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Letters to the Editor

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THE DISCONNECT BETWEEN THE SCIENCE ON CANNABIS AND PUBLIC HEALTH CAMPAIGNS

The review of cannabis-induced psychosis by Ian Hamilton in *Addiction* [1] raised key issues that warrant further exploration.

In focusing upon the literature up to the turn of the 21st century, the author omitted the subsequent explosion of scientific inquiry into a causal relationship between cannabis use and chronic psychotic disorders. The author pointed towards one type of relevant research, citing the seminal dose–response work of Andréasson [2] while disregarding recent publications on this topic [3,4], as well as other important criteria which have been applied to test causation: prospective studies on the timing of use and psychosis [5]; age of onset of schizophrenia in users versus non-users [6]; percentage exhibiting psychotic symptoms from clinically administered Δ^9-tetrahydrocannabinol [7]; conversion from temporary to chronic psychosis from cannabis versus other drugs [8]; likelihood of cannabis use in those with a family history of psychosis [9]; cannabis-induced psychosis without such a family history [10]; rates of psychosis in siblings discordant for cannabis [11]; course of recovery from psychotic breaks in non-users versus those who used and quit, or continued use [12]; and whether family history of psychosis affects recovery from cannabis-induced psychotic breaks [13]. Collectively, this literature supports cannabis having a causal impact on chronic psychotic disorders.

The second key issue is the rarity of the outcome. A disease is considered rare in the United States if it afflicts one person per 1500 (Rare Disease Act) or, in Europe, one per 2000 (European Commission on Public Health). As the recent literature supports a fourfold increase in risk for chronic psychosis in heavy users [14], and given that the life-time morbid risk for schizophrenia is, on average, 0.7% [15], the outcome would be rare by no generally accepted standard. It appears plausible that in asserting cannabis-induced psychosis to be relatively rare, the author was influenced by the publication of Hickman [16] reporting on the number needed to abstain from using the lower-strength cannabis prevalent in the last century in order to prevent one case of schizophrenia [number needed to prevent (NNP)], and showing that 2800 was the NNP for males aged 20–24 years. What is often not understood is that the NNP was per year, i.e. to prevent one schizophrenia case per year in each age group studied [17]. If treatment programs are geared towards a couple of decades of prevention, the NNP for today's cannabis across the age groups/genders combined can be estimated to be far fewer than 100 individuals.

The author raised a third key issue, that being the reason for lack of acceptance of a causal impact of cannabis on psychosis, correctly placing some blame on the outcome being difficult to discern at the population level. Indeed, discernable impacts on whole populations depend upon tracking both the variable and the outcome; on the population size interacting with the variable; and on whether there are changes in untracked variables over time that influence outcome. The involvement of gene–environment interactions and the failure to identify a mechanism for how cannabis induces psychosis have also been cited by some scientists as reasons for downplaying the likelihood of a causal basis.

Conversely, cannabis use causing chronic psychosis should be no different than carcinogens causing cancer, where an agent suspected of being a carcinogen based on dose–responsive cancer rates in large case–control or prospective studies will move from the category of suspected carcinogen to known carcinogen if it is also shown to cause cancerous or pre-cancerous lesions in the laboratory. Whether or not genes influence the development of the cancer is considered a moot point in that designation, as all cancers involve some type of gene–environment interaction. With tobacco smoking, 72–84% of individuals (depending on where they live) can smoke for most of their lives without developing lung cancer [18]. Despite the reasons for differential susceptibility not being well understood and the exact mechanism of the carcinogenesis still being determined, large public health campaigns have been directed against tobacco use for more than half a century. In contrast, no widely broadcast public health advisories or Surgeon General's warnings have been issued about the mental health impact of cannabis, at a time when it is undoubtedly the most well-studied and well-replicated finding for an environmental factor promoting psychotic outcomes.

Declaration of interests

None.

Keywords Cannabis, causal, clinically administered Δ^9-tetrahydrocannabinol, family history, gene-environment, other drugs, psychosis, recovery, schizophrenia, siblings.

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RESPONSE TO MILLER (2017): COMMUNICATING THE ROLE OF CANNABIS IN THE RISK OF DEVELOPING PSYCHOSIS

The points made by Dr Miller [1] raise further important issues we need to consider in thinking about the relationship between cannabis and psychosis.

With regard to later research, I accept that much of the research published after the millennium was not included in my original paper, as the focus was upon how work prior to this time had evolved and how well it had stood the test of time.

The second point highlighted by Dr Miller about the definition of rarity is helpful. Unfortunately, any calculation used to suggest that cannabis psychosis crosses this threshold relies upon epidemiological data that have significant limitations, a point acknowledged by McGrath and colleagues, whose work Dr Miller cites [2]. Any calculation based on estimates of 'heavy users' is problematic; although used frequently in the literature, it has no standardized meaning.

Dr Miller raises the role of treatment programmes in preventing use of cannabis. Prevention would need to extend beyond treatment programmes if any impact on uptake of cannabis use at a population level were to be realized. Unfortunately, positive evaluations of drug prevention programmes meet most people's definition of 'rare' [3]. Economic evaluation of such programmes are rarer still. This is unlikely to entice policymakers to invest in such programmes as they currently stand.

Dr Miller refers to the strength of cannabis that was prevalent in the last century. We need to be careful when referring to lower-strength cannabis, as the increasing cannabis potency narrative has developed without much empirical analysis. Most of what we know about cannabis potency is based on proxy measurement which used the analysis techniques of the day [4]. More sophisticated methods of analysis, such as gas chromatography, have been developed in recent years [5]. Add to this that even the specific studies exploring cannabis and psychosis restrict definitions of cannabis exposure to used or not used, with no detail about potency or type.