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No changes in densities of cannabinoid receptors in the superior temporal gyrus in schizophrenia

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Abstract: Objective In recent years, abnormal changes in the endocannabinoid system have been found in schizophrenia. The superior temporal gyrus (STG) is strongly implicated in the pathophysiology of schizophrenia, particularly with regards to auditory hallucinations. In this study, we investigated the binding density of cannabinoid CB1 receptors in the STG of schizophrenia patients compared to control subjects. **Methods** Quantitative autoradiography was used to investigate the binding densities of [^{3}H]SR141716A (a selective antagonist) and [^{3}H]CP-55940 (an agonist) to the CB1 receptors in the STG. Post-mortem brain tissue was obtained from the NSW Tissue Resource Centre (Australia). **Results** Contrasting to previous findings in the alterations of CB1 receptor densities in the prefrontal, anterior and posterior cingulate cortex of schizophrenia, which were suggested to be associated to impairment of cognition function, no significant difference was found between the schizophrenia and control cases in both [^{3}H]SR141716A and [^{3}H]CP-55940 binding. **Conclusion** We suggest that CB1 receptors in the STG are not involved in the pathology of schizophrenia and the auditory hallucination symptom of this disease.

Keywords: schizophrenia; cannabinoid receptor; autoradiography; superior temporal gyrus

1 Introduction

A number of studies have reported that both acute and chronic exposure of cannabis may induce symptoms similar to those seen in schizophrenia, including delusions, paranoid, impairments in cognitive function, and occasional hallucinations etc.[1,2]. These psychiatric effects of cannabis are likely to be mediated by cannabinoid CB1 receptor, since many of the psychological effects of marijuana are reversed by SR141716A, a selective CB1 receptor antagonist[3]. In addition, cannabis consumption may precipitate psychosis among vulnerable individuals, and provoke relapse and exacerbates symptoms among those who have already developed schizophrenia[1,4]. Following the finding of central cannabinoid receptors and endogenous cannabinoids[5,6], a cannabinoid hypothesis has been proposed[7,8].

In recent years, abnormalities of the endocannabinoid system have been found in schizophrenia. For example, two endogenous cannabinoid agonists (anandamide and palmitylethanolamide) were found to be significantly higher in the cerebrospinal fluid of schizophrenic patients[9,10]. In post-mortem brain studies, it has been reported that schizophrenic patients had an increased density of cannabinoid CB1 receptors in the dorsolateral prefrontal cortex[11], and anterior and posterior cingulate cortex[12,13].

There is substantial evidence to support that the superior temporal gyrus (STG) is involved in the pathology of schizophrenia, particularly in auditory hallucination[14]. It is also well known that hallucination is a common side effect of cannabis use, especially following excessive dosages[4]. Since high densities of cannabinoid CB1 receptors have been identified in the STG[15], these receptors may play a role in the hallucination symptom associated with schizophrenia. However, to date, no study has been performed to reveal the possible alteration of cannabinoid receptors of the STG in pathology of schizophrenia. In this

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Article ID: 1673-7067(2007)06-0341-07
CLC number: R749.3
Document code: A
Received date: 2007-06-05

study, we have examined the cannabinoid CB1 receptor density in the STG of schizophrenic patients compared to control subjects using quantitative autoradiography.

2 Materials and methods

2.1 Subjects and preparation of post-mortem brain tissue

Postmortem brain tissue was obtained from the NSW Tissue Resource Centre (TRC) through brain donor programs, including 8 schizophrenic patients and 8 controls. Cases, which had a significant history of neurological disorder, head injury, or with post-mortem intervals (PMI) over 48 h, were excluded. In all cases, the tissue was taken from the left-brain hemisphere of superior temporal gyrus (Brodmann's 22). Once brain tissue collected, it was immediately dissected into small blocks (10 mm thick) and stored in a –80 °C freezer until sectioning. At this store condition, brain tissue could be stored for many years without degradation. This study was approved by the Human Research Ethics Committee, University of Wollongong, Australia, and complied with the NHMRC National Statement on Ethical Conduct in Research Involving Humans 1999. The Demographic and clinic data of all these cases were presented in Tab. 1.

The diagnosis of schizophrenia was established after reviewing all available medical records by using the Item Group Checklist of the Schedules for Clinical Assessment in Neuropsychiatry (SCAN)[16] and the Diagnostic Instrument for Brain Studies (DIBS)[17]. The diagnosis of schizophrenia was confirmed according to the Diagnosis and Statistical Manual of Mental Disorders (DSM-IV). These schizophrenia cases were matched with 8 control cases by gender (all males), age and PMI. For control case, medical records were reviewed to exclude any history of major psychiatric disorders, and if necessary to exclude possible psychopathology. All of the following procedures for autoradiography and quantitative analysis were taken at blind of diagnosis.

The brain tissue was cut into 14 μm sections using a

Tab. 1 Demographic and radioligand data of schizophrenia subjects and controls

Subject No.	Age (year)	PMD (h)	pH	FRD	THC	SUBS	Cause of death	Medication at death
Schizophrenia								
1	51	21	6.02	400	ND	No	Ischaemic heart disease	Thioridazine, mesoridazine
2	27	27	6.25	314	ND	Yes	Suicide: hanging	Nil
3	67	5	6.36	1300	ND	No	Ischaemic heart disease	Thioridazine, risperdal
4	57	35.5	6.35	275	ND	No	Myocardial scarring	Thioridazine, sertraline
5	27	30	6.22	198	ND	Yes	Myocarditis	Clozapine
6	27	9.5	6.17	649	ND	Yes	Clozapine toxicity	Clozapine
7	30	24	6.60	325	ND	No	Suicide: Carbon monoxide poisoning	Clozapine
8	44	35	6.55	500	D	Yes	Suicide: hanging	Nil
Mean ±SEM	43.5±5.2	19.9±2.6	6.31±0.07	495±354				
Control								
8	55	20	N/A				Cardiac arrest	
10	37	21	N/A				Ischaemic heart disease	
11	60	13	N/A				Acute myocardial	
12	61	24	6.52				Ischaemic heart disease	
13	18	33	N/A				Hypertrophic	
14	37	11	5.25				Pulmonary embolism	
15	37	24	6.37				Electrocution	
16	43	13	6.43				Thrombotic coronary artery occlusion	
Mean ±SEM	41.3±5.6	23.4±4.0	6.14±0.30					

Abbreviation: PMI, post-mortem interval; FRD, final recorded antipsychotic drug use (chlorpromazine equivalents per day); THC, blood D^9-tetrahydrocannabinol levels; ND, not detected; D, detected in urine; SUBS, substance (marijuana) users.

cryostat at –17 °C and the sections were mounted on polysine-coated slides. All sections were stored at –20 °C until they were thawed at room temperature prior to incubation. All sections from schizophrenia and control cases were processed simultaneously to minimize experimental variance.

2.2 [^{3}H]SR141716A binding autoradiography [^{3}H] SR141716A binding was performed as previously described[12]. Briefly, all sections were incubated for 60 min at room temperature in 50 nmol/L Tris-HCl buffer (pH7.4) containing 1.5 nmol/L [^{3}H] SR141716A (specific activity 52 Ci/mmol, Amersham, UK) and 0.1% bovine serum albumin (BSA) in the presence or absence of 100 μmol/L of the potent cannabinoid receptor agonist HU210. After incubation, the sections were washed 2 times in ice-cold Tris-HCl buffer containing 0.1% BSA for 30 min each, and followed with a rapid dipping in ice-cold distilled water to remove buffer salts. These sections were quickly dried with a stream of warm air.

2.3 [^{3}H] CP-55940 binding autoradiography [^{3}H]CP-55940 binding was performed as previously described. In brief, sections were pre-incubated for 30 min at room temperature in 50 mmol/L Tris-HCl (pH 7.4) containing 5% BSA . Sections were then incubated in the same buffer with the addition of 10 nmol/L [^{3}H]CP-55940 (168 Ci/mmol, PerkinElmer) for 2 h at room temperature in the presence or absence of 10 μmol/L CP-55940. Following incubation, the sections were washed three times. The first wash was in 50 mmol/L Tris-HCl (pH 7.4) containing 1% BSA, for 1 h at 4 °C. The second wash was in 50 mmol/L Tris-HCl (pH 7.4) for 3 h at 4 °C, and the third wash was in the same buffer for 5 min at 4 °C. Finally, the sections were dipped briefly ice-cold distilled water and air-dried.

2.4 Quantification and statistical analysis The slices were exposed to Kodak BioMax MR film for 120 d with [^{3}H] microscales (Amersham) as standards. The films were developed at room temperature in Kodak X-ray developer and fixer. Autoradiographic images were digitized with a GS-690 Imaging Densitometer (Bio-RAD, USA) for quantitative analysis using a Multi Analyst program (Bio-RAD, USA). Then optical density measurement was converted into fmoles [^{3}H] ligand per milligram tissue equivalent (TE) by comparing to the standards. A set of sections from all cases were stained with cresyl violet and used for confirmation of anatomical structure. Comparisons between two groups (schizophrenia and control) on radioligand binding, age, PMI, and brain pH were made using Student *t*-test. Analysis of covariance (ANCOVA) was performed for controlling age, PMI and brain pH for binding results. Binding densities between marijuana and non-marijuana users were analysed using Mann-Whitney U test. Pearson correlation was used to assess the relationship between binding density and the final recorded antipsychotic drug use (FRD).

3 Results

3.1 Subjects There were no differences in age ($t = –0.296$, $df = 14$, $P = 0.77$), PMI ($t = 0.739$, $df = 14$, $P = 0.47$) and brain pH ($t = 0.77$, $df = 10$, $P = 0.46$) between the schizophrenia and control groups. Examples of [^{3}H]SR141716A and [^{3}H] CP-55940 binding in the STG are presented in Fig. 1.

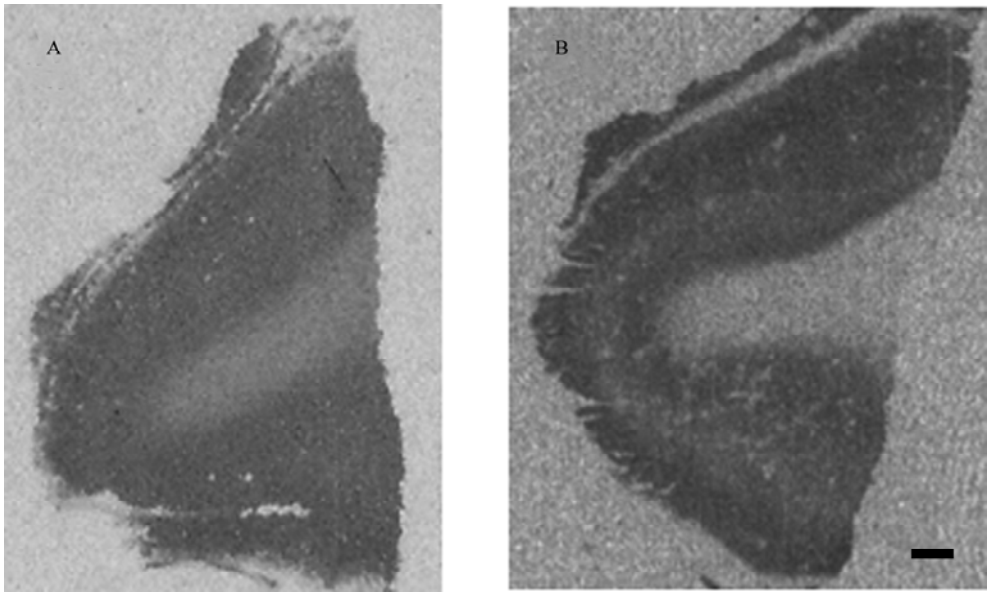

Fig. 1 Examples of autoradiograms to show (A) [^{3}H] SR141716A and (B) [^{3}H] CP-55940 bindings in the superior temporal gyrus. Scale bar: 1 mm.

3.2 [^{3}H] SR141716A binding [^{3}H]SR141716A binding was homogeneously distributed through all layers of the STG in both schizophrenic and non-schizophrenic subjects (Fig. 1A). No significant difference was found between the schizophrenia and control cases in [^{3}H]SR141716A binding ($F_{1,15}$ = 0.443, P = 0.518) (Fig. 2A). Furthermore, there were also no significant effects of age ($F_{1,15}$ = 0.541, P = 476), PMI ($F_{1,15}$ = 2.564, P = 0.135) and brain pH ($F_{1,15}$ = 0.57, P = 0.817) on [^{3}H]SR141716A binding. In the schizophrenic cases, there were no relationship between the [^{3}H] SR141716A binding density and the final recorded dose of antipsychotic drugs (r = 0.381, P = 0.352).

3.3 [^{3}H] CP-55940 binding [^{3}H]CP-55940 binding sites showed a laminar distribution pattern in the STG and three binding bands were observed (Fig. 1B). The upper band corresponded to cortical layers I-II, the middle band corresponded to cortical layers III-IV, and the deeper band corresponded to cortical layers V-VI. Compared to the middle layers, there were a greater binding density in the upper (t = 9.819, df = 15, P < 0.001, Two-tailed paired t-test) and deep layers (t = –8.693, df = 15, P < 0.001) (Fig. 2B). However, there were no significant difference in [^{3}H]CP-55940 binding densities between the schizophrenia and control cases in all three binding layers (upper layers: $F_{1,15}$ = 0.124, P = 0.730; middle layers: $F_{1,15}$ = 0.249, P = 0.627; deeper layers: $F_{1,15}$ = 0.163, P = 0.693). There were also no significant effects of age (P > 0.05 in all layers), PMI (P > 0.05 in all layers) and brain pH (P > 0.05 in all layers) on [^{3}H]CP-55940 binding densities. Furthermore, there was no significant correlation between binding density and the final recorded dose of antipsychotic drugs in the schizophrenic cases in all [^{3}H]CP-55940 binding layers (–0.326 < r < –0.498; all P > 0.05).

3.4 No effect of marijuana use on CB1 binding As shown in Tab. 1, half of the schizophrenia patients were marijuana users. The CB1 receptor densities in the tissues from these marijuana users were not significant from the non-marijuana users in both [^{3}H]SR141716A and [^{3}H]CP-55940 bindings (Tab. 2).

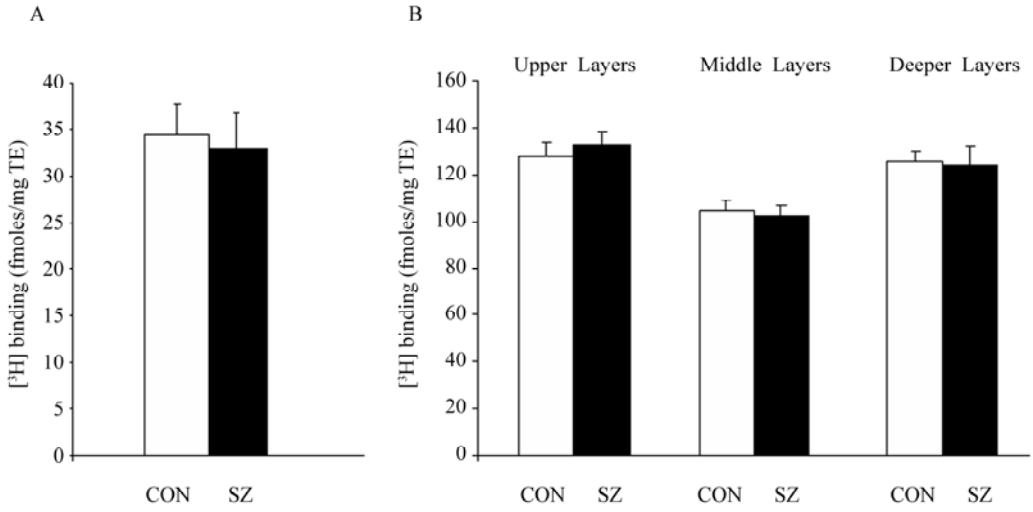

Fig. 2 No significant difference in (A) [^{3}H] SR141716A and (B) [^{3}H] CP-55940 binding densities (Mean ± SEM) in the superior temporal gyrus between schizophrenia (SZ) and control groups (CON). TE, tissue equivalent.

Tab. 2 Comparison of [^{3}H]SR141716A and [^{3}H]CP-55940 bindings between the schizophrenia patients with and without a history of using marijuana

	[^{3}H]SR141716A (fmole/mg TE; mean ± SEM)	[^{3}H]CP-55940 (fmole/mg tissue; mean ± SEM) Upper layers	Middle layers	Deeper layers
Marijuana user (n = 4)	34.30±8.00	140.40±9.67	108.14±4.67	135.45±12.37
Non-marijuana user (n = 4)	31.51±2.44	123.74±5.76	97.19±4.10	112.59±5.64
Mann-Whitney U test	P = 0.486	P = 0.343	P = 0.200	P = 0.486

4 Discussion

This study found that, similar to other cortical regions, [^{3}H]CP-55940 presented a laminar binding pattern in the STG, but [^{3}H]SR141716A binding showed a homogeneous labelling through all cortical layers of the STG[12,15]. Even though the two ligands showed different binding patterns in the cortex, previous studies have shown that both of them are suitable to reveal the alteration of CB1 receptor density in schizophrenia[11-13].

A previous study in our laboratory identified an increase in the binding density of [^{3}H]SR141716A in the anterior cingulate cortex of schizophrenia patients[12], utilising tissue mostly obtained from subjects used in the present study (all 8 schizophrenia patients and 7 of 8 control cases). However, in the present study, no difference was identified in the binding density of cannabinoid CB1 receptors in the STG of schizophrenia patients compared to controls. Similarly, it was previously reported that compared to controls there are no changes in the binding density of [^{3}H]CP-55940 in the hippocampus of schizophrenia patients[11], although the same study reported an increase of [^{3}H]CP-55940 binding in the dorsolateral prefrontal cortex in schizophrenia. Therefore, an alteration of endogenous cannabinoid receptors in the cortex of schizophrenia may be region specific and possibly related to specific symptoms.

The effect of cannabis use on CB1 receptor density is also regional specific. Dean *et al*. has found that recent cannabis ingestion (close to death) increases the density of CB1 receptor in the caudate-putamen, but not in the dorsolateral prefrontal cortex and hippocampus using [^{3}H] CP-55940 (CB1 receptor agonist) binding[11]. In this study, blood Δ^9-tetrahydrocannabinol level was detected only in once case (No. 8, Tab. 1) indicating cannabis ingestion close to death, in which the CB1 receptor density showed no difference comparing to other schizophrenia subjects. However, it is possible that acute and chronic cannabis use have different effects on cannabinoid receptors. In animal studies, chronic cannabinoid administration (1-2 week) has been reported to decrease [^{3}H]CP-55940 binding in the rat striatum, cerebellum and limbic forebrain of rats, but not in the ventral mesencephalon[19,20]. The binding densities in the tissues obtained from the schizophrenia patients with a history of marijuana use were not significantly different to those in non-marijuana users in this study (Tab. 2). It suggests that the CB1 receptor density in the STG may not be influenced by cannabinoid level.

Antipsychotic medication is another possible factor to influence receptor binding, however we know little about the effects of medication on cannabinoid receptor. A recent study has found that only clozapine, but not antipsychotic drugs (e.g. haloperidol, chlorpromazine and olanzapine), decreased CB1 density (examined by [^{3}H]CP-55940) in nucleus accumbens of rats[21]. More importantly, all of these drugs did not affect CB1 density in the frontal cortex and other brain regions tested (hippocampus and striatum)[21]. This report suggests that effect of antipsychotic drugs on CB1 receptor binding is also regional specific, and that various antipsychotics have different effects. Furthermore, it is also consistent with our findings in this study that no correlation between the CB1 binding density in the STG and final recorded antipsychotic drug use. Therefore, although all of the schizophrenia cases had been treated with antipsychotic drugs in this study, these medications should not influence our results.

It has been postulated that alteration in the endocannabinoid system is associated with impairment of cognitive function, a subgroup of schizophrenia syndromes[7]. Increased cannabinoid CB1 receptor density in the dorsolateral prefrontal, anterior and posterior cingulate cortex supported this postulation[11-13]. The STG is involved in the pathology of schizophrenia, particularly in auditory hallucination[14,22-24]. Due to the lack of change in CB1 receptor binding densities in the STG, this study does not support the idea that CB1 receptors in the STG are involved in the pathology of schizophrenia and the auditory hallucination symptom of this disease. Instead, hallucination induced by excessive cannabis use may be via other neural pathways, such as an elevation in the mesolimibic dopaminergic pathway[25,26]. Recently CB1 receptor has been proposed as a novel target for treating schizophrenia and other psychiatric disorders[27], therefore further research in the regional and symptom-specific alterations of cannabinoid receptors in schizophrenia may provide novel approaches for the treatment of this disease.

Acknowledgements: This study was supported by a University of Wollongong URC grant to C. Deng and the Schizophrenia Research Institute, Australia, utilizing infrastructure funding from NSW Health. The Schizophrenia Research Institute is formerly known as the Neuroscience Institute of Schizophrenia and Allied Disorders (NISAD). Tissues were received from the NSW Tissue Resource Cen-

tre which is supported by the Schizophrenia Research Institute, National Institute of Alcohol Abuse and Alcoholism and NSW Department of Health, University of Sydney. We thank Dr K. Newell for her critical comments and Miss K. Weston-Green for her help in proof-reading of the manuscript.

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精神分裂症患者颞上回的大麻素受体密度没有显著性变化

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摘要: 目的 近年来研究发现，在精神分裂症患者的内源性大麻素递质系统会出现异常变化，而颞上回在精神分裂症的病理生理机制中和幻听症状密切相关。因此，对照正常人群，我们研究了精神分裂症患者颞上回大麻素CB-1受体的密度变化。**方法** 采用定量放射自显影技术，通过[^{3}H]SR141716A(CB-1受体选择性拮抗剂)和[^{3}H]CP-55940(CB-1受体激动剂)检测颞上回CB-1受体密度水平。死后脑组织由澳大利亚新南威尔士州组织资源中心提供。**结果** 先前研究发现，精神分裂症患者与认知功能失常相关的额前叶，前、后扣带回皮质的CB-1受体密度水平有异常改变. 与此相反，本研究发现在精神分裂症患者的由[^{3}H]SR141716A和[^{3}H]CP-55940检测的颞上回大麻素受体密度水平和对照组比较没有显著变化。**结论** 我们认为颞上回大麻素CB-1受体和精神分裂症患者的发病及幻听症状无关。

关键词：精神分裂症；大麻素受体；放射自显影技术；颞上回