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Commentary

Precision psychiatry: thinking beyond simple prediction models, without dismissing them

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Precision medicine; causal inference; big data; machine learning methods; prognostic/prediction modelling.

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Response

Krishnadas and colleagues make an important and timely case for strengthening precision psychiatry through causal prediction, with a particular focus on personalised treatment: using individual-level information to determine which intervention might improve an individual patient's outcome.[1] However, much of the subsequent debate[2,3] appears to stem from a more central definitional issue: whether precision or personalised psychiatry should be understood broadly, or narrowly as causally informed individual treatment selection. This narrow framing also underlies the second disputed claim that non-causal prediction models 'may be futile at best and actively harmful at worse'. Seyedsalehi and colleagues were right to challenge this latter claim.[2] However, the subsequent exchange left unresolved the more fundamental definitional issue underlying the dispute, thereby inviting a further response.[3]

Precision or personalised medicine is generally used as a broad term for moving beyond the 'average patient' by tailoring prediction, prevention, diagnosis, treatment or care to individual characteristics, including clinical, molecular, environmental, lifestyle and digital information.[4,5] Precision psychiatry, as a field within this broader agenda, should therefore not be reduced to only one of its components: causal personalised treatment selection. Krishnadas et al invoke the phrase 'the right intervention to the right patient and at the right time' to defend this narrower framing. However, this phrase is a legitimate aspiration for precision medicine, not a licence to exclude all risk-stratified or prognostic tools from the field. Their response[3] to Seyedsalehi et al therefore risks resolving the debate by narrowing the definition of precision psychiatry, rather than addressing whether non-causal prediction models have legitimate roles within the broader precision medicine agenda.

Krishnadas and colleagues correctly note that non-causal prediction models should not be interpreted as estimating causal effects: changing a covariate in such a model should not be read as evidence of what would happen under an intervention. Yet it does not follow that non-causal prediction models are futile. They answer a different question: who is likely to experience an outcome under the care patterns represented in the data? That question can be clinically valuable for prognosis, risk communication, triage, referral, monitoring, resource allocation, trial enrichment and risk-stratified prevention. Examples include the Oxford Mental Illness and Suicide tool,[6] which estimates suicide risk in people with severe mental illness, and PsyMetRiC 2.0,[7] which estimates cardiometabolic risk in young people with psychosis spectrum disorders. Such tools can guide clinical decisions about monitoring, referral, preventive care and prioritisation while remaining prognostic models rather than estimates of the causal effect of a specific intervention.

The way forward is not to choose between prediction and causality as competing camps, but to choose the modelling approach according to the clinical question being addressed and what is practical for the clinical setting. A risk model may support risk stratification and preventive decisions under 'usual care' assumptions, but it should not be presented as answering a 'what-if' treatment question. Conversely, causal prediction may be needed for intervention-specific questions, but even a causal model can be incomplete if it considers only one intervention or a restricted subset of actionable interventions. For example, a causal model estimating the effect of a medication alone may miss better outcomes achievable through psychological therapy, physical health intervention, social support or combined care. Such restriction can lead to misaligned decisions and worse achievable outcomes even when the effects of included interventions are correctly identified and estimated.[8] Moreover, causal modelling remains difficult to operationalise because it depends on specifying an appropriate causal structure, which is often hard to establish in complex clinical settings, and on assumptions that are often untestable; ground-truth causal labels are also rarely available for validation.[9] These challenges limit the immediate clinical readiness of causal prediction models. Precision psychiatry will therefore be better served by consistently foregrounding these distinctions: what outcome is targeted, what interventions are included or omitted, what heterogeneity is captured, what assumptions are required, what is practically feasible and what claims the model can and cannot support. This preserves the legitimate contribution of risk prediction while recognising causal personalised treatment as an important, but not exclusive, goal of the field.

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Declaration of interest

None.

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