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Drug Induced Psychosis or Schizophrenia?

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Editorial

Cannabis, cocaine, amphetamines and derivative substances so as hallucinogens and other drugs with psychotomimetic properties has reached an extensive misuse often producing psychotic reactions similar to primary psychotic disease like schizophrenia. In particular cannabis and cocaine use by young people has been increased during these years, and the age of first use is dramatically decreased [1,2].

However, cannabis, or marijuana, is the most common illegal substances used in the most of the countries, including UK and USA. About 20% of young people now report its use every week or heavy use (use on major than 100 occasions). The use of such substances has dramatically increased particularly during early adolescence, when the brains go through a developing phase and can be especially sensitive to environmental exposures. Experimental studies and surveys of users show strong evidence that cannabis intoxication can initially produce temporary effects, usually mild, but also psychotic and affective symptoms and with time it can induce a chronic psychopathological picture that can persist, or can occur independently of acute intoxication effects [3].

On the other hand cannabis is on sale, at present, in a more potent form and synthetic compounds, also known as "new psychoactive substances" increasing globally and the phenomena may be under valued. Synthetic cannabinoids (SCs) result among the most new psychoactive substances used. They are usually purchased as marijuana-like drugs, marketed as herbal blends and considered risk-free by the inexperienced users. Furthermore, contrary to Δ^9 -tetrahydrocannabinol, SCs may lead more frequently to severe health consequences like tachycardia, anxiety, hallucinations, including violent behaviour and psychosis. Reports suggest that SCs may either exacerbate previously chronic psychotic symptoms at least in sensible individuals or trigger new-onset psychosis in individuals with no previous history of psychosis [4,5].

As a whole, preclinical data seem to support the hypothesis that adolescent exposure to cannabinoids might represent a great risk for the development of psychotic-like symptoms in adulthood since it interferes with maturational events occurring in the adolescent brain. This can leads to alterations affecting brain connectivity and functionality similar to those present in

schizophrenic patients. The most likely mechanism underlying these effects involves the disruption of maturational events within the endocannabinoid system. Indeed, the impairment of the endocannabinoid system maturation might have an impact on the correct neuronal evolution peculiar of the adolescent brain leading to altered adult brain functionality and behavior.

In particular cannabis derivatives produce their CNS effect through activation of the endocannabinoid (eCB) system, a recently discovered signaling system comprising the cannabinoid CB1 and CB2 receptors, their intrinsic lipid ligands, endocannabinoids (eCBs), such as the N-arachidonylethanolamide (anandamide, AEA) and the 2-arachidonoylglycerol (2-AG), and the associated enzymatic process (biosynthetic, transporters and catabolic enzymes).

The biological actions of delta-9-tetrahydrocannabinol and synthetic cannabinoids are mediated primarily by CB1 and CB2 receptors which both belong to the super family of G protein-coupled receptors (GPCRs) [6].

Dependence by substances and schizophrenia are both associated with dopaminergic dysfunction. In other words, It has been proposed that the association between cannabis use and psychosis including schizophrenia is mediated by an alteration of dopamine (DA) transmission. In other words, chronic cannabis use is associated with reduced DA synthesis and introduces the hypothesis that cannabis increases the risk of psychosis by inducing the same DA alterations of schizophrenia. Preclinical studies indicate that the acute administration of Δ^9 -tetrahydrocannabinol, the main psychoactive component of cannabis, increases DA mesolimbic neuron firing rates via endocannabinoid CB1 receptor agonism. CB1 agonists inhibit striatal dopamine reuptake, selectively increase tyrosine hydroxylase expression, and increase DA release and synthesis in the majority of, studies [7].

Furthermore, several studies have reported a link between cannabis use in adolescent and the development of stable psychosis in early adulthood. This condition is further complicated by the high rate of concomitant substance abuse by subjects primarily affected by a psychotic illness which, especially in young patients with an early-phase psychotic disorder, can make the diagnosis very problematic. The literature data concerning the characteristics of psychotogenic drugs and the psychotic symptoms can induce to discuss the

association between substance abuse and psychosis with particular emphasis on the differential diagnosis of a primary, some authors speaking directly of schizophrenia, and substance-induced psychotic disorder.

On the other hand co-occurrent drug abuse is a very frequent condition among patients presenting their first episode of schizophrenia, prevalence rates ranging between 25% and 60% [8].

The comorbidity of schizophrenia and substance abuse is associated with more frequent relapses, more positive symptoms and depression, cognitive impairment, and a poorer outcome and treatment response. It has been hypothesised that substance abuse could trigger psychotic symptoms in vulnerable individuals, furthermore substances might be used to self-medicate symptoms of schizophrenia. Formulating a psychiatric diagnosis in patients who experience the onset of psychotic symptoms during episodes of current or recent psychoactive substance use is often challenging, even though some key predictors, such as differences in demographic, family, and clinical domains, could help emergency clinicians to correctly classify early-phase psychotic disorders that co-occur with substance use [8].

Actually, despite the several study limitations, such distinction between primary and secondary drug induced psychosis tends to vanish over a long-time showing that drug induced psychotic disorders are often followed by development of persistent psychotic conditions even if it seems always difficult to speak of schizophrenia. Further researches on larger samples and primarily on longer period of time is needed to clarify such an important issue by medical, social and political implications.

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