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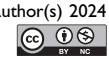

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That men should put an enemy in their mouths to steal away their brains: Reconsidering the origins of model psychosis

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Abstract

The promises of the Prozac century have fallen short; the number of novel, therapeutically significant medications successfully completing development shrinks every year; and the demand for better treatments constantly grows. Answering these hardships is a renewed optimism concerning the efficacy of controlled psychedelic therapy, a *renaissance* that has seen the resurgence of a familiar concept: intoxication as model psychosis. And yet, little has been made of where this peculiar idea originates. Why did we come to liken psychosis to intoxication, and why is this an idea we find so hard to shake? Questioning the conventional narrative that identifies the concept as emerging in the mid 19th century, this article seeks to uncover the conceptual foundations underlying what is now intended by 'model psychosis'. This investigation begins with an assessment of both Moreau de Tours's concept of hashish madness in 1845 and Emil Kraepelin's study of artificial insanity in the 1880s–90s. In seeking to contextualize these ideas, this article further considers the deeper historical association between intoxication and psychosis, instead proposing that intoxication represents an originary conception of madness. Bringing this examination into the 19th century, it becomes apparent that perceptions of intoxicants, and intoxication, were immanently participatory in the emerging understanding of psychosis. The contemporary understanding of model psychosis comes into focus when these elements coalesce with the advent of psychological modelling. Ultimately, the goal is not merely to understand how and why model psychosis became thinkable, but to examine how overlooked concepts have engendered new ways of being neuro-psychiatric subjects.

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Introduction

'Model psychosis' initially appears to be fairly simple to define. It refers to the use of intoxicants, especially hallucinogens, to artificially produce symptoms 'sufficiently similar' to those of acute mental illnesses (Langlitz, 2013: 139). It is 'the suggestion that […] drugs could be used to scientifically model the phenomenon of psychosis' (Friesen, 2022: 592). Each definition circumscribes model psychosis in accordance with its novel, experimental comparison of mental states with intoxicated states. Yet, for all this seeming simplicity, the question of the first example of model psychosis remains a controversial one. Kurt Beringer first used the terms 'artificial model of psychosis' in 1927 to describe mescaline intoxication, a refinement on the concept of 'experimental psychoses through mescaline' expounded in 1922/3 (Beringer, 1923, 1927). The language of psychomimetics was introduced 30 years later, in 1957, by Humphry Osmond, who compared the reported experience of schizophrenia with the effects of LSD (Corlett *et al.*, 2011; Osmond, 1957). But there are earlier examples that frequently make their way into the history sections of contemporary scientific studies on model psychosis, most notably Moreau de Tours's study on hashish madness and Emil Kraepelin's research on the psychometrics of intoxication. Where does model psychosis truly begin?

This uncertainty surrounding the first emergence of something that might be referred to as proto-model psychosis persists in historical studies as well. Langlitz's boldly titled 'Ceci n'est pas une psychose: Toward a Historical Epistemology of Model Psychoses' tips its hat to the enterprising efforts of Moreau and Kraepelin as the first to develop the concept of model psychosis, only to localize its 'historical epistemology' to the 'empirical concept and practice of the hallucinogen model of psychosis as an aspect of the 'regional epistemology' of psychopharmacology' (Langlitz, 2006: 160). Other historians, among them Steinberg and Himmerich, simply presume that Kraepelin developed the concept (Steinberg and Himmerich, 2011). The appeal of limiting discussions of model psychosis to the hallucinogen model is clear. One can proceed with the surefooted knowledge that their study treads the very context in which the specific language of 'model psychosis' emerged. There is also the possibility of a simple incongruence between contemporary lay, and even professional, perceptions of psychosis and the historical concept over the course of its development. In societies running on caffeine and increasingly amenable to marijuana legalization, the suggestion that tea or even hashish produces anything likenable to psychosis inspires incredulity. Hallucinogens make for a snugger fit. There is little difficulty in describing their potent, perception-bending effects as 'pathological'. But what of Kraepelin, Moreau, or those who might have come before them? It would seem fidelity to the hallucinogen model fails to bring us much closer to unearthing the conditions underlying the appearance of model psychosis in the first place, to understanding why model psychosis is thinkable at all.

Returning to our initial definition, overemphasizing the hallucinogen model loses sight of the broader historical sense of 'model psychosis'. Moreau de Tours's hashish is hardly a hallucinogen in the sense that mescaline might be considered, and Kraepelin's extensive research on the psychometrics of intoxication mustered an array of intoxicants—none of them 'hallucinogens' (Kraepelin, 1892). Could these intrepid experimentalists have set the path for later, hallucinogenically minded researchers to realize model psychosis, in full? In seeking to understand how model psychosis became thinkable at all, this investigation begins by tracing the early history of the relationship between intoxication and madness, followed by an effort to situate the role of intoxicants in the 19th century emergence of 'psychosis' and the development of experimental psychological models. It is only then that we might start at taking a look at the cases of Moreau and Kraepelin as would-be inventors of model psychosis. Yet it is only in finding the case for either figure wanting that this article seeks to unravel the constitutive elements of the 'model psychosis' concept, bringing to light the foundations of that which makes model psychosis possible: the comparison between intoxication and madness; the category of psychosis; and the development of experimental models of psychological states.

In doing so, it becomes clear that model psychosis is not what it first seemed, that the 'innovation' behind model psychosis is not the comparison between intoxicated states and mental disturbances at all. Model psychosis is—instead—realized as one of the central organizing concepts of psychiatric science, fording the rift between antiquated madnesses and contemporary neuropsychiatric illnesses. It is a through line that enjoins and orients many of the disparate elements that make up modern psychiatry. Without the long-established connection between intoxication and insanity, there is no way of knowing whether we would have the concept of psychosis, and even familiar means of experimentally modelling psychopathological states. Let us begin to uncover this narrative by considering the two chief examples of the concept of intoxication as experimental mental illness—*ante litteram.*

Madness and intoxication: A familiar story

Even in the most cursory of reviews, it becomes apparent that neither Moreau nor Kraepelin can be credited with the realization of intoxication as a temporary form of insanity. The association between intoxication and madness has an ancient pedigree. Euphoria, intoxication, and madness possessed a porous association in the Greco-Roman world—enjoined in symbolic and praxic horizontality via the bacchanal worship of Dionysus (Sevilla-Sadeh, 2021). In the *Phaedrus,* Plato's Socrates presents intoxication as one of the four types of divine madness, taking the form the Dionysian mystical rites (Plato, 2005). Nor was this impression limited to religious, cultural, or philosophical life, further entering into Greco-Roman medical thought through the works of Hippocrates-Galen. Based on his ascription of *De diaeta in morbis acutis* to Hippocrates, Galen asserted that wine affected the mind as its hot-dry nature spread throughout the body, relying on Hippocrates' purported reputation as the ancient world's foremost oenologist (Jouanna and Allies, 2012; Kokoszko and Jagusiak, 2020).[1] Galen similarly framed mental disturbances in humorological terms, understanding affectations of the mind to be the effects of humoral imbalances in the brain (Dols and

Immisch, 1992). In medieval European medicine, Galen's continued authority ensured that humoral imbalance and intemperate drinking, rather than sin or possession, were perceived as being among the foremost causes of disturbances of the mind (Kroll and Bachrach, 1984). Wine, along with other intoxicants, were thus both analogously and aetiologically interconnected with illnesses stemming from a humorological disequilibrium in the head.

Moving into the Early Modern Period, the connection between intoxication and insanity began to assume a variety of different forms. In 1531, Heinrich Stromer defined frequent drunkenness as sickness affecting the brain and veins, resulting from an alternation in the established pressure of brain tissue, a cause of mental disturbances (Spode, 1993). By the 17th and 18th centuries, a 'close connection between insanity and intoxication' was broadly established, 'with the latter being regarded as either a cause or a species of the former' (Loughnan, 2012: 177). It was in this period that English criminal courts began to see intoxication presented as an 'informal plea for exculpation', 'as it was held to render the defendant 'disturbed in her mind', or 'out of his mind', or to ensure that he 'did not know what he did'' (ibid.). Intoxication, already long encountered as a madness, had been wreathed in the politico-religious legitimacy of the law, a product of the increasingly technical methods of Early Modern administration.

The language of madness further featured in the theories of habitual drunkenness that arose toward the end of the 18th and into the 19th century. As part of what is arguably the earliest example of a disease theory of alcoholism, Benjamin Rush described drunkenness as a kind of transient 'madness' (Rush, 1791: 4; 1805: 6). Thomas Trotter and Brühl-Cramer, other early advocates for the disease theory of alcoholism, independently defined drunkenness as 'a temporary madness' (Trotter, 1804: 129; Brühl-Cramer, 1819; Perkins-McVey, 2023b).[2] Trotter makes this comparison explicit, finding in cases of yellow fever and drunkenness demonstrations of the singular nature of physical and mental illnesses (Trotter, 1804: 42–3). Each shared in a unitary Brunonian—or Brunonian adjacent—definition of illness as a discrepancy in the quantum of excitability, understanding alcohol, opium, and other intoxicants to be powerful stimulants capable of acutely stimulating the vital force (Perkins-McVey, 2023b).

Though far from exhaustive, this cursory review is sufficient to support the argument that intoxication and madness have a long-standing relationship, one that reached far beyond the 19th century. Even as the definitions and conceptual nature of terms such as 'drunkenness', 'intoxication', or 'madness' underwent constant development over the course of many centuries, intoxication and madness enjoyed a more or less constant closeness, one seemingly rooted in common elements dispersed across the symbolic-cultural imaginaries of various European societies.[3] In this sense, Moreau's and Kraepelin's suggestions that intoxicated states might be likenable to psychopathological states were not particularly radical. Each of these claims, at a rudimentary level, relied on an association between intoxication and madness that had undergone a process of continuous reconceptualization and symbolic normalization since, at least, antiquity. As we will soon see, what would appear to distinguish their respective arguments is an effort to engender a shift from the notion that intoxication was *like* madness to the idea that intoxication *was* madness. But even this arguably quite defensible position is complicated by the examples just provided from intoxication's use as an informal plea

in English criminal court to the early disease theories of alcoholism. In the former case, intoxication was a kind of madness, at least in a functional sense, which was sufficient to the English court. In the latter, the madness of drunkenness assumes the status of factical illness by precisely the same aetiological mechanisms that produced mental illness.

Before there was such a concept as a 'model', intoxication was a way of knowing insanity, both as a reflection and as a direct expression, or type, of madness. Before the *disease* of madness even existed, intoxication served as a reflection, a hermeneutic, and a vehicle for madness itself. What appears to be the case is that intoxication is, in this sense, what might be provisionally designated as an originary expression, or originary type, of madness.[4] This is not to ignore the situated nature of concepts like madness, intoxication, and embodiment. Concepts are meaningful, as Ian Hacking puts it, as 'words in their sites;' knowledges are situated (Hacking, 2002: 68).[5] This is what makes the persistent conceptual association between intoxication and madness all the more significant. Despite constant and profound adjustment to the specific constellations of meaning independently surrounding madness and intoxication, the two groups of concepts scarcely strayed from each other's orbits. This is perhaps precisely because both concepts designate something linguistically intractable: intoxication and madness are both tacit, experiential knowledges. It is imaginably their shared residence in spaces of embodied awareness that ultimately bind the concept of intoxication and madness together.

What does this mean for the legacy of our founding figures, Moreau and Kraepelin? Perhaps they drew on a pre-established association but singularly led the effort to establish this connection in a psychiatric, medical context? Now that our discussion has reached the long 19th century, we can begin to take a look at the foundations of the modern conception of model psychosis. Far from exceptional in their medical interest in the affiliation between intoxicants and mental pathology, Moreau and Kraepelin participated in a broader milieu of figures similarly compelled by the conceptual relationship. Here, it becomes clear that this long-standing association between intoxication and insanity directly participated in the constitution of those concepts situated within the efforts to define and categorize mental illness that spanned the long 19th century. To this end, let us finally look to the 19th-century association between intoxicants and madness, that we might get a sense of how they participated in the formation of emerging concepts in the study of mental medicine.

Madness and civilization: Intoxication and insanity in the 19th century

The very same year that Moreau published *Hashisch*, the Austrian physician Ernst von Feuchtersleben published his highly influential *Lehrbuch der ärtzlichen Seelenkunde* (Feuchtersleben, 1845).[6] Remarkably formative for modern psychiatry, the *Lehrbuch* popularized both the terms 'psychosis' (Psychose) and 'psychopathy' (Ban, 2006; Feuchtersleben, 1845; Mason, 2006).[7] Feuchtersleben came up in the tradition of the *Naturphilosophen*, which took from Schelling the principle that *Seele* was incorruptible by illness (Beer, 1995; Schelling, 1858[1799]: 3).[8] After the fashion of *Naturphilosophie*,

Feuchtersleben saw mental phenomena as emergent of the dialectic between mind and body (Beer, 1995; Feuchtersleben, 1845). For Feuchtersleben, mental illnesses were truly illnesses of the personality (*Persönlichkeitskrankheiten*), as it was the personality that represented the holistic, experiential relationality between self and world, between mind and body (Feuchtersleben, 1845).[9] Feuchtersleben, thus, elected to use Canstatt's language of 'psychosis', now coupled with 'psychopathy', as a means of linguistically overcoming the conflict between somaticists and alienists (Beer, 1995; Feuchtersleben, 1845). The former group, represented chiefly by Wilhelm Griesinger, preferred the language of *Geisteskrankenheit*, while others made use of *Seelenstörungen*, which greatly emphasized the mind (Beer, 1995).

This association with *Naturphilosophie* is relevant in light of the close relationship between German Romantic thought and intoxicating substances. In his *Naturphilosophie,* Schelling educed that the perceived effects of opium were uniquely demonstrative of the true nature of the dialectical principle of excitability behind all life (Schelling, 1858[1799]: 3). Intoxication was the perceived effect of an acute stimulation of the vitalistic dialectic between body and world (ibid.).[10] The phenomenon of intoxication provided direct insight into the fundamental nature of all living things. Opium's effects were even selected to be Schelling's prime example of bodily causation, the soporific effects of opium being merely the mirror image of the vital, as well as galvanic, excitation elicited by opium's stimulating properties (ibid.). Thus, for Feuchtersleben, as for other *Naturphilosophen,* intoxicants, and intoxication, figured centrally within their broader medical philosophy.

In keeping with Schelling and the *Naturphilosophen,* Feuchtersleben understood the effects of intoxicants in Brunonian terms: opium, hashish, wine, and other intoxicating vital stimulants produced an immediate state of excitation, followed by a corresponding degree of stupefaction, which was the direct result of vital exhaustion (Feuchtersleben, 1845). Intoxicating vital stimulants, Feuchtersleben reasoned, 'operate psychically in that they promote psychical function through a feeling of uninhibited organic vitality' (ibid.: 365).[11] Their curative potential in the treatment of psychoses lay in psychosis's character as an illness emergent of a dysregulation in the proper balance between organic vitality and the mind (Feuchtersleben, 1845). This accorded vital stimulants both a therapeutic and a causative role in psychosis. But, more than that, it would appear that Feuchtersleben recognized intoxication and psychosis as sharing some fundamental similitude.

Here, Feuchtersleben turns to his 'clinical' experience. In the patient, 'a demonic being seems to take possession of man, his state is one of a *waking dream*' in which one finds 'all transitional states as various nuances of a single kind—dreaming, *intoxication*, dizziness, magnetism, even delirium and hyperesthesia—meeting and finding their focal point in an exalted fantasy, as in a concave mirror' (Feuchtersleben, 1845: 258).[12] Just like intoxication, psychotic delirium was the effect of inequality in the dialectic relationality between the mind and body. States of intoxication, much like dreams, are but liminal expressions, even reflections, of madness.

Further still, Feuchtersleben and Moreau shared more than just a year of publication, a vocation, and a professional interest in substances of intoxication. Feuchtersleben also experimented with the use of hashish and other intoxicants, with sufficient ardour to

have published a book of poetic verse on the topic of drinking and drugging in 1843/4 (Feuchtersleben and Schwind, 1978[1875]). Here, as in his subsequent psychiatric textbook, Feuchtersleben compares dream states and intoxicated states:

> Dreams like to seduce,
>
> feigning at the appearance of lawlikeness—
>
> impressing forms
>
> and yet they are mere swindles
>
> As Nirot's fragrant plant
>
> And the brown juice of Arabia
>
> In an enigmatic dance
>
> Create colourful fantasies. (Feuchtersleben and Schwind, 1978: 9)[13]

Elsewhere, Feuchtersleben's 'brown juice of Arabia' pulls the reveler into a waking dream of the 'Sultan's delight', 'twisting the smoke into images which only the artist's sense recognizes' (Feuchtersleben and Schwind, 1978: 9, 10, 11).[14] Hashish, wine, and opium bring into the light of day that which is relegated to the domain of the sleeper, mirroring how von Feuchtersleben would describe psychosis the following year.

It would seem that Feuchtersleben's direct experiences with intoxication, dovetailed with the extant Romantic interest in both intoxicants and experiential science, likely had some influence on approach to the concepts of dreaming and psychosis. Feuchtersleben's poetic report on the effects of intoxicants might even be compared with the self-experimentation of figures like Moreau. The experimentation carried out by Moreau, after all, relied on 'personal experience [as] the criterium of truth' (Moreau, 1845: 4).[15] The 'experiment' was always to understand insanity by way of one's own phenomenal experience of intoxication, an epistemology of experiment that borders on including all manner of a posteriori reflections on occasional inebriation.[16] Regardless of the extent to which Feuchtersleben's experiences are to be regarded as experimental in nature, it is nevertheless apparent that intoxication was central to the emerging concept of psychosis, as presented in Feuchtersleben's *Lehrbuch.*

The suggestion of a porous interface between intoxication and early psychosis would be difficult to maintain had it ended with Feuchtersleben, but the language of psychosis almost immediately caught on with Carl Flemming, who reviewed Feuchtersleben's work in 1846 and published the first textbook on psychosis in 1854 (Beer, 1996; Flemming, 1846, 1859). Flemming has been referred to as part of the somatic school, though Flemming understood psychosis 'as the psychological aspect of a neurosis', which in turn was a disease of the nerves (Beer, 1995: 275).[17] Responding directly to the likes of Feuchtersleben and Moreau, Flemming understood that 'the dream is, like

intoxication, a delirium' (Flemming, 1859: 80).[18] This comparator appears to have been fairly illustrative for Flemming. Citing the observations that the effects of alcohol, and presumably alkaloids, are predicated on the entry of the substance into the blood mass and thereby into the nerve fiber, Flemming openly speculates on the notion that the seat of mental disturbance is in brain tissue (ibid.: 189–90).[19] In this sense, Flemming recognizes 'the temporary mental disturbance of intoxication', going as far as suggesting that intoxication can, 'in some cases increase to mania' and 'can, if repeated often and over a great duration, create a chronic mental disorder' (ibid.: 114).[20] *Delerium tremens* was thus identified as a form of psychosis, and Flemming speculated that the misuse of hashish and opium precipitated mental effects in a similar way (ibid.: 117).

From its inception, the language of psychosis was relative to the concept of intoxication. For Feuchtersleben, intoxication was not merely a reflection of psychotic delirium, but a more fundamental expression of excitability as the dialectical synthesis underlying organic phenomena.[21] Much as intoxication was the effect of a temporary inequilibrium in the vital process, psychosis was the perceptual by-product of an inequilibrium in the dialectic between mind and body—self and world. Even as Flemming sought to extricate the language of psychosis from the trappings of *Naturphilosophie,* psychosis was nevertheless framed using the terminology of dreams, delirium, and intoxication.

The next significant development in the relationship between intoxication and psychosis was Paul Julius Möbius's categorization of psychoses into the parallel categories of endogenous and exogenous psychoses. It is this dichotomy between endogenous and exogenous that Steinberg identifies as Möbius's foremost innovation, and from whence Kraepelin would derive the language of exogenous and endogenous psychoses in describing both intoxication as a form of artificial mental disturbance and the constitutive entities of his nosology (Steinberg, 2005).

By some measures, Möbius's duality of exogeny and endogeny only rightly came to the fore in 1892, though Möbius first applied the division in 1886 (Beer, 1996; Bürgy, 2008). But this is somewhat misleading. For, as early as 1882, Möbius's *Die Nervosität* explicitly argued that 'acute alcohol intoxication or intoxication is a temporary insanity' (Möbius, 1882: 103).[22,23] This statement is then extended specifically to states of intoxication engendered by the consumption of opium, morphine, or chloral, although Möbius presumably imagines this concept to be universally applicable to all intoxicants (ibid.: 104). Evidently, intoxicants such as morphine, alcohol, hashish, and cocaine, each engender acute, temporary mental pathologies—psychoses. Even tobacco, coffee, and tea —which Möbius considers significantly less problematic substances—'can also bring about a state of nervous overexcitation' (ibid.).[24] The etiology of intoxication as a temporary insanity is, then, at the very least analogously comparably with the etiologies of what Möbius would come to term endogenous psychoses. For Möbius, it thus appears that substances of intoxication and mental illness were understood as essentially equitable, even before he had developed the specific concepts behind exogenous and endogenous psychoses. By 1886–92, with the division between endogenous and exogenous psychoses fully developed, Möbius would explicitly identify intoxication as a type of exogenous psychosis (Steinberg, 2005).

With the association between intoxication and madness backgrounding the development of the concept of psychosis, it should be unsurprising that, from Feuchtersleben

and Flemming to Möbius and Kraepelin, the developing concept of psychosis was shaped by the influence of intoxicants and intoxication. The case could be made that the development of the concept of psychosis was part of a gradual effort to define and classify the symptomology of delirium by way of intoxication as an 'originary type' of madness. Much like Flemming, Möbius kept with Feuchtersleben's initial conceptions of intoxication as essentially comparable with mental illnesses via the conceptual association between dream-likeness and delirium (Möbius, 1882: 104). While some, notably Feuchtersleben, applied the term psychosis more broadly, nearly every iteration shares an association with the symptomology of delirium, culminating in the effective exclusivity of such a relationship by the late 19th century.

With this, we have sufficiently established that the characterization of intoxication as some form of temporary, artificial mental pathology, readily likenable, if not an explicit form of, mental illness has deep roots, reaching well beyond the work of Moreau or Kraepelin. Intoxication may represent an originary type of madness, long-realized as a means of knowing and giving expression to the murky depths of mental disturbance. This is readily seen in the examples pulled from before the turn of the 19th century. And yet, with the 19th-century advent of psychosis, we once again find intoxicants coming to the fore as ready interlocuters. The waking-dreams of intoxicated delirium helped to shape and define the emerging concept of psychosis, from its Romantic nascence in the works of Feuchtersleben to Möbius's influential duality of exogenous and endogenous psychoses. With such reflections in hand, we can now bring them to bear on the classic history of model psychosis, in an examination of model psychosis in the thought of Kraepelin and Moreau de Tours.

Moreau de Tour's hashish madness and Kraepelin's artificial insanity

Concerning the history of model psychosis, the earliest formulation of the concept is often attributed to Moreau de Tours. The mid-20th-century French psychiatrist Henri Ey regarded Moreau to be both the father of modern psychiatry and the indisputable inventor of experimental psychosis (Ey, 1970: 2; Revuelta and Moreno, 2021b). Cause for such lofty accolades are largely afforded on account of Moreau's 1845 text: *Du Hachisch et de l'aliénation mentale*. Moreau's *Hachisch* drew on his exploits over the course of an eleven-month journey through the Near East in 1836, bolstering his testimony with the contents of his own experiences consuming hashish upon returning to France (Moreau, 1845). It was Moreau's 1836 voyage, and its aftermath, which placed the relationship between intoxication and madness at the centre of his efforts to understand the nature of insanity and discover a cure (Moreau, 1841).

Prior to publishing *Hachisch,* the contents of Moreau's experiences in the Middle East was the subject of two papers, titled 'Mémoire sur le traitement des hallucinations par le Datura stramonium' (1841) and 'Recherches sur les aliénés en Orient' (1843).[25] The groundwork for much of what Moreau would later espouse in *Haschish* can be found in his 1841 'Mémoire'. Over the course of his travels, Moreau witnessed the effects to two substances: hashish and datura. Both datura and hashish are cited as causes of

delirium and madness in the course of Moreau's travels, with Moreau having taken hashish and reported euphoria, hallucinations, and finally delirium (Moreau, 1841: 644–5; 1843: 111). In seeing these effects, Moreau took it upon himself to test the effects of datura on his patients in order to throw a 'light on the etiology of nervous-intellectual disorders, and on the nature of the treatment which suits them best' (Moreau, 1841: 645).[26]

The theoretical rationale was simple. In keeping with faculty psychology, phrenology, and the notion of unitary insanity, the origin of hallucinations was presumed to be the nervous system (Moreau, 1841: 643).[27] This is also true of the delirium and eventual paralysis that comes with the progression of insanity, which almost always begin with mania and hallucinations before developing into paranoia and delirium, before terminating in paralysis (Moreau, 1841). It appeared to Moreau that intoxicants seemed to interact with the same faculties as those affected in cases of insanity, going as far as suggesting that in the course of intoxication one experiences a progression from mania, to hallucinations, and finally delirium (ibid.). Moreau had already noted how vivid hallucinations, unfettered throws of passion, and exaltation of the imagination can accompany even a few drops of spirits, or a couple grains of opium (ibid.). It was no wonder, Moreau remarked, that chronic alcohol use was common among those brought to the asylum (ibid.). Hashish was no different. He himself experienced how hashish intoxication progressed from euphoric mania to hallucinations and then paranoid delirium (ibid.; Revuelta and Moreno, 2021b). If intoxicants produced in otherwise healthy individuals what lesions produce in the mentally ill, what effect would intoxicants have on the mentally ill?

It was on these grounds that Moreau began medicating his patients with datura pills in water (Moreau, 1841: 674). Datura was selected because of its unique propensity to rapidly induce hallucinations and delirium (Moreau, 1841). With the exception of those patients already suffering from dementia, Moreau reported that sustained use of datura gradually calmed the patient's delirium, reduced hallucinations, and ultimately brought them to a renewed state of health (ibid.). A patient who had suffered for over a year was gradually cured, weaned off the datura, and released back to his family (ibid.: 675). Moreau theorized this was only possible through a direct cerebral modification (ibid.: 647). If all mental illnesses were the effect of lesions, it followed that intoxicants acted on the same areas of the nervous system (Moreau, 1841). Intoxicants, in this sense, were theorized to 'cure' insanity through the targeted modification of those areas affected by lesions (ibid.). Datura's therapeutic effects were, thus, 'the result of some kind of substitution of one disease for another disease', after which the patient could be weaned back to a baseline state (ibid.: 642).[28]

With Moreau's 'Mémoire' in hand, one can begin to make sense of the concepts underlying the considerably more substantial *Hashisch,* which began with a programmatic declaration:

> I had seen in hashish, or rather in its action on the moral faculties, a powerful, unique means of exploration in matters of mental pathogenesis; I had convinced myself that through it one should be able to be initiated into the mysteries of alienation, to go back to the hidden source of these disorders, so numerous, so varied, so strange that we are in the habit of designating under the collective name of madness. (Moreau, 1845: 29–30)[29]

Hashish in particular, but intoxication in general, would be the tool that would finally uproot the primordial nature of madness, bringing into the light of day the observation that the waking dream of insanity is 'a true *dream state*, albeit a dream *without sleep*' (Moreau, 1845: 37).[30] Starting with the effects of lower doses and gradually increasing, Moreau tracks how hashish produces effects of euphoria, excitation, dissociation of ideas, perceptions changes in time and place, as well as paranoid delirium (Moreau, 1845). As with datura in 1841, Moreau finds that 'there is no elementary or constitutive fact of madness which is not found in the intellectual modifications developed by hashish' (ibid.: 35).[31] Just as delirium is identical to the dream state, hashish allows one to 'fall asleep without ceasing to be awake' (Moreau, 1841: 645; 1845: 37).[32] Moreau makes similar observations about the effects of nitrous oxide, opium, alcohol, and other narcotics (Moreau, 1845).

Concerning the underlying cause of mental illness, Moreau maintains that all mental disturbances, however varied and specific, develop out of an originary intellectual modification, or excitation (Moreau, 1841; 1845: 36, 98). Excitations refer to modifications or molecular disintegrations of the intellect, which are the embryonic state of all mental disturbance (Moreau, 1845: 98).[33] Heredity factors, including Lamarckian transmission of acquired characteristics, predispose the individual to cerebral alteration (Moreau, 1845). In this sense, diagnoses do little to describe the *actual* causes of disturbances, although it remains important to group symptomatologies in localizing the initial cause (ibid.: 44). This is, further, Moreau's case for the universal applicability of his case study. All of these observations, Moreau contends, can be deduced by reflecting on the effects of hashish on the psyche (Moreau, 1845). For, were mental disturbances not the product of some cerebral alteration, how else could hashish so perfectly mirror the state of madness?

The approach to the concept of artificial insanity pioneered by Kraepelin differed from that of Moreau's in just about every way. August 1, 1881, Kraepelin wrote to Wilhelm Wundt with an informal habilitation proposal: he hoped to use Wundt's methodology for the study of reaction time to observe the effects of various '*Nervina'* on consciousness.[34] The principle underlying this research proposal was that, in experimenting with the psychometrics of intoxication, the rudimentary psychological nature of mental disturbance could be brought to light.[35] Wundt enthusiastically accepted Kraepelin's proposal and, before the year was out, Kraepelin was running experiments on the influence of alcohol, amyl nitrate, chloroform, morphine, chloral, and ether on various kinds of reaction time (Kraepelin, 1881/2b, 1883a, 1883b). Shortly after, with the 1881/2 publication 'Ueber psychische Zeitmessung', Kraepelin first presented his own 'model psychosis', advancing the argument that intoxication engendered transient pathologies in one's psychological processes, which mirrored the effects of naturally occurring mental disturbances (Kraepelin, 1881/2b; Müller, Fletcher, and Steinberg, 2006: 135; Steinberg and Müller, 2005).[36] Kraepelin's pharmapsychological research program would expand in scope and sophistication over the subsequent decade, culminating in the development of the Dorpat Psychological Society, the supervision of doctoral theses on the psychometrics of intoxication, and the publication of history's first book on the experimental psychology of intoxication in 1892 (Kraepelin, 1892, 1983; Müller, Fletcher, and Steinberg, 2006).

With its nascence in the early years of Wundt's psychological laboratory, Kraepelin's experimental approach was structured in accordance with the methodological strictures of experimental physiology. Kraepelin's own research program was additionally a response to the shortcomings of prior efforts to analyse the psychometrics of intoxication, those of Exner in 1873 and Dietl and Vintschgau in 1877 (Dietl and Vintschgau, 1877; Exner, 1873; Kraepelin, 1881/2a, 1881/2b).[37] This meant that, unlike Moreau, Kraepelin's research program was rigorously controlled and narrowly focused on measuring simple mental processes, rather than macro changes in apparent affect (Kraepelin, 1881/2a). In Kraepelin's context, the effects of intoxicants were only meaningful insofar as they could be translated into quantifiable measurements. This is why Kraepelin's experiments were largely focused on the influence of different intoxicants on simple, choice, and discrimination reactions, although he did produce further measurements on the effects of intoxicants on reading speed, arithmetic speed, and other higher order mental operations (Kraepelin, 1881/2a, 1881/2b, 1883a, 1883b, 1892). Even still, these relatively simple higher order mental operations were not regarded as meaningful in themselves (Kraepelin, 1892). It was only in conjunction with the psychometrics of simple mental processes that more complicated tasks could be understood.

Kraepelin's concept of exogenous psychosis outwardly appears to be the direct function of the operative definition of mental illness. A follower of Wundtian psychology, Kraepelin understood the unitary experience of consciousness to be composed of discrete subprocesses: perception, apperception, and will (Kraepelin, 1881/2a, 1881/2b; Wundt, 1874). If these subprocesses make up the totality of experienced consciousness, the visible symptoms of mental illness entailed distortions in the conscious process. Disruption in the regular course of conscious life occurred somewhere in the processes of perception, apperception, or will. This further suggested that, where it possible, the outward presentation of mental disorder could be reproduced through modification in these processes. Enter substances of intoxication: materials whose defining quality was their apparent effect on consciousness. Taking on the Wundtian definition of consciousness, intoxicants evidently produced their effects by modifying cognitive subprocesses. On a theoretical level, this made substances of intoxication attractive potential interlocuters for any endeavour to excavate the hidden character of mental disturbances. This was another practical element of Kraepelin's focus on simple mental processes. By limiting the bulk of his measurements to the most basic of psychological times, Kraepelin was theoretically able to isolate the effect of specific intoxicants on different aspects of cognition.

The extent to which Kraepelin's research program was successful remains the topic of protracted debate within historical scholarship (Steinberg and Müller, 2005). This is not to say Kraepelin's experiments failed to produce consistent results. Kraepelin was able to demonstrate, for example, that substances such as amyl nitrate consistently lengthened reaction times by affecting apperception, while those such as morphine could shorten reaction time by affecting the strength of perceptions (Kraepelin, 1892). This made intoxicated states experimentally comparable with regular mental disturbances, insofar as differences in symptomology accorded with affectations at different levels of the conscious process. In this sense, Kraepelin's pharmapsychological research program could

even be called programmatically successful, even if Kraepelin struggled to translate his experimental findings into practical application.

Moreau's hashish madness and Kraepelin's exogenous psychosis—this discussion has served to provide a basic outline of the two principle early claimants on the concept of model psychosis. We could mull over the extent to which Moreau's undertaking was not truly experimental, but rather *merely* empirical, even by the standards of Kraepelin's time. And yet, pinning model psychosis to the lapel of either researcher does not tell us very much about where idea of model psychosis comes from, about where it figures in the history of medicine and madness. From the outside, both Moreau's and Kraepelin's approaches to model psychosis develop with some degree of conceptual necessity. How novel, really, were their comparisons between intoxicated states and mental disturbances? It has already become apparent that the innovation behind model psychosis, if there is an innovation to be identified, is not the claim intoxicated states are comparable with any number of mental disturbances. This is as much the case for earlier notions of madness as it was for the 19th-century conception of psychosis. Where they were merely unwitting participants in a greater story about intoxication and mental illness? Where does this leave our history of model psychosis? For the answer to this question, we can find a clue in the phrase 'model psychosis' itself. If we are to cast inquiring pebbles into the murky origins of model psychosis, we must do so in the direction of the history of psychological modelling.

Experimental psychological modelling

The function of the preceding overview has been twofold. (1) It has attempted to tentatively outline the broad sense in which intoxication and madness have long shared a conceptual affinity, likening intoxication to an 'originary type' of madness. (2) It has sought to anchor these ideas relative to emergence of the concept of psychosis, of apparent significance to the concept 'model psychosis', and which was itself profoundly shaped by interaction and interface with substances of intoxication. If intoxication is to be understood as an original type of mental disturbance, the 'missing piece' of the modern history of model psychosis is the history of psychological modelling. If there is any innovation to be found, it lies with the translation of the strong association between madness and intoxication into a reified experimental model, that of model psychosis.

Looking back upon our discussion up to this point, a general history of modelling in association with intoxicants is already beset with difficulty. One approach is to apply a 'big-tent' definition of 'modelling'. This would entail considering historical claims concerning the illustrative, imitative, or exemplative potential of intoxicants with respect to mental disturbance. But we have already demonstrated that a simple equivalence between intoxicated states and mental illness is not merely lacking in novelty but borderline normative, despite continuous changes in the functional definitions of mental disturbances and intoxication. Even caveating this definition with an inclusive concept of 'experimentation', it does little to shorten the list of possible candidates, as reflected in the comparability of the experimental attitude of Moreau and the 'experimentation' of Feuchtersleben. Where, then, does model psychosis begin and mere intoxication as madness end? This question is already greatly clarified through the observation that

'model psychosis', for as long as the term has existed, has been intended in accordance with the ideas of experimental psychological modelling. Understanding the history of model psychosis, thus, entails looking into the superficially separate history of psychological modelling. Here, too, intoxicants proved remarkably influential.

The development of psychological models through experimentation is co-original with, if not historically prior to, the earliest continental ventures in experimental psychology, with intoxicating substances inhabiting a privileged place from the outset. Roughly a year before Wilhelm Wundt published *Grundzüge der physiologischen Psychologie* and more than half a decade before the founding of Wundt's psychological laboratory, Sigmund Exner published his 1873 'Experimentelle Untersuchung der einfachsten psychischen Processe, Erster Abhandlung' (Exner, 1873: 609). An experimental physiologist concerned with the physiology of sensation, Exner was interested in Friedrich Bessel's discovery that isolated, concurrent astronomical observations constantly yielded slight, mathematically meaningful variations in chronological measurements, a phenomenon that Bessel referred to as the 'persönliche Gleichung' (personal equation; Exner, 1873: 606' Hoffmann, 2006: 172). Drawing on Helmholtz and Baxt's research on the relativity of nerve propagation velocity and the methodology of Donders, Exner sought to reduce the personal equation to its most basic physiological processes by measuring the shortest time required to acknowledge and respond to an external stimulus, coining the term 'reaction time' (*Reactionzeit*; Exner, 1873). Reaction time never strove to approximate the total sum of cohesive processes underlying an individual 'reaction'. Rather, it sought to reduce reactions to a theoretically isolatable loop of physiological processes, which could, then, stand in for, or represent, the cohesive totality of embodied perception (ibid.). Exner took the real-world phenomenon of Bessel's personal equation and redeveloped this concept as a physiological model, packaging a series of specific processes into a modality that could then be translated into new circumstances, between peoples, cultures, and contexts.

Exner's approach to reaction time as a model becomes all the more apparent when the experimental significance of intoxicants is also considered. Although Exner was primarily concerned with studying the simplest forms of reaction time, there were secondary observations that he hoped to understand. Exner had quickly come to realize that exhaustion appeared to lengthened reaction times, but wanted for a consistent, experimentally controllable method of inducing an exhausted state (Exner, 1873). To this end, Exner's 1873 study introduced tea, wine, and morphine into the experimental process, not as direct objects of study, but rather as means of expelling and engendering physical states (ibid.).[38] The state of intoxication itself was outside of the conceptual scope of the experiment. Intoxicants were taken up as tools, extensions, and *models* of experimental conditions beyond their immediate effects. From the very moment that intoxicants were interpolated within the heuristic framework of experimental physiology they assumed the conceptual function of a model: morphine and wine as model exhaustion, and tea as model excitation.

Morphine, alcohol, and tea did not produce Exner's desired effects and ultimately elected to exclude those measurements from his final results (Exner, 1873). But it would be remiss to call this a methodological failure, as Exner's introduction of intoxicants as models of different physiological states was soon taken up by other

physiologists, as well as those in other contexts. A few years later, in 1877, Michael Dietl and Maximilian von Vintschgau's 'Das Verhalten der physiologischen Reactionszeit unter dem Einfluss von Morphium, Caffée und Wein' attempted to recreate Exner's experiments involving intoxicants in order to resolve his uncertain findings (Dietl and Vintschgau, 1877). Primarily hoping to attain some clarifying result, Dietl and Vintschgau considerably simplified on Exner's experiment design, in addition to substituting tea for coffee and wine for champagne (ibid.: 318–19).[39] They were able to determine 'that the reaction time can be remarkably shortened by coffee' and that subcutaneous morphine 'very quickly lengthens reaction time' (ibid.: 368, 357).[40,41] Here, too, Dietl and Vintschgau attended to the notion developed by Exner that these different intoxicants could be used to experimentally modify physiological states, such as exhaustion.

Reaction time had, at once, come to denote some kind of negative phenomenon, an experimental methodology, a research program, and a theoretical model, the distillate of interactions between theories, concepts, epistemic comportments, apparatuses, and experimental methodologies.[42] Reaction time does not outwardly appear to be a model in same the way that a biologist might construct a model of an amino acid out of wire and colourful foam balls.[43] Yet the tangible materiality of this example is deceptive. Hacking is right in asserting that models are not 'literal pictures of how things really are' (Hacking, 1983: 22). The colourful Styrofoam balls are not transubstantiated into oversized carbon, hydrogen, or nitrogen atoms. Models do, however, reinforce and shape what we perceive, what we interact with, and how we understand 'how things really are'.

Writing on animal behavioural genetic models in addiction research, Nicole Nelson employs the metaphor of 'epistemic scaffolds' to describe how psychological models come to be supported and ultimately reinforce the rudimentary assumptions brought to other aspects of the human sciences (Nelson, 2018). The 'epistemic scaffold', as described by Nelson, is 'a series of increasingly risky claims about a model's knowledge production capacities' (ibid.: 86). Unlike specific observations of experimental phenomena, 'behavioral models are shared entities used by many practitioners in the scientific community' (ibid.: 87). In this sense, the epistemic scaffolding that supports a given model represents a collective praxic and theoretical work space, where different groups of researchers converge and carry out the work of scientific research. Much like Imre Lakatos's concept of negative heuristics, the epistemic scaffolds themselves become objects of negotiation and renegotiation in knowledge production, with some researchers moving to add or subtract from the existing scaffolding without explicitly attacking the central claims represented in the model (Lakatos, 1970; Nelson, 2018).

A fundamental difference between models and observations or concepts is their ready translatability, or portability. In yet another metaphor, we might further turn to Bruno Latour's example of map-making as knowledge production (Latour, 1988). Taking from map-making in the age of European exploration, Latour describes the map as the product of cycles of knowledge accumulation, as peripheral observations and knowledges are mustered at a central site where the map, as a form of knowledge claim, is actualized (ibid.). But the map is 'not a representation of the world but an inscription that does (or sometimes does not) work in the world' (November, Camacho-Hübner, and Latour,

2010). Far from simply describing an environment, maps set claims upon the world, engendering diversities of new, and unanticipated, forms of activity (Latour, 1988). Maps can, both literally and figuratively, move between spaces and inform different kinds of activity. Like maps, models are not mere representations but inscriptions. They make the world, carrying over into unforeseen spaces and forms of knowledge production.

We might take from these ideas in coming to understand the development of model psychosis, and so distinguish model psychosis from earlier conceptions of intoxication as madness. Bessel's personal equation had referred to consistent personal latencies in time measurements. The simultaneous development of the conceptual model of reaction time, and the experimental method of encountering it, transformed the negative phenomena of Bessel's personal equation into a discrete, positive phenomena. The interplay between physiological speculation and experiment engendered the formation of a model that constituted the basis for a new kind of physiological event: reaction time. Reaction time may not be an entity in the sense that an amino is often taken to be, and yet, by way of modelling, it attains a certain entity-likeness.

When the methods of experimental physiology were brought to bear on psychology by the likes of Wundt, modelling with intoxicants came with them, taking on a new form. For Wundt, the physiological sub-processes that comprised the reaction time model were replaced by a combination of physiological and psychological sub-processes: the physiological events of initial sensation and eventual firing of the muscles were bookends on either side of a psychological process entailing perception, apperception, and will (Wundt, 1874). Using Donders's subtraction method, Wundt was able to strip away the layers of the psychic process by modifying the nature of the reaction itself, primarily through the development of discrimination and choice reactions (ibid.). Truly having to choose whether a stimulus was correct or discriminate between stimuli, Wundt suggested, produced variations in reaction time relative to baseline levels because of latencies engendered by the lengthening of discernible components of the conscious process (ibid.).

What Wundt had adopted from Donders and Exner was not merely reaction time as a methodology, but reaction time as a conceptual model, one in which Wundt was able to represent the structure of consciousness as such. Attacking the 'epistemic scaffolding' rather than the model itself, Wundt problematized the structure surrounding the model of reaction time without attacking the model itself, likewise translating the reaction time model into a different setting where it set about doing different kinds of work. Even the spatially and temporally circumscribed experimental apparatus, the empirical crucible to which reaction time owed its entire existence, had effectively been divorced from conceptual model of reaction time, the likes of which had already been freely translated across apparatuses, bodies, and minds.

Identifying with Wundt's framework, it was in response to Dietl and Vintschgau's 1877 paper that Kraepelin proposed a habilitation project to Wundt as early as 1881, which would centre on the psychometrics of intoxication.[44] Kraepelin was explicit about his intentions to import the methodological pairing of intoxication with reaction time first introduced by Exner, and given additional explication by Dietl-Vintschgau, and further develop it within a psychological, rather than physiological, framework (Kraepelin, 1881/2a, 1881/2b, 1892).[45] Anointing his research program with the name

'pharmapsychology', Kraepelin proceeded on the premise that intoxicants produced their perceivable effects through modification of one or more components of Wundt's conscious process, much as Exner, Dietl, and Vintschgau operated on the presumption that intoxicants would interfere with some component of the physiological process (Kraepelin, 1881/2a, 1881/2b, 1892).[46] A psychiatrist by vocation, Kraepelin immediately came to approach different states of intoxication as temporary modifications in the constitutive elements of Wundt's conscious process, thus creating the possibility of approximating 'natural' mental disturbances (Kraepelin, 1881/2a, 1881/2b).[47] Reaction time models had gone from a physiological event, to a means of modelling consciousness, and then, by extension, to a method of modelling psychopathological states.

The genesis of model psychosis as an experimental model is, in this sense, attached to the emergence of reaction time as the premier experimental model in psychology. Sure enough, the conceptual basis of the claim that madness could be likened to intoxication had roots stretching deep into antiquity, but the advent of model psychosis proper appears to be grounded in the late 19th century. From here, we have a relatively clear view of how the modern conception of model psychosis came to be. The modern sense of model psychosis is generally, but not always, divorced from the classic study of reaction time, but, as scientific research changed and developed over the 20th century, one can see how model psychosis came to be supported by new 'scaffolding' as it was carried forward into ever diversifying contexts. Nor has the legacy of model psychosis limited itself to acute mental states in human subjects. To once more turn to Nelson's study, the 'pharmacological argument', as she terms it, is one of the most prominent pieces of scaffolding supporting the elevated plus maze as a model of rat—and, by extension, human—anxiety (Nelson, 2018: 88). Much as one might validate that a rat experienced anxiety because of their response to benzodiazepines, the treatment of anxiety and depression-like symptoms in rats undergoing cocaine withdrawal with an anti-depressant might be used to establish the efficacy of a particular anti-depressant in treating anxiety and depression (Barbosa Méndez and Salazar-Juárez, 2019). Model psychosis, broadly defined, has arisen as one of the foremost supports of animal behaviour models in modern experimental science.

This has not been an effort to anoint Kraepelin as the originary developer of the model psychosis concept. Kraepelin as a particular individual no doubt greatly impacted the early formation of the concept, but there is a sense in which he merely happened to be a willing interlocuter, conveniently located nearby when the constitutive elements of the model psychosis concept finally began to coalesce. Intoxicants and their role in the early history of experimental psychological modelling are the main focus here. On this point, it has at the very least become clear that intoxicants, and the states they engender, were central participants in the early development of experimental psychological modelling, in particular reaction time.

What then is be surmised about the narrative we have just considered? From the outset, it has been apparent that the historic novelty of model psychosis never lay in the comparison between intoxicated states and mental disturbances. We could glibly retort that this framing of model psychosis is contingent upon some form of experimentalism, that experimental intervention is a load bearing wall in the conceptual architecture of model psychosis. But this quickly leads into a game of assessing the earliest sufficiently

contemporary understanding of experimentation, since definitions of what is and what is not properly experimental changed considerably over the course of the 19th century.

What this reconstruction has attempted to do is situate the concept of model psychosis within the development of those concepts that appear to define it: intoxication, psychosis, and experimentation. Far from a conceptual innovation within the broader development of psychopathology, the etiology of mental illness, or psychological modelling, nascent expressions of the concept of model psychosis were party to the emergence of each of these elements. Intoxication formed an 'originary type' of madness, a conceptual basis for the definition of psychosis, participated in the formation of reaction time as a psychological model, and became the earliest experimental psychological model of mental disturbance. At this point, it becomes tenable to suggest that model psychosis represents one of the earliest forms of experimental modelling in psychiatry, if not the earliest of its kind. Even still, this is not owing to the transformative innovation of model psychosis, but, as has been shown, because of intoxicants have shaped and defined some of psychiatry's most influential developments over the last 200 years, converging on the advent of psychological modelling at the close of the 19th century.

Conclusion

It has been suggested that model psychosis is entering a new phase of prominence, that the psychedelic 'renaissance', already underway, is once more bringing model psychosis into the foreground of novel experimental research (Corlett *et al.*, 2011). This does not mean model psychosis ever 'left'. To find common uses of the model psychosis concept, one need look no further than prevalent animal models of mental illness. The advent of animal models in the 20th century created a wealth of ethical and experimental latitude for those tinkering with mental pathology, but raised practical questions such as 'How do you give a mouse schizophrenia or depression?' Historically and to this very day, pharmacological intervention has been at the forefront of animal models of mental illness. 'Hyper-locomotion induced by amphetamine' has been treated as a 'predictively valid model of schizophrenia', much as 'psychostimulants, such as cocaine or amphetamine', have been used to produce 'mania-like behaviour' (Kaiser and Feng, 2015; Nestler and Hyman, 2010). Just two of many examples, pharmacologically based animal models of psychiatric illnesses are functional and factical expressions of model psychosis.

As for the history of this idea, it has hopefully become apparent that the concept of 'model psychosis' encompasses, at once, a relic of bygone centuries and something radical and pathbreaking, in many ways a centrally organizing concept in psychiatric science. Having begun with the thesis that the novelty of model psychosis was found in the identification of intoxicated states with mental disturbances, this review set out by considering those have been often by counted as model psychosis's primary developers. It found that Kraepelin and Moreau, though highly influential experimentalists with inscrutable contributions to the history of both general psychiatry and model psychosis, were far from the first to equate intoxicated states with mental disturbances. Intoxication is what might be called an 'originary type' or 'expression' of madness, the tendrils of which spread all the way to the modern concept. But—more than that—the constitutive

elements of the modern understanding of 'model psychosis' appear to have been formed in the womb by intoxicants. Experimental psychological modelling owes a great deal to the intercession of intoxicants for the modelling for pathological states. A presumptive association between intoxication and mental disturbances would appear to be the Argo, upon which so much of psychiatric medicine has set sail.

Outwardly, this article has been an effort to reframe the present academic understanding of model psychosis in the hopes that we might come to understand it not as the invention of a modern world fascinated with psychopharmacy but as a concept with far deeper roots. Yet, at its heart, it has been an enterprise with far bolder aspirations, identifying model psychosis as a constellation of material and conceptual associations at the very heart of the modern medical world. Necessarily limited in scope, it leaves many unanswered questions about the historical comparison between intoxication and madness: Are there other historical instances where the identification of intoxication with madness had a significant historical impact? What of other spheres of cultural influence? Was intoxication viewed in a comparable way in the Indian subcontinent, or China? Are there other circumstances in which concepts adjacent to model psychosis affected experimental models? All these questions, and many more, are left to subsequent investigations. And yet, they reflect upon the unconsidered scope of influence attached the concept of model psychosis.

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Notes

1. This led to the organization of different wines according to the extent of their effects. Galen attributed hotter effects to straw-yellow wines, which thus produced greater effects on the mind (Jouanna and Allies, 2012). Those with a hot nature were told to avoid wine, so as not to get sick.
2. The later examples of Rush, Trotter, and Brühl-Cramer understood both intoxication and insanity in Brunonian terms (Perkins-McVey, 2023b). It is worth noting that Rush, Trotter, and Brühl-Cramer were unfamiliar with each other's work and lived in America, Great Britain, and Russia respectively, but nevertheless developed similar conclusions in similar terms.
3. Consideration of cultures outside of the European sphere are, sadly, outside of the scope of this, already overbroad, discussion.
4. Originary in the sense of historical co-originality, as well as in its tacit expression.
5. This is not to explicitly align with Hacking's definition of concepts here, but rather to broadly share in his approach to situated knowledge. As much as 'sites' always designate a 'larger site

of neighborhood, institution, authority, language', Hacking's analytic-linguistic definition, here, could be seen as de-emphasizing tacit, embodied knowledge such as the state of intoxication itself (Hacking, 2002: 68).

6. Although the prefix 'von' in von Feuchtersleben denoted a hereditary title rather than an honorific, as in, for example, 'Hermann von Helmholtz', I standardize it in-text to 'Feuchtersleben'.
7. Karl Friedrich Canstatt has come to be recognized for coining the term 'psychosis' before Feuchtersleben, introducing the term in his 1841 *Handbuch der Medizinischen Klinik* (Bürgy, 2008). Feuchtersleben likely received credit for coining the term because his work was considerably more influential, and thus he nevertheless is credited with popularizing the term. For Canstatt, 'psychosis' (*Psychose*) referred to irritability of the nervous system that engendered delirium (Bürgy, 2012; Canstatt, 1843: 1). Canstatt recognized toxins, such as intoxicants, as potential causes of delirious psychosis (Canstatt, 1843: 1, 368). Ban makes the case that Feuchtersleben was the first to distinguish psychiatry from neurology as the study of mental pathology, rather than the nervous system (Ban, 2006).
8. For Schelling, *Seele* was but one part of the dialectic that underlies living beings. It is worth further considering that Schelling was a Brunonian, and thus all illness was in effect the product of a disequilibrium in excitability, which was, for Schelling, nothing less than a description of the dialectic of the organic itself (Perkins-McVey, 2023a; Schelling, 1858[1799]: 3).
9. Feuchtersleben nevertheless maintained the independence of some neuroses.
10. The conceptual basis for this idea was the Brunonian system of medicine, as formulated by Andreas Röschlaub (Perkins-McVey, 2023a).
11. Stimulants 'wirken psychisch, indem sie die psychische Funktion durch ein Gefühl ungehemmter orga nischer Lebensthätigkeit fördern'. All translations belong to the author.
12. 'Ein dämonisches Wesen (§.84) scheint sich des Menschen zu bemächtigen, sein Zustand ist ein waches Träumen; und wir sehen hier, als vielfache Nüancen Einer Art, die angeführten Erscheinungen aller Übergangszustände, des Träumens (§.58), Rausches (§.59), Schwindels (§.60), Magnetismus (§. 84), ja des Deliriums (§.88) und der Hyperästhesie (93 u . w .) sich begegnen, und in einer exaltirten Fantasie, wie in einem Hohlspiegel, ihren Erennpunkt finden.' The formatting of the quote has been simplified for readability. Emphasis added.
13. 'Träume möchten gern verführen, / heucheln des Gesetzes Schein, – / Formen wollen imponiren, / Und sind doch nur Gankelei'n / Wie sie Nirot's duft'ige Pflanze / Und Arabiens brauner Saft / Bei dem räthselhaften Tanze / Bunter Phantasien schafft.' Citations refer not to the page number, but the number of the poem. The translation has been prepared literally to aid in its comprehension, rather than for poetic ends.
14. 'Arabiens brauner Saft'. 'Sultans Hochgenuss'. 'Schlingt der Rauch sich zu Gebilden, / Die nur Künstlers Sinn erkennt.'
15. 'L'expérience personnelle est ici le criterium de la vérité.'
16. The border is, at the very least, highly ambiguous.
17. This was consistent with Cullen's definition of neurosis.
18. Flemming was aware of delirium tremens and understood chronic alcohol use to be the cause of some psychoses, and a trigger of others (Flemming, 1859).
19. Flemming cites Rudolf Virchow as proposing that narcotics produce an ischemic condition in the brain, while further highlighting the ambiguity of this proposal (Flemming, 1859: 189).
20. The 'vorübergehenden Seelenstörung des Rausches' going as far as suggesting that intoxication can, 'in eizelnen Fällen bis zur Manie steigert' and 'kann, wenn sie sich oft und lange Zeit hindurch wiederholt, eine chronische Seelenstörung erzeugen'.

21. *Naturphilosoph par excellence* Lorenz Oken declared that 'excitability is the most general phenomenon of organic matter, and it belongs to plants and animals' ('die Erregbarkeit ist das allgemeinste Phänomen der organischen Masse, und kommt Pflanzen und Tieren zu'), in keeping with Schelling's description in *Erster Entwurf eines Systems der Naturphilosophie* (Oken, 1810: 134; Schelling, 1858: 3).
22. 'Die akute Alkoholvergiftung oder der Rausch ist ein vorübergehendes Irresein.'
23. Concerning endogenous psychosis, Bürgy suggests that, as early as 1875, Möbius had introduced endogenous psychosis as a concept (Bürgy, 2008). That the concept of endogenous psychoses existed in some form quite early on is hardly surprising in light of Möbius's indebtedness to Magnan and French degeneration theory (Beer, 1996; Steinberg, 2005).
24. 'auch durch ihn ein Zustand nervöser Ueberreiztheit herbeigeführt werden kann'.
25. Moreau's background is important here. Moreau studied in Chinon and Tours, the General Hospital in Tours being where Moreau enrolled in surgery with Pierre-Fidèle Bretonnau in 1824 (Revuelta and Moreno, 2021a). Moreau then interned at the Hospital de Charenton in Paris with Jean-Étienne Dominique Esquirol, at the recommendation of Bretonnau, possibly via Trousseau. Resisting nosological distinctions, Moreauian observations employed generic terms, such as 'madness', alongside Esquirol's symptomological 'categories of mania, lypemania, monomania, dementia or idiocy' (Haustgen, 2018; Revuelta and Moreno, 2021a: 164). Moreau would go on additional trips around Europe, some of which greatly affected his outlook on treatment methods, though it was his voyage to the Near East that undoubtedly had an indelible effect on his career. In his doctoral thesis, Moreau argued that 'moral causes, the passions, can determine insanity but only through alterations in the brain, which are the origin of other physical alterations in the mentally ill, and thus the influence of moral treatment on the physical is through the brain' (Revuelta and Moreno, 2021a: 164). Each case of insanity could therefore be understood as having predisposing components, causative elements, entailing courses of development, and determinable cures (Haustgen, 2018; Moreau, 1830; Revuelta and Moreno, 2021a). A patient might develop a case of insanity on account of some physical alteration of the brain, perhaps the result of an underlying pathogenic or hereditary condition (Haustgen, 2018; Moreau, 1830). Over the course of the decade that followed, Moreau went on a series of trips along with patients throughout Europe and Middle East, at the insistence of Esquirol, which served as extended case studies in addition to being therapeutic for the patients (Mahone and Vaughan, 2007). This was the basis of Moreau's trip to the Near East and the theoretical basis for his use of hashish.
26. 'Ce résultat, comme on le verra plus tard, a des rapports étroits avec l'objet principal de ce mémoire, et me semble propre à jeter une vive lamière sur l'étiologie des troubles nerveux-intellectuels, et sur la nature du traitement qui leur convient le mieux.'
27. For this reason, Moreau never proceeds from *folie* to any kind of nosology (Revuelta and Moreno, 2021b).
28. 'le résultat d'une sorte de substitution d'une maladie à une autre maladie.'
29. 'J'avais vu dans le hachisch, ou plutôt dans son action sur les facultés morales, un moyen puissant, unique, d'exploration en matière de pathogénie mentale; je m'étais persuadé que par elle on devait pouvoir être initié aux mystères de l'aliénation, remonter à la source cachée de ces désordres si nombreux, si variés, si étranges qu'on a l'habitude de désigner sous le nom collectif de folie.'
30. 'un véritable *état de rêve*, mais de rêve *sans sommeil*!' Emphasis is found in the original. A nearly identical quote about hashish intoxication can be found in Moreau (1841: 645).

31. ‘Il n’est aucun fait élémentaire ou constitutif de la folie qui ne se rencontre dans les modifications intellectuelles développées par le hachisch.’
32. ‘Je dirais que l’on s’endort sans cesser d’être éveillé.’
33. In the period from 1827 to 1828, Moreau appears to develop an interest in phrenology and faculty psychology, pathologizing patients’ symptoms in light of an affectation of one faculty over another (Haustgen, 2018; Revuelta and Moreno, 2021a). In 1830, Moreau attained his doctorate with a thesis titled ‘De la influence de physique relativement au désordre des facultés intellectuelles et en particulier dans cette varietés du délire désignée par M. Esquirol sous le nom de monomania’. Although the dissertation itself was dedicated to Moreau’s teachers Esquirol and Bretonnau, it distanced his work from that of his former mentors, arguing for the somatic nature of insanity in a clear departure with alienist theory (Haustgen, 2018; Ritti, 1887; Revuelta and Moreno, 2021a; Sémelaigne, 1930). His doctoral thesis represented the beginning of Moreau’s lifelong support for the somatic theory of insanity. It was in keeping with the dual influences of faculty psychology and phrenology that Moreau advanced a somatic theory of insanity (and intoxication), which he understood as a modification in the nervous tissue. In his doctoral thesis, Moreau argued that ‘moral causes, the passions, can determine insanity but only through alterations in the brain, which are the origin of other physical alterations in the mentally ill, and thus the influence of moral treatment on the physical is through the brain’ (Moreau, 1830; Revuelta and Moreno, 2021a: 164).
34. Kraepelin, E. (1881) Letter to Wilhelm Wundt, Munich, 1 August 1881, available at: https://home.uni-leipzig.de/wundtbriefe/viewer.htm.
35. Kraepelin was both a long-time follower of Wundt and a psychiatrist, so his interest from the outsets seems to have been that the study of intoxicants would help develop the limited understanding of mental illness. Wundt, by comparison, had little interest in such practical extensions of his research program.
36. Kraepelin would later make explicit experimental comparisons between ‘drug-induced model psychoses and real neuropsychiatric diseases … in Dresden and Dorpat’ (Müller, Fletcher, and Steinberg, 2006; Steinberg and Angermeyer, 2001).
37. Exner, Dietl, and Vintschgau were not interested in intoxication as a modification of the conscious process or as a psychomimetic (Dietl and Vintschgau, 1877; Exner, 1873). Their work is discussed later in the text.
38. This is noteworthy relative to some of the discussions about the effects of intoxicants found in this paper. It is possible that Exner understood intoxication to be likenable to the effects of mental illness and thought it a relatively unproblematic secondary characteristic of the intoxicants used for the purposes of his experiment. It is far more likely, however, that Exner understood intoxicants as a physiologist in the school of Helmholtz, and thus was solely concerned with physio-chemical relationships that could be explained experimentally. The later accords with Exner’s physiological definition of reaction time.
39. Exner compared simple reaction times across a variety of points on the body (hands, feet, etc.) because his primary objective was the establishment of reaction time itself (Exner, 1873). Dietl and Vintschgau were interested in the effects of intoxicants on physiological reaction time and so used a single reaction vector, in addition to using their own apparatus design (Dietl and Vintschgau, 1877).
40. ‘dass durch den Caffée die Reactionszeit auffallend verkürzt werden kann’.
41. ‘verlängert sehr rasch die Reaktionzeit’.
42. ‘Negative phenomenon’ here refers to the inconsistency or latency in individual measurements witnessed by Bessel, a considerably more amorphous ‘event’ than what reaction time would

later come to reference. It is far from the scope of this article to historicize the 'reaction' itself. What can be said here is that the identification of 'reactions' as a distinct psycho-physical, or merely physiological, phenomena has a distinct history, which—imaginably—has some interaction with the experience of 'reacting' itself.

43. This is an allusion to an example given by Ian Hacking in *Representing and Intervening* (Hacking, 1983: 22).
44. Kraepelin, E. (1881) Letter to Wilhelm Wundt, Munich, 1 August 1881, available at: https://home.uni-leipzig.de/wundtbriefe/viewer.htm.
45. Ibid.
46. Ibid.
47. As has already been discussed, Möbius, a significant influence on Kraepelin, had by this point directly identified intoxicated states with mental illnesses. Kraepelin would soon after adopt Möbius's language of endogenous psychosis to denote 'natural' psychoses and exogenous psychoses to describe intoxicated states. Müller *et al.* (2006) credit Kraepelin's 1881/2 'Ueber psychische Zeitmessung' with the creation of the 'model psychosis' concept (Müller, Fletcher, and Steinberg, 2006: 135; Steinberg and Angermeyer, 2001).

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