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Cannabis Use and the Etiology of Schizophrenia: Evaluating Necessary, Sufficient, and Contributory Causes

The increasing prevalence of cannabis use, especially among adolescents and young adults, has sparked growing concern about its potential role in the development of psychiatric disorders, and most notably, schizophrenia. As we seek to understand the complex origins of schizophrenia, applying the concepts of necessary, sufficient, and contributory causes can help clarify cannabis's role in the disorder’s etiology.

To begin, cannabis is not a necessary cause of schizophrenia. This means that schizophrenia can and does occur in individuals who have never used cannabis. The disorder has been documented in populations and time periods where cannabis use was rare or nonexistent, indicating that the presence of cannabis is not required for schizophrenia to manifest.

Similarly, cannabis use is not a sufficient cause of schizophrenia. Not all individuals who use cannabis, regardless of frequency or potency, go on to develop schizophrenia. In fact, the majority do not. This demonstrates that cannabis alone cannot account for the development of the disorder and that other biological, environmental, and psychosocial factors must be involved.

However, the strongest evidence suggests that cannabis functions as a contributory cause of schizophrenia. A contributory cause increases the likelihood that a disorder will develop, though it is neither necessary nor sufficient on its own. Numerous studies have shown a clear association between frequent cannabis use and an elevated risk of developing psychotic symptoms or full-blown schizophrenia, particularly in genetically vulnerable individuals. Di Forti et al. (2019) found that daily use of high-potency cannabis was associated with a fourfold increase in the risk of psychosis, and in some cities, such as London and Amsterdam, cannabis use explained a significant proportion of new schizophrenia diagnoses.

This finding aligns with the diathesis-stress model of mental illness, where genetic vulnerability (the diathesis) interacts with environmental stressors, like cannabis use to trigger the onset of psychiatric disorders. Individuals with a family history of psychosis, or specific genetic variants such as the COMT gene, may be more likely to develop schizophrenia following cannabis exposure. This interaction helps explain why cannabis is a risk factor for some, but not for others.

In conclusion, while cannabis is neither a necessary nor sufficient cause of schizophrenia, the evidence strongly supports its role as a contributory cause and one that can significantly increase risk, particularly in individuals with preexisting vulnerabilities. Understanding cannabis’s place within the broader etiology of schizophrenia is essential for public health education, clinical prevention efforts, and more informed decision-making around cannabis policy and mental health support.