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ИНДУЦИРОВАННЫЕ ПСИХОАКТИВНЫМИ ВЕЩЕСТВАМИ ИЛИ АССОЦИИРОВАННЫЕ С НИМИ ПЕРВИЧНЫЕ ПСИХОЗЫ? ПРОДОЛЖЕНИЕ ДИСКУССИИ. ОТВЕТ И.А. ФЕДОТОВУ И СОАВТОРАМ

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Это письмо в редакцию продолжает дискуссию о сходствах и различиях между вторичным психозом и шизофренией, начатую авторами статьи «Индуцированные наркотическими веществами психозы и шизофрения: точки соприкосновения» (Федотов И.А., Кватрон Д., Шустов Д.И. Индуцированные наркотическими веществами психозы и шизофрения: точки соприкосновения // Российский медико-биологический вестник имени академика И.П. Павлова. 2020. Т. 28, №4. С. 593-604. doi:10.23888/PAVLOVJ2020284593-604).

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SUBSTANCE-INDUCED OR SUBSTANCE-ASSOCIATED PRIMARY PSYCHOSES? CONTINUING THE DISCUSSION. A RESPONSE TO I.A. Fedotov, et al.

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This letter to the editor continues the discussion about the similarities and differences between secondary psychosis and schizophrenia, which was initiated by the authors of the article «Substance-induced psychosis and schizophrenia: the interaction point» (Fedotov I.A., Quattrone D., Shustov D.I. Substance-induced psychosis and schizophrenia: the interaction point. *I.P. Pavlov Russian Medical Biological Herald*. 2020;28(4):593-604. doi:10.23888/PAVLOVJ2020284593-604).

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Substance induced psychotic disorder (IPD) is defined by the presence of delusions and/or hallucinations reflecting the brain effects of a psychotropic agent (substance of abuse and/or medication), based on evidence from the clinical interview, physical examination, and laboratory findings. It is a severe and a common complication of drug use (1), which can be manifested during intoxication or with drawal phases (2) and be induced by several different substances, including alcohol, cocaine, cannabis, amphetamines, and hallucinogens (3).

The relationship between IPD and 'primary' psychoses (in particular, schizophrenia) has long been a topic of interest and controversy due to the relative paucity of scientific evidence revealing the natural history of induced psychotic disorders and clear markers that can differentiate them from schizophrenia. Indeed, the current Diagnostic and Statistical Manual of the American Psychiatric Association (DSM-5) proposes a distinction that is only based on timing the persistence of psychosis beyond one month after last exposure to the implicated substance would exclude IPD and suggest schizophrenia (4). To complicate conceptual, diagnostic, and treatment approaches to psychosis and substance use, it is well known that certain substances, in particular cannabis, are risk factors for the onset and complication of schizophrenia (5).

Schizophrenia and IPD may both interact and diverge in multiple aspects, as it has been suggested in a recent publication at this journal (6). I.A. Fedotov, et al. highlighted evidence showing neurobiological mechanisms common to both disorders: Most psychoactive substances directly or indirectly produce dopamine increases in the striatum, activating a neural circuitry responsible for the 'positive' psychotic symptoms of (hallucinations and delusions), which are both characteristic of IPDs and schizophrenia of (hallucinations and delusions), which are both characteristic of IPDs and schizophrenia. In addition, some substances may induce 'negative' symptoms, even though these have not been described as characteristic of IPD: for instance, PCP and ketamine, through the increase of glutamate transmission, have been shown to reduce prosocial activity and induce other negative symptoms (7). All this makes it more difficult for clinicians to differentiate between IPD and a schizophrenic episode in acute presentations. As a help in clinical decisions, I.A. Fedotov, et al. offered an array of typical differences between both disorders in patients' sociodemographic characteristics. They suggested that patients with IPD tend to present with later onset of illness and comorbid antisocial personality disorder, while patients with schizophrenia would more typically present with more 'florid' positive and negative symptoms as well as family history of schizophrenia (6).

In this letter to the editor, we would like to add further discussion to the topic, stressing the diagnostic difficulties to differentiate IPD and schizophrenia that also arise taking into account additional multidisciplinary research in psychosis and substance use, as well as potential implications for early management of psychotic episodes in patients using substances. To start with, there is emerging evidence that supports IPD and schizophrenia as two clearly defined separate entities. First, on a neurobiological level, a positron emission tomography (PET) study published in 2014 shown hypermetabolism in the posterior cingulum and precuneus in patients with induced psychosis but not in those with schizophrenia in spite of having taken the same substance; this seems to support the known predominance of positive symptoms in IPD (8). Secondly, vulnerability to schizophrenia is strongly determined by genetics, while IPD requires the exposure to substances as a necessary cause (i.e., it is a more 'environmental' condition). For instance, for schizophrenia there is a 50% concordance rate in monozygotic twins (9), and about 40 candidate genes have already been identified, supporting the neurodevelopmental hypothesis of schizophrenia (2). These findings suggest that while schizophrenia and IPD cannot be differentiated simply through observing symptoms and demographic characteristics,

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they seem to represent distinctive conditions in terms of etiology (7).

Yet this does not shed enough light to justify the high co-occurrence of substance abuse and schizophrenia. Indeed, the prevalence of substance use disorders in individuals with schizophrenia is significantly higher than in the general population (10). A drug abuse institute in Thailand found that 25% of their patients had schizophrenia (11). A first theory to explain this overlap involves the « two-hit» genetics-environment model, which posits that a pre-existing neurobiological vulnerability would synergically interact with environmental factors, such as substances, inducing schizophrenia (12). Another theory proposes that individuals with schizophrenia are at higher risk of abusing substances because of their poor cognitive and social functioning, as well as social (poverty) and other cumulative factors (13). Conversely, the «Reward Deficiency Syndrome» proposed in 1999 assumes a common dysfunction in the reward circuitry, which would lead patients to « self-medicate» with substances as a means of compensation (14). Similarly, the « self-medication» model proposes that individuals with psychosis seek the effects of specific substances to alleviate painful symptoms or medication side-effects (15). Finally, we would like to highlight an unifying postulated by Khokhar J.Y., et al.: according to them, there is a shared vulnerability for substance use in patients who are at risk of developing psychosis; the synergy of genetic risk and early environmental insults would produce dysfunctional mesocortico-limbic brain reward circuits, which would make ‘pre-psychotic’ (high-risk) adolescents more prone to using substance; in the last instance, the use of those substances would trigger the onset of schizophrenia in those vulnerable individuals (16).

These theories, while needing more empirical support, show promise in the understanding of the complicated interrelation between IPDs and schizophrenia, and are open to considering psychosis as a spectrum rather than a single entity (17). In fact, there is a high conversion rate of IPD to schizophrenia. A Swedish national registry-based study revealed that 11% of individuals diagnosed with IPD were later diagnosed with schizophrenia (18). In another study with a sample of 6788 individuals, the 20-year conversion rate to either schizophrenia or bipolar disorder for patients with IPD was 32.2% (19). These elevated rates of transition make even more compelling the need to establish early diagnosis differentiating between schizophrenia and IPD if it is possible at the moment of the first clinical presentation.

On the other hand, in a study conducted in China, which followed individuals presenting for IPD, persistent psychotic symptoms were more common in those with a positive family history of mental illness, an earlier age of onset of illicit drug use and a longer history of illicit drug use (20). In addition, both the nature of the abused substance and the severity of substance use were found to influence the progression to schizophrenia (18).

We want to stress, nevertheless, that the absence of such factors does not rule out the diagnosis of primary psychotic disorders, and we definitely count on scarce evidence to allow for accurate diagnosis and prognosis, importantly about the prediction of the risk of transition to schizophrenia. Why is it so important to make this prediction? Schizophrenia is a debilitating diagnosis severely affecting the quality of life of millions of people worldwide (21), and associated with high morbidity and early mortality (22). Delays in the start of effective treatments can have unfortunate, irreversible consequences, in part associated with an increased risk for progressive brain damage that could make prognosis bleak (23).

As a final reflection, and inviting colleagues to add to the discussion, one wonders whether every first episode psychosis with the involvement of substances should be treated as a primary psychosis.

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