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Treating the individual: moving towards personalised eating disorder care

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Abstract

Eating disorders (EDs) are complex and heterogeneous conditions, which are often not resolved with conventional, manualised treatments. Arguments for the development of holistic, person-centred treatments accounting for individual variability have been mounting amongst researchers, clinicians and people with lived experience alike. This review explores the transformative potential of personalised medicine in ED care, emphasising the integration of precision diagnostics and tailored interventions based on individual genetic, biological, psychological and environmental profiles. Building on advancements in genomics, neurobiology, and computational technologies, it advocates for a shift from categorical diagnostic frameworks to symptom-based and dimensional approaches. The paper summarises emerging evidence supporting precision psychiatry, including the development of biomarkers, patient-reported outcomes, predictive modelling, and staging models, and discusses their application in ED research and clinical care. It highlights the utility of machine learning and idiographic statistical methods in optimising therapeutic outcomes and identifies key challenges, such as ethical considerations, scalability and implementation.

Plain english summary

Traditional eating disorder (ED) treatment approaches often use a "one-size-fits-all" method, despite the fact EDs are complex and can vary greatly from person to person. This review discusses how personalised treatment can transform care for people with EDs. Personalised care tailors treatment to each person's unique biology, mental health, and life circumstances, with the understanding that a more flexible and individualised approach could lead to better outcomes. We explore new discoveries in genetic research, machine learning, and advanced tracking methods to predict how someone might respond to specific treatments and identify what works best for them. We also emphasise the importance of addressing changes in the illness experience over time and including patients' perspectives in their care. While these approaches show great promise, challenges remain, such as ensuring we have evidence to guide effective personalisation, and that treatments are ethical, widely available and easy for clinicians to use. The paper highlights a future where ED treatments are more precise, effective, and adapted to the individual, offering new hope for recovery.

Keywords Precision psychiatry, Eating disorders, Personalised medicine, Individualised medicine, Precision diagnostics, Holistic care, Idiographic, Person-centred

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Introduction

Every person is a universe: such is the tenet of personalised medicine, which moves away from the tried and tested "one-size-fits-all" healthcare model, towards precision diagnosis and treatment based on an individual's unique genetic, biological, and environmental profile. Personalised medicine (also referred to as 'precision' or 'individualised' medicine) aims to optimise therapeutic outcomes, minimise adverse effects of unsuitable treatments, and address the complex heterogeneity of disease. The approach can be traced back to the early 20th century and the 1990s Human Genome Project, which by mapping and sequencing the entire human genome allowed for the identification of genetic markers associated with various diseases [1–3]. However, personalised medicine has swiftly evolved in recent years. Advances in multi-omics data (including genomics, proteomics, metabolomics), the emergence of molecular diagnostics, integration of computational technologies and the development of formal idiographic statistical models has enabled a more comprehensive understanding of disease mechanisms and the development of more precise diagnostic and therapeutic approaches [4]. The approach is now an integral part of many aspects of healthcare. In oncology, for instance, personalised medicine approaches have led to the development of numerous targeted therapies and immunotherapies. Additionally, the use of genetic testing in routine clinical practice has become more common, allowing for more personalised treatment plans for a wide range of diseases, including cancer, cardiovascular disease and rare genetic disorders.

The application of personalised medicine to psychiatric disorders is a newer concept, complicated by limited objective measurements and a lack of understanding of illness mechanisms. Advancements in machine learning, computational technologies and big data has the potential to change the face of psychiatry, perhaps more than any other field of medicine. Psychiatry is the only medical speciality without reliable biomarkers and thus relies heavily on self-report and psychometric instruments to furnish polythetic diagnostic classification systems, where an individual must meet a certain number of unweighted criteria in order to be categorised according to a diagnosis [5]. The necessity of diagnoses based on clusters of symptoms that ostensibly share a common cause, rather than on an obvious biomarker, means individual variability in illness profile is overlooked. Given that evidence does not support the categorical approach to psychopathology in terms of reliability, validity and clinical utility [6–9] and does not provide a clear-cut distinction between health and illness [8, 10], for some time researchers & clinicians have argued for a paradigm shift, where heterogenous symptoms that 'quantitatively deviate' from a state of health should define mental illness.

Mental disorders are systems of many different processes that interact across time to form a unique pattern of symptoms experienced by each individual [11]. At an observational level the fit of the categorical model is poor: risk factors and symptoms are continuously distributed in the population [12] and mental disorders are experienced as highly comorbid – at least 50% of people with a mental health diagnosis have more than one [13–18]. There may, therefore, be utility in thinking dimensionally and symptom-based rather than categorically when it comes to illnesses which exist on a spectrum and are largely unresponsive to current treatments [6, 7, 9, 19].

Eating disorders (EDs) is an illness group which may benefit substantially from this paradigm shift. Diagnostic classification systems for EDs are complicated by the normalisation of disordered eating behaviour in Western cultures [20–22]; an illness continuum ranging from disordered to sub-clinical to clinical eating pathology; and 'gold-standard' psychometric measures that lack specificity, are unable to measure qualitatively distinct features of illness and are poorly validated in diverse populations. More heterogeneous than they are homogenous, EDs affect a spectrum of people with diverse biological and psychological phenotypes from a myriad of sociocultural, economic and environmental backgrounds who will naturally respond differently to different treatments. Poor to modest treatment outcomes with current manualised treatments (only 31% of people with Anorexia Nervosa and 68% of those with Bulimia Nervosa will recover within 9 years) [23] suggest we do not fully understand the mechanisms of illness in EDs, and arguments for the development of holistic, person-centred treatments accounting for individual variability have been mounting amongst researchers, clinicians and people with lived experience alike. An individualised, precision medicine approach may be key to closing the translational gap currently facing psychiatric research. The individualised approach is relatively new to psychiatry and has rarely been used in EDs. Recent research in other mental illness groups including psychotic disorders, major depression and chronic pain has sought to develop client-specific outcome measures and markers of treatment progress [24–26]; machine learning prediction models for future disability in people at high-risk of psychosis or with recent-onset depression [27]; models incorporating imaging, clinical, cognitive and biological data to predict psychosis onset and outcome [28]; individualised models to examine change during treatment [29–31]; as well as bespoke individualised treatments [32–34]. Fernandez et al. (2017) conceived a semiautomated process that both develops individualised models and generates suggestions for best-fit interventions to target key symptoms of the internalising disorders (i.e., depression and anxiety)

[35]. The first trial to use this method [34] resulted in large average improvements but did not include a comparison group. Williams et al. (2016) has proposed the conceptualisation of mental disorders as disorders primarily of brain circuit dysfunction, suggesting treatments should target an individual's circuit signature (or biotype) in a granular fashion [36]. Marquand et al. (2016) introduce 'normative modelling' using Gaussian process regression to solve the wicked problem of heterogeneity in mental illness [37].

Researchers across mental health are calling for a shift in our approach [7, 38, 39] and specific methods and frameworks have been proposed. Wium-Andersen et al. (2017) identified six domains to stratify individual presentations of illness: Phenomenological features, Clinical risk factors, Biomarkers, Molecular markers, Genetic markers and Neuroimaging markers [40]. We may also extend this to include social and interpersonal domains, such as functional impairment and social difficulties. Williams et al. 2022 suggest three strategies for developing an individualised/precision approach in psychiatry:

1) *Precise classification*: understand the pathophysiology of each individual patient and develop subtype profiles that integrate biological, psychological, and experiential factors;

2) *Precise treatment planning*: craft interventions that are specifically tailored to an individual's unique clinical profile, reducing dependence on conventional trial-and-error methods or broadly applied standard treatments; and.

3) *Precise prevention*: implement individualised strategies to pre-empt the onset or progression of psychiatric disorders [41].

In this vein, the extant literature on individualised medicine in EDs can be broadly separated into four overarching categories: understanding the idiographic manifestation and maintenance of eating disorder symptoms; measuring heterogeneity in clinical presentation; characterising heterogeneity using statistical modelling and methods; and treating heterogeneity in clinical treatment trials. In this article we summarise the emerging evidence-base for personalised medicine in ED treatment and outline considerations for integrating idiographic methods in research, clinical case conceptualisation and clinical care.

Understanding heterogeneity – factors and processes

Eating disorders (EDs) have significant variability in their onset, course, and response to treatment. This variability arises from a complex interplay of biological, psychological, socio-environmental and illness factors. Breaking down this heterogeneity is the first step to a more nuanced approach to conceptualisation and individualised intervention. Prognostic considerations, including illness chronicity, severity at onset, presence of psychiatric and medical comorbidities, and prior treatment responsiveness, are critical in determining individual pathways to recovery. Staging models provide a valuable heuristic for distinguishing between early stages of illness and entrenched, refractory forms, thereby informing phase-specific interventions. Furthermore, inter-individual differences in sociocultural context and in core psychopathological processes—such as cognitive rigidity, compulsivity, emotion dysregulation, and temperament—contribute to differential treatment needs. Emerging evidence also implicates genetic predispositions, neurobiological dysregulation, metabolic alterations, and gut-brain axis interactions in shaping illness expression and treatment response.

Staging & prognostic considerations

Staging defines the extent of progression of a disease and is a concept aligned with precision psychiatry [42]. It refers to the idea that clinical presentation evolves and changes over time, from prodrome to syndrome and then on a continuum of severity (mild to severe enduring). Processes in EDs such as neuroprogression, neuroadaptation and social isolation contribute to the evolution of the disorder into the severe enduring stage [43, 44].

Understanding progression of illness is not only important for early identification and early intervention but also to understand the complex illness factors impacting treatment efficacy and acceptability, enable improvements in case management and match treatments to stage of illness [42, 45]. Staging has been proposed as a way to overcome the challenges associated with conventional diagnostic practice, in particular its poor capacity to provide meaningful information about severity or prognosis [42]. A four stage model of illness severity has been proposed for a number of mental disorders, including mood disorders [46], panic disorder [47], alcohol use disorder [48] and schizophrenia [49]. McGorry et al. (2006) suggest that the ideal staging model is clinicopathological, whereby clinical features are augmented by objective measures that link to pathophysiology (i.e., biological markers of illness) [42]. Provisional staging models have also been investigated for EDs. In accordance with other staging models in mental health, evidence supports a staging heuristic for AN, but has limited utility in BN/BED [43]. Treasure, Stein & Maguire (2015) proposed a four-stage model for EDs (comprising high-risk, early syndrome, full syndrome and severe enduring illness) for use in the management, treatment and prognosis of EDs and Maguire et al. (2017) developed an illness-specific, four-stage model of severity for AN [43, 50] [50].

Consideration of the course of an illness is integral to realising the full potential of an individualised approach. EDs almost always have a prodromal period, typically (but not always) in adolescence, with slower progression to more severe disorders (Treasure, Stein & Maguire 2015) [43]. However, some sub-threshold disorders resolve on their own. Whether or not a person goes on to have a diagnosable ED, and what type of ED that is, might be influenced by genetic or other risk factors. It follows that certain biomarkers could help to predict level of intervention, type of intervention and likely treatment response for an individual at that earliest possible opportunity [43]. There is a need for further research to empirically validate clinical staging models across EDs [51].

Other illness factors

Some of the earliest treatment trials of psychotherapy for people with EDs demonstrated the relevance of 'individualised' stratification, at that time commonly based on duration of illness. Russell et al. in 1987 [52] and then Eisler et al., 2007 [53] compared post-inpatient family-based therapy (FBT) and individual therapy at 1- & 5-years following discharge and found differential response to family therapy based on illness duration (< vs. > 3 years). Research conducted since has consistently shown that FBT is most effective when delivered to adolescents in the early stages of the illness, providing support for the tailoring of treatment to illness stage at a bare minimum, to optimise treatment outcome [43, 54, 55]. However, a 2020 systematic review and meta-analysis found that duration of illness did not influence treatment outcome [56], therefore evidence for the prognostic value of illness duration is mixed.

Other researchers have explored predictors of treatment outcome in EDs based on certain risk factors or illness features. Vall & Wade (2015) captured all types of treatment ($n = 126$ studies) in a meta-analysis and found baseline predictors such as higher BMI, fewer binge/purge behaviours, fewer psychiatric comorbidities, better interpersonal functioning, fewer familial problems, greater motivation to recover, lower depression, and lower shape/weight concern predicted better treatment outcome in people with EDs [57]. Linardon, Garcia & Brennan (2017) synthesised the results from 65 studies in their meta-analysis on predictors, moderators, and mediators of outcome following CBT [58]. Early treatment response/change was found to be a consistent mediator of better outcomes across all EDs, however, there was no strong evidence for any moderators or predictors, leading the authors to suggest that it is 'unclear how and for whom this treatment works.' In more recent years and with aforementioned technological and modelling advancements, there is a clear trend toward more granular, sophisticated personalised treatment and research approaches in the ED literature.

Sociocultural and environmental factors

Sociocultural factors significantly influence the onset, maintenance, and trajectory of EDs [59]. Western beauty ideals, social media, and family dynamics shape illness outcomes, with the internalisation of the thin ideal—reinforced by media and peer environments—linked to disordered eating and greater illness severity [60, 61]. Systematic reviews indicate that sociocultural pressures around body image, particularly in Western societies, contribute to overvaluation of weight and shape, exacerbating psychopathology and prolonging illness chronicity [62, 63]. Social comparison processes further reinforce these cognitions, particularly among adolescents and young adults who are highly susceptible to media messaging [64]. The rise of social media has intensified these effects, with platforms such as Instagram and TikTok amplifying appearance-based comparisons, normalising restrictive eating, and reinforcing perfectionism—factors directly linked to more entrenched symptomatology and poorer recovery outcomes [65, 66]. Beyond media and peer influences, family and cultural dynamics shape illness trajectory and recovery. While parental criticism, enmeshment, and weight-related teasing are associated with greater symptom severity and lower motivation for change [67–69] strong familial and social support promote treatment engagement and better psychological outcomes [70]. Sociocultural stigma further complicates help-seeking, particularly in groups where disordered eating is normalised within cultural or athletic subcultures [71, 72]. Cross-cultural research also highlights variations in symptom presentation and risk factors, emphasising the need for culturally sensitive individualised interventions [73].

Environmental factors, including trauma, bullying, school experiences, and weight stigma, further contribute to ED risk. Childhood trauma, including physical, emotional, and sexual abuse, is strongly associated with ED development, with meta-analyses showing significant links between early adversity and restrictive or binge-purge behaviours [74]. Bullying, particularly weight-related teasing, heightens body dissatisfaction and increases vulnerability to disordered eating [75]. School environments that emphasise academic or athletic achievement can reinforce perfectionistic tendencies, a known risk factor for EDs [76, 77]. Additionally, weight stigma—both interpersonal and institutional—has been linked to disordered eating, increased dietary restraint, and body image concerns, with longitudinal studies suggesting that experiences of weight-based discrimination predict greater ED severity over time [78, 79].

Biology and genomics

There is growing interest in integrating biological markers, such as genetic, neurobiological, and physiological factors, into treatment strategies, however while theoretical advancements in personalised medicine for EDs show promise, their clinical application in routine practice remains limited [80]. Most biologically-oriented personalised medicine studies in ED have focused on AN, with significant efforts directed towards understanding its genetic underpinnings [81, 82]. However, there is the potential of many other approaches including multi-omic approaches (including metabolomics and microbiome research), in identifying biomarkers that could inform more tailored treatments. These areas are still in the early stages of discovery, with more research needed to bridge the gap between bench and bedside [80, 83, 84]. Some large-scale studies using multi-omic approaches are currently underway including EDIFY (Eating Disorders: Delineating Illness and Recovery Trajectories to Inform Personalised Prevention and Early Intervention in Young People) at Kings College London [85] and Holistic Understanding at the Australian Eating Disorders Research & Translation Centre [86]. Both of these studies aim to collect a large suite of physiological and genetic data, neuroimaging, cognitive tests and psychological/social data from individuals with EDs in order to better understand risk factors, illness mechanisms and trajectories, and targets for personalised intervention.

Recent advancements in neuroscience have the potential to enhance traditional diagnostic classification systems by considering how interactions between neurobiological substrates and psychosocial variables act to modulate symptomatology. These scientific developments offer new methods to address the heterogeneity observed within and across mental disorders, thereby facilitating more nuanced and precise therapeutic decision-making. By establishing connections between clinical phenotypes and an individual's unique biosignature (or biotype), these insights may allow for the customisation and selection of therapeutic interventions that specifically target distinct clinical features and biotypes. Such interventions may encompass a diverse array of therapeutic options, including pharmacological agents, behavioural therapies, neuromodulation techniques, and innovative therapeutic approaches, all of which hold the potential to modify the underlying biological mechanisms that give rise to symptoms and phenotypes [41].

There are multiple precision medicine approaches to EDs incorporating ongoing work in metabolic and psychiatric genomics [82, 83, 87]. This includes application of Genome-Wide Association Study (GWAS) findings to clinical assessment and treatment targeting; the identification of implicated biological pathways that may inform staging and other illness models; targeted prevention and tailored intervention; development of pharmacologic agents that target the core biology of the illnesses; and the use of polygenic risk scores to predict developmental phenotypic manifestations of illness and gene–environment associations to inform risk assessment [82, 83]. Hubel et al. (2021) argue that the heterogeneity inherent in genetic profiles among individuals necessitates a more nuanced approach to treatment—one that tailors therapeutic interventions to the genetic and phenotypic idiosyncrasies of each patient [88].

Adams et al. (2023) explored the genetic architecture of AN through an innovative multi-omic approach that prioritised risk genes by integrating different data modalities across 14 tissue types [89]. Employing a combination of transcriptome-wide association studies (TWAS), proteome-wide association studies (PWAS), and splicing association analyses (spliceWAS), the research identified 134 genes with genetically predicted mRNA expression associated with AN. Fine-mapping analysis highlighted an overrepresentation in the pathway associated with immune system regulation, specifically involving the genes MST1, TREX1, PRKAR2A and PROS1. WDR6, which is implicated in cell growth, proliferation and autophagy, emerged as a particularly salient candidate gene. This comprehensive analytical framework not only refines our understanding of AN's genetic landscape but also illuminates the potential causal role of immune system pathways in its pathogenesis, underscoring the utility of multi-omic data in disentangling the biological complexity of EDs [89].

Burstein et al. (2023) undertook a genome-wide investigation into the genetic determinants of binge eating, employing a model derived from a binge eating phenotype [90]. They identified several loci associated with binge eating behaviours. The findings contribute to a more nuanced understanding of the genetic predispositions underlying these behaviours, suggesting a potential overlap in the genetic risk factors for binge eating and anorexia nervosa. Research in this area extends our understanding of the shared and unique genetic underpinnings of various eating disorder phenotypes [90]. By identifying the neurobiological pathways underlying genetic variations—such as reward system dysregulation (dopaminergic pathways), impulsivity and executive dysfunction (serotonergic regulation), or metabolic and appetite control deficits (leptin and insulin signalling)—genetic findings could facilitate a more refined classification of ED subtypes. This, in turn, has significant implications for treatment stratification, as pharmacological interventions could be tailored to specific neurobiological profiles, with dopaminergic agents addressing compulsive overeating, serotonergic medications targeting emotional dysregulation, and metabolic-targeting drugs modulating appetite dysregulation. Likewise,

psychological interventions could be personalised, with cognitive-behavioural therapies being prioritised for those with cognitive control deficits and metabolic-focused interventions being more appropriate for individuals with genetically driven metabolic impairments.

Johnson et al. (2023) examined the translational potential of genetic findings in AN by mapping identified genetic variants to specific clinical phenotypes [91]. The study correlates these genetic markers with distinct clinical traits, such as body mass index (BMI), anxiety, and obsessive-compulsive behaviours, bridging the gap between genetic susceptibility and observable clinical presentations, and demonstrating the critical importance of elucidating the genotype-phenotype nexus in the pursuit of personalised therapeutic strategies [91].

Measuring heterogeneity – the importance of comprehensive data collection

The systematic collection and analysis of psychometric and outcome measures are essential for elucidating the heterogeneity of mental illness. Consistent assessments provide a means to systematically capture this complexity by identifying distinct symptom clusters through data-driven clustering techniques. By leveraging advanced statistical and machine learning methods, researchers can move beyond homogenous diagnostic categories and delineate biologically or psychologically meaningful subtypes that may respond differentially to treatment [92]. Such approaches also enable the distinction between core psychopathological features and more peripheral or co-occurring symptoms, refining theoretical models of illness and allowing for the identification of specific factors driving symptomatology. By providing empirically derived subtypes and treatment response profiles, data-driven clustering enhances the precision of clinical decision-making, ensuring that interventions are tailored to the specific needs of individual patients. When integrated with biological, neurocognitive, and behavioural data, these measures further contribute to mechanism-based treatment approaches, supporting the broader aims of precision psychiatry. For instance, if interoceptive deficits emerge as a key factor influencing treatment response in AN, interventions targeting bodily awareness could be prioritised for individuals exhibiting this phenotype. Clinically, the longitudinal monitoring of psychometric and outcome measures may foster a more dynamic and responsive approach to care, moving away from rigid, one-size-fits-all treatment models toward individualised, empirically informed interventions that optimise therapeutic efficacy and improve patient outcomes.

Routine outcome measures

The International Consortium for Health Outcomes Measurement (ICHOM) has developed a comprehensive set of patient-centred outcome measures specifically for EDs [93]. This initiative involved collaboration among leading clinicians, researchers, and individuals with lived experience to identify outcomes that hold the greatest significance for patients. The standardised set encompasses various domains, including ED symptoms (assessed via instruments such as the Eating Disorder Examination Questionnaire for individuals aged 15 and above, and the Children's Eating Attitudes Test for those aged 8–14), symptoms of avoidant/restrictive food intake disorder (ARFID), co-occurring mental health conditions (such as depression, anxiety, and suicidal ideation), and quality of life and social functioning. By implementing these standardised measures, healthcare providers can systematically evaluate treatment outcomes, thereby facilitating the identification of effective, personalised interventions for diverse patient populations.

In Australia, the absence of a nationally comprehensive and consistent dataset for EDs has historically impeded the ability to monitor treatment outcomes and inform service delivery [94]. To address this gap, the Inside-Out Institute for Eating Disorders, with support from the Australian Government Department of Health, has developed a national minimum dataset (MDS) for EDs [94]. This MDS was established through a consensus-driven process involving a diverse group of stakeholders, including clinicians, researchers, and individuals with lived experience. The implementation of this standardised dataset aims to enhance the understanding of patient presentations and treatment outcomes across various healthcare settings, thereby supporting the development of tailored interventions and improving the quality of care for individuals with EDs in Australia.

Personalised outcome measures

The Psychological Outcome Profiles (PSYCHLOPS) is the only patient-generated measure of psychological distress and therapeutic change that has been validated in EDs. Unlike standardised patient-reported outcome measures, PSYCHLOPS allows individuals to identify and rate their own key problems, providing a personalised and responsive assessment of mental health outcomes. It is commonly used in therapy and research settings to capture subjective experiences of psychological well-being and treatment progress. The measure consists of three main sections: problems, functioning, and well-being, with an additional section for post-treatment reflection. Austin et al. (2021) tested the efficacy and validity of the personalised patient-reported outcome measure (PROM) in the context of ED treatment [95]. The study involved 278 emerging adults with first episode eating disorders, using PSYCHLOPS alongside two standard ED PROMs, the Eating Disorder Examination Questionnaire (EDE-Q) and the Clinical Impairment Assessment (CIA), over

a 12-month period. The authors found that PSYCHLOPS exhibited satisfactory psychometric properties, including adequate internal reliability (Cronbach's alpha ranging between 0.73 and 0.84) and strong concurrent validity (Spearman's coefficient of -0.69, $p < 0.01$) when compared to patient-reported recovery [95], echoing the findings of previous validations in primary care settings [96]. More than half the participants identified concerns not captured by the EDE-Q or CIA, particularly issues related to depression, anxiety, academic problems, and treatment concerns, highlighting the limitations of standardised PROMs in fully addressing nuance and underscoring the importance of personalised PROMs for providing a more comprehensive assessment of a person's needs [95].

Characterising heterogeneity – data, models and methods

Multilevel models, which are currently the most common model used for the study of within-person processes in psychology, are not truly person specific or idiographic. While these models can capture within-person variations and account for individual differences through random effects, they rely on estimating an average fixed effect along with its variance. Individualised, or personalised models are truly idiographic as they allow each person's parameter values to be entirely independent of those from other participants in the sample [97].

There are several idiographic modelling techniques designed to personalise the study of psychopathology, most of which involve intensive longitudinal data (i.e., time-series data collected from a single individual). Among the most widely used are variations of vector autoregression models (VAR). Examples include Unified Structural Equation Modelling (uSEM), which integrates structural equation modelling with VAR, Graphical VAR, which merges VAR with Gaussian graphical models, and Dynamic Structural Equation Modelling (DSEM), which combines structural equation modelling, multilevel modelling, and time-series analysis.

Other commonly used methods include the p-technique, which uncovers the dynamic structure (such as the number of factors and the pattern of factor loadings) of an individual's multivariate time series data while keeping factor associations contemporaneous rather than lagged. Dynamic Factor Analysis (DFA) expands on the p-technique by incorporating structural paths across waves. Lastly, network analysis treats the variables being studied as an interconnected system. Each of these methods increases the flexibility of the modelling process (e.g., contemporaneous associations, structured residuals) and enhances the potential informational yield from multivariate time series data [97]. Some of the more common models and methods employed thus far in ED treatment are discussed below.

The network approach

Complex network approaches, which are currently being developed at the crossroads of various scientific fields, have the potential to provide a way of thinking about disorders that does justice to their complex organisation. In such approaches, disorders are conceptualised as systems of causally connected symptoms rather than as effects of a latent disorder. Using network analysis techniques, such systems can be represented, analysed and studied in their full complexity. The study of network structures thus yields several new possibilities to go beyond the conventional classification of psychiatric disorders. This may be especially helpful for the study of the interaction of phenotype, neural development, environmental input and behaviour [98].

Roefs et al. (2022) outline a network-based framework for understanding and treating mental disorders, recognising that they are constituted by dynamically interacting symptoms and other relevant variables, which together form a complex network unique to each individual [7]. The framework is based on three interconnected pillars: *mapping dynamic networks* across a large population with a variety of mental disorders (to capture the full spectrum of symptoms and their interactions) using methods like EMA (which allows for the capture of real-time data on how symptoms fluctuate and interact over time); *zooming into causal mechanisms* that underlie the relationships between elements in these networks (using experimental and pre-clinical studies to identify transdiagnostic processes that drive the interactions within these networks); and *targeting interventions*, or developing and testing personalised, network-informed interventions, and then comparing the effectiveness of these network-based interventions against traditional, evidence-based treatments to assess their efficacy [7].

There are of course challenges within the Network approach, including the selection of variables for EMA, considerations of statistical power in network analyses, and the practicalities of implementing network-informed interventions in clinical practice. There is also a need to train therapists in using these network models to guide treatment, which would be a significant shift from current therapeutic practices. But researchers argue that this approach could lead to better long-term outcomes for patients and a more efficient allocation of resources within the mental health system [7].

Several researchers have used the network approach in EDs, typically to estimate centrality or the core features or symptoms of ED psychopathology using between-person networks [99–104]. Christian et al., 2020, observed that even where 'central' symptoms were able to be identified, significant differences in network structure (i.e., how symptom pathways are connected) emerged across age groups. The authors suggested that even where

symptom severity does not increase over time, symptom interconnectivity might [105].

Levinson (2020, 2021) has conducted numerous studies examining idiographic networks using time-series data such as EMA (ecological momentary assessment). In 2020, her group conducted longitudinal group-level (*addressing symptom interrelations across people and across time; between-subjects, contemporaneous, and temporal networks*) and intra-individual (*symptom interrelations within each person and across time; contemporaneous and temporal networks*) network analyses using prospective 4-year data in at-risk adolescents and young adults, finding that individual networks were highly variable across individuals [106].

Predictive modelling & machine learning

Predictive modelling in psychiatric research leverages advanced statistical and machine learning (ML) techniques to forecast mental health outcomes, personalise treatment, and enhance clinical decision-making. Until now, predictions in psychiatry have been constrained by the limitations of linear models and subjective clinical judgement, which often fall short in addressing the complexity and variability intrinsic to psychiatric disorders [107]. With the advent of predictive modelling, researchers can now interrogate vast, multidimensional datasets—encompassing genetic, neuroimaging, ecological momentary assessment (EMA), and electronic health records— allowing for the identification of intricate patterns and individualised risk factors. This approach not only enhances the precision of predictions related to disease progression and therapeutic outcomes but also supports the development of personalised treatments tailored to the specific needs of individual patients.

Researchers have begun to explore predictive modelling in EDs. With its ability to handle large datasets and complex variable interactions, ML could significantly improve predictions of long-term outcome in EDs [108]. Haynos et al. (2021) conducted a longitudinal analysis of 415 females with full or subthreshold ED diagnoses, comparing ML-based elastic net regularised logistic regression to traditional logistic regression in predicting ED outcome at one- and two-year follow-ups. The ML models demonstrated superior predictive accuracy (AUC = 0.78 vs. 0.67), with results remaining robust even when key predictors were removed or alternative ML techniques were applied. Key predictors included baseline ED diagnosis (especially bulimia nervosa), psychological factors such as dietary restraint and shape concern, and clinical variables like psychiatric hospitalisation history and lower baseline BMI, which strongly predicted underweight status at follow-up. These findings highlight the promise of ML in refining psychiatric prognosis, enabling more precise risk assessment and personalised intervention strategies. The study advocates for further research integrating multimodal and real-time data to enhance predictive accuracy and clinical applicability.

Anderson et al. (2023) looked at the potential of causal discovery analysis (CDA) as a novel tool for advancing precision medicine in the treatment of EDs, arguing that CDA may provide fine-grained, individualised models of causal relations among symptoms and behaviours, generating person-specific models of psychopathology [109]. Unlike traditional statistical methods that often focus on correlations, CDA aims to uncover the underlying causal structures that govern relationships between variables. They presented a series of case studies where CDA was applied to real-world data, demonstrating its utility in developing personalised treatment models, and found that CDA could successfully identify individual-specific causal pathways that maintain ED psychopathology. For example, in one case study, CDA revealed that negative affect was a direct cause of binge eating in some individuals, suggesting that targeting emotion regulation could be particularly effective for these people [109].

Arend et al. 2023 employed a correlation-based ML approach using idiographic item subsets collected through Ecological Momentary Assessment to predict binge eating episodes on an individual level. EMA was used to gather real-time data on participants' emotions, thoughts, and behaviours [110]. The idiographic item subsets demonstrated a high degree of predictive accuracy for binge-eating episodes, achieving a mean area under the curve (AUC) of 0.80 across a sample of 13 patients. In several cases, sensitivity approached 100%, successfully identifying every reported binge episode while maintaining a relatively high level of specificity—that is, accurately predicting the absence of binge episodes when none occurred. The authors noted that predicting binge-eating episodes based on psychological and contextual states was very feasible and precise, underscoring the idiographic nature of these predictor sets and supporting a paradigmatic shift from exclusively nomothetic models toward idiographic approaches in both prediction models and theoretical conceptualisations [110].

ML has demonstrated considerable promise in psychiatry, particularly for atheoretical prediction, enabling the identification of absolute risk (e.g., poor prognosis) and relative risk (e.g., momentary binge-eating vulnerability) with high accuracy. By leveraging complex, high-dimensional patterns, ML can enhance early detection and intervention strategies, such as predicting chronicity in EDs or utilising digital phenotyping to monitor real-time fluctuations in ED risk [111]. However, despite its predictive power, ML is inherently correlational rather than mechanistic, often functioning as a "black box"

that captures nonlinear associations without elucidating causal pathways [112]. This limitation is particularly relevant for identifying individual mechanistic targets, as ML alone cannot disentangle whether observed risk markers are causal drivers of binge eating or merely downstream correlates of broader psychopathology. To address this, future research should integrate ML within multimodal frameworks that incorporate causal inference methodologies and theory-driven experimental approaches, such as computational psychiatry, to bridge the gap between predictive modelling and mechanistic understanding. Such an approach would optimise the clinical utility of ML, ensuring that its predictive capabilities translate into more precise, mechanistically informed interventions in eating disorders.

Espel-Huynh et al. (2021) examined whether ML models could better predict ED treatment response trajectories than a simpler multinomial logistic regression approach. Researchers analysed data from 333 women in residential ED treatment, identifying three trajectories: Rapid Response (24%), Gradual Response (58%), and Low-Symptom Static Response (18%), using self-reported assessments from the first two weeks of treatment. The best ML model (radial support vector machine) achieved an AUC of 0.94, while logistic regression performed nearly identically (AUC of 0.93), indicating no meaningful advantage of ML. The most predictive variables—baseline symptom severity, percent change in symptoms, and early trajectory slope—suggest that simple early markers are sufficient for prediction. Given the negligible performance difference, logistic regression may be preferable for its simplicity, interpretability, and lower computational burden. While early trajectory prediction could help tailor treatment, ML did not enhance predictive accuracy in this context. The study is limited by its focus on female patients in a specific setting, but overall, it suggests that logistic regression remains a highly effective tool for predicting ED treatment response.

Krug et al. (2023) compared ML techniques to conventional logistic regression in predicting ED onset and diagnostic differentiation between AN and bulimia nervosa (BN), finding that while ML approaches did not surpass logistic regression in predictive accuracy, they produced more parsimonious models that may refine screening and early intervention efforts [113]. Similarly, Forrest et al. (2023) investigated the predictive value of ML models in forecasting treatment outcomes for binge-eating disorder (BED), concluding that neither traditional regression nor ML approaches provided consistently robust predictions, though ML models offered marginal improvements in handling complex data structures [114]. In contrast, Svendsen et al. (2023) demonstrated the utility of ML in predicting non-response to ED treatment using privacy-preserving synthetic data, showing that Random Forest models reduced classification errors by 31.3%, though additional data from later treatment stages yielded only minor gains in predictive accuracy [115].

Monaco et al. (2024) have introduced an Artificial Intelligence Platform aimed at addressing the challenges in treating EDs [116]. The primary aim of the study is to develop a personalised treatment platform, referred to as the Master Data Platform (MDP), that leverages AI to improve patient outcomes and provide real-time, data-driven insights into ED treatment. The platform will integrate data from various sources—such as clinical assessments, patient demographics, and physiological data—using AI algorithms to identify risk factors, plan treatment strategies, and predict relapse risks. Additionally, the platform includes a chatbot to engage with patients, providing educational support and guidance throughout the recovery process [116]. The platform utilises a combination of machine learning algorithms and deep learning architectures to process large-scale datasets. These datasets include neuroimaging data, social and behavioural information, and patient self-reports. The use of Natural Language Processing (NLP) models allows the system to analyse patient diaries and social media interactions, which can reveal critical insights into a patient's mental state, adherence to treatment, and potential relapse risks [116]. It also integrates sensors and smart devices (e.g., smartwatches) to collect physiological data, such as heart rate and sleep patterns, which are then analysed by AI to monitor patient progress and adjust treatment plans accordingly. Furthermore, AI decision-support algorithms aid clinicians in making informed treatment decisions, reducing the likelihood of operator-dependent errors [116].

Treating heterogeneity – exploring personalised treatment

Existing clinical treatment trials

Traditional treatment models typically follow standardised protocols and manuals, which cannot account for the unique biological, psychological, and environmental factors influencing a person's illness. Some clinical treatments incorporating basic personalisation have been trialled, providing early data on how the approach can improve patient engagement, symptom reduction, and recovery rates.

McFarlane et al. (2015) investigated the effectiveness of an individualised day hospital (DH) treatment program for individuals at Toronto General Hospital. The study compared 655 patients (standard group treatment $n = 446$, individualised treatment $n = 209$) who were admitted between 2007 and 2014 [117]. The individualised program incorporated two individualised therapy sessions per week alongside structured independent homework, aiming to better accommodate the unique

needs of participants. These one-on-one cognitive-behavioural sessions were designed to address barriers impeding progress in symptom cessation as well as subtle maladaptive eating behaviours, and to facilitate in-depth exploration and resolution of body image disturbances. People were assigned to the individualised treatment on clinical recommendation. They were typically older, had a longer duration of illness and had engaged with treatment more often than those in the standard treatment group. Individualised patients also had more severe psychopathology, including higher levels of depression, anxiety, and obsessive-compulsive symptoms. They were more likely to be diagnosed with AN-Binge Purge subtype (AN-BP) or Eating Disorders Not Otherwise Specified EDNOS (now Other Specified Feeding or Eating Disorder OSFED) [117]. Despite these challenges, individualised patients had comparable retention rates to standard patients, with no significant differences in premature discharge. The duration of treatment was slightly shorter for individualised patients, but the groups had similar rates of rapid response to treatment (defined as achieving significant symptom reduction within the first four weeks). This suggests even patients with more complex presentations can respond well to treatment when individualised care is provided [117]. Both groups experienced substantial reductions in ED symptoms, including binge eating and vomiting. However, patients in the individualised group were less likely to achieve complete abstinence from these symptoms by the end of treatment, particularly regarding vomiting. Additionally, patients in the individualised group who required weight gain were less likely to achieve a healthy post-treatment body mass index (BMI) compared to those undergoing standard treatment. Research shows that patients with more severe EDs and comorbid conditions often experience residual symptoms at the end of treatment, leaving them vulnerable to relapse, therefore this may not have been a result of the individualised protocol.

Haynos et al. (2016) compared standardised and individualised caloric prescriptions in inpatient weight restoration for Anorexia Nervosa (AN) [118]. In a natural experiment ($n = 70$), they found that the standardised approach led to faster, curvilinear weight gain (peaking at ~4.5 lbs/week in week three) compared to the gradual, linear gains of the individualised approach (~3.5 lbs/week in week four). By one month, the standardised group had gained two additional pounds on average. While hospitalisation length and discharge BMI were similar, activity restriction (e.g., bedrest) was 30% lower in the standardised group, suggesting greater efficiency. No cases of refeeding syndrome requiring medical intervention were observed, challenging the traditional "start low, advance slow" model. Thus, when it comes to nutritional rehabilitation at least, a standardised approach may optimise weight restoration within the constraints of short inpatient stays. However, it is important to note the researchers did not assess biochemical markers of refeeding syndrome nor did they collect qualitative data on tolerability [118].

The individualised treatment approach described by McFarlane et al. (2015) is distinct from the conventional use of clinical intuition in routine practice, as it is systematically guided by empirical principles and tailored to individual patient data. In their study, the researchers implemented an adaptive treatment strategy, wherein the therapeutic interventions were dynamically adjusted based on patient response, rather than relying on a fixed, standardised protocol. This approach aligns with a precision medicine framework, emphasising iterative decision-making informed by measurable clinical outcomes (DeRubeis et al., 2014). While it is true that individualised treatment is common in clinical settings, such practices are often based on heuristics and subjective judgment rather than structured, evidence-informed modifications. Indeed, research indicates that most individuals receiving treatment for psychological disorders do not benefit from empirically supported interventions, as clinicians frequently deviate from best-practice guidelines in favour of intuitive decision-making (Waller & Turner, 2016). The model described by McFarlane et al. (2015) differs by explicitly integrating empirical data into treatment personalisation, ensuring that therapeutic adaptations are systematically evaluated rather than based on clinician preference alone. This distinction is critical, as it underscores the contrast between the individualised, data-driven methodology under discussion and the prevailing treatment-as-usual paradigm, which often lacks empirical grounding.

Levinson et al. (2021) collected data via ecological momentary assessment (5 times per day for 15 days – or 75 total measurement points) to investigate the best methods for informing the selection of personalised treatment targets using idiographic network analysis [119]. The research showed how symptom assessment, symptom selection, model type and statistic could influence which targets are selected for treatment, and how targets could influence treatment planning and ordering of interventions. Treatment targets and symptom profiles were highly heterogenous, with less than 50% of individuals endorsing central symptoms related to weight and shape. No single target was identified as most important, highlighting the need for individualised treatment methods. The heterogenous manifestation of symptoms within one construct (e.g., body dissatisfaction, drive for thinness, fear of weight gain performing as different manifestations of overvaluation of weight and shape) found by the study demonstrates the need to tailor or focus

treatments on symptoms rather than global pathological constructs [119].

The same team is now conducting a pilot randomised controlled trial comparing 20 sessions of transdiagnostic network-informed personalised treatment (T-NIPT-ED) with 20 sessions of CBT-E over 20 weeks [120]. The study incorporates ecological momentary assessment (EMA) to gather intensive longitudinal data for NA and generate idiographic networks to identify and target core symptoms that maintain ED pathology within individuals [120].

Challenges inherent in the individualised approach

The personalised approach to the study of psychopathology faces similar challenges to other intensive longitudinal research, including issues with low temporal resolution in our theories, making it difficult to identify the timescale on which the relevant processes occur; complications in linking variables that happen at different times and on varying timescales throughout the day; and having to make decisions around the intensity and duration of assessments necessary to create a dependable individual model [97]. Homogenous psychometric instruments that lack specificity and lack of standardisation of these instruments in routine clinical practice, further complicates matters.

Consideration of whether individualised psychopathology models will capture idiosyncratic manifestations of the same pathology ('surface personalisation') or truly individualised processes ('deep personalisation') will be important [97]. These models need to be validated and directly tested against established approaches (e.g., cross-sectional self-report) / TAU (comparator groups) [121, 122]. Barriers to the widespread adoption of idiographic research include concerns over generalisability and the challenges associated with scaling such research to large participant samples. While personalised psychopathology models lack generalisability by design, being tailored to an individual's unique processes is precisely what makes them valuable. However, personalised models also offer a more direct and potentially accurate measurement of contextualised dynamic processes, which could serve as foundational components for constructing bottom-up models of psychopathology that are generalisable [97]. Ideally, this approach would enable the estimation of person-specific models across large participant groups to identify reliable, shared characteristics [97].

Use of self-reported data (as is most common in methods like EMA) can introduce biases, as participants may underreport or overreport their experiences, however this has long been a challenge in psychiatric research [123–125]. Additionally, generalisability of findings may be limited due to the small and specific sample sizes required for idiopathic research [110].

The complexity of developing and integrating individualised care platforms into existing healthcare systems cannot be underestimated. Technical issues, such as data interoperability and cybersecurity concerns must be addressed to ensure successful implementation. This may be mitigated by the advancement of AI, which offers powerful tools for data analysis and decision-making, but equally, over-reliance on AI could introduce vulnerabilities such as system failures or biases within algorithms. This may compromise patient care [116].

Further, there are ethical and safety challenges inherent in the individualised model/approach. Researchers and clinicians must hold responsibility for ensuring transparency and generalisability of individualised models; consider the appropriate communication of risk estimates; ensure data protection and privacy; and foster the equitable distribution of mental health care regardless of severity, risk, stage or phenotype [126].

In our eagerness to reconceptualise early or staged interventions, it will be essential to balance the tension between early intervention and safety. The development of individualised models must carefully consider both their potential applications and misapplications [45]. There's a risk of misapplying models and treatments suited for more advanced stages to earlier stages of illness, and vice versa [126]. Additionally, the use of staging and individualised models may over-pathologise an already highly stigmatised mental disorder. People might face discrimination based on perceived "severity," risk, biotype, or genetic profile. For example, individuals with Stage 1 anorexia nervosa (AN) could be denied treatment for being "too mild," whilst those with Stage 4 might be refused treatment due to their "intractability" or difficulty to treat. Ongoing work in this area will need to be approached with great care and sensitivity to these concerns.

Another critical issue is the ethical implications of using both AI and continuous psychophysiological measurements in mental health care [110]. There is a need for strong data governance to protect patient privacy related to consent, data ownership, and the potential misuse of sensitive health information [116].

In sum, emerging individualised approaches to conceptualising and treating EDs while promising, remain in the early stages of development, and their superiority over existing methodologies has yet to be empirically established. It is crucial to recognise that demonstrating incremental improvements in predictive modelling, such as enhancements achieved through machine learning over traditional regression techniques, does not necessarily translate into meaningful advancements in clinical decision-making. Moreover, despite the data-driven nature of these approaches, human judgment remains integral to their development and application. Researchers

play a critical role in selecting variables for inclusion in machine learning, network and biological models, inherently shaping the scope of analysis based on existing theoretical and empirical perspectives. This subjectivity raises the risk that clinically relevant factors lying outside of prevailing research paradigms may be overlooked. Additionally, the quality and completeness of input data vary significantly across studies, introducing further constraints on the reliability and generalisability of findings derived from these methodologies. Acknowledging these limitations is essential to ensuring that the enthusiasm for novel approaches is tempered with critical appraisal of their practical utility and epistemological constraints.

Conclusions and future research

The history of personalised medicine reflects a gradual but significant shift from a "one-size-fits-all" approach to a more nuanced understanding of individual variability in health and disease. From early discoveries in pharmacogenetics to the current era of genomics and big data, personalised medicine aims to provide more effective and tailored treatments for patients. This may be revolutionary in EDs, where current understandings and treatments are limited [38, 127, 128]. Deepening our understanding of illness and mechanisms of disease/change as well as developing more comprehensive multi-axial assessment measures will require detailed profiling, phenotyping and genotyping of illness using a mixed-methods approach marrying biological, neurological, genetic, psychological, social and environmental factors. Defining and measuring constructs should be conducted through a co-production process and treatment tailored in a data-driven way (development of novel measures and novel treatments could occur in parallel, with adaptations made according to response). The study of critical transitions of illness can also be facilitated by longitudinal profiling of bio-psycho-social risk, prodromal factors and how features of illness change/respond to treatment.

To advance precision ED research, it will be imperative to establish clinically relevant quantitative metrics, standardised psychometric instruments that reflect these and normative data. Concurrently, there is a pressing need for multisite, longitudinal cohort studies that adhere to standardised protocols within clinical settings, incorporating diverse measurement modalities. These studies should also provide the ability to assess the potential of precision psychiatry tools to guide prospective treatment assignments [41]. Equally critical is the creation of an infrastructure that supports the simultaneous investigation of a broader spectrum of therapeutic interventions using the same set of measures and precision psychometric instruments. The conventional research paradigm, which typically funds studies focusing on a single treatment in isolation, limits the ability to perform direct comparisons across different interventions. As a result, there remains a significant gap in identifying which measures and tools are uniquely tailored to specific treatments, indications, or biotypes [41]. Furthermore, rigorous examination of the impact of model specification decisions on inferential outcomes is required, alongside the development and dissemination of nonstationary models that do not presuppose uniformity over time [97]. The feasibility and acceptability of individualised modelling procedures will need to be assessed for both patients and clinicians, as this will play a crucial role in the successful implementation of health system change [97]. Funding model changes and a new strategic direction is required at a government level, evidenced so far by such initiatives as the National Institute of Mental Health launching of the Research Domain Criteria and Precision Medicine Initiative in the US.

Individualised models, which demand substantial resources in terms of data, time, and analytical complexity, must be rendered accessible and interpretable to clinicians who may not possess expertise in these specialised areas [129]. To achieve the widespread implementation of personalised treatments for ED, it is essential for idiographic researchers to collaborate closely with clinicians, software developers, and engineers. This interdisciplinary effort should focus on designing an intuitive and practical system capable of translating idiographic models into a format that is easily comprehensible and seamlessly integrated into clinical practice, while also incorporating valuable input from clinicians to ensure its efficacy and relevance [35, 119].

Currently, no standard personalised treatments have been successfully implemented in clinical settings for EDs, reflecting a gap between theoretical models and practical application [80]. To ensure that innovative ED treatments addressing nutrition, metabolism, psychopathology, pharmacology and related clinical areas are grounded in evidence rather than driven by unsubstantiated novelty or hype [87, 126], it will be imperative to establish rigorous, standardised measures for methods. Additionally, we must look at the complexities of nonlinear dynamic interactions within network models and the like and continue to critically interrogate theoretical frameworks of EDs [119, 130].

Author contributions

EB: conceptualisation, methodology, formal analysis, writing—original draft; PM, ST, SM: conceptualisation, writing—review and editing, supervision; KG, SBo, MP, SBa: writing—review and editing.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Human ethics and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
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References

1. Collins FS, Morgan M, Patrinos A. The human genome project: lessons from Large-Scale biology. Science. 2003;300(5617):286–90.
2. Venter JC, Adams MD, Myers EW, Li PW, Mural RJ, Sutton GG, et al. The sequence of the human genome. Science. 2001;291(5507):1304–51.
3. Garrod A. The incidence of alkaptonuria: a study in chemical individuality. Lancet. 1902;li:1616–20.
4. Mirnezami R, Nicholson J, Darzi A. Preparing for precision medicine. N Engl J Med. 2012;366(6):489–91.
5. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. Vol. 5. 2013.
6. Holmes EA, Craske MG, Graybiel AM. Psychological treatments: A call for mental-health science. Nature. 2014;511(7509):287–9.
7. Roefs A, Fried EI, Kindt M, Martijn C, Elzinga B, Evers AWM, et al. A new science of mental disorders: using personalised, transdiagnostic, dynamical systems to understand, model, diagnose and treat psychopathology. Behav Res Ther. 2022;153:104096.
8. Hyman SE. Psychiatric disorders: grounded in human biology but not natural kinds. Perspect Biol Med. 2021;64(1):6–28.
9. Kapadia M, Desai M, Parikh R. Fractures in the framework: limitations of classification systems in psychiatry. Dialogues Clin Neurosci. 2020;22(1):17–26.
10. Borsboom D, Cramer AOJ, Kalis A. Brain disorders? Not really: why network structures block reductionism in psychopathology research. Behav Brain Sci. 2018;42:e2.
11. Durstewitz D, Huys QJM, Koppe G. Psychiatric Illnesses as Disorders of Network Dynamics [Internet]. arXiv; 2018 [cited 2025 Feb 15]. Available from: http://arxiv.org/abs/1809.06303
12. Taylor MJ, Martin J, Lu Y, Brikell I, Lundström S, Larsson H, et al. Association of genetic risk factors for psychiatric disorders and traits of these disorders in a Swedish population twin sample. JAMA Psychiatry. 2019;76(3):280–9.
13. Lilienfeld SO. The research domain criteria (RDoC): an analysis of methodological and conceptual challenges. Behav Res Ther. 2014;62:129–39.
14. Cramer AOJ, Waldorp LJ, van der Maas HLJ. Comorbidity: A network perspective. Behav Brain Sci. 2010;33(2–3):137–50.
15. Kessler RC, Chiu WT, Demler O, Merikangas KR, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National comorbidity survey replication. Arch Gen Psychiatry. 2005;62(6):617–27.
16. Kim H, Eaton NR. The hierarchical structure of common mental disorders: connecting multiple levels of comorbidity, bifactor models, and predictive validity. J Abnorm Psychol. 2015;124(4):1064–78.
17. Nolen-Hoeksema S, Watkins ER. A heuristic for developing transdiagnostic models of psychopathology: explaining multifinality and divergent trajectories. Perspect Psychol Sci J Assoc Psychol Sci. 2011;6(6):589–609.
18. Sauer-Zavala S, Gutner CA, Farchione TJ, Boettcher HT, Bullis JR, Barlow DH. Current definitions of transdiagnostic in treatment development: A search for consensus. Behav Ther. 2017;48(1):128–38.
19. Layard R, Clark DM, Thrive. How better mental health care transforms lives and saves money. Princeton University Press; 2015. p. 390.
20. Hesse-Biber S, Leavy P, Quinn CE, Zoino J. The mass marketing of disordered eating and eating disorders: the social psychology of women, thinness and culture. Womens Stud Int Forum. 2006;29(2):208–24.
21. Brunner R, Resch F. Dieting Behavior and Body Image in Societal Change. In: Herpertz S, de Zwaan M, Zipfel S, editors. Handbook of Eating Disorders and Obesity [Internet]. Berlin, Heidelberg: Springer; 2024 [cited 2024 Nov 28]. pp. 9–14. Available from: https://doi.org/10.1007/978-3-662-67662-2_2
22. Faw MH, Davidson K, Hogan L, Thomas K. Corumination, diet culture, intuitive eating, and body dissatisfaction among young adult women. Pers Relatsh. 2021;28(2):406–26.
23. Eddy KT, Tabri N, Thomas JJ, Murray HB, Keshaviah A, Hastings E, et al. Recovery from anorexia nervosa and bulimia nervosa at 22-Year Follow-Up. J Clin Psychiatry. 2017;78(02):184–9.
24. Jones S, Hurrell E. A single case experimental design: how do different psychological outcome measures capture the experience of a client undergoing CBT for chronic pain. Br J Pain. 2019;13(1):6–12.
25. Kaiser T, Laireiter AR. Process-symptom-bridges in psychotherapy: an idiographic network approach. J Pers-Oriented Res. 2018;4(2):49–62.
26. Smith JD, Eichler WC, Norman KR, Smith SR. The effectiveness of collaborative/therapeutic assessment for psychotherapy consultation: A pragmatic replicated Single-Case study. J Pers Assess. 2015;97(3):261–70.
27. Koutsouleris N, Kambeitz-Ilankovic L, Ruhrmann S, Rosen M, Ruef A, Dwyer DB, et al. Prediction models of functional outcomes for individuals in the clinical High-Risk state for psychosis or with Recent-Onset depression: A multimodal, multisite machine learning analysis. JAMA Psychiatry. 2018;75(11):1156–72.
28. Tognin S, van Hell HH, Merritt K, Winter-van Rossum I, Bossong MG, Kempton MJ, et al. Towards precision medicine in psychosis: benefits and challenges of multimodal multicenter Studies—PSYSCAN: translating neuroimaging findings from research into clinical practice. Schizophr Bull. 2020;46(2):432–41.
29. Boswell JF, Anderson LM, Barlow DH. An idiographic analysis of change processes in the unified transdiagnostic treatment of depression. J Consult Clin Psychol. 2014;82(6):1060–71.
30. Boswell JF, Bugatti M. An exploratory analysis of the impact of specific interventions: some clients reveal more than others. J Couns Psychol. 2016;63(6):710–20.
31. Hartogs BMA, Bartels-Velthuis AA, Van der Ploeg K, Bos EH. Heart rate variability biofeedback stress relief program for depression*.. A replicated Single-Subject design. Methods Inf Med. 2017;56(6):419–26.
32. Boswell JF, Schwartzman CM. An exploration of intervention augmentation in a single case. Behav Modif. 2023;47(6):1219–41.
33. Brake CA, Sauer-Zavala S, Boswell JF, Gallagher MW, Farchione TJ, Barlow DH. Mindfulness-Based exposure strategies as a transdiagnostic mechanism of change: an exploratory alternating treatment design. Behav Ther. 2016;47(2):225–38.
34. Fisher AJ, Soyster P. Generating Accurate Personalized Predictions of Future Behavior: A Smoking Exemplar [Internet]. OSF; 2019 [cited 2024 Sep 13]. Available from: https://osf.io/e24v6
35. Fernandez KC, Fisher AJ, Chi C. Development and initial implementation of the dynamic assessment treatment algorithm (DATA). PLoS ONE. 2017;12(6):e0178806.
36. Williams LM. Precision psychiatry: a neural circuit taxonomy for depression and anxiety. Lancet Psychiatry. 2016;3(5):472–80.
37. Marquand AF, Rezek I, Buitelaar J, Beckmann CF. Understanding heterogeneity in clinical cohorts using normative models: beyond Case-Control studies. Biol Psychiatry. 2016;80(7):552–61.
38. Touyz S, Bryant E, Dann KM, Polivy J, Le Grange D, Hay P, et al. What kind of illness is anorexia nervosa? Revisited: some preliminary thoughts to finding a cure. J Eat Disord. 2023;11(1):221.
39. Purgato M, Singh R, Acarturk C, Cuijpers P. Moving beyond a 'one-size-fits-all' rationale in global mental health: prospects of a precision psychology paradigm. Epidemiol Psychiatr Sci. 2021;30:e63.
40. Wium-Andersen IK, Vinberg M, Kessing LV, McIntyre RS. Personalized medicine in psychiatry. Nord J Psychiatry. 2017;71(1):12–9.

41. Williams LM. Special Report: Precision Psychiatry—Are We Getting Closer? Psychiatr News [Internet]. 2022 Sep [cited 2024 Nov 28];57(9). Available from: https://psychiatryonline.org/doi/full/10.1176/appi.pn.2022.09.9.23.
42. McGorry PD, Hickie IB, Yung AR, Pantelis C, Jackson HJ. Clinical staging of psychiatric disorders: a heuristic framework for choosing earlier, safer and more effective interventions. Aust N Z J Psychiatry. 2006;40(8):616–22.
43. Treasure J, Stein D, Maguire S. Has the time come for a staging model to map the course of eating disorders from high risk to severe enduring illness? An examination of the evidence. Early Interv Psychiatry. 2015;9(3):173–84.
44. Treasure J, Russell G. The case for early intervention in anorexia nervosa: theoretical exploration of maintaining factors. Br J Psychiatry. 2011;199(1):5–7.
45. Maguire S, Grange DL, Surgenor L, Marks P, Lacey H, Touyz S. Staging anorexia nervosa: conceptualizing illness severity. Early Interv Psychiatry. 2008;2(1):3–10.
46. Fava GA, Kellner R. Prodromal symptoms in affective disorders. Am J Psychiatry. 1991;148(7):823–30.
47. Fava GA, Kellner R. Staging: a neglected dimension in psychiatric classification. Acta Psychiatr Scand. 1993;87(4):225–30.
48. Chung T, Martin CS, Grella CE, Winters KC, Abrantes AM, Brown SA. Course of alcohol problems in treated adolescents. Alcohol Clin Exp Res. 2003;27(2):253–61.
49. Agius M, Goh C, Ulhaq S, McGorry P. The staging model in schizophrenia, and its clinical implications. Psychiatr Danub. 2010;22(2):211–20.
50. Maguire S, Surgenor LJ, Le Grange D, Lacey H, Crosby RD, Engel SG, et al. Examining a staging model for anorexia nervosa: empirical exploration of a four stage model of severity. J Eat Disord. 2017;5(1):41.
51. Hyam LE, Phillips M, Gracie L, Allen K, Schmidt U. Clinical staging across eating disorders: a scoping review protocol. BMJ Open. 2023;13(11):e077377.
52. Russell GFM, Szmukler GI, Dare C, Eisler I. An evaluation of family therapy in anorexia nervosa and bulimia nervosa. Arch Gen Psychiatry. 1987;44(12):1047–56.
53. Eisler I, Simic M, Russell GFM, Dare C. A randomised controlled treatment trial of two forms of family therapy in adolescent anorexia nervosa: a five-year follow-up. J Child Psychol Psychiatry. 2007;48(6):552–60.
54. Le Grange D, Lock J, Agras WS, Moye A, Bryson SW, Jo B, et al. Moderators and mediators of remission in family-based treatment and adolescent focused therapy for anorexia nervosa. Behav Res Ther. 2012;50(2):85–92.
55. Datta N, Hagan K, Bohon C, Stern M, Kim B, Matheson BE, et al. Predictors of family-based treatment for adolescent eating disorders: do family or diagnostic factors matter? Int J Eat Disord. 2023;56(2):384–93.
56. Radunz M, Keegan E, Osenk I, Wade TD. Relationship between eating disorder duration and treatment outcome: systematic review and meta-analysis. Int J Eat Disord. 2020;53(11):1761–73.
57. Vall E, Wade TD. Predictors of treatment outcome in individuals with eating disorders: A systematic review and meta-analysis. Int J Eat Disord. 2015;48(7):946–71.
58. Linardon J, de la Piedad Garcia X, Brennan L, Predictors. Moderators, and mediators of treatment outcome following manualised Cognitive-Behavioural therapy for eating disorders: A systematic review. Eur Eat Disord Rev J Eat Disord Assoc. 2017;25(1):3–12.
59. Barakat S, McLean SA, Bryant E, Le A, Marks P, National Eating Disorder Research Consortium. Risk factors for eating disorders: findings from a rapid review. J Eat Disord. 2023;11(1):8.
60. Stice E, Shaw HE. Role of body dissatisfaction in the onset and maintenance of eating pathology: a synthesis of research findings. J Psychosom Res. 2002;53(5):985–93.
61. Rodgers RF, Slater A, Gordon CS, McLean SA, Jarman HK, Paxton SJ. A biopsychosocial model of social media use and body image concerns, disordered eating, and Muscle-Building behaviors among adolescent girls and boys. J Youth Adolesc. 2020;49(2):399–409.
62. Levine MP, Smolak L. The role of protective factors in the prevention of negative body image and disordered eating. Eat Disord. 2016;24(1):39–46.
63. Schaefer LM, Burke NL, Thompson JK, Dedrick RF, Heinberg LJ, Calogero RM, et al. Development and validation of the Sociocultural attitudes towards appearance Questionnaire-4 (SATAQ-4). Psychol Assess. 2015;27(1):54–67.
64. Fardouly J, Vartanian LR. Social media and body image concerns: current research and future directions. Curr Opin Psychol. 2016;9:1–5.
65. Perloff RM. Social media effects on young women's body image concerns: theoretical perspectives and an agenda for research. Sex Roles. 2014;71(11):363–77.
66. Wilksch SM, O'Shea A, Ho P, Byrne S, Wade TD. The relationship between social media use and disordered eating in young adolescents. Int J Eat Disord. 2020;53(1):96–106.
67. Latzer Y, Rozenstain-Hason M, Kabakov O, Givon M, Mizrachi S, Alon S, et al. Childhood maltreatment in patients with binge eating disorder with and without night eating syndrome vs. control. Psychiatry Res. 2020;293:113451.
68. Latzer Y, Hochdorf Z, Bachar E, Canetti L. Attachment style and family functioning as discriminating factors in eating disorders. Contemp Fam Ther. 2002;24(4):581–99.
69. Treasure J, Schmidt U. The cognitive-interpersonal maintenance model of anorexia nervosa revisited: a summary of the evidence for cognitive, socio-emotional and interpersonal predisposing and perpetuating factors. J Eat Disord. 2013;1(1):13.
70. Pisetsky EM, Utzinger LM, Peterson CB. Incorporating social support in the treatment of anorexia nervosa: special considerations for older adolescents and young adults. Cogn Behav Pract. 2016;23(3):316–28.
71. Griffiths S, Castle D, Cunningham M, Murray SB, Bastian B, Barlow FK. How does exposure to thinspiration and fitspiration relate to symptom severity among individuals with eating disorders? Evaluation of a proposed model. Body Image. 2018;27:187–95.
72. Mond JM, Robertson-Smith G, Vetere A. Stigma and eating disorders: is there evidence of negative attitudes towards anorexia nervosa among women in the community? J Ment Health. 2006;15(5):519–32.
73. Becker AE, Franko DL, Speck A, Herzog DB. Ethnicity and differential access to care for eating disorder symptoms. Int J Eat Disord. 2003;33(2):205–12.
74. Caslini M, Bartoli F, Crocamo C, Dakanalis A, Clerici M, Carrà G. Disentangling the association between child abuse and eating disorders: A systematic review and Meta-Analysis. Psychosom Med. 2016;78(1):79–90.
75. Puhl R, Suh Y. Stigma and eating and weight disorders. Curr Psychiatry Rep. 2015;17(3):10.
76. Sundquist J, Ohlsson H, Winkleby MA, Sundquist K, Crump C. School achievement and risk of eating disorders in a Swedish National cohort. J Am Acad Child Adolesc Psychiatry. 2016;55(1):41–e461.
77. Prnjak K, Jukic I, Tufano JJ, Perfectionism. Body satisfaction and dieting in athletes: the role of gender and sport type. Sports. 2019;7(8):181.
78. Hunger JM, Smith JP, Tomiyama AJ. An Evidence-Based rationale for adopting Weight-Inclusive health policy. Soc Issues Policy Rev. 2020;14(1):73–107.
79. Brewis A, SturtzSreetharan C, Wutich A. Obesity stigma as a globalizing health challenge. Glob Health. 2018;14(1):20.
80. Norris ML. Exploring biologically oriented precision mental health initiatives for the care of patients with eating disorders: A narrative review. Eur Eat Disord Rev [Internet]. 2023 [cited 2024 Aug 11];n/a(n/a). Available from: https://onlinelibrary.wiley.com/doi/abs/10.1002/erv.3114
81. Watson HJ, Yilmaz Z, Thornton LM, Hübel C, Coleman JRI, Gaspar HA, et al. Genome-wide association study identifies eight risk loci and implicates metabo-psychiatric origins for anorexia nervosa. Nat Genet. 2019;51(8):1207–14.
82. Breithaupt L, Hubel C, Bulik CM. Updates on Genome-wide association findings in eating disorders and future application to precision medicine. Curr Neuropharmacol. 2018;16(8):1102–10.
83. Bulik CM, Coleman JRI, Hardaway JA, Breithaupt L, Watson HJ, Bryant CD, et al. Genetics and neurobiology of eating disorders. Nat Neurosci. 2022;25(5):543–54.
84. Malcolm A, Phillipou A. Current directions in biomarkers and endophenotypes for anorexia nervosa: A scoping review. J Psychiatr Res. 2021;137:303–10.
85. Hemmings A, Sharpe H, Allen K, Bartel H, Campbell IC, Desrivières S, et al. EDIFY (Eating disorders: delineating illness and recovery trajectories to inform personalised prevention and early intervention in young People): project outline. BJPsych Bull. 2022;47(6):328–36.
86. Australian Eating Disorders Research & Translation Centre. Gaining a Holistic Understanding of Eating Disorders: Phenotyping and Genotyping Illness [nternet] [Internet]. 2025. Available from: osf.io/dpbqr.
87. Bulik CM, Carroll IM, Mehler P. Reframing anorexia nervosa as a metabo-psychiatric disorder. Trends Endocrinol Metab TEM. 2021;32(10):752–61.
88. Hübel C, Abdulkadir M, Herle M, Loos RJF, Breen G, Bulik CM, et al. One size does not fit all. Genomics differentiates among anorexia nervosa, bulimia nervosa, and binge-eating disorder. Int J Eat Disord. 2021;54(5):785–93.
89. Adams DM, Reay WR, Cairns MJ. Multiomic prioritisation of risk genes for anorexia nervosa. Psychol Med. 2023;53(14):6754–62.

90. Burstein D, Griffen TC, Therrien K, Bendl J, Venkatesh S, Dong P, et al. Genome-wide analysis of a model-derived binge eating disorder phenotype identifies risk loci and implicates iron metabolism. Nat Genet. 2023;55(9):1462–70.
91. Johnson JS, Cote AC, Dobbyn A, Sloofman LG, Xu J, Cotter L, et al. Mapping anorexia nervosa genes to clinical phenotypes. Psychol Med. 2023;53(6):2619–33.
92. Tozzi L, Zhang X, Pines A, Olmsted AM, Zhai ES, Anene ET, et al. Personalized brain circuit scores identify clinically distinct biotypes in depression and anxiety. Nat Med. 2024;30(7):2076–87.
93. Austin A, De Silva U, Ilesanmi C, Likitabhorn T, Miller I, Sousa Fialho M et al. da L,. International consensus on patient-centred outcomes in eating disorders. Lancet Psychiatry. 2023;10(12):966–73.
94. Bryant E, Broomfield C, Burrows J, McLean S, Marks P, Maloney D, et al. Gaining consensus on clinical quality outcomes for eating disorders: framework for the development of an Australian National minimum dataset. BMJ Open. 2023;13(4):e071150.
95. Austin A, Potterton R, Flynn M, Richards K, Allen K, Grant N, et al. Exploring the use of individualised patient-reported outcome measures in eating disorders: validation of the psychological outcome profiles. Eur Eat Disord Rev J Eat Disord Assoc. 2021;29(2):281–91.
96. Ashworth M, Evans C, Clement S. Measuring psychological outcomes after cognitive behaviour therapy in primary care: a comparison between a new patient-generated measure PSYCHLOPS (Psychological outcome Profiles) and HADS (Hospital anxiety and depression Scale). J Ment Health. 2009;18(2):169–77.
97. Wright AGC, Woods WC. Personalized models of psychopathology. Annu Rev Clin Psychol. 2020;16:49–74.
98. Borsboom D, Cramer AOJ. Network analysis: an integrative approach to the structure of psychopathology. Annu Rev Clin Psychol. 2013;9:91–121.
99. DuBois RH, Rodgers RF, Franko DL, Eddy KT, Thomas JJ. A network analysis investigation of the cognitive-behavioral theory of eating disorders. Behav Res Ther. 2017;97:213–21.
100. Forrest LN, Jones PJ, Ortiz SN, Smith AR. Core psychopathology in anorexia nervosa and bulimia nervosa: A network analysis. Int J Eat Disord. 2018;51(7):668–79.
101. Solmi M, Collantoni E, Meneguzzo P, Degortes D, Tenconi E, Favaro A. Network analysis of specific psychopathology and psychiatric symptoms in patients with eating disorders. Int J Eat Disord. 2018;51(7):680–92.
102. Monteleone AM, Mereu A, Cascino G, Criscuolo M, Castiglioni MC, Pellegrino F, et al. Re-conceptualization of anorexia nervosa psychopathology: A network analysis study in adolescents with short duration of the illness. Int J Eat Disord. 2019;52(11):1263–73.
103. Calugi S, Sartirana M, Misconel A, Boglioli C, Grave RD. Eating disorder psychopathology in adults and adolescents with anorexia nervosa: A network approach. Int J Eat Disord. 2020;53(5):690–701.
104. Schlegl S, Smith KE, Vierl L, Crosby RD, Moessner M, Neumayr C, et al. Using network analysis to compare diagnosis-specific and age-specific symptom networks in eating disorders. Int J Eat Disord. 2021;54(8):1463–76.
105. Christian C, Perko VL, Vanzhula IA, Tregarthen JP, Forbush KT, Levinson CA. Eating disorder core symptoms and symptom pathways across developmental stages: A network analysis. J Abnorm Psychol. 2020;129(2):177–90.
106. Levinson CA, Cash E, Welch K, Epskamp S, Hunt RA, Williams BM, et al. Personalized networks of eating disorder symptoms predicting eating disorder outcomes and remission. Int J Eat Disord. 2020;53(12):2086–94.
107. Chekroud AM, Bondar J, Delgadillo J, Doherty G, Wasil A, Fokkema M, et al. The promise of machine learning in predicting treatment outcomes in psychiatry. World Psychiatry. 2021;20(2):154–70.
108. Haynos AF, Wang SB, Lipson S, Peterson CB, Mitchell JE, Halmi KA, et al. Machine learning enhances prediction of illness course: a longitudinal study in eating disorders. Psychol Med. 2021;51(8):1392–402.
109. Anderson LM, Lim KO, Kummerfeld E, Crosby RD, Crow SJ, Engel SG, et al. Causal discovery analysis: A promising tool in advancing precision medicine for eating disorders. Int J Eat Disord. 2023;56(11):2012–21.
110. Arend AK, Kaiser T, Pannicke B, Reichenberger J, Naab S, Voderholzer U, et al. Toward individualized prediction of Binge-Eating episodes based on ecological momentary assessment data: item development and pilot study in patients with bulimia nervosa and Binge-Eating disorder. JMIR Med Inf. 2023;11(1):e41513.
111. Bzdok D, Meyer-Lindenberg A. Machine learning for precision psychiatry: opportunities and challenges. Biol Psychiatry Cogn Neurosci Neuroimaging. 2018;3(3):223–30.
112. Shatte ABR, Hutchinson DM, Teague SJ. Machine learning in mental health: a scoping review of methods and applications. Psychol Med. 2019;49(9):1426–48.
113. Krug I, Linardon J, Greenwood C, Youssef G, Treasure J, Fernandez-Aranda F, et al. A proof-of-concept study applying machine learning methods to putative risk factors for eating disorders: results from the multi-centre European project on healthy eating. Psychol Med. 2023;53(7):2913–22.
114. Forrest LN, Ivezaj V, Grilo CM. Machine learning V. traditional regression models predicting treatment outcomes for binge-eating disorder from a randomized controlled trial. Psychol Med. 2023;53(7):2777–88.
115. Svendsen VG, Wijnen BFM, De Vos JA, Veenstra R, Evers SMAA, Lokkerbol J. A roadmap for applying machine learning when working with privacy-sensitive data: predicting non-response to treatment for eating disorders. Expert Rev Pharmacoecon Outcomes Res. 2023;23(8):933–49.
116. Monaco F, Vignapiano A, Piacente M, Pagano C, Mancuso C, Marenna A et al. An advanced Artificial Intelligence platform for a personalised treatment of Eating Disorders. 2024.
117. McFarlane T, MacDonald DE, Trottier K, Olmsted MP. The effectiveness of an individualized form of day hospital treatment. Eat Disord. 2015;23(3):191–205.
118. Haynos AF, Snipes C, Guarda A, Mayer LE, Attia E. Comparison of standardized versus individualized caloric prescriptions in the nutritional rehabilitation of inpatients with anorexia nervosa. Int J Eat Disord. 2016;49(1):50–8.
119. Levinson CA, Hunt RA, Keshishian AC, Brown ML, Vanzhula I, Christian C, et al. Using individual networks to identify treatment targets for eating disorder treatment: a proof-of-concept study and initial data. J Eat Disord. 2021;9(1):147.
120. Butler RM, Ortiz AML, Pennesi JL, Crumby EK, Cusack CE, Levinson CA. A pilot randomized controlled trial of transdiagnostic network-informed personalized treatment for eating disorders versus enhanced cognitive behavioral therapy. Int J Eat Disord. 2023;56(8):1674–80.
121. Fusar-Poli P, Yung AR, McGorry P, van Os J. Lessons learned from the psychosis high-risk State: towards a general staging model of prodromal intervention. Psychol Med. 2014;44(1):17–24.
122. Fusar-Poli P, Davies C, Rutigliano G, Stahl D, Bonoldi I, McGuire P. Transdiagnostic Individualized Clinically Based Risk Calculator for the Detection of Individuals at Risk and the Prediction of Psychosis: Model Refinement Including Nonlinear Effects of Age. Front Psychiatry [Internet]. 2019 May 9 [cited 2024 Sep 14];10. Available from: https://www.frontiersin.org/journals/psychiatry/articles/https://doi.org/10.3389/fpsyt.2019.00313/full
123. Cuijpers P, Li J, Hofmann SG, Andersson G. Self-reported versus clinician-rated symptoms of depression as outcome measures in psychotherapy research on depression: A meta-analysis. Clin Psychol Rev. 2010;30(6):768–78.
124. Boyle GJ. Self-report measures of depression: some psychometric considerations. Br J Clin Psychol. 1985;24(1):45–59.
125. Kreitchmann RS, Abad FJ, Ponsoda V, Nieto MD, Morillo D. Controlling for Response Biases in Self-Report Scales: Forced-Choice vs. Psychometric Modeling of Likert Items. Front Psychol [Internet]. 2019 Oct 15 [cited 2024 Nov 28];10. Available from: https://www.frontiersin.org/journals/psychology/articles/https://doi.org/10.3389/fpsyg.2019.02309/full
126. Fusar-Poli L, Rutten BPF, van Os J, Aguglia E, Guloksuz S. Polygenic risk scores for predicting outcomes and treatment response in psychiatry: hope or hype? Int Rev Psychiatry. 2022;34(7–8):663–75.
127. Miskovic-Wheatley J, Bryant E, Ong SH, Vatter S, Le A, Aouad P, et al. Eating disorder outcomes: findings from a rapid review of over a decade of research. J Eat Disord. 2023;11(1):85.
128. Monteleone AM, Pellegrino F, Croatto G, Carfagno M, Hilbert A, Treasure J, et al. Treatment of eating disorders: A systematic meta-review of meta-analyses and network meta-analyses. Neurosci Biobehav Rev. 2022;142:104857.
129. Ralph-Nearman C, Rae J, Levinson CA. Using clinician and patient input to assess utility, accuracy, efficiency, and therapeutic implementation of a new data-driven digital therapeutic for personalized clinical eating disorder treatment: awaken digital guide. Psychother Res. 2024;0(0):1–14.
130. Fried EI. Lack of theory Building and testing impedes progress in the factor and network literature. Psychol Inq. 2020;31(4):271–88.

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