

Two Clinical Case Reports: Supporting “D-Cell Hypothesis” of Mental Illnesses

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Introduction

The post-mortem brain research is an essential way for mental diseases. The author established “D-cell hypothesis of schizophrenia” induced from post-mortem brain studies of patients with schizophrenia, which showed cellular and molecular basis of paranoid-hallucinatory state, progressive pathophysiology of schizophrenia, linking the neural stem cell (NSC) dysfunction hypothesis and dopamine (DA) hypothesis [1,2]. The D-cell is the aromatic L-amino acid decarboxylase (AADC=DDC: dopa decarboxylase)-containing neuron lacking DA and serotonin, and produces trace amines, including β -phenylethylamine, tyramine and tryptamine. The D-cell hypothesis shows the importance of subventricular NSC functions in relation with striato-accumbal D-cell (D15~16) function and mesolimbic DA system, whereas NSC functional suppression due to stress, aging and alcohol, being the cause of paranoid-hallucinatory state [2]. D-cell hypothesis also indicates prospectiveness of TAAR1 (trace amine-associated receptor, type 1) medicinal chemistry [3]. Here, I briefly report two clinical cases to verify D-cell hypothesis of pathophysiology of mental illnesses.

Case I

1st episode schizophrenia following the Great East Japan Earthquake

Male 50's Following the Great East Japan Earthquake in March 2011, he complained that “Yakuza is catching me up!”. This persecutory delusion gradually aggravated, so he consulted to a mental clinic. The doctor examined him, diagnosed his symptoms as schizophrenia, and prescribed antipsychotic drugs. As he insisted on the existence of restless noisy sounds in his 1st floor residence of a building, his family was obliged to move from the 1st floor to the 4th floor, according to his request. Seven years later, he admitted our general hospital *via* the Emergency Center, because of loss of consciousness and cardiac arrest. After recovery from his consciousness loss, the doctor of internal medicine consulted to our department of psychiatry to confirm if his abulia was a symptom of schizophrenia or residual symptom of hypoxic brain injury of cardiac arrest. Four times of plain brain CT did not show any specific findings.

Case II

Paranoid-hallucinatory state only during hospital stays in an elderly female patient

Female 80's In her 2nd hospital stay due to lumbago, the doctor of orthopedics consulted to Psychiatry to manage with her paranoid-hallucinatory, of which onset was the 1st admission to a hospital ward 2 year before. She complaint that there was annoyance from some people around her, but there was no evidence of such annoyance. Her daughter explained that similar persecutory delusion was seen in her 1st hospital stay, but she stopped to complain of annoyance when she returned to his house. So, her daughter stopped to give the patient antipsychotic drugs prescribed by a medical doctor in her inpatient period. During her 2nd hospital stay, her persecutory delusion recurred, however, her complaint disappeared just after her return to her house. One mg Risperidon was needed only her hospital stay. Plain brain CT showed slight brain atrophy considered her age.

Cellular and molecular basis of paranoid hallucinatory state in stress vulnerable state is shown in Figure 1. The highlight is that D-cell hypothesis is the sole hypothesis explaining cellular and

molecular mechanisms of the initiation of paranoid hallucinatory state, where NSC, D-cell, trace amine and TAAR1 is involved [2].

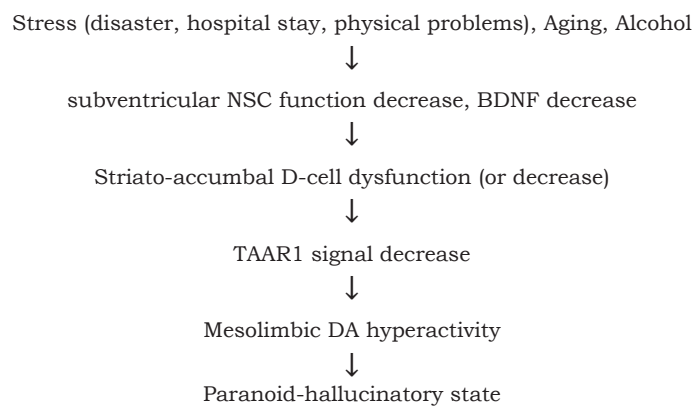


Figure 1: Paranoid-hallucinatory state evoked by stress, aging or alcohol: Explanation based by D-cell hypothesis.

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