In this mission, to get further insights into the molecular basis of the novel diagnosis and treatments for schizophrenia, our research team will intend to explore "the social brain markers" associated with maturation of the specific social behavior that is disturbed in schizophrenia from the pharmacological and developmental views. The schizophrenomimetic actions of dopamine agonists and NMDA type glutamate receptor antagonists may be explained by the possibility that these drugs disturb the molecular and cellular equipment in an information processing system or neuron circuit that specifically malfunctions in schizophrenia. Although the exact causative mechanism for schizophrenia remains unclear, the pathophysiological changes in the information processing system might be associated with the development-dependent nature of schizophrenia and a schizophrenomimetic-induced mental symptoms. Thus, the onset of schizophrenia typically occurs after adolescence. It has been consistently reported that schizophrenomimetic drugs often induce psychotic symptoms in adults, but not in children. In experimental animals, the psychotomimetic effects of dopamine agonists and NMDA receptor antagonists have also been observed after the critical period around postnatal week three. The late-developing features of schizophrenia and its pharmacological models indicate that maturation of the specific neuronal systems could be required for their onsets. The hypothetical human system and its animal homologue should contain molecules that are responsive to the schizophrenomimetic drugs only after the adolescence and the critical period for the animal model of schizophrenia. We therefore will focus our efforts on seeking such critical period-regulated genes and on clarifying their expressional and/or genomic features to obtain candidates for "the social brain markers."