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| **Testo di partenza** | **Testo tradotto dal candidato** | **Spazio a disposizione del correttore** | **Punteggi** |
| **The Brain in Schizophrenia** |  |  |  |
| If dopamine cannot account well for schizophrenia, what is the missing link? A critical clue came from the effects of another abused drug: PCP (phencyclidine), also known as angel dust. In contrast to amphetamine, which mimics only the positive symptoms of the disease, PCP induces symptoms that resemble the full range of schizophrenia’s manifestations: negative and cognitive and, at times, positive. These effects are seen not just in abusers of PCP but also in individuals given brief, low doses of PCP or ketamine (an anesthetic with similar effects) in controlled drug-challenge trials. |  |  |  |
| Such studies first drew parallels between the effects of PCP and the symptoms of schizophrenia in the 1960s. They showed, for example, that individuals receiving PCP exhibited the same type of disturbances in interpreting proverbs as those with schizophrenia. More recent studies with ketamine have produced even more compelling similarities. Notably, during ketamine challenge, normal individuals develop difficulty thinking abstractly, learning new information, shifting strategies or placing information in temporary storage. They show a general motor slowing and reduction in speech output just like that seen in schizophrenia. Individuals given PCP or ketamine also grow withdrawn, sometimes even mute; when they talk, they speak tangentially and concretely. PCP and ketamine rarely induce schizophrenia-like hallucinations in normal volunteers, but they exacerbate these disturbances in those who already have schizophrenia. |  |  |  |
| The ability of PCP and ketamine to induce a broad spectrum of schizophrenia-like symptoms suggests that these drugs replicate some key molecular disturbance in the brain of schizophrenic patients. At the molecular level the drugs impair the functioning of the brain signaling systems that rely on glutamate, the main excitatory neurotransmitter in the brain. More precisely, they block the action of a form of glutamate receptor known as NMDA receptor, which plays a critical role in brain development, learning, memory and neural processing in general. This receptor also participates in regulating dopamine release, and blockade of NMDA receptors produces the same disturbances of dopamine function typically seen in schizophrenia. Thus, NMDA receptor dysfunction, by itself, can explain both negative and cognitive symptoms of schizophrenia as well as dopamine abnormalities at the root of the positive symptoms. |  |  |  |
| One example of the research implicating NMDA receptors in schizophrenia relates to the way the brain normally processes information. Beyond strengthening connections between neurons, NMDA receptors amplify neural signals, much as transistors in old-style radios boosted weak radio signals into strong sounds. By selectively amplifying key neural signals, these receptors help the brain respond to some messages and ignore others, thereby facilitating mental focus and attention. |  |  |  |