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Commentary

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Beyond problematic binaries in mental healthcare: a commentary on Read & Moncrieff – depression: why drugs and electricity are not the answer

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Read and Moncrieff (2022)'s discussion of antidepressant medications and electroconvulsive therapy (ECT) is contextualized within an approach that takes a binary view of psychiatric illnesses and their treatment. The characterization of depression as a 'medical illness' is understood by them to imply that depression is caused by biological dysfunctions (with social and psychological factors relegated to a secondary status) and that treatments work by correcting said dysfunctions. It is further stated this view of depression as a medical illness excludes the possibility of meaning and agency. This is contrasted with their own view of depression as an 'emotional and meaningful response to unwanted life events and circumstances', with the implied rejection of biological causes. This problematic binary framing facilitates erroneous discussion of psychiatric concepts and practices (Aftab, 2020). It fails to take into account the variety of ways in which disorder concepts are understood philosophically (Phillips et al., 2012), many of which do not conceptualize psychiatric conditions as biological diseases. The dichotomy gives the misleading impression that we must choose between either viewing depression as a medical illness or as a 'normal' response to life events. In our opinion, it is a mistake to debate within these binary terms and we intend to outline why this is the case.

A sharp and binary distinction between biological and psychological explanations is untenable in light of our best understanding of cognitive-affective science and neuroscience. Our minds are embodied, embedded, and enacted. The involvement of biological factors can take many different forms in explanations of depression: (i) biological *dysfunctions* (e.g. hypothyroidism, stroke, HPA axis abnormalities, etc); (ii) biological *risk factors* (e.g. genetic variants, inflammatory processes, etc), (iii) biological *mechanisms* (e.g. brain circuits involved in the regulation of mood). There is extensive literature that supports this, summarized in most psychiatry textbooks. The exact nature of involvement will vary from person to person; for some individuals there may very well be no biological dysfunctions or biological risk factors, but due to the embodied nature of mind, there will still be biological mechanisms involved. Understanding biology in this manner also provides a rationale for biological intervention that doesn't necessarily rely on disease processes; biological mechanisms do not have to be dysfunctional for us to successfully intervene on them to produce desired effects. Read and Moncrieff fail to account for another crucial aspect: the tremendous heterogeneity of depression. It follows from the heterogeneity that there is likely no single etiological account of depressive syndromes (e.g. depression as a response to life circumstances) because depression is not one thing.

Read and Moncrieff describe a dichotomy between a 'disease-centered' and 'drug centered' model of drug action; in the case of former, medications are assumed to correct an underlying biological dysfunction; in the case of latter, 'psychiatric drugs change mental states and behavior through the modification of normal brain functions'. This dichotomy fails to capture the complex ways in which medications are employed and their mechanisms of actions understood in both general medicine and psychiatry (Huda, 2019). Some medications do target specific disease processes, but many are often prescribed to reduce or manage symptoms and do not correct underlying biological dysfunctions (Huda, 2019, p.291). In many cases in medicine, medications will act to alter 'normal' processes, e.g., antipyretics and antitussives produce symptom relief by interfering with 'normal' mechanisms of temperature regulation and cough. In other instances, which is often the case in psychiatry, medications are utilized because they are known to be effective, but the mechanisms are unknown or poorly understood. In a survey of UK consultant psychiatrists regarding their attitudes to prescribing psychiatric medication, around 85% adhered to an outcome-centered model for antidepressants for depression in which psychiatrists thought there was evidence of benefit but not necessarily reversal of a disease process (Huda, 2019, p.110). It may well turn out to be, although it remains to be

established, that the efficacy of antidepressants doesn't depend on correction of a biological dysfunction: contemporary researchers are exploring hypotheses such as the enhancement of synaptic neuroplasticity by antidepressants, which would facilitate reorganization of brain networks guided by environmental stimuli (e.g. psychotherapy), resulting in therapeutic improvement (Casarotto et al., 2021).

Not only do Read and Moncrieff ignore traditional psychiatric accounts such as descriptive and psychodynamic approaches, they also do not consider developments of the last decade. These include, among others: (a) symptom network models, which conceptualize depressive syndrome as a self-sustaining cluster of symptoms in the absence of a common cause or biological disease (Borsboom, 2017); (b) the Hierarchical Taxonomy of Psychopathology (HiTOP), which conceptualizes syndromes such as depression dimensionally, reflecting differences in degree rather than in kind, with no natural boundary that distinguishes normal from abnormal (Kotov et al., 2017); (c) enactive approaches (De Haan, 2020), which conceptualizes depression as a disorder of 'sense-making', arising from an interaction of physiological, experiential, socio-cultural, and existential dimensions.

Read and Moncrieff's preferred approach to depression as a 'meaningful response to unwanted life events and circumstances' doesn't satisfy the basic desiderata of a scientific explanation. 'Meaningful' remains woefully undefined and offers no basis for discrimination between what is and is not 'meaningful'. It accounts poorly for instances of depression that bear no manifest causal relationship to life events, or where the causal relationship to life events is just one factor in a web of causes. The hypothesis is too vaguely specified and too flexible to be testable or falsifiable. It does not explain why some depressive reactions present with severe symptoms such as psychosis and catatonia, while others do not even come to clinical attention. And it fails to consider the role neurobiological mechanisms (such as the HPA axis) play in mediating responses to stressful and traumatic life events.

Read and Moncrieff emphasize the importance of socio-political action, the implication being that such interventions are somehow incompatible with the medical approach. They write, 'Classifying anxiety, depression and other emotional reactions as mental diseases or disorders obscures the relation between our moods and our circumstances. It leads society to believe that social structures are unchangeable.' This is manifestly incorrect, and evidence to the contrary is easily available by looking at the history of robust public health interventions in response to bona fide diseases such as tuberculosis. The on-going COVID-19 pandemic itself is proof enough of the importance of socio-political action for disease prevention and control. While social determinants of health have been neglected in psychiatric practice, their existence is not in dispute in the medical model. Just last year the president of the American Psychiatric Association made addressing social determinants of health a priority of her presidency (Pender, 2021), while remaining firmly within the medical bio-psycho-social model. Therefore, we reject the implication by Read and Moncrieff that addressing socio-political determinants of health requires giving up the medical model in the broad and pluralistic manner we understand it.

In this brief commentary, we have restricted ourselves to conceptual and philosophical issues that provide the context for Read and Moncrieff's article, without delving into the empirical shortcomings pertaining to antidepressants and ECT. In our opinion, the numerous conceptual deficiencies of Read and Moncrieff's article indicate that it cannot be taken seriously as a guide to practice by clinicians, researchers, patients/service users, legislators, and the public at large.

Conflict of interest. None.

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