BJPsych | The British Journal of Psychiatry (2026)
1–4. doi: 10.1192/bjp.2026.10802

Feature

Self-medication as metaphor: why psychiatry should revise an *(in)*convenient narrative about substance use and mental disorders

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Summary

The term *self-medication* remains an influential narrative to explain why some patients with mental health problems use psychoactive substances. This Feature offers a conceptual critique of its broad use in psychiatry. We argue that self-medication often functions less as a testable explanatory model and more as a rhetorical metaphor: clinically convenient, yet scientifically under-specified and normatively loaded. Reviewing large epidemiological studies, ecological momentary assessment and longitudinal sequencing, we noted that while evidence supporting coping motives and negative reinforcement exist, they do not support adopting self-medication as the main mechanism for comorbidity or as a therapeutic interpretation of substance use. Our concern is that over-reliance on this metaphor can subtly shift clinical stance from curiosity to assumption, carrying three main clinical risks: (a) fostering paternalism, (b) diagnostic drift through reverse inference and (c) explanatory distraction. Moreover, medicalising substance use as self-medication does not necessarily reduce stigma and may paradoxically reinforce narratives of biological defectiveness. We propose a few safeguards for using this metaphor within rigorous clinical formulation: replacing assumptive language with structured motive-inquiry, recognising plural mechanisms and prioritising evidence-based addiction interventions over pharmaco-centric responses.

Keywords

Mental disorders; substance use; dual diagnosis; self-medication; diagnostic reasoning.

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Language and implicit assumptions

Words do not merely describe reality; they organise it. Insisting on linguistic seriousness is not semantic pedantry; it is an argument about respect, clarity and epistemic power.

We argue that in contemporary clinical and scientific psychiatry talk, the phrase *self-medication* often functions less as a testable mechanism and more as a metaphor, with ethical and epistemic implications.

The use of prescribed medications and non-prescribed psychoactive substances is interpreted differently across communities, substances and contexts. The same or similar psychoactive effect may therefore acquire different meanings depending on whether it occurs within a prescription, self-directed use of medication, recreational use, ritual practice or an illicit market. Within healthcare systems, prescribed medication is ideally embedded in an institutionally authorised clinical practice involving assessment, diagnosis, prescription, professional accountability, regulatory safeguards and therapeutic expectations.[1] By contrast, substance use is often interpreted through less stable and more contested frameworks, including individual choice, dependence, risk, illegality and negative prognostic attributions, such as loss of control, relapse, chronicity, reduced agency or difficult treatability.[2,3]

This distinction is not absolute. Prescribed medication can be ineffective, harmful, misused or shaped by commercial and institutional interests; conversely, non-prescribed psychoactive substance use may be socially accepted, culturally meaningful, subjectively helpful or embedded in collective practices. Nevertheless, the word medication generally carries a therapeutic and institutional register. When attached to substance use through the term self-medication it may import that register into behaviours whose meanings are multiple and not necessarily medical. The resulting narrative can sound empathic – *they are coping* – and mechanistic – *they are correcting symptoms* – while quietly narrowing psychological formulations about the patients' motives, contexts and constraints. This narrative also appears intuitively coherent, yet its coherence is largely rhetorical.

Our argument is not that self-medication never occurs, nor that clinicians routinely misuse the term, but that the term should not be asked to carry more explanatory and clinical meaning than the evidence can support.

What the self-medication paradigm claims and what it leaves vague

The self-medication hypothesis was influentially formalised by Khantzian (1997) as a psychodynamic hypothesis, suggesting that individuals use specific substances to relieve painful and intolerable affective states.[4] Indeed, high comorbidity between mental disorders and substance use disorders has been consistently documented and linked with worse outcomes than either alone.[5] Yet high co-occurrence does not specify mechanism. Self-medication can be a patient-valid description and sometimes a risk marker, but it is a poor default mechanism.

If self-medication is treated as more than a metaphor, it implies at least some empirical assumptions:

(a) Intentionality: the person primarily uses a substance to manage an initial problem, such as distress, insomnia, anxiety or inattention. This may include continuing medication beyond medical advice or obtaining non-prescribed substances when access to prescribed treatment is limited. However, although some substance use may be motivated by attempts to manage negative affect or other perceived problems, this cannot be assumed for all people, all substances or all episodes of use.[6]

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(b) Specificity: the substance is preferentially selected because of a perceived fit between its pharmacology and the symptom profile (e.g. stimulants for inattention). This is one of the most problematic assumptions, since drug choice is influenced by many factors, including availability, cost, social context, habit, expected pleasure, peer networks, previous exposure and polysubstance use.[7]
(c) Perceived benefit: the substance is experienced as helping to manage the perceived problem. Even prescribed medication does not invariably improve symptoms or functioning; however, perceived short-term benefit does not, by itself, establish sustained symptom improvement, functional benefit or therapeutic specificity.[8]
(d) Temporal direction: symptoms precede use, and use persists because it is experienced as symptom-relieving. This temporal sequence may occur in some cases. However, it should not be generalised to all patients or all forms of substance use, especially when mental health symptoms, intoxication, withdrawal and social adversity may influence one another bidirectionally over time.[9]

The core scientific problem is that the paradigm often mixes motive, mechanism and clinical interpretation without keeping these levels distinct. A patient may report using a substance to cope; this does not by itself establish why the disorder and substance use co-occur, nor whether the substance has therapeutic meaning.

Self-medication is commonly treated as established despite limited direct, prospective, mechanism-specific evidence. Some longitudinal epidemiological studies have supported self-medication as a predictor of later substance use disorder.[9,10] Conversely, ecological momentary assessments (EMAs) of motives of substance use research have cautioned that trait-like motive questionnaires and momentary motives may not measure the same construct.[11] Taken together, these findings suggest different risk pathways, of which self-medication is only one of the possible subjective accounts.

Self-medication is normatively loaded

Epistemic inflation

Earlier reports have raised concerns about the limited empirical support for broad applications of self-medication to explain psychoactive substance use among individuals with mental disorders.[12,13] Still, the narrative remains a recognisable explanatory frame in patient accounts, clinical discourse and research literature.[2,14] We argue that the narrative carries an inferential risk, as it can make one possible interpretation of substance use appear more coherent than the available evidence warrants.

The self-medication story tends to overgeneralise from three kinds of findings:[9,15,16] (a) people endorse coping motives, (b) coping is associated with worse outcomes and (c) longitudinally, self-identified self-medication predicts later disorder.

Acute psychoactive relief may reflect withdrawal relief, sedation, stimulation, cognitive narrowing or short-lived affect suppression with long-term rebound. Similar acute relief may also occur with prescribed psychoactive medication, for example during a short course of benzodiazepines. However, in clinical prescribing, expected benefits are ideally situated within the therapeutic relationship, in which indication, formulation, dose, duration, monitoring, adverse effects and functional outcomes, can be made explicit and reviewed.[17] By contrast, self-medication reasoning can retrospectively convert subjective relief into evidence that the substance was treating an underlying disorder.[18]

The smoking literature illustrates this inferential risk, particularly when the self-medication hypothesis is applied loosely in clinical discourse. Smokers often experience cigarettes as psychologically beneficial, yet a large systematic review and meta-analysis found cessation is associated with improvements in depression, anxiety, stress and quality of life, suggesting that perceived self-treatment can sometimes be misattributed relief from withdrawal cycles rather than symptom remediation.[19] Therefore, acute relief can be clinically plausible and pharmacologically real, but it does not by itself establish therapeutic action, disease specificity or diagnostic meaning.

Agency and paternalism: 'fun' is not an afterthought

The phrase self-medication sounds patient-centred, but it can be patronising in at least two ways.

First, it attributes a medical logic to behaviour that may be driven by many logics: pleasure, belonging, curiosity, performance pressure, habit, despair or a varying mixture of these. Empirical work on motives for substance use repeatedly found that coping is only one motive among several, and not always the dominant one. For alcohol, a large meta-analysis (229 studies; >130 000 participants) found that enhancement motives – drinking to increase positive affect – and coping motives – drinking to reduce negative affect – are among the strongest predictors of drinking problems, with enhancement motives especially strongly associated with alcohol use itself.[20] Therefore, pleasure seeking and positive reinforcement are central, often as central as coping.

Second, self-medication frames the patient as a quasi-clinician who attempts treatment – but also as someone who does so incompetently. The patient is simultaneously credited with insight – *they are treating symptoms* – and discredited as unsafe – *they choose the wrong drug*. This ambivalence is not neutral; it structures the clinical encounter and flattens clinicians' curiosity about patient experiences. Clinical knowledge may make the term self-medication descriptively more appropriate in some cases, for example when a clinician uses a psychoactive medication for themselves. However, knowledge alone does not eliminate concerns about objectivity, nor does it replace clinical formulation. Literature on psychiatrists' self-treatment suggested that, despite professional knowledge, self-medication may be shaped by concerns about confidentiality, social and professional stigma and help-seeking attitudes.[21] Thus, even in informed users, self-medication can remain a clinical interpretation that risks overlooking complex motives.

Stigma

Self-medication can be invoked to reduce blaming accounts of substance use. In the addiction field, the brain disease model has similarly been promoted partly as a destigmatising framework.[22] However, medicalised causal stories do not necessarily reduce stigma in a straightforward way. Previous work suggested that neurobiological narratives may reshape, rather than remove, stigmatising identities, whereby reduced blame may coexist with greater prognostic pessimism, perceived dangerousness or assumptions of reduced agency.[23]

If self-medication is used as a quasi-biological story – e.g. dopamine deficit to cocaine as correction – it may inadvertently increase perceptions of chronic defectiveness, while obscuring social determinants and personal agency.

ADHD and stimulants: a test case

Convergent epidemiological findings associate attention-deficit and hyperactivity disorder (ADHD) and stimulant abuse.[24] This substantial overlap warrants attention, not interpretive shortcuts.

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The ADHD-stimulants story is often presented as self-medication because it is pharmacologically plausible: cocaine is a stimulant; stimulant medication treats ADHD; therefore, cocaine use is framed as improvised pharmacotherapy. Examples of this reasoning can be found in research reporting that clinical suspicion of adult ADHD may arise from the role of cocaine in symptom control, or that a paradoxical calming reaction to cocaine may increase diagnostic suspicion of co-occurring adult ADHD.[25,26]

In real-world clinical reasoning, our concern is that, when the self-medication frame is used loosely, reports of stimulant-related relief may acquire excessive diagnostic weight: stimulant use may become a proxy marker for ADHD – diagnosis-by-substance – rather than a hypothesis to be tested against developmental history, impairment trajectories and the confounding effects of intoxication/withdrawal. This is where metaphor becomes an unreliable method, and where risk accumulates.

Clinical risk of overpromoting the metaphor

The following risks are not presented as empirically proven causal effects of the use of the term self-medication itself. Rather, they are conceptual and inferential risks that may be logically derived from the evidence reviewed above.

Explanatory distraction and shadowing of real problems

Overpromoting self-medication risks funnelling research into a single motivational lane (i.e. negative affect relief), while underweighting positive reinforcement, social learning, cue reactivity, habit formation, trauma-related processes, socioeconomic and network determinants and the pharmacology of withdrawal and rebound. It distracts from competing mechanisms and from multilevel causation. This narrowing is difficult to justify given evidence that substance-use motives are heterogeneous,[6] momentary motives may differ from trait-like reports[11] and patterns of use challenge substance-specific explanations.[7]

Shadowing may also occur at the level of suffering. If drugs are treated as medication, clinicians may under-attend to what else is being managed: trauma symptoms, shame, loneliness, precarious housing, workplace demands or other personal problems.

Inadvertent legitimation of substances as coping tools

From an ethical perspective, we should be aware of the risk of iatrogenic communication. Overreliance on labelling and suggestive language in psychiatry and psychotherapy may lead patients to literalise the metaphor such that it permanently transforms their self and world perceptions into fixed views.[27] Additionally, the descriptor may promote excuse making, in that individuals may avoid responsibilities and justify their behaviour through evoking their psychiatric condition.[13]

Diagnostic drift

The self-medication narrative can promote diagnostic shortcuts: stimulant use to ADHD, cannabis use to psychosis vulnerability and alcohol use to social anxiety. This can produce overdiagnosis – by diagnosing a comorbid disorder based on the substance used – and missed diagnosis – by attributing problems to self-medication rather than assessing them on their own terms.

Additionally, interpreting substance use as a misguided attempt to treat an underlying illness may create a discursive environment where pharmacological substitution feels logically mandatory and overprescription becomes plausible, with consequent ethical and social implications.[8]

This can happen as a result of a fundamental confusion between two conceptual models of psychopharmacology: the drug-centred model and the disease-centred model.[28] When a patient experiences temporary relief from distress or inattention after using a stimulant (e.g. cocaine), this is a predictable, non-specific psychoactive effect of the drug on mental states (the drug-centred perspective). However, self-medication reasoning often retrospectively filters this altered state through a disease-centred lens, erroneously assuming the substance is bridging a pathological deficit or correcting an underlying chemical imbalance.[29] This rationale is then used to justify the prescription of medically substitute medications. Ultimately, reducing the complex cycle of addiction to a missing neurochemical deficit often leads to suboptimal, if not unsafe, pharmacological outcomes.

Towards an alternative: formulation over metaphor

We do not propose banning the phrase self-medication, nor do we assume that clinicians do not already ask about motives or formulate substance use broadly. We emphasise the importance of using self-medication critically, as one possible patient account or clinical hypothesis, and being guarded against its use as a diagnostic or prescribing shortcut.

Practically, we suggest strengthening three safeguards:

(a) Replace motive assumptions with structured motive inquiry: ask explicitly – *What do you like about it? What does it change? What does it cost you? When is it worth it, when not?* This keeps pleasure, sociality and relief on the table without moralising.
(b) Acknowledge plural mechanisms: motives are dynamic and not reliably captured by trait labels. Stimulant use does not diagnose ADHD; it may reflect availability, culture, reinforcement and individual preference, until a developmental formulation says otherwise.
(c) Keep addiction science in view: for example, in the context of cocaine use disorder, meta-analytic evidence suggested that contingency management was associated with objective reduction in use, with pharmacotherapy effect remaining more limited.[30] Leaning too heavily on psychiatric medication narratives may risk privileging medicalising substance use even when integrated interventions with stronger evidence for substance use outcomes should be offered.

Closing perspective

Self-medication is attractive because it sounds compassionate and mechanistic at once. That is precisely why its uncritical use may be misleading: it can transform empathy into epistemic closure. An alternative standpoint does not deny that some patients use substances to manage distress or symptoms. It insists that we do not assume this motive, romanticise it as treatment or let it dictate diagnosis or prescribing. We can validate coping without turning drugs into a clinical allegory. We owe patients something more respectful than a metaphor: careful questions, multiple hypotheses and language that preserves agency without abandoning compassion.

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First received 20 May 2026 UTC, final revision 22 Jul 2026 UTC, accepted 16 Aug 2026 UTC

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Acknowledgements

The authors used ChatGPT-Edu (version 5.5) to improve English grammar and phrasing in limited sections of the manuscript, without generative use.

Data availability

This article did not generate or analyse any data-sets; no new code was created.

Author contributions

M.M. and G.M.G. contributed to the conceptualisation of the study. M.M. contributed to writing the original draft. M.M. and G.M.G. contributed to writing, reviewing and editing the manuscript. G.M.G. contributed to supervision. The authors approved the final version of the manuscript.

Funding

This research received no specific grant from any funding agency, commercial or not-for-profit sectors.

Declaration of interest

None.

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