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Odors identification differences in deficit and nondeficit schizophrenia

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ABSTRACT

Background: There is evidence that deficit schizophrenia (DS) is associated with neuroanatomical changes in structures including those involved in olfaction. Olfactory dysfunction, which includes impaired odor identification, is found in patients with schizophrenia and their family members.
Methods: 82 patients with DS and 72 patients with NDS (nondeficit schizophrenia), somatically healthy and without acute psychotic symptoms undertook a smell identification test using the 16-item Sniffin' Sticks ID test. Demographic and psychometric data were collected.
Results: No differences in the course of the illness, perinatal history and demographic data were found between the DS and NDS groups. No differences in the number of correctly identified odor samples were found. Some differences in the qualitative identification of samples between DS and NDS were found in the groups of female (fewer correct identifications of cinnamon and pineapple smells in DS) and male patients (fewer correct identifications of the smell of rose and more correct identifications of the smell of orange than in NDS).
Conclusions: No overall differences between DS and NDS regarding odors identification have been found. The results seem to indicate some specific deficits in the identification of markers of rose, pineapple, orange and cinnamon.

Introduction

Schizophrenia is one of the most disabling mental diseases. A number of factors that may affect its clinical picture have been analyzed. Despite its prevalence (1% in the general population) and many years of research (since 19th c.), no sufficiently valid markers of schizophrenia have been found to date. The development of the illness can be understood as global spectrum of impairment, including social, emotional, cognitive impairment as well as disturbed thought processes. Researchers have focused on specifying potential points of clinical examination that may be conducive to quicker and more accurate diagnosis and implementation of more efficacious treatment and rehabilitation of patients.

In some patients, primary negative symptoms occur throughout the course of disease. Such symptoms are not drug-induced, cannot be linked to depressive symptoms, are not due to extreme social exclusion or schizophrenic disintegration. In 1988, Carpenter referred to this condition as the deficit syndrome [1]. Primary, persistent negative symptoms of the deficit syndrome determine the clinical course of the disease and have been associated with poor clinical outcomes [2] as well as with poor response to treatment and worse functioning [3–5]. The symptoms are stable in the course of the illness and retrospective and prospective

Abbreviations: ALT, alanine transaminase; AST, aspartate transaminase; CNS, central nervous system; DS, deficit schizophrenia; DSM IV, Diagnostic and Statistical Manual of Mental Disorders, Forth Edition; DUP, duration of untreated psychosis; GABA, γ-aminobutyric acid; ICD10, International Statistical Classification of Diseases and Related Health Problems, Tenth Edition; MINI, Mini International Neuropsychiatric Interview; NDS, non deficit schizophrenia; OPCRIT, Operational Criteria for Psychotic Illness; PANSS, Positive and Negative Syndrome Scale; SDS, Schedule for the Deficit Syndrome; S'S, Sniffin Sticks; TSH, thyroid-stimulating hormone.

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studies demonstrate an 80 per cent consistency between the first and successive diagnoses [3,6]. The prevalence of deficit schizophrenia (DS) – 15% of patients with a first episode of schizophrenia, 25–30% in patients suffering from chronic schizophrenia [2,4]. Patients with DS have poorer premorbid adjustment, and experience a slow, difficult to recognize, insidious onset of the disease [7]. Given the above described course of the illness, neurostructural changes in the brain should be a focus in future research attempts. Also genetic background is investigated [8,9]. Patients with the deficit syndrome more frequently suffer from neurological disorders (sensory integration, impaired motor coordination, etc.) [10]. Functional neuroimaging studies demonstrate the inhibition of glucose metabolism, reduced cerebral blood flow and exacerbated neuron loss. Post-mortem examinations found an increased density of the interstitial cells of the white matter in the frontal and temporal cortex in patients with DS compared to healthy controls [11,12]. Observations on the neuroanatomical underpinnings of the condition have implicated structures involved in olfactory processes [13]. Neurotransmitters (which play a prominent role in the pathogenesis of schizophrenia), including γ-aminobutyric acid (GABA), glutamate and dopamine are also involved in olfactory processes [14,15].

Olfactory dysfunctions, including a raised odor detection threshold or impaired odor identification occur in the early stages of CNS diseases. Such impairments were diagnosed in Parkinson's, Alzheimer's, Huntington's diseases, multiple sclerosis [16], before [17] and after the onset of schizophrenia (early onset deficits, progressive character of olfactory dysfunction, odor identification deficits present in family members) [18]. While olfactory deficits can be seen and diagnosed early in the course of schizophrenia [19], they are positively correlated with the course of the disease [20]. They are to be observed both in patients and their family members [21]. Neuroleptic drugs [22], tobacco addiction, cognitive dysfunction and the severity of schizophrenia are thought not to trigger olfactory dysfunction [23]. A raised odor detection threshold and smell impairment in schizophrenia are combined with negative symptoms [18,24,25]. Studies [26,27] demonstrated that olfactory impairments were related to the deficit syndrome in schizophrenia. In the study by Strauss et al., lower accuracy in smell identification was negatively correlated with SDS symptom severity, and valence ratings for pleasant odors were positively correlated with SDS diminished emotional range. In view of the above evidence, odor identification may be a useful biomarker for the schizophrenic process, as literature reports seem to indicate that olfactory impairments originate at CNS level [28]. There are no olfactory studies on Polish population to date in the described group of patients. Consequently, the available literature contains no reports on the subject.

Materials and methods

Subject

154 unrelated Caucasians, Polish nationals (84 men, 70 women) with schizophrenia was recruited for the study. The average age of patients was 39.3 ± 10.98.

All the subjects were fully informed (aims and the protocol) and all of them expressed their written informed consent.

The protocol was approved by the Bioethics Committee of the Pomeranian Medical University of Szczecin.

All the subjects were treated according to the guidelines for the psychopharmacological treatment of schizophrenia [29–33]. In both groups the patients received typical or atypical antipsychotics. The groups did not differ as regards the type of administered neuroleptics, i.e. there was no advantage of the use of one type of antipsychotics over the other. Chlorpromazine equivalent doses: in DS-699 mg per day, in NDS-727 mg per day. The difference was not statistically significant ($p = 0.69$).

Inclusion/exclusion criteria

The following inclusion criteria were applied: patients diagnosed with schizophrenia according to ICD-10 for a minimum of 18 months, 18–60 years of age, ability to understand the study procedures. Patients diagnosed with other mental diseases, including affective disorders, suffering from dementia or significant organic brain injury, epilepsy, alcohol addiction or substance abuse, as well as patients with general poor somatic health were excluded from the study.

The somatic conditions of the subjects were tested based on the following chemical tests: complete blood count, blood glucose level tests, TSH, AST, ALT, ionogram, urea level, creatinine level. The results of patients qualified for the study were within laboratory normal ranges. Also, they were subjected to a complete physical examination. The above exclusion criteria were supposed to help in drawing more precise conclusions after selecting a group of patients with deficit schizophrenia, i.e. to minimize the possible influence of other, organic mental disorders.

At the time of smell examination the patients were healthy, their nasal ducts were free of discharge, with no present signs of sinusitis, cold or fever.

Questionnaires and psychometric scales

Additional data were obtained with the use of demographic data questionnaires, OPCRIT (the operational criteria checklist for psychotic and affective illness) [34] and MINI (the Mini-International Neuropsychiatric Interview) [35].

We have also applied psychometric scales, PANSS (Positive and Negative Syndrome Scale) [36] and SDS (Schedule for the Deficit Syndrome). SDS-6 core items of the deficit syndrome, with both item and anchor point descriptions. Ratings were made on a five-point scale, on each of six items: restricted affect, diminished emotional range, poverty of speech, curbing of interests, diminished sense of purpose, diminished social drive in accordance with their primary or secondary character, persistence and intensity. To meet the criteria for the deficit syndrome, an individual must demonstrate a moderate or higher level of severity on at least 2 of these symptoms.

Information was collected from all available sources as well as from direct observation of the patients with the a general rule that if data were in conflict, evidence from external informants such as patient's emotional range, interests and social drive were considered of greater weight than these obtained from the patient [37].

Deficit/nondeficit subgroups of patients

Based on clinical symptoms, medical history of problems in functioning, symptomatology, and SDS outcome, the cohort was divided into 2 subgroups:

patients with deficit schizophrenia (DS) $n = 82$; (female $n = 29$, male $n = 53$);
patients with nondeficit schizophrenia (NDS) $n = 72$; (female $n = 41$, male $n = 31$).

Olfactory testing

A battery of Sniffin' Sticks (S'S), an olfactory test originally designed for Europeans, was used in smell identification examinations [38,39]. The reliability and relevance of S'S used to evaluate odor identification impairments has been validated in many neurological and psychiatric studies [40,41]. A 16-item S'S