB135	FAM19A2
	ALDH3A1	PRICKLE2	FAM110B	C12orf68
	ODF4	ENTPD3	MAL2	HCFC2
	TSPAN16	RNF186	SARDH	RPL10L
	CCNE1	EIF4G1	NUP188	PRKAB2
	TGFA	UGT2B4	C9orf171	CA12
	SATB2	RASSF6	TMEM27	C15orf2
	SLC45A1	FGA	XAGE3	ZP2
	IL1RL2	PAICS	CUL4B	SALL1
	BBS5	SH3RF2	PNCK	C16orf46
	CTLA4	UBD	SMIM9	SLC5A2
	EPPIN	ECM1	ERAS	GPT2
	OSGIN1	HOXD11		
Down-regulated genes (2)	IQCF6	POM121L12		

(p < 0.05 with a fold change >2).