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Program Abstracts

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Autism, Care, and the Limits of Destigmatization

Sarah Arnaud and Quinn Gibson

Over the past two decades, the perception of autism has moved away from pathologizing and towards recognition as a social identity. Advocates within the neurodiversity (ND) movement have been promoting the destigmatization of autism and the inclusion of autistic people in discourse and research related to autism. Like any political stance, this perspective has faced opposition. We identify the following strands in this backlash, which we believe are misdirected:

- a) The imputation to the ND movement of the assumption that autism is not harmful.
- b) The claim that the ND movement will obscure the scientific reality of autism.
- c) The claim that following the prescriptions of the ND movement will cause autistic people to lose access to therapeutic or social care and accommodations.
- d) The claim that following the prescriptions of the ND movement will lead to overdiagnoses of autism and a subsequent dilution of its significance.

In this presentation, we aim to analyze these reactions to demonstrate that they rest on misapprehensions about the ND movement. Nevertheless, we do think that there is inherent risk that the ND movement could be 'captured' by the elite or embraced merely for the purpose of virtue signaling. Here too, we argue that the best way to mitigate these risks is to properly understand the ND movement. Our goal is to show that without a reorientation of their focus, the most prominent criticisms of the ND movement overlook the path to emancipation that is beginning to unfold through autism activism.

As not all autistic people participate in shaping the demands of the ND movement, those more adept at socially dominant forms of communication are more likely to gravitate towards the roles of spokespersons and decision-makers, which makes the ND movement especially vulnerable to *elite capture*. In general, elite capture is what happens 'when the advantaged few steer resources and institutions that could serve the many toward their own narrower interests and aims.' (Táiwò 2022, 22). Those with greater facility in social communication who gravitate to leadership positions within the movement can easily, just in virtue of being those who speak on

behalf of those who do not, come to represent the movement as a whole.

This is also manifest when well-meaning people outside of the movement mistake such demands as purely cultural and contribute to their trivialization by repeating them in culturalized, neutered form. Sometimes, this is virtue signaling: once culturalized, those in the broader culture, “allies”, engage with the movement primarily using easily shared and reproduced memes. But while often unintentional, virtue signaling runs the risk of important failures in care- providing; we will show this through the framework of care-ethics as defined by Joan Tronto (1998).

While the ND movement is properly understood as acknowledging both the scientific reality of autism, and the fact that it *can* be harmful, the elite capture of the movement contributes to the appearances of minimizing the negative and obscuring the objective. It is in the interests of both the elites in the movement, and those outside the movement contributing to the neutralization and culturalization of the movement’s demands, that the difficult and scientifically grounded reality of autism not be placed center stage. But this is very different from saying that the ND movement somehow rests, at its core, on the problematic assumptions that autism is not harmful or that it is, in fact, interested in obscuring the scientific reality of autism. Such are misapprehensions. Consequently, the practical worries (c) and (d) should gain no support from (a) and (b).

Dualism, Reductionism, and Medical Explanation: Addressing the “Metaphysical Morass”

G. Scott Waterman

Despite advances of medical science and practice, many patients leave physician appointments without diagnoses of their complaints (O’Leary, 2018). And many who *are* assigned diagnoses are considered to be suffering from conditions such as chronic fatigue syndrome, fibromyalgia, or others for which medical explanation is unavailable. It should not be surprising that understandings of all human maladies have not been achieved, but the problem of “medically unexplained” symptoms and syndromes – in particular, confusion about their relation to psychiatric disorder – extends beyond the scientific/empiric realm and into the conceptual arena. This presentation will introduce philosopher Diane O’Leary’s (2021) formulation of this “metaphysical morass.” While affirming a major premise of her argument, as well as the importance of its motivation, it will raise questions about its conclusion.

Patients whose complaints are “medically unexplained” frequently feel unheard and dismissed by their doctors’ presumptions that their problems are more psychological than biomedical. Even worse, some are eventually diagnosed with serious conditions for which earlier

recognition and treatment might have obviated extended periods of suffering. In the above-referenced article, O’Leary argues compellingly that medicine’s (including psychiatry’s) collective understanding of mind-body dualism is incoherent. She lays the blame for this philosophical muddle at the feet of George Engel, whose biopsychosocial model (BPSM) remains the proclaimed theoretical foundation of psychiatric practice – if not that of medicine as a whole. In his effort to address the shortcomings of the biomedical model and replace it with a holistic approach to clinical problems, Engel introduced several conceptual and definitional errors. The most egregious and consequential of them is the equation of mind-body dualism with biological reductionism. O’Leary is motivated to correct that misconception by a very practical concern: that it poses a threat to public health. Specifically, she argues that the (mis)understanding of holism that sees it as the antithesis of dualism (the latter spuriously conflated with reductionism, holism’s *actual* opposite) entails an injunction against distinguishing “biomedical conditions” from psychiatric ones. And she believes that the resultant “deliberate diagnostic vagueness” at what she considers the body-mind border endangers patients.

In a recent interview with Awais Aftab (2023), O’Leary explains further the distinction she draws – and asserts is required for good clinical care – between “mind problems” and “body problems”: “... [M]ind problems are caused by experience, while body problems are caused by purely biological states.” She explicitly abjures substance dualism and thus stipulates that “all experiences are correlated with brain states,” but she nevertheless perceives a fundamental difference between those conditions for which the relevant brain (body) state is causally tied to experience (“mind problems”) from those which purportedly arise “all on their own” (“body problems”). This formulation, and its stated motivations, raise several questions:

1. Does taking the ontological status of experience/subjectivity seriously entail the existence of two fundamental kinds of problems for physicians to distinguish?
2. Does consideration of risk factors and pathogenesis, along with etiology, change this two-kinds-of-problems taxonomy? How does one categorize conditions for which experiences are predisposing but not causal, or for which experiences affect pathogenesis, illness expression, or outcome?
3. Does adoption of the two-kinds-of-problems formulation improve patient care? Should allocation of clinical care among specialties be based on causes?
4. What are “medical explanations” and what can be inferred when they are unavailable?

The conclusion will be drawn that O’Leary’s plea to reify the distinction between medical and psychiatric illnesses is better expressed as an entreaty to take symptoms (experiences) as seriously as signs (abnormal findings). Conceptual/linguistic recommendations will be made to facilitate clearer communication between physicians and philosophers in the hope of resolving the confusion, and its serious consequences, that she rightly identifies.

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Dysfunction Framings of Mental Illness and the Strawsonian Objective Standpoint

Sera Schwarz and Justin Garson

In the 1990s, some mental health theorists and advocacy groups held out hope that biomedical framings of mental health problems – those that likened mental illnesses to diseases such as diabetes or cancer – would positively impact patients’ lives. They expected them to alleviate stigma, in particular, by showing that the mentally ill person is not responsible for their strange thoughts or extreme moods. Recently published longitudinal data (Pescolido et al 2021; Schomerus et al 2022), however, has shown that some forms of stigma toward serious mental illness have actually increased over the last three decades — for example, the desire for social distance (DSD). In fact, Schomerus and colleagues argue that this latter stigma is actually a *product* of biomedical framings, a conjecture supported by independent psychological data (Haslam and Kvaale 2015). One hypothesis that has been suggested to explain these findings is that, if I view your thoughts and feelings as the byproduct of a brain disorder, I’m more inclined to see you as unpredictable and frightening (see Schomerus et al. 2013; Lebowitz and Applebaum 2019 for discussion). But why this might be so remains an open question.

Here, we draw upon the tools of twentieth-century analytic philosophy to offer a novel explanation of the emerging link between biomedical models and stigma. Our argument is twofold. *First*, we suggest that the core feature of biomedical models responsible for sustaining stigma is their commitment to what we call a “dysfunction-as-brokenness” framing. Such a framing construes thoughts and feelings associated with mental illness not only as having biological causes, but also — crucially — as caused by some *broken* inner thing. We present preliminary empirical data to support this claim.

Second, we suggest that a dysfunction-as-brokenness framing tends to perpetuate stigma by virtue of precipitating the suspension of the Strawsonian “participant standpoint” and the assumption of the “objective standpoint” toward the mentally ill person. To briefly recap: Strawson (1962) famously distinguished between two different stances that one can take up toward others. The *participant* standpoint is our ordinary, default mode of interpersonal engagement, by which we treat others as fellow citizens of a human world—as persons with

whom we can, to various degrees and in various possible ways, come to share our thoughts, feelings, and lives. The *objective* standpoint, on the other hand, is defined by a systematic retreat from the participant attitudes. When we assume such a standpoint, we precisely refuse to engage with and react to others as rational and moral participants in human relationships; we instead regard them as we would regard any other things in the world — as things, objects, or empirical “material” to be dispassionately understood and efficaciously handled.

We argue that there is an intimate conceptual link between seeing a person’s thoughts, feelings, and choices as the byproduct of a dysfunction and assuming the objective standpoint toward them. Our suggestion, in brief, is that when I explain your thoughts, actions, and moods in terms of some broken inner thing (e.g., explain your anger as due to a chemical imbalance or unresolved trauma), I direct attention specifically *toward* the sub-personal components of the mechanisms that underlie psychological life, and *away* from ordinary agent-centered explanations (e.g., of your anger toward me as due to some perceived slight on my part). Moreover, by framing your choices and actions as the byproduct of *broken* “parts,” I foreground explanatory factors that are inexplicable in terms of functions or purposes. In this way, I block, or discourage, any further attempt to explain those choices and actions in ordinary, agent-centered terms. But this is just to suspend the participant standpoint toward you. Stigma (qua DSD) represents the natural sequela of this suspension of the participant standpoint.

We close by considering and responding to two objections to our argument. We consider, first, whether one can somehow occupy both participant and objective standpoints simultaneously, successively, or only in carefully discriminated contexts; and second, whether *non*-dysfunction framings (say, function framings or neurodivergence framings) are equally likely to precipitate the suspension of the participant attitudes.

Our ultimate goal is not to argue that dysfunction framings are never appropriate or warranted. Rather, we want to suggest a way of closing a gap in the existing empirical literature regarding the causes of stigma, and to draw attention to one unacknowledged and systematic possible harm of dysfunction-based framings.

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Scientific Underdetermination and Psychiatric Diagnosis

Matthew Valiquette

Why does psychiatry seem to struggle more than other forms of medicine in classifying pathologies? What makes diagnosis particularly challenging in the case of mental disorders? Consider several excerpts from a study conducted by O'Connor et al., which describe people's experiences of continuously cycling through different diagnoses: one participant recounts how they were diagnosed with "*depression and then generalized anxiety disorder and eating disorder not otherwise specified*"; another participant relays how clinicians "*just kept throwing diagnoses at me [and] that... I was kind of along for the ride*"; yet another describes how clinicians "*diagnosed me as three different types of depression, personality disorders, complex PTSD, lots of different labels that they just kept throwing at me and every time I'd say there was something else wrong [...] they'd just label it with... personality disorder*" (2022, p.6-7). These narratives are echoed by empirical findings in the literature—authors report how nearly one third of patients diagnosed with schizoaffective disorders have their diagnosis changed upon reassessment (Santelmann et al., 2016), and others find that many first-admission patients displaying psychotic symptoms are misdiagnosed even up to two years after initial hospitalization (Bromet et al., 2011). Clearly, psychiatrists struggle to make diagnostic judgments that are reliable on both empirical and temporal grounds. The question is, what is at the root of these often-occurring changes?

One issue identified in the psychiatric literature concerns the problem of the underdetermination of evidence. Briefly, scientific underdetermination refers to the idea that the evidence available to us at any given time cannot singlehandedly specify what beliefs we ought to hold—that is to say, the evidence 'does not speak for itself' (Stanford, 2009). Most authors who discuss scientific underdetermination do so rather peripherally, often in the capacity that psychiatric categories are never fully substantiated by the evidence available. Generally, authors either argue that the evidence does not clearly delineate between different categories of mental disorder (Kempf et al., 2005), that the evidence fails to decisively favour one nosology over another (Kendler 2022; Solomon, 2022), or that historically, psychiatric categories are mistakenly neglected or proposed on the basis of underdetermined evidence (Garrett, 2022). To be sure, the questionable validity of competing nosologies and systems of classification creates problems for reliable diagnosis. However, and somewhat strangely, few authors have more thoroughly engaged with the question as to how the underdetermination of evidence more directly affects diagnostic decision-making on a case-by-case basis, apart from how limited evidence bears on the reliability and validity of psychiatric categories writ large.

As such, my aim is to explore in what sense diagnostic judgments are underdetermined by the scientific evidence available to practitioners. I will argue that diagnosis is underdetermined, not necessarily because the validity and reliability of psychiatric categories are contested broadly, but because the modes of evidence appealed to for diagnosis are particularly prone to ambiguity in psychiatric contexts, more than for other forms of medicine. First, psychiatrists often rely on behaviour and other surface manifestations of mental disorders for diagnosis, yet these are not necessarily reliable indicators of underlying pathology; neither can they decisively differentiate between instances of psychiatric symptoms and non-pathological behaviour (Bortolotti, 2011). Second, the complexity of patient experiences, coupled with the causal heterogeneity of mental disorders, do not straightforwardly map onto neurobiological data and evidence generated by neuroimaging, suggesting that biomarkers alone cannot straightforwardly distinguish psychopathologies from one another (Muang, 2016). Last, psychiatry's extensive reliance on self-reports makes professionals' diagnostic judgments especially prone to underdetermination, given the difficulty associated with interpreting utterances and forming accurate linguistic representations of what patients communicate.

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Psychiatry's Second Validity Crisis? The Problem of Disparate Validation

Nicholas Zautra

In response to the “crisis in confidence” in the validity of the DSM’s diagnostic categories, psychiatry has seen a proliferation of alternative research frameworks for studying and classifying psychiatric disorders in new ways. The “big three” alternative approaches, which include the Hierarchical Taxonomy of Psychopathology (HiTOP), the Network Approach to Psychopathology, and the Research Domain Criteria (RDoC) have been characterized as a healthy response to the DSM’s “crisis of validity.”

A yet unexplored aspect of psychiatry’s “validity crisis” is related to disagreements regarding the *standards* of validity. Disagreements regarding standards of validity that amount to multiple distinct senses of validity point to a thornier methodological problem for psychiatry that I term “the problem of disparate validation.” This two-part problem can be summarized as follows: scientific psychiatry aims at achieving empirically informed classifications that demonstrate “validity” in the sense that they correspond to “real” attributes of psychopathology. To achieve this, alternative research frameworks are now approaching the conceptualization, testing, organizing, and validation of different features of psychopathology by their own standards in the hopes of one day informing more valid systems of psychiatric classification. The first problem is, given a system of classification, by whose standard of validity should such a system be validated? Is there a single validation procedure by which validation should proceed, or some other combination thereof? Second, when we attempt to validate classifications informed by differing standards of validity, will any such validation be capable of assessing a unified fundamental sense of “validity” that exists across the various frameworks, or will they only be “valid” in their own narrow sense?

In this talk, I offer an assessment of the problem of disparate validation through faithful reconstructions of the “holy quadrinity” of distinct senses of validity in psychiatry: starting with diagnostic validity (DSM) and proceeding with psychometric validity (HiTOP), network psychometric validity (the Network Approach), and etio-pathophysiological validity (RDoC). I introduce commonalities across frameworks that have not been previously addressed, including

how each framework employs *expert curation*, being the selection and justification of certain elements into their model based on compromises, and how the goal of each framework eventually becomes a return to the *original* validators of Robins and Guze to evaluate prognosis, biomarkers, and etiology of psychiatric classifications.

By evaluating psychiatry's distinct senses of validity, I argue that despite an appearance of a shared goal of informing more valid classifications, the existence of multiple frameworks in which each employs their own standards of validity and validation is ultimately the worst possible situation methodologically speaking for trying to do any kind of unified validation work. At its core, fundamental disagreements concerning 1) the underlying phenomenon that researchers are attempting to make inferences about; 2) standards of validating evidence; and 3) the very nature of validity and validation, move each framework further and further toward a state of *unrecognized pluralism*, being that we have yet to fully realize to what extent these frameworks are really not at all talking about the same thing and are in fact engaged in different projects with different aims.

I conclude with a positive program that suggests in what ways such different frameworks with distinct validation procedures, although ultimately incompatible, might come to inform one another in a kind of "patchwork" plurality. Given the inability of establishing a unified sense of validity, I compare several solutions for psychiatry's problem of disparate validation, from selecting and orienting one's validation work around what is evidenced to be the "most fruitful framework" and sticking to it, to psychiatry abandoning its emphasis on validity and validation in favor of differing notions of "testability," and other solutions in between.

Looking into Psychiatric Symptoms: The Case of Anhedonia and its Measurements

Daniel Andrés Montero Espinoza

Some of the current attempts to overcome the several challenges faced by the DSM have encouraged a gradual abandonment of DSM-based research to focus, instead, on the development of alternative frameworks for investigating psychopathology. Some research initiatives –most notably the Research Domain Criteria (RDoC)—have endorsed a transdiagnostic research approach in which psychopathological phenomena are investigated on the basis of psychobiological constructs (e.g., attention and working memory) that cut across the traditional DSM diagnoses. Although transdiagnostic research is not a unified program, a common goal of these initiatives –and certainly one of RDoC goals— is to free researchers from the traditional DSM categories by providing an alternative framework that aims to connect pathophysiology with psychiatric *symptoms* (Insel et al. 2010; Cuthbert & Insel 2013).

Concerns about the DSM-diagnoses' lack of validity are ubiquitous in the psychiatry literature, however, as Fellowes notes, those concerns seldom extend to psychiatric *symptoms*, the very constituents of the DSM-diagnoses (2021: 4503). In fact, some critics of the DSM have called for the abandonment of psychiatric diagnoses while still talking of traditional symptoms as

something that future research should focus on (ibid). In a similar vein, a former director of RDoC declared that “the concern about the current diagnostic environment has not been so much with the symptoms themselves” (Cuthbert 2014: 32), and later on, that “it is the grouping of symptoms into what have turned out to be overly heterogeneous syndromes that poses the problems for research” (Cuthbert 2015: 94).

In my talk I suggest that the default attitude adopted by current transdiagnostic research initiatives –such as RDoC— towards psychiatric symptoms is unwarranted. Firstly, I argue that the default attitude *generally* presumes that symptoms are (1) fairly straightforward study-objects whose measures are interchangeable in the research context, and (2) that symptoms are transdiagnostic in nature. To flesh out what I call the “default attitude in transdiagnostic research” I rely on evidence provided by Fried’s study (2017) of depression-measurement, and on the foundational papers of RDoC. Later, I challenge (1) by showing how two different and widely-used measures of *anhedonia* differ from each other in significant and clinically relevant ways: while one the measures portrays anhedonia as a long-lasting trait-like symptom, the other measure does not discriminate between a long-lasting and a transient-type of anhedonia. A consequence of this difference is that they fail to identify reasonably coextensive groups of individuals as having anhedonia, which replicates the problem of heterogeneity found at the level of diagnoses. Later, I rely on the same case-study to show that one of the measures of anhedonia was deliberately developed to “eliminate the effects of depression”, i.e., the scale was devised with the explicit goal of discriminating between a depressive-type of anhedonia and a schizophrenic-type of anhedonia (Chapman et al. 1976: 376). I conclude that my case-study provides evidence that while symptoms such as anhedonia can in principle be found in more than one diagnostic category in the DSM, some of the instruments used for its assessment were developed and calibrated targeting specific DSM-categories. This, I claim, challenges one of the main tenets of transdiagnostic research, namely, (2) that symptoms are largely independent from specific DSM-disorders. Furthermore, I claim that RDoC’s overall goal of “free[ing] research from constraint by current diagnostic entities” (Sanislow et al. 2010: 637) becomes implausible due to its *de facto* reliance on symptom-measures that are strongly DSM-based.

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Keynote Address

BioSocial Futures: Toward a Community Ecology of Health

Helena Hansen

This talk is based on over a decade of participant observation in the field of Translational Social Science and the use of social technologies in relation to health inequalities. It provides a case study in the author's research on race and the development and marketing of new opioids that led to the contemporary opioid crisis. The study of opioids revealed "technologies of whiteness" - neuroscience, new biotechnology development, regulation and marketing - that explain the racial symbolism and demographics of opioid use. The talk ends by interrogating the magic bullet ideology underlying the persistent lack of investment in social and structural determinants of health equity, including the biomedicalization of addiction in the era of white opioids, while offering approaches – from “structural competency” to BioSocial research and community ecological medicine – to addressing the institutional and policy drivers of the overdose crisis.

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Scientific Ignorance and Expert Trustworthiness

Manasa Gopakumar

It is commonly assumed that ignorance among experts undermines the public's trust in their expertise. This assumption, for instance, motivates Owen Whooley's (2019) sociological study of psychiatry, which traces the history of American psychiatry as a record of the profession's collective attempts at "managing" its ignorance of the nature of mental illness. Whooley's discussions, despite resting on several other questionable assumptions—e.g., that psychiatric ignorance is somewhat unique compared to other scientific fields (cf. Kourany and Carrier 2020) and that scientific disagreements necessarily amount to ignorance or crisis in the field (cf. Solomon 2014)—raise important normative questions about ignorance and expert trustworthiness that merit consideration in light of the widespread public distrust in psychiatry. This paper seeks to address two such questions: (1) Does ignorance necessarily undermine psychiatry's epistemic trustworthiness? and (2) How should the psychiatric community respond to its ignorance?

Contrary to the common assumption, I argue that ignorance in itself does not undermine expert trustworthiness; however, certain dispositions toward one's ignorance may either undermine or, in some cases, even enhance one's epistemic trustworthiness. This paper draws from the fields of agnotology (i.e., the study of ignorance) and feminist epistemologies of ignorance to develop an account of a normatively appropriate disposition toward one's ignorance, which can improve one's epistemic trustworthiness and facilitate responsible knowledge production. I build on the feminist notion of "loving ignorance" (Tuana 2006), which is a disposition characterized by an acceptance of the limitations of one's situated knowledge and one's epistemic dependence on differently situated others. Therefore, it is a form of epistemic humility that is attuned to the relational aspects of knowing and concerns the epistemic and ethical responsibility involved in producing knowledge about others. I argue that cultivating this epistemic virtue is crucial to enhancing psychiatry's epistemic trustworthiness and improving its epistemic practices.

I discuss the application of this concept by focusing on the various advocacy movements, such as the Mad Pride and neurodiversity movements, that have been challenging psychiatry's professional authority. I argue that these movements ought to be understood as epistemological movements seeking to reclaim epistemic agency for those who have historically been denied it and that it is important for the psychiatric community to demonstrate loving ignorance in this context. While many in recent years have been calling for greater integration of patient and advocacy groups in psychiatric research—which would certainly be a step in the right direction—I argue that these arguments nevertheless fall short of capturing the difficulty of establishing trust in the context of relations of power and what it means to respect the epistemic agency of minoritized groups in such contexts. Acknowledging the epistemic agency and authority of others goes beyond regarding them as mere sources of information or evidence—it also involves active engagement in the form of scrutinizing, checking, and

questioning one's perspective. This is important for establishing relations of epistemic trust between professional psychiatrists and patients/service users in collaborative or participatory research, which in turn is necessary for knowledge production.

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Justifying New Uses of Non-diagnostic Psychiatric Constructs: The Case of Insight

Derek Braverman

In contemporary psychiatry, psychosis is understood as a loss of contact with reality that may be caused by a mental illness, as in schizophrenia's hallmark symptoms of delusions and hallucinations. People diagnosed with schizophrenia and other psychotic disorders are often assessed as lacking awareness regarding their own mental illness: they may, for example, deny they are ill or affirm their delusions. In the mid-19th century, psychiatrists began to describe the different ways a person could possess awareness of their psychotic disorder as manifestations of a phenomenon called 'insight' (Marková 2005). But insight received little attention beyond clinicians specializing in psychosis treatment until Anthony S. David's (1990) influential proposal to standardize the construct. According to David, insight is a phenomenon with three dimensions: awareness that one has a mental illness, awareness that one's symptoms are pathological, and treatment adherence. So characterized, insight has recently enjoyed a surge of research and its clinical applications have expanded well beyond its initial scope of psychosis (David 2020; Oyebode 2023). In this presentation, I examine two of these new uses of insight: as an object of general psychiatric research, and as a factor whose absence licenses involuntary hospitalization. I argue there is insufficient justification for the continuation of either use, and I draw out some broader lessons for evaluating and refining psychiatric constructs.

General psychiatric research on insight is not limited to particular mental illnesses like psychotic disorders but instead is expected to have relevance across psychiatry. Examples include research

on the neural mechanisms that may underlie insight and the development of clinical interventions aimed at improving (i.e., increasing the presence of) insight (Williams, Olfson, and Galanter 2015; Oyeboode 2023). Meanwhile, an assessment that someone lacks insight can now provide an independent reason in favor of involuntary hospitalization. In some jurisdictions, lack of insight by itself warrants involuntary hospitalization (Radovic, Eriksson, and Dahlin 2020).

To ascertain whether these two uses are justified, I propose a minimal justificatory standard specific to each use: there should be some benefit to studying insight's three dimensions together rather than separately in general research, and insight assessments should provide non-redundant information pertinent to involuntary hospitalization. I then canvas the reasons that have been adduced in favor of these two uses, namely: correlations between lack of insight and negative patient outcomes; insight's relevance to decision-making capacity; insight's intrinsic value as a species of self-knowledge; and the results of mechanistic research on insight. Ultimately, I contend that none of these putative justifications meets the minimal standard for continuing each use. Furthermore, I explain how cultural variation on insight assessments would present a further challenge for the justification of both uses (Jacob 2010).

I conclude with two general points. First, I underscore how the minimal justificatory standards for different uses of a single psychiatric construct may vary dramatically, especially between clinical and research applications. Accordingly, I suggest that the justification of specific uses of non-diagnostic constructs deserves greater attention; compare, for example, the emphasis on validating diagnostic constructs. Second, I submit that the theory-avoidance of contemporary psychiatry (Decker 2007; Wakefield 2022) is a key factor that has allowed for the entrenchment of David's three-dimensional insight construct and the proliferation of its unjustified uses (Wimsatt 2007; Eronen and Bringmann 2021). I gesture toward an iterative approach (Chang 2004; Kendler 2012) to mid-level or middle-range theorizing (Alexandrova 2017; Cartwright 2020) that could start to rectify this problem, and I highlight promising instances of this approach in research that distinguishes clinical insight from cognitive insight (Beck et al. 2004) and separates insight in psychosis from insight in obsessive-compulsive disorder (Marková, Jaafari, and Berrios 2009).

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Karl Jaspers Award

The Epistemic Prerequisites of Reliable Abstinence in Addiction

Arthur Krieger

The distinction between first- and second-order ability in the philosophy of action enables new clarity in the discussion of behavioral control in addiction. Addiction involves automated patterns of thought and behavior that undermine the (first-order) ability to reliably abstain. However, addicts retain a second-order ability to reliably abstain if they remain able to learn the

“epistemic prerequisites” of reliable abstinence, including addiction-specific metacognitive skills and cue-avoidance strategies. The second-order ability to reliably abstain depends to a significant extent on socio-economic situation and access to the right social resources. This “Epistemic Prerequisite Model” supports the view that addiction is a compulsion, and suggests that moral responsibility in addiction depends on factors relating to both orders of ability.

TUESDAY, MAY 7

Normativity, Deviation, and Mental Disorder

Andrew Evans

The most common theoretical approaches to defining mental disorder are: *naturalism* which characterizes mental disorders as biological dysfunctions (Kendell 1975; Boorse 1976; Boorse 1977), *normativism* which defines mental disorders in terms of what society deems harmful (Sedgwick 1973; Cooper 2002; Bolton 2008), and *hybridism* which asserts that mental disorders are both socially disvalued and biologically dysfunctional (Wakefield 1992; APA 2013). Naturalism and normativism are often portrayed as diametrically opposed, with naturalism grounded in objective science and normativism grounded in social convention and values. Hybridism is seen as a way of combining the two. However, all three approaches share a common feature in that they conceive of mental disorders as deviations from norms. Naturalism concerns biological norms; normativism concerns social norms; and hybridism, both biological and social norms.

The fact that these theories all conceive of mental disorders as norm deviations brings the following two questions into view: (a) Are biological and social norms the only sorts of norms that are relevant to considerations of mental disorder? (b) Should addressing norm deviations continue to be a major focus of mental healthcare, or are there other objectives that the field ought to pursue? In Section 2, I begin by arguing for the claim that naturalism, normativism, and hybridism are best understood as theories of norm deviation. In Section 3, I introduce the concepts of “psychological norms,” “individual norms,” and “welfare norms.” A condition deviates from psychological norms when it inhibits one’s ability to navigate their psychosocial world (Plutynski 2023; Leder & Zawidzki 2023). A condition deviates from individual norms when it is a marked change from the person’s baseline (Bolton 2008). And a condition deviates from welfare norms when it decreases one’s state of well-being. I argue that mental disorders often deviate from these other types of norms, and so they should be considered alongside biological and social norms. In Section 4, I call into question mental healthcare’s focus on addressing norm deviations in the first place. Advocates of the neurodiversity paradigm and social model of disability have argued that deviation is not inherently negative, and have called for social changes to accommodate those who diverge from the norm (Walker 2013; Kinn 2016; Chapman 2019). Supporters of Mad Studies and the Mad Pride movement have argued that

psychological difference should be celebrated rather than treated medically (Beresford & Russo 2016; Rashed 2018). Drawing on these approaches, I argue that addressing norm deviations ought not be a major objective of the mental health field. I end by suggesting other possible objectives such as reducing suffering, providing holistic support, and facilitating growth.

When is Drug Use an Addiction?

Bennett Knox

In this presentation I will explore and defend a necessary condition for drug use to count as an addiction: it must undermine a person's well-being. Further, I will argue that for the purposes of these judgments, we ought to understand well-being according to Valerie Tiberius's subjectivist value fulfillment theory (2018). According to this theory, we may judge that a person's drug use is undermining well-being (and therefore a candidate to count as an addiction) when drug use constitutes a value that is inappropriate to that person. I will first present the value fulfillment theory, highlighting the aspects which make it relevant for making judgments about drug use and addiction. Then I will explore the implications of this theory for various kinds of drug use, in order to show that the value fulfillment theory both gets obvious cases right and can give us guidance in more contentious and puzzling cases.

The heart of the value fulfillment theory is the claim that well-being consists in the fulfillment of one's values across a lifetime. Values are understood as robust patterns of desires and emotions, which individuals see as giving reasons for action relevant to living their lives over time. Though the theory is ultimately subjectivist about well-being, it also gives an account of how to assess and critique the appropriateness of values for an individual. Appropriate values must be well-suited to the person, reflectively endorsed, and capable of being fulfilled across a lifetime.

In these terms, my central claim is that drug use can count as an addiction only if the value of using drugs is inappropriate to the individual in question—that is, if that value is undermining their well-being according to the value fulfillment theory. Though this claim may seem uncontroversial or even uninteresting at first blush, following its implications leads to some controversial (but, I argue, correct) conclusions.

There are many cases of drug use which are obviously not addictions (i.e. do not undermine well-being), and many which obviously are. But there are many cases where the judgment about whether or not drug use is undermining well-being is less clear. In cases where drug use undermines the fulfillment of a person's other important values, we can judge that such a person has a drug addiction (i.e. their valuing of drug use is inappropriate).

But there are also cases where others may judge that a person's drug use constitutes an addiction, but where drug use is actually appropriate given the person's circumstances and values. To explore this possibility, I will discuss cases where value of drug use does not undermine fulfillment of other values, and may even enhance fulfillment of values like social

connection and belonging; where giving up drug use would involve a radical personal transformation, such that the resulting person is importantly different from the original person; and where social circumstances are such that drug use is the least bad option for a person. In these cases I argue that we cannot judge that the person's value of drug use as inappropriate, and so as I have defined things they would not count as having a drug addiction. I will conclude by offering some implications that my view has for how psychiatry ought to engage with cases like these, and with drug use more broadly.

Fleishman is in Trouble: On Sadness, Irrealis Moods, and Existential Choice in Psychiatry

Harini Sridhar and Laila Knio

This session will position existential sadness as stemming from the dilemma that alternatives exclude, and will explore ways in which mental health providers can recognize, attend to, and address this suffering. We will begin with a close reading of the final scene from the miniseries *Fleishman is in Trouble*. The show, based on Brodesser-Akner's novel of the same name, is set in Manhattan and explores the challenges encountered by recently-separated physician Toby Fleishman. Toby's best friend, the non-omniscient narrator, poignantly describes the essence of the story during a 3-minute segment that will be played at the beginning of the session: "It's about life, and marriage and money and dissatisfaction and lifelong friendship, and how all these things coalesce in middle age and make you miserable right at the exact point that you're supposed to have everything set."

The piece will serve as an entry point into the proposal that much of the refractory sadness that affects us and our patients stems from our multipotentiality and the realization that by virtue of choice-making, many of our potentials remain unfulfilled. We interweave Sartre's notion of existential choice, with the ultimate recognition that these choices shape identity. In particular, themes from the session will engage this portion of text: "Nothing could make her unmake the choices that she made. She just didn't know when she was making the choices that they were gonna limit all the other choices that she could make in the future... It's just, how can you live when you used to have unlimited choices, and you don't have them anymore?" Just as synaptic pruning molds the ever-evolving brain, choice pruning molds the ever-evolving self.

We further that loss of future possibility, and grief over this loss, is deeply embedded in language. In his latest work *Homo Irrealis*, Aciman describes the way in which the irrealis moods – "the might-be and the might-have-been" – are critical to the way we talk about, remember, and yearn for the past, present, and future (these verb tenses include the conditional subjunctive, the optative, and the imperative). This language structure allows for a kind of reflection Aciman terms "retro-prospection":

"the script of roads not lived and of lives that have been cast adrift, unlived, or misspent... [T]he life we're still owed and cannot live transcends and outlasts everything, because it is part yearned for, part remembered, and part imagined, and it cannot die and it cannot go away because it never, ever really was."

Drawing on what Heidegger calls “being-towards-death,” we position mortality – and our recognition of it – as a necessary condition of retro-prospection and existential choice. Given that we have a singular life to live, the pressure to make the ‘right’ choice (for instance about life, marriage, money, friendships) can be overwhelming. With precision and breathless brevity, it comes down to this: “things fade: alternatives exclude” (John Gardner in his novel *Grendel*).

Ultimately, we situate psychiatry and palliative care/hospice medicine as two specialties particularly well situated to intersect with and address existential suffering. In end-of-life care as well as chronic and refractory mental illness, we suggest that a nuanced attunement to irrealis moods, retro-prospection, and existential choice in the narratives our patients tell us can better illuminate the conditions that warp our patients’ suffering. For instance, we suggest that this kind of attunement might help us better understand – and treat – treatment-refractory depression and existential pain. Finally, in recognizing the intersubjectivity of the clinical encounter as well as the permeable membrane between our professional and personal lives, we point to ways in which this kind of attunement might palliate our yearning for our own pasts, presents, and futures.

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The Dodo Bird Verdict and Psychotherapies as Placebos

Julia A. Harzheim and Dien Ho

Although the benefits of psychotherapeutic modalities when compared to no treatment are widely recognized, there has been growing empirical evidence that all conventional psychotherapies provide roughly the same benefits. Dubbed the “Dodo Bird Verdict” by Saul Rosenzweig in his 1936 paper “Some implicit common factors in diverse methods of psychotherapy,” the worry is that the theoretical foundations of radically different conventional psychotherapies matter little in clinical outcomes and that incidental factors such as empathy and therapeutic alliance perform much, if not all, of the therapeutic work. Couple that with Grünbaum's definition of placebo effect as those caused by incidental features of a treatment, the natural conclusion is that the benefits of psychotherapy are largely placebogenic.

A number of scholars have expressed dismay at the prospect of psychotherapy-as-placebo. Their worry is that it undermines both the legitimacy of psychotherapeutic practice and its corresponding training. In this paper, we examine the threat of illegitimacy and argue that the worry conflates a number of concepts. For instance, one concern is that psychotherapy works not

as various approaches intend; instead, if the Dodo Bird Verdict is correct, the benefits are caused by incidental factors and thus non-specific. Any treatment that is non-specific is medically illegitimate, so the argument goes. We note that the causal power of a treatment is conceptually different from its specificity. Bygone medical practices (e.g., hot baths to sweat out syphilitic poison) might have no causal power but they are nevertheless specific treatments. This echoes the old demarcation debate: bad science can still be science. Moreover, incidental features such as empathy and therapeutic alliance do not exist in a theoretical vacuum. To initiate a working therapeutic alliance, therapists must recognize the existence of a pathology that is the cause of the patient's distress. This latter determination depends on the theoretical orientation of the therapist. In this respect, even if incidental features such as empathy shoulder much of the therapeutic work, it does not follow that the theoretical foundation of a therapist's approach is of no clinical importance. To urge that one jettisons allegiance to a particular psychotherapeutic school in favor of the Contextual Model, as Bruce Wampold and Zac Imel advocate in their 2015 *The great psychotherapy debate: the evidence for what makes psychotherapy work*, is to presuppose that other psychotherapeutic modalities are false. The evidence for the latter is thin. Indeed, there appears to be significant challenges designing a randomized clinical trial that compares a psychotherapeutic modality against a placebo control that is also a bona fide treatment.

Finally, even if it were the case that the benefits of a therapeutic intervention are wholly or largely placebogenic, it does not follow that their continual prescriptions violate epistemic or ethical norms. Many conventional therapies provide tremendous benefits vis-à-vis placebogenic actions (e.g., vertebroplasty). A quick rejection of them without providing a placebo alternative robs patients of effective treatments. Here, the tension rests on broad questions about the very goal of medicine; that is, should we privilege effectiveness defined as the outperformance against placebo controls (a particular kind of scientific effectiveness) or focus instead on a treatment's superiority against no-treatment (a pragmatic orientation)?

Understanding Produces Expectation: Ricoeur's Narrative Theory and the Open-label Placebo

Jennifer Hauptman

Ricoeur's narrative theory demonstrates how phenomenological hermeneutics can produce an understanding a text, or event, rather than an explanation. In this paper, I propose that the placebo effect is likewise generated through the process of generating a narrative understanding of the therapeutic potential of the placebo, and that this occurs through employment within the context of Ricoeur's narrative theory.

My argument is that the placebo effect requires a therapeutic expectation to be conferred on to a therapeutically inert agent. However, I reject the requirement of deception to produce this expectation. This is because studies (see Charlesworth et al, 2017) have demonstrated that even

when individuals are explicitly told that they are receiving a therapeutically inert agent, the placebo effect is observed. As this is the case, I propose that the placebo effect is the product of an expectation of a possible outcome, rather than a knowledge of an outcome that is anticipated by another. This is because the placebo effect is observed in individuals who have not received an explanation of the therapeutic effects of the agent, as described above.

So this expectation must be generated by the individual within the context of an absent expectation of a therapeutic outcome. As such, I argue that this expectation occurs in response to the event itself, rather than an explanation of the event provided by another. Ricoeur's phenomenological hermeneutics demonstrates a how understanding is generated by an individual through their own interpretation of the experience of a text, or events. I propose that this is also how expectations are generated for open-label placebos. In this example, I argue that this expectation is produced through the act of emplotment.

In this case, the event subject to emplotment is the encounter with the placebo. In *Time and Narrative*, Ricoeur outlines how emplotment occurs via the process of threefold mimesis. Emplotment for the event of the placebo occurs in the same way. And for the individual receiving the placebo, this emplotment generates a narrative understanding of the therapeutic potential of this substance.

In the threefold mimesis of the placebo effect, mimesis₁ represents the preunderstanding of the effect of the therapeutically inert agent based on the individual's own prior understanding of the agent and its context. Key to mimesis₁ is that this preunderstanding includes both consciously and unconsciously held beliefs, or impressions. Mimesis₂ is the process of confrontation with another perspective and the process of configuration, and in this example represents the event of the encounter with the placebo itself. Mimesis₃ represents a re-figuration of the pre-understanding informing mimesis₁, including the impressions of the event encountered in mimesis₂. This represents a concordant discordant emplotment of the event in which an understanding of possibly contradictory events can be produced. In this example, it may be the consciously held belief that the agent is therapeutically inert, and the unconscious impression of a possible encounter with a therapeutically active agent.

As proposed in *Time and Narrative*, the act of emplotment resolves the issue of *distentio animi* for Augustine's account of temporality. For the placebo, threefold mimesis demonstrates how emplotment produces an expectation or anticipation of future events, as well as a narrative understanding of past and current events. I argue that the effect of the placebo is produced in response to resolve the expectations generated via emplotment.

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Bayesian Brain Modeling in Placebo and Medical Studies: Unifying Theories and Enhancing Predictive Insights

Kevin Jumaa

Historically, placebo responses have predominantly been construed within the framework of Pavlovian and operant conditioning. In more recent times, open-label paradigms have illuminated the many psychosocial factors that influence the placebo response. These factors collectively contribute to the current conceptualizations of the placebo response. Despite the acknowledgment of these components, contemporary considerations tend to isolate them. What is needed to further the understanding of placebo responses is a unified theory that can incorporate diverse conceptualizations of placebo responses. This would facilitate the establishment of standardized controls, assist in ethical decision-making in RCTs, integrate psychobiological, neurobiological, and contextual factors, enhance mind-body control, and quantify expressions and beliefs for the purpose of measuring their impact on outcomes.

An approach in probability theory, Bayes' theorem, has been gaining popularity in explaining brain phenomena with recent application in placebo research. The Bayesian Brain Model (BBM) explains placebo responses in a way that successfully integrates current leading theories of the brain. According to the BBM, the predicted likelihood of an event occurring is constantly being updated in the brain by integrating past and present experiences, external cues, and internal cues, in terms of probabilities. The brain is continuously generating predictions of events through top-down processing, and it consistently revises these assumptions during bottom-up processing. Top-down and bottom-up processing often conflict with each other, and since the goal of the brain is to maintain coherence, it takes the path of least resistance during these conflicts. The path of least resistance is a cognitive strategy employed by the brain to conserve mental resources and streamline decision-making processes when it is receiving conflicting inputs. For example, in OLP studies that occur in clinical settings, patients knowingly receive placebos in clinical settings. The conflicting top-down processes of being in a clinical setting, which signals a healing environment, and knowingly receiving an inert substance, conflict with each other. The brain reconciles the conflict of knowingly receiving a placebo by endogenously produced analgesics secondary to the healing environment (clinical setting), signaling recovery in the brain. In the face of conflicting top-down and bottom-up processing, choosing the path of least resistance allows the brain to quickly reconcile discrepancies and maintain a coherent narrative.

Studies have begun to use the BBM to predict placeboogenic responses in analgesic studies with great predictive capabilities¹⁻². These studies show that the BBM is able to integrate and operationalize the different components of placebo responses to accurately predict placebo responses. Because of this, I argue that the implementation of the BBM in placebo studies not only addresses the complexities of psychosocial factors, but also provides a crucial bridge between disparate theories, creating a unified framework for understanding placebo responses. By integrating Pavlovian and operant conditioning theories in open-label paradigms, the BBM facilitates a more holistic approach to studying placebos. This comprehensive perspective allows for the establishment of standardized controls in experimental settings, aiding

researchers in mitigating confounding variables and ensuring the reliability of results in Randomized Controlled Trials (RCTs). The ethical implications of placebo administration are also more systematically addressed with the BBM, as it enables a nuanced consideration of the contextual factors influencing participants' responses. In the realm of medical studies, the Bayesian Brain Model's application extends beyond placebo research to offer valuable insights into various health-related phenomena. Researchers can leverage the BBM to gain a deeper understanding of the mind-body connection, shedding light on how psychological and neurological processes interact to influence health outcomes. This nuanced understanding holds immense potential for refining treatment approaches, tailoring interventions to individual needs, and advancing personalized medicine.

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Keynote Address

Unsettling Evidence-Based Medicine: Three Challenges from Placebo Studies

Phoebe Friesen

The rapidly emerging field of placebo studies is causing ripples across medicine. The placebo effect is now receiving recognition as a real and powerful phenomenon that plays a significant role within medical encounters, as evidence increasingly demonstrates how expectations, conditioning, and aspects of the patient-physician relationship can mediate changes in the brain and body leading to improved clinical outcomes, but only in some, often psychosomatic, symptoms (e.g., acute pain, nausea) and conditions (e.g., chronic pain, mood disorders, functional syndromes). This presentation will explore three significant and underexplored challenges for evidence-based medicine (EBM) that emerge from the burgeoning field of placebo studies. First, placebo scholarship challenges the boundaries of EBM. A close look at the mechanisms underlying placebo effects indicates that practitioners working outside of these boundaries, within the domain of complementary and alternative medicine, may be especially capable of producing relief through placebo effects. Second, placebo research undermines the methods of EBM. Treatments that utilize placebo effects are unlikely to be deemed efficacious within randomized-control trials, leading to the efficacy paradox, in which the best available treatments for some placebo-responsive conditions cannot make it to market. Finally, the field of placebo studies threatens to reveal and reinforce longstanding biases within medicine. The conditions that tend to be most responsive to, and most examined, within placebo studies, are psychosomatic ones, in which patients are often seen as untrustworthy and unreliable witnesses,

and patients often unheard and disrespected. Placebo research can serve to reinforce the notion that these forms of suffering take place ‘all in the head.’

“You’re Gonna Love This Drug”: The Ethics of Psychiatric Salesmanship

Zach Schwartz and W. Alexander Bennion

Should psychiatrists intentionally exaggerate the benefits, and downplay the risks, of the medications they prescribe? On its face, this may seem wildly unethical; but in clinical practice it is extremely common (or so we believe, based on our experience as psychiatric trainees). Psychiatrists will frequently tell patients, “I think X will do a great deal to relieve your Y,” when they know, from large-scale clinical trials, that the probability Y responds to X for any given patient is fractional. Likewise, psychiatrists may intentionally minimize treatment risks (“Some patients who begin taking SSRIs develop sexual side effects,” rather than, “The majority of patients who begin taking SSRIs develop sexual side effects”) or neglect to mention certain risks altogether, out of concern for exacerbating nocebo effects. This kind of “salesmanship” appears at odds with the neutral accounting of risks/benefits expected in non-psychiatric medicine and, indeed, with the principles of informed consent.

The question, then, is whether psychiatric salesmanship is ethically justified. There are certainly grounds to think it is not. First, and most obviously, it is dishonest, and honesty is valuable to the doctor-patient relationship for many reasons. Second, by manipulating the information patients receive about their treatment options, psychiatric salesmanship would seem to infringe on autonomy. Third, one might question whether psychiatrists are improperly motivated or have a conflict of interest in “selling” prescriptions to their patients. Lastly, one might raise concerns about unintended, downstream effects of widespread psychiatric salesmanship, particularly on the overvaluation of pharmacological strategies for improving mental health.

On the other hand, there are compelling reasons to think psychiatric salesmanship *is* ethically justified. First and foremost, psychiatric salesmanship is likely effective. While no clinical trials have directly demonstrated this, indirect evidence about placebo, nocebo, and related phenomena strongly suggests that rhetorical exaggeration of benefit, and minimization of harm, leads to improved treatment outcomes (i.e. relief of suffering) for patients with psychiatric disorders. Next, it’s conceivable that psychiatric patients constitute a special population in regard to the ethics of informed consent, for two related reasons: 1) psychiatric disorders tend to be uniquely belief-responsive, i.e., what patients believe about their prognosis is causally relevant to their actual prognosis, and 2) psychiatric disorders can predispose patients to a heightened awareness of negative experience, as well as excessively pessimistic predictions regarding future experience. By ignoring these unique features of psychiatric illness, and trying to neutrally inform patients of “just the facts,” providers may inadvertently support biased reasoning. Thus, we might conceive of psychiatric salesmanship as a necessary corrective measure in the service of a patient’s best interests.

After evaluating each of these arguments, we conclude that psychiatric salesmanship is ethically justified in many, but not all, discussions of treatment. The ethical justifiability for any particular case would seem to depend on the nature of the disorder, the harms of treatment versus non-treatment, and the availability of other treatments. We suspect these are the factors that good psychiatrists already take into account (if only subconsciously) when choosing their rhetoric, but we believe there is value in making such considerations more explicit.

Do No Harm: Applying Nocebo Research for Non-maleficence

Mariève Cyr and Jay Olson

Non-maleficence — doing no harm — is a core principle of bioethics in medicine. Harm caused by negative expectations, however, is rarely considered. Medical treatments have two types of negative effects: side effects, such as those caused by the active ingredient in the medication; and nocebo effects, caused by contextual factors such as negative expectations, verbal suggestions, and social observation. For example, patients report more pain when clinicians use negative rather than neutral language, such as “You will feel a small pinch” rather than “I will now place the IV” (Lang, 2012). Such nocebo effects are well documented in randomised controlled trials, with Papadopoulos and Mitsikostas (2011) finding that 18 to 79% of patients in placebo control groups report various side effects. Such side effects can worsen health outcomes (e.g., Howe et al., 2019), for example by reducing treatment adherence or increasing anxiety.

In clinical practice, side effects and nocebo effects are entangled and fall under the umbrella term of adverse events. Yet, medicine generally focuses on the biological causes of adverse events while neglecting the contextual and psychosocial factors that modulate them. Indeed, healthcare professionals generally receive little training on how to leverage such factors to minimise nocebo effects. Clinician instructions thus often contain unintended negative suggestions which can promote nocebo effects, such as “This is the worst part of the procedure” or “You are a high-risk patient”. Ignoring these contextual factors can result in preventable harm to patients and, we argue, can thereby go against non-maleficence.

Fortunately, many of these negative effects are easily preventable. Studies have demonstrated that simple changes to clinicians' behaviour, which often do not require additional time or resources, can reduce these adverse events and their associated consequences. In order to fully adhere to the "do no harm" principle, we argue that clinicians should be trained on how to avoid contextual factors that cause unintended harm. However, there are currently no comprehensive reviews offering specific and feasible recommendations for clinicians on how to reduce nocebo effects in clinical settings.

With the goal of developing such recommendations, we conducted a pre-registered systematic review of the literature. We first searched several databases for nocebo-related terms (MEDLINE, PsycINFO, Embase, Web of Science, and Cochrane Reviews). Raters then screened the resulting 1542 abstracts to include only peer-reviewed randomised trials. We focus on studies that manipulate at least one situational and controllable independent variable (e.g., credibility cues, specific verbal suggestions) in which (1) at least 20 people receive at least one level of the manipulated contextual factor, (2) there is at least one comparison group, and (3) at least one nocebo-related or clinically relevant outcome is reported.

After compiling the results, we plan to complete a narrative and qualitative synthesis focusing on the feasibility of the contextual factors, the original authors' interpretation of the results, and the quality of the studies. Finally, we will compile a list of feasible and concrete recommendations for clinicians, which can serve as the basis for training on reducing nocebo effects in healthcare settings. We hope that this project will allow healthcare providers to reduce preventable negative outcomes and thereby adhere to a broader definition of non-maleficence.

Subverting the Diagnostic Treatment Commensurability Paradigm: Approaching an Ethical Framework for Placebo Inclusion in Research and Clinical Care

Julia Kolak

It is generally taken to be a core tenet in research ethics that what makes an intervention appropriate to incorporate within a therapeutic clinical arsenal is that it has been empirically substantiated as efficacious through a rigorous process of testing and research. As the gold standard for clinical trials, placebo controlled randomized trials (RCTs) primarily aim to demonstrate the efficacy of an intervention by way of assay sensitivity—namely, distinguishing active from inactive controls (Temple and Ellenberg 2000). However, in the context of psychopharmacological therapies, the endpoints which factor into the assessment of efficacy is a metric fundamentally anchored to self-reported changes in symptoms. While this is in large part informed by the fact that the diagnostic categories which constitute the reference class of any investigative psychiatric drug are primarily descriptive (i.e., etiologically neutral) in nature, their status as pharmacological targets are also informed by the harms associated with the clinical sequelae of higher-order symptom space.

For this reason, if a perceived impact on these psychiatric symptom clusters can be brought about by inert substances or “sugar pills” alone, and that change is both statistically and clinically significant, it is not clear why placebo effects in psychiatry should be discounted as efficacious interventions. Importantly, this proscription does not intrinsically derive from the empirical norms of the investigative environment; the ability of placebos to produce significant effects which may outstrip an active control have been widely documented—they have even been called one of the most powerful therapies in psychiatric medicine (Shorter, 2011). Rather, their rejection is standardly grounded on a mistaken interpretation of the ethical standards of medical practice which I term the diagnostic-treatment commensurability paradigm.

Broadly construed, I define this paradigm as the expectation that a therapeutic modality must be capable of directly interfacing with the biological basis or underlying causal mechanisms of a disorder or disease to be considered an ethically acceptable medical intervention. Described in a naturalist or essentialist mien, those who implicitly or explicitly subscribe to this framework often do so to marshal the claim that a given application of biomedicine bypasses evaluative bias or normative contamination—a narrative that biological psychiatry is especially eager to adopt amidst the ongoing crisis of substantiating diagnostic kinds (Tabb, 2017).

Despite the logical disconnect between what is clinically significant and what is causally apt, any favorable symptomatic changes brought about by an *active* control are interpreted as the direct impact of the pharmacological agent on the causal underpinnings of a mental disorder. Though much attention has recently been paid to the limitations of this approach and the question of whether and to what extent the assay sensitivity of psychopharmacological agents are reliable (e.g., Moncrieff, Cooper, Stockman et al., 2022; Lacasse and Leo, 2005; Kirsch 2009, 2014; Wang et al. 2016; Jakobsen et al. 2017), those who discredit placebo inclusion in research and practice primarily do so because of their conviction that only those drugs which have been shown to outperform placebo controls license causal claims about their efficacy, and ipso facto, their clinical appropriateness.

Accordingly, I systematically unpack the erroneous assumptions on which the diagnostic treatment commensurability paradigm is based and gesture towards an ethically sound foundation for the prospective application of placebos in clinical practice. Importantly, this approach transcends the expectation that effective treatments must causally interface with the underlying biological basis of a diagnostic category, building upon recent developments which emphasize the clinical burden of higher-order symptom space, rather than the etiological profile, of psychiatric disorders (e.g., Gauld, 2022). Given the important ballast to the proliferation of ineffective treatments that assay sensitivity represents, I conclude by identifying alternative means of supporting the continued use of the concept in clinical research by distinguishing between “inactive” and “therapeutically inert” controls.