Comprehensive Psychiatry 131 (2024) 152468

Contents lists available at ScienceDirect

Comprehensive Psychiatry

journal homepage: www.elsevier.com/locate/comppsych

Staging models in eating disorders: A systematic scoping review of the literature

E. Tomba *, L. Tecuta, V. Gardini, G. Tomei, E. Lo Dato

Department of Psychology, University of Bologna, Bologna, Italy

ARTICLE INFO

Keywords:
Staging
Eating disorders
Anorexia
Bulimia
Binge eating disorder
Clinimetrics

ABSTRACT

Eating Disorders (ED) are characterized by low remission rates, treatment drop-out, and residual symptoms. To improve assessment and treatment of ED, the staging approach has been proposed. This systematic scoping review is aimed at mapping the existing staging models that explicitly propose stages of the progression of ED.

A systematic search of PubMed, PsycINFO, Scopus was conducted with the terms *staging, anorexia nervosa, bulimia nervosa, binge-eating disorders, eating disorders*. Eleven studies met inclusion criteria presenting nine ED staging models, mostly for anorexia nervosa. Three were empirically tested, one of which was through an objective measure specifically developed to differentiate between stages. Most staging models featured early stages in which the exacerbation of EDs unfolds and acute phases are followed by chronic stages. Intermediate stages were not limited to acute stages, but also residual phases, remission, relapse, and recovery. The criteria for stage differentiation encompassed behavioral, psychological, cognitive, and physical features including body mass index and illness duration. One study recommended stage-oriented interventions. The current review underscores the need to empirically test the available staging models and to develop and test new proposals of staging models for other ED populations. The inclusion of criteria based on medical features and biomarkers is recommended. Staging models can potentially guide assessment and interventions in daily clinical settings.

1. Introduction

Eating disorders (ED) are complex psychiatric illnesses characterized by dysfunctional eating or weight-control behaviors, with serious consequences both on physical health and psychological functioning [1]. The causes behind ED are multifactorial and they involve social, psychological, and biological processes [2]. According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5-TR) [3] and the International Classification of Diseases (ICD-11) [4] the main ED diagnoses include anorexia nervosa (AN), bulimia nervosa (BN), binge-eating disorder (BED) and other specified eating disorders (OSFED). However, half of the patients observed in clinical practice show atypical forms of ED, often severe and long-lasting, characterized by a mixed clinical picture that does not meet the standard diagnostic criteria [2].

Indeed, exclusive reliance on conventional diagnostic classification systems based on the medical model has been challenged in ED for various reasons [5]. The medical model is traditionally limited to observable signs and symptoms of diseases at the time of the assessment [6,7], and it does not concern itself with the trajectories and the longitudinal course of symptoms, individual differences, and possible combinations of symptoms or comorbidities [8]. Ignoring such clinical features may hamper the identification of efficacious treatments and the definition of outcomes in ED, whose clinical configuration is complicated by frequent diagnostic cross-over among ED diagnoses, concomitant medical ED-related complications, and high comorbidity rates with other psychiatric disorders [9-11].

Despite the existence of evidence-based treatments for ED, such as enhanced cognitive-behavioral therapy (CBT-E) [12], EDs remain difficult to treat, drop-out rates are high and some patients actively resist attempts to help them [2,13]. Only 40–60% of patients recover irrespective of the ED diagnosis [14] and residual symptomatology often persists even after standard [15], integrated (Mitchell et al., 2002; Grilo et al., 2011; Allen et al., 2012; MacDonald et al., 2021) and sequential treatments [16]. Consequently, the ED research field has extended its focus to investigate long-term outcomes of the illness, as well as the multiple factors that might influence prognosis to improve and properly plan ED treatment [17,18].

A possible complementary conceptualization of disease that may also

* Corresponding author at: Department of Psychology, University of Bologna, Viale Berti Pichat 5, Bologna 40122, Italy.
E-mail address: elena.tomba@unibo.it (E. Tomba).

https://doi.org/10.1016/j.comppsych.2024.152468

Available online 4 March 2024

aid in the conceptualization of clinical-psychological models of EDs, such as the transdiagnostic model [12], is the staging model of psychiatric conditions [19]. This approach, while having its roots in the medical field, overcomes the limits of the medical model [20] and categorical diagnoses classification [3,4]. Staging considers the longitudinal trajectory of an illness and has been applied to a wide range of psychiatric disorders, such as mood and anxiety disorders, schizophrenia, and alcohol use [19,21-27]. Its application allows the identification of discrete phases (or stages) that characterize a certain disease and allows clinicians to allocate patients into specific phases in the continuum of a disorder based on established criteria. This approach has the advantage of better differentiating between early and mild clinical phenomena of the same disorder and better recognizing nonspecific forms of mental disorders (i.e., mental disorders of mild or moderate severity that do not reach the full diagnosis based on DSM diagnostic criteria) [28], as well as identifying features of progression and chronicity of a disease [15].

Additionally, the staging model might be useful for treatment planning and guiding the selection of more precise treatments based on the specific stage of illness of the client [19,29], as has been shown in various clinical populations [28,30,31,32]. Staging models operazionalized in other clinical populations have supported stage-oriented interventions, proposing specific stage-based treatments [28,29,30,32].

An active debate on staging models applied to EDs is also emerging in the literature. Numerous longitudinal studies on the long-term outcomes and progression of ED can support the utility of its application in this clinical population [17,18,33-35]. Staging might represent the appropriate framework through which longitudinal ED data could be better conceptualized also in reference to relapse [36,9], recovery [37,38] and chronicity [17,39].

Despite the proliferation and breadth of longitudinal data supporting staging in ED, the use of staging conceptualizations as an assessment tool to decipher the phases of illness of patients with ED in routine clinical setting is still scarce [40,41]. To further support the clinical application of staging models in ED, the aim of the current study was to systematically review and identify models of staging that explicitly propose classifications of progression of ED illness in distinct stages, along with identifying their applications and potential knowledge gaps to improve research on staging in ED.

2. Methods

The present systematic scoping review was conducted in accordance with the Joanna Briggs Institute [42] guidelines and the Preferred Reporting Items for Systematic Reviews and Meta-analyses extension for scoping reviews guidelines (PRISMA-ScR; [43]). Systematic scoping reviews are particularly useful when the available literature on a specific topic has not been comprehensively reviewed yet or, such as in this case, when it has a heterogeneous nature which makes it difficult to address through a more precise systematic review [42].

The following research questions have been posed:

a) How many staging models are available in the literature that explicitly propose a classification of the progression of the ED illness in distinct stages and in reference to which ED diagnostic groups? What are the characteristics of the articles that present such staging models of ED?
b) How do the proposed staging models differ in their criteria of stage differentiation? Do they use any objective measures developed specifically to differentiate between stages or other objective measures as empirical evidence of the suggested staging models?
c) What stage-specific interventions, if any, are recommended in the proposed staging models?

2.1. Search strategies

Studies were selected through a systematic literature search on Pubmed, Scopus and PsycINFO, combining the following keywords: *anorexia nervosa* OR *bulimia nervosa* OR *binge eating disorder* OR *eating disorder* AND *staging*. Titles and abstracts were screened by two authors. Articles that appeared potentially relevant for the purpose of the study were independently reviewed and assessed by the same two authors who found consensus for eligibility. In case of disagreement, multiple rounds of full-text revisions and discussions were held until agreement was reached by all the authors.

2.2. Eligibility criteria

Eligible articles were in the English language and published in peer-reviewed journals. Since the staging model has been conceptualized and tested in ED clinical populations only in the last two decades [44], a temporal restriction criterion was applied to only include studies published from January 2000 to May 2023. Following the guidelines for scoping reviews [42] both empirical studies, reviews of the literature, and other type of sources (summaries, essays, panel studies and theoretical proposals) were included in the present study. Papers presenting staging models in other clinical populations or that only briefly mentioned the staging approach without providing a classification of the progression of the illness in ED in distinct stages were excluded (See Fig. 1).

2.3. Data extraction

Data were extracted from the articles included in the scoping review by two of the authors. Inclusion and exclusion criteria and data extraction were based on patient, intervention, comparison, outcome, and study design (PICOS) criteria [45], when applicable. Please see Table 1 for the criteria used for data extraction details.

3. Results

3.1. Selection of sources of evidence

The literature search yielded a total of 175 records, of which 128 were from Scopus, 25 from Pubmed and 22 from PsycINFO. After duplicates removal, a total of 134 articles were selected. A first screening of title and abstracts led to the exclusion of an additional 115 works, mainly because they proposed staging in clinical populations other than ED. Eighteen articles underwent full-text screening, leading to the exclusion of a further seven works. Therefore, the review included eleven articles (Fig. 1), including three empirical, five reviews, one essay, one panel study and one theoretical proposal.

For empirical studies, the following data were extracted: sample characteristics, study methodology, definitions of staging, criteria used for the classification of staging, treatment and relevant outcomes. For reviews and other type of sources, data concerning diagnostic groups, methodology, definitions of staging, criteria used for the staging classification and relevant outcomes were also extracted (see Table 2).

3.1.1. *How many staging models are available in the literature that explicitly propose a classification of the progression of the ED illness in distinct stages and in reference to which ED diagnosic groups? What are the characteristics of the articles that present such staging models of ED?*

In the 11 articles included in this scoping review, nine different staging models that explicitly proposed specific stages of the progression of the ED illness were identified. Six staging models on AN (66.6%) were identified in seven papers [52,56,46,44,50,54,57], one on BN (11.1%) in one paper [46], one on BED (11.1%) in one paper [58], and one on mixed ED populations (11.1%) presented in three papers [49,47,48].

Among the 11 included articles, the majority were literature reviews

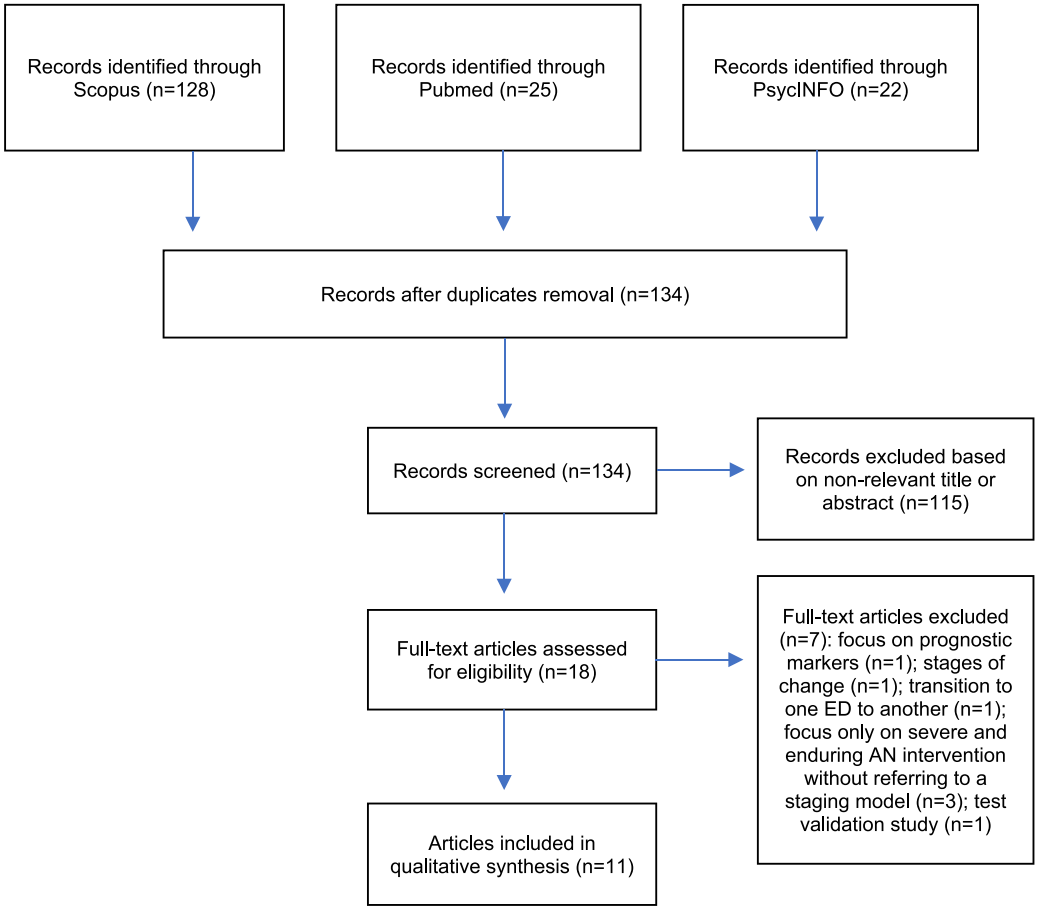

Fig. 1. PRISMA flow-diagram: identification, screening, eligibility and inclusion of data sources for the study.

(*N* = 5, 45.45%). Three of these (60%; [46,49,47]) were defined by the authors as systematic, however any reference to systematic review procedure guidelines (such as PRISMA or COCHRANE manuals; [59,60]) was lacking. Two (40%) were defined as non-systematic [44,48]. Additionally, one of the included sources (9.09%) was an essay [56], one (9.09%) a panel study [57], and one (9.09%) was a theoretical proposal [58]. The remaining three works (27.27%) were empirical studies, all with a longitudinal design [52,50,54] (see Table 2).

With regard to sample characteristics, the three empirical studies [52,50,54] included mostly female patients (96.30%), with a mean age of 25.34 years and age ranges reported in only two of the studies (16–58 years in [50] and 13–53 years in [54]). Ethnicity was reported only in the study by Ambwani et al. [52], in which 97.5% of the participants were white, 1.3% mixed white/black and 1.3% Asian (see Table 2).

3.2. How do the proposed staging models differ in their criteria of stage differentiation? Do they use any objective measures developed specifically to differentiate between stages or other objective measures as empirical evidence of the suggested staging models?

The number of identified stages differed among the models, with half of them (50%) proposing four stages (see Table 2). A set of heterogeneous criteria have been used to differentiate between the various stages (see Table 2). These included illness severity criteria based on psychological, physical, and social symptoms (N = 5, 55.5%) [56,46,50,57,47], duration of the illness criteria based on length in years (N = 5, 55.5%) [52,56,50,54,57], biometric criteria based on Body Mass Index (BMI) (*N* = 2, 22.2%) [50,57] and cognitive dysfunctions criteria, such as reward processing system alterations (*N* = 1, 11.1%) [58]. Only one of the studies [50] used an objective measure specifically developed to differentiate between stages, namely the *Clinician Administrative Staging Instrument for Anorexia Nervosa* (CASIAN; [51]), whereas two studies [52,54] used other objective measures to allocate patients into stages, namely the *Depression and Anxiety Stress Scale* (DASS; [53]) and the *Clinical Impairment Assessment* (CIA; [55]) as empirical evidence of the suggested staging models.

In the paragraphs that follow, results are presented separately for studies that proposed models that explicitly suggested specific stages of illness in AN patients only, in BN patients only, in BED patients only and in mixed ED diagnoses. See Table 2 for more details.

3.2.1. Anorexia nervosa

Six different staging models were proposed among the seven articles focusing on staging proposals in AN patients (Table 2). Four of these identified four stages [56,46,44,50]. In their essay, Beumont and Touyz [56] proposed a staging model according to which the clinical manifestations of the progression of AN were hypothesized to be organized in the following four stages: (1) acute episode; (2) partial or complete remission; (3) partial or complete relapse; (4) final outcome (including recovery, fatality, chronicity or residual phase) (Fig. 2). Two main criteria, based on the available literature at that time – though not explicitly reported – were suggested to be used to differentiate the four proposed staging phases: illness severity (physical, psychological, and social symptoms), and duration of the illness. Following the acute phase, if partial or complete remission is not reached, patients may show an exacerbation of physical, psychological, and social symptoms leading to partial or complete relapse, which in turn may lead to mortality, chronicity, a residual phase, or recovery. When relapsing into chronicity, isolation and lack in autonomy are frequently reported, paranoid and

Table 1
Inclusion and exclusion criteria and data extraction based on PICOS criteria.

PICOS	Inclusion criteria	Exclusion criteria	Data extraction
Population	• All ages • Female, male or mixed gender studies • Any ED diagnosis	• Sample included only other psychiatric diagnoses • Sample composed by patients with other medical illnesses not related to EDs	• Number of participants • Gender • Mean age • ED diagnosis • Other psychiatric diagnoses when present
Intervention	• All types of psychological or medical interventions		• Treatment information when provided
Comparison group	• Studies with and without comparison groups		• Allocate into groups where applicable
Outcome	• ED-related symptoms • Body Mass Index • Comorbid psychiatric or psychological symptoms • Neuropsychological and cognitive functioning	• Studies do not provide an ED staging model where specific phases of illness progession are proposed	• Differences among patients or modifications of outcome variables over time
Study design	• Empirical studies (prospective or retrospective cohort, cross-sectional, case-control, or RCT) • Reviews • Summaries • Essays • Panel studies • Theoretical proposals • English language		• Study design • Study setting

obsessive thoughts might be manifest, and body emaciation and dysfunctional weight loss behaviors can become life-threatening. According to Beumont and Touyz [56], longer duration of the illness indicated more severe cases, with the identified mean duration of AN being seven years. BMI levels below 14 or 15 were also indicated as significant markers of aggravation. Furthermore, even patients who eventually recover are unlikely to fully return to their normal health, with adverse physical, psychological and social effects often persisting.

In the non-systematic review by Maguire et al. [44] a four-stage model defining the severity progression of AN was recommended with the following stages: (1) mild or incipient; (2) moderate; (3) severe; (4) extremely severe (Fig. 2). The proposed stages of AN were described by Maguire et al. [44] based on severity progression using criteria taken from the available literature at that time [61-65]. AN severity progression based on psychological, behavioral, and physical symptoms, which largely varied across their proposed stages and among individuals was suggested despite the criteria having not been specified and provided for each single stage. Among the proposed psychological symptoms, Maguire et al. [44] included body image disturbances (i.e., drive for thinness and fear of weight), whereas for behavioral symptoms they included both weight reduction behaviors (i.e., extreme dietary restriction) and compensatory behaviors, which might lead to significant physical consequences such as loss of menses, hypophosphatemia and cardiac dysfunctions.

The four-stage model proposed by Maguire et al. [44] was empirically tested by the same authors [50] on a sample of 171 young women with full and subthreshold forms of AN using the scores from the *Clinician Administrative Staging Instrument for Anorexia Nervosa* (CASIAN; [51]), an objective measure specifically developed to differentiate between AN stages (Fig. 2). The CASIAN is a 34-item clinician administered psychometric interview for the assessment of AN severity and encompasses seven dimensions: weight/weight history, onset and duration of illness, dietary control, compensatory behaviors, psychological status, physical status, and ego-syntonic features [51,50]. The CASIAN appeared to be able to discriminate between the milder stages (stages 1 and 2) of Maguire et al. [44]'s staging model of AN from the more severe forms (stages 3 and 4). Specific cut-off scores are presented in Table 2 and Fig. 2.

Cosci and Fava [46] in their review proposed a four-stage model of AN which included: (1) prodromal stage; (2) acute manifestation; (3) residual stage; (4) chronic stage, either in attenuated or persistent form. The stages of AN (Fig. 2) proposed by Cosci and Fava [46] specifically defined symptomatologic characteristics of AN, including behavioral, physical, cognitive, social, and psychological symptoms as criteria, based on the available literature at the time of publication [66,67,68,44].

Despite these models sharing the same number of stages and described in terms of psychological, physical, and social symptoms severity, they differ in both the identified phases and the criteria used for stage differentiation. In terms of stage identification, the model by Beumont and Touyz [56] provides possible subtypes of the four proposed stages (i.e., it recognizes different possible outcomes of the illness, including relapse and remission). Moreover it is the only one identifying both illness duration and BMI levels as severity markers, whereas Cosci and Fava's [46] staging model offers a more detailed description of symptoms for each stage based on the available literature. However, only one model is empirically tested through an objective measure that differentiates between stages [44,50] (Fig. 2).

The two remaining staging models of AN identified two stages of the illness [52,54]. Ambwani et al. [52] proposed a two-stage model of progression of AN, which was empirically tested in a sample of 187 young women with AN in a longitudinal study. The recommended staging model consisted of an (1) early stage and a (2) severe and enduring AN (SE-AN) stage (Fig. 3). A time criterion, namely duration of the illness, was used by Ambwani et al. [52] to allocate patients in either of these two stages (< 3 years for early stage and ≥ 7 years for SE-AN) based on previous studies [69-72]. Additionally, due to the relevance of psychological distress in eating symptomatology, Ambwani et al. [52] defined SE-AN also based on severity of psychological distress criteria assessed through the *Depression and Anxiety Stress Scale* (DASS; [53]), establishing a cut-off score ≥ 60 based on previous studies [73].

Ramos et al. [54] proposed and empirically tested a two-stage model of AN, distinguishing between (1) non SE-AN and (2) SE-AN (Fig. 3) in a longitudinal study with 139 AN patients. A time criterion, duration of the illness, was used to allocate patients in either of these two stages ($<$ 7 years for non SE-AN and ≥ 7 years for SE-AN). Through an exploratory analysis, Ramos et al. [54] also tested the role of an adjunctive criterion for allocating patients in the aforementioned stages, that is clinical impairment as assessed through the *Clinical Impairment Assessment* (CIA; [55]), considering a cut-off score ≥ 16 [54]. The CIA is a 16-item self-report measure that assesses clinical impairment secondary to eating disorders in three domains: personal, social, and cognitive. When also considering clinical impairment as a criterion for their proposed staging classification, significant differences emerged between the non-SE and SE stages of AN, with SE-AN patients endorsing worse eating and depressive symptomatology and worse emotion dysregulation. The two-stage models share and empirically test the same conceptualization of the illness as progressing from an early/non-severe phase to a severe and enduring manifestation based on illness duration and general psychological distress/impairment in both cases assessed through the use of objective measures (Fig. 3).

In the panel study by Steinglass et al. [57], a five-stage model of AN progression was proposed (Fig. 4). To find consensus on a definition of

Table 2
Article characteristics.

Authors	Country	Methodology	Diagnostic group and sample characteristics	Definition of staging	Criteria for staging	Outcomes or relevant findings
Reviews						
Maguire et al. [44]	Australia USA UK New Zealand	Review	AN	Four-stage model of AN: (1) mild or incipient; (2) moderate; (3) severe; (4) extremely severe AN.	Total symptomatic severity of AN, including psychological, physical and behavioral symptoms.	The use of a standardized staging model for AN might serve to accommodate the heterogeneous presentations of the illness within the same diagnosis, distinguishing between different sub-groups of patients based on symptomatic and prognostic factors.
Cosci and Fava [46]	Italy U.S.A.	Systematic review	AN and BN	Four-stage model: (1) prodromal phase; (2) acute manifestation; (3) residual phase; (4) chronic phase (in attenuated or persistent form).	Descriptive characteristics of the illness based on behavioral, physical, cognitive, social and psychological symptoms taken from the available literature.	The staging model allows to assess the longitudinal development of mental disorders. It also offers an alternative to the traditional diagnostic classification, since it allows not only to determine the progression of a disorder, but also to estimate where a certain patient is located along a continuum of severity.
Treasure et al. [47]	UK Israel Australia	Systematic review	Mixed ED diagnoses	Four-stage model of ED according to McGorry et al. [29] conceptualization of staging for psychosis: (1) high risk; (2) early syndrome (including subsyndromal forms); (3) full syndrome; (4) severe enduring illness.	Descriptive characteristics of the illness based on behavioral, physical, neuropsychological and social symptoms taken from epidemiological studies and neuropsychological findings.	The existence of stages is supported in patients with AN and it allows to determine prognosis and implement optimal interventions. Accessible interventions in the early stages of the illness might attenuate the symptomatology and tailored interventions at different stages of the illness might improve outcomes.
[48]	UK	Summary	Mixed ED diagnoses	Four-stage model of ED according to McGorry et al. [29] conceptualization of staging for psychosis: (1) high risk; (2) early syndrome (including subsyndromal forms); (3) full syndrome; (4) severe enduring illness	Descriptive characteristics of the illness based on behavioral, physical, neuropsychological and social symptoms taken from epidemiological studies and neuropsychological findings.	Specific interventions should be implemented depending on the stage of the illness. Specifically, whereas guidelines already exist for the treatment of the first stages of the illness, less is known about the treatment of SE forms of eating disorders. These might include cognitive and cognitive-behavioral interventions, exposure therapy and brain stimulation.
Hay and Touyz [49]	Australia	Systematic review	Mixed ED diagnoses	Four-stage model of ED by Treasure et al. [47], applied to SE-AN.	Descriptive characteristics of the illness based on behavioral, physical, neuropsychological and social symptoms taken from epidemiological studies and neuropsychological findings.	The use of the staging approach is useful in AN to improve outcomes and treatment approaches, whereas further investigations are needed for others ED diagnoses. Severe and enduring AN might be treated through goals modification, de-emphasization of weight gain and focus on maintaining factors.
Empirical studies						
Maguire et al. [50]	Australia USA UK	Longitudinal study	171 patients with full and subthreshold forms of AN, aged 24.39 (±8.05), range 16–58, 100% females, treated either with an intensive	Four-stage model of AN: (1) mild or incipient; (2) moderate; (3) severe; (4) extremely severe AN.	Cut-off points on the *Clinician Administrative Staging Instrument for Anorexia Nervosa* (CASIAN) [51] scores as follows: < 34.50 for stage 1; ≥ 34.50 for stage 2; ≥ 48.50 for stage 3; ≥ 52.50 for stage 4.	The identified staging model and the cut-off scores used are able to detect patients at different stages of the illness. Patients with more severe stages engaged in more intensive treatment options,

(*continued on next page*)

Table 2 (*continued*)

Authors	Country	Methodology	Diagnostic group and sample characteristics	Definition of staging	Criteria for staging	Outcomes or relevant findings
			program or in an outpatient setting.			such as hospital treatment, whereas patients at a milder stage were less likely to be treated in an inpatient setting.
Ambwani et al. [52]	UK	Longitudinal study	87 patients with AN (97.5% white, 1.3% mixed white/black, 1.3% asian), aged 27.81 (± 9.80), 96.8% females, treated in a NHS outpatient setting for eating disorders.	Two-stage model of AN: (1) early stage; (2) severe and enduring (SE) anorexia nervosa.	Two criteria: (1) duration of the illness (< 3 years for early stage and ≥ 7 years for SE); (2) distress levels (only for SE-AN) assessed through the *Depression and Anxiety Stress Scale* (DASS; [53]) (≥60)	Patients in the SE stage of AN reported more lifetime hospitalization, worse disordered eating symptoms and more impaired work and social well-being. The clinical utility of using the 7-years threshold together with distress levels as a marker of severity has been supported. Different treatment efforts at different stages of the illness are needed.
Ramos et al. [54]	Portugal	Longitudinal study	139 patients with AN, aged 23.82 (± 8.70), 92.09% females, treated both in outpatient and inpatient settings in two different hospitals specialized in ED treatment.	Two-stage model of AN: (1) non-SE-AN and (2) SE-AN.	One main criterion: illness duration (<7 years for non-SE-AN and ≥7 years for SE-AN). As an adjunctive exploratory analysis, two criteria: (1) illness duration; (2) impairment levels assessed through the *Clinical Impairment Assessment* (CIA; [55]) (≥16) in three domains: personal, social, and cognitive.	Patients in the SE stage of AN (only based on illness duration) reported a higher number of previous hospitalizations than those in the non-SE stage. When also considering clinical impairment, patients in the SE stage of AN reported significantly higher levels of eating and depressive psychopathology and emotion dysregulation.
Others						
Beumont and Touyz [56]	Australia	Essay	AN	Four-stage model of AN: (1) acute episode; (2) partial or complete remission; (3) partial or complete relapse; (4) final outcome (recovery, fatality, chronicity, residual).	Two criteria: (1) illness severity (nutritional disturbance, medical problems, psychopathology, behavioral abnormalities and psychosocial functioning); (2) illness duration, for which criteria are not specified but that might represent a severity marker (mean duration of the illness 7 years)	Thinking of AN as an illness progressing through different stages might help personalizing treatments. In particular, it might be beneficial for patients in the most severe stages of the illness, in which severe emaciation and life-threatening behaviors are present.
Steinglass et al. [57]	USA	Panel study. 31 experts in AN from different fields have been longitudinally interviewed to achieve consensus on a staging model of AN.	AN	Five-stage model of AN: (1) subsyndromal; (2) full syndrome/early AN; (3) persistent illness; (4) partial remission; (5) full remission.	Three criteria: (1) illness duration (≤ 1 year for early stage; > 3 years for persistent AN; >1 year without symptoms for remission); (2) eating-disordered symptoms, including body-image disturbance; (3) BMI levels (≤18.5 in defining illness).	Restrictive eating is the central behavioral characteristic of all the stages, whereas its absence is central to all the stages of recovery. BMI has been indicated as an important biological marker of illness in the full syndrome.
Bodell & Racine [58]	Canada	Theoretical proposal	BED	Three-stage model of BED: (1) at risk; (2) recent-onset; (3) established BED.	One main criterion: cognitive dysfunctions levels based on dysfunctional reward processing alterations.	As novel treatments for BED patients target reward processing, considering and testing reward alterations changes over stages of the illness might be useful to target specific reward processing alterations at different phases, as well as preventing the development of BED in individuals with high reward sensitivity.

staging in this clinical population, thirty-one experts in the treatment of AN from different disciplines (psychiatry, psychology, internal medicine, adolescent medicine, social work and nutrition) were interviewed. The final proposed staging model of AN included five stages: (1) subsyndromal; (2) full syndrome; (3) persistent illness; or (4) partial remission; (5) full remission. From full syndrome a patient may progress to persistent illness or alternatively obtain partial remission and finally full remission with a possible repetition of the cycle of illness. Stages were defined based on illness duration and AN symptomatological (behavioral, cognitive, and biological) criteria. Concerning time frames, consensus was met in assigning an illness duration ≤ 1 year for the early stage, > 3 years for persistent AN (with a time range varying from 3 to 10 years), >1 year without symptoms for partial remission and > 3 years without symptoms for full remission. Regarding the behavioral

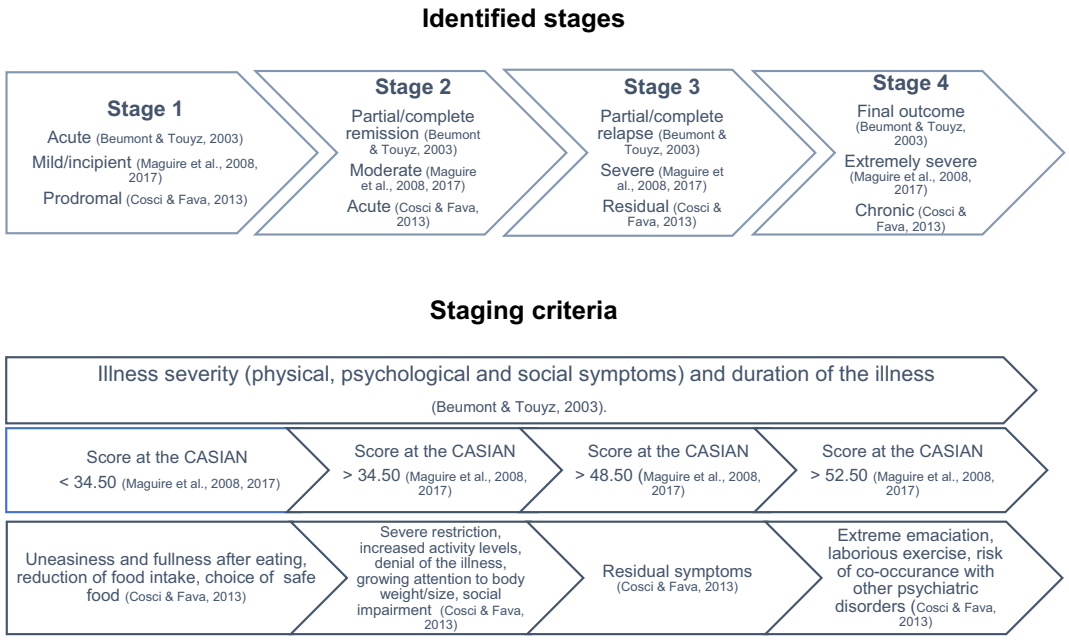

Fig. 2. Four-stage models of AN.

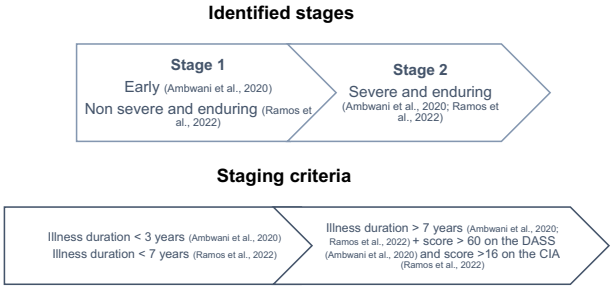

Fig. 3. Two-stage models of AN.

components, the panel agreed in recognizing restrictive eating as characterizing almost all stages of the illness. Specifically, mild to moderate restrictive eating behaviors were observed in the subsyndromal phase, whereas a criterion of no more than minimal restriction was required for partial remission and for complete remission, together with abstinence from binge-purging behaviors. A high consensus was met in recognizing the existence of a subsyndromal phase, characterized by body image disturbances and mild restrictive eating, without significant weight loss. From a cognitive perspective, all stages were characterized by body image disturbance. In terms of medical components, BMI was identified as an important biological marker, with levels ≤ 18.5 defining persistent illness. However, the exact BMI score needed to determine whether an individual is at a low risk of relapse and more likely to have an improvement in ED symptoms is still unclear.

3.2.2. *Bulimia Nervosa*

Only one of the included articles proposed a specific staging model for BN [46]. Similarly to the staging model proposed for AN patients, Cosci and Fava [46] in their review suggested a four-stage model of BN, including: (1) prodromal stage; (2) acute manifestation; (3) residual stage; (4) chronic stage, either in attenuated or persistent form. Several symptomatologic criteria, including behavioral, physical, cognitive, social, and psychological symptoms, taken from the available literature [74,75,76], were proposed to describe the BN characteristics in each stage (Fig. 5).

3.2.3. *Binge-eating disorder*

Among the 11 included articles, only one as a theoretical proposal [58] suggested a staging model for BED, including three stages: (1) at risk; (2) recent-onset; (3) established BED (Fig. 6). The main criterion for their stages classification was represented by the level of cognitive functioning, in particular the reward processing alterations in this clinical population, based on existing reward processing models of addiction (i.e., [77]) and binge-type eating disorder (i.e., [78,79]). Specifically, it was suggested that the at-risk stage was characterized by a hypersensitivity to both the anticipatory (*wanting*) and the hedonic (*liking*) aspects of food-related and non-food-related rewards, whereas with the progression of the illness a gradual increase in the value attributed to binge cues (*wanting)* and a stabilization or a decrease of *liking* might be observed. See Fig. 6 for more details.

3.2.4. *Mixed ED diagnoses*

Three of the included papers [47-49], presented the same staging

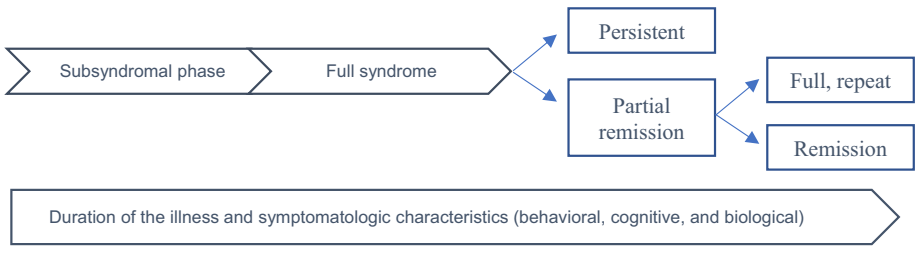

Fig. 4. Steinglass et al., (2020) staging model of AN [57].

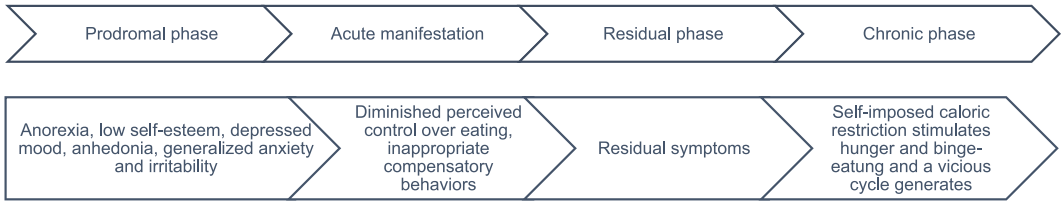

Fig. 5. Cosci and Fava [46] staging model of BN.

Fig. 6. Bodell and Racine [58] mechanistic staging model of BED (Bodell & Racine, 2022).

model for mixed ED diagnoses. Treasure et al. [47,48], in line with the staging model of McGorry et al. [29], proposed in their review and summary respectively a four-stage model of ED including the following stages: (1) high risk; (2) early syndrome; (3) full syndrome; (4) severe enduring illness (Fig. 7). Based on epidemiological studies, neuropsychological findings, treatment responsivity and prognosis in ED, a longitudinal progression of ED illness was suggested according to a four-stage conceptualization. Predisposing factors, differentiated for AN, BN and BED, such as obsessive-compulsive traits, attentional bias and cognitive inflexibility in AN and a family history of obesity in BN and BED, are predominant during the initial stages of the illness of individuals at risk and might increase the vulnerability to develop an ED. Subsequently, the early stage of the illness is characterized by initial weight loss that might be followed by restrictive eating and/or compensatory behaviors. With the progression of the illness, consequences on brain plasticity and neuroadaptation might manifest as a result of starvation or prolonged dysfunctional eating behaviors and might be associated with social isolation and loneliness that in turn might lead to the development of a full syndrome. A chronic phase, severe and enduring illness, might follow, with serious medical, psychological, and social consequences.

3.3. What stage-specific interventions, if any, are suggested in relation to the proposed staging models?

Only three of the included papers [47-49] suggested specific stage-oriented treatments in reference to their proposed staging models for ED. In Treasure et al. [47,48] an overview of stage-oriented treatments for both AN, BN and BED was presented. For the at-risk stage of AN, preventive interventions, such as media literacy, resilience to fat talk, and approaches targeting cognitive dissonance, were recommended. Family-based therapy (FBT) was recommended for the early forms of AN, whereas for the severe and enduring phase a combination of medication, psychotherapy, and inpatient care, together with interventions to improve quality of life, have been shown to be effective. For at-risk stages of BN and BED, interventions targeting body dissatisfaction have been shown to be useful, while cognitive-behavioral therapy (CBT) is recommended across all stages of the illness [47,48].

In the systematic review by Hay and Touyz [49], the aforementioned staging model of ED [47] was adopted with a specific focus on the SE-AN stage. In their work, Hay and Touyz [49] highlighted the clinical utility of reducing psychosocial and neurocognitive disturbances and of de-emphasizing weight regain in the SE-AN stage. More specifically, the use of cognitive-behavioral therapy for severe and enduring AN (CBT-SE) was also supported to improve quality of life, social adjustment, and depression, as well as BMI levels. Other treatment options included transcranial magnetic stimulation (rTMS) and cognitive remediation therapy (CRT), targeting neurocognitive inflexibility, combined with intensive multidisciplinary usual care. Furthermore, an outpatient treatment option named the Anorexia Nervosa Intensive Treatment Team (ANITT; [80]), consisting of a combination of supportive care and specific schema psychological therapies targeting inflexibility, was mentioned for the specific SE-AN stage of AN illness [49].

4. Discussion

The present systematic scoping review aimed to map the existing staging models that explicitly proposed a classification of the progression of the ED illness in distinct stages and to identify their applications and potential knowledge gaps. Nine staging models have been identified in the available ED literature, the majority of which concerned patients with AN [46,50,52,54,56,57], followed by mixed ED diagnoses [47,48], BN [46] and BED [58]. Empirical data on these staging models is still scarce and most of the literature on the topic included theoretical proposals, with only three empirically tested staging models, all of them for AN.

Overall in the available proposed staging models of ED, a consensus on the definition and number of stages and on the criteria used for their conceptualization was lacking. The number of stages ranged from two to five, although most studies identified four phases. The identified proposals of staging models in ED are in line with the view that staging represents a useful approach to better conceptualize the longitudinal progression of an illness, as proceeding along a continuum of severity that goes from an early phase to chronic manifestations of the illness. The inclusion of early and/or subsyndromal stages also allows the identification of non-specific forms of the disorder, which represents one

Fig. 7. Treasure et al., (2015) staging model of eating disorders (AN, BN and BED) (from [47], p. 179; [49]).

of the goals of the staging approach [28]. Detecting at-risk individuals for a certain disorder might indeed be clinically useful to prevent illness progression, as early successful treatments might radically change the prognosis of the disorder [29].

Indeed, more than a half of the nine staging models available (75%) took into account early or high-risk stages in ED. The earlier stages of the illness, such as prodromal [46], subsyndromal [57], mild/incipient [50], early syndrome [52,47], or at-risk [58] were mostly characterized by body-image disturbances or moderate eating restrictions. A premorbid high-risk stage characterized by the presence of predisposing factors (i.e., obsessive-compulsive traits in patients with AN and personal or family history of obesity in BN and BED) has also been proposed [47].

The early phases of the illness are thought to progress to an acute manifestation [46], a full syndrome [47,57], a moderate stage [50], or an acute episode [56], corresponding to full diagnostic criteria. Two models only [46,56] proposed a residual phase following the acute manifestation, as a possible consequence of a partial persistence of the disorder or of an aggravation of pre-existing personality traits as a response to stressful events [46]. Alternatively, at this point of the illness, during which treatment is usually provided, partial or complete remission can be observed as a stage of the ED illness. The remission stage has been proposed in only two of the reviewed staging models [56,57], and described as an absence of restrictive eating and/or binge-purging behaviors for at least 1 year (partial remission) or 3 years (full remission) [57]. Remission might be followed by a relapse phase, either partial or complete, which however has been proposed in one model only without a detailed description of its characteristics [56]. Conversely, in the majority of the staging models it has been acknowledged that in the case of remission failure a more persistent form of the illness might take hold, specifically defined as severe and enduring [47,48,52,54], severe [50], persistent [57], chronic [46,56], or established [58]. One model only proposed a recovery stage, though without providing a detailed description of this stage and its criteria [56].

In line with the existing evidence for other clinical populations with mood disorders and schizophrenia [81], all the proposed staging models of ED included physical/medical, cognitive, behavioral, and social symptoms as criteria of progression of ED illness in all the stages. In terms of physical/medical criteria, BMI has been identified as the only biometrical marker [50,57], whereas a cognitive marker – the level of reward processing alterations – was used as a criterion for the staging classification in BED patients [58]. In a earlier study [47] the persistence of symptoms of ED illnesses has been indeed considered as a consequence of neurobiological mechanisms, as dysfunctional eating behaviors, including fasting, feasting and the oscillation between these behaviors might influence brain development and functioning [47]. Behavioral criteria included for example dietary restriction for AN and binging/purging behaviors for BN [44,46], whereas social symptoms included isolation and failure in establishing autonomy [46,56].

Illness duration has also been identified as a shared severity marker criterion in some proposed staging models in ED. Even though a general consensus was not met on specific time thresholds, authors have found that the mean duration of AN before successful recovery corresponded to seven years, and that the presence of symptoms for more than seven years might lead to or imply the progression to the severe and enduring stage of the illness [52,54,56], whereas specific time thresholds have not been indicated for the other ED diagnoses.

In line with previous studies in other clinical populations, most of the included studies have not empirically tested their staging proposals. Few studies of the proposed ED staging models, in particular of AN, used objective measures specifically developed to classify the stages or other objective measures as empirical evidence of the suggested staging models. The CASIAN was the only objective measure specifically developed and tested for the assessment of staging in AN [50]. Interestingly, in the staging model by Ambwani et al. [52] psychological distress levels were also evaluated through the DASS [53] as a criterion to allocate AN patients in the severe and enduring stage. Similarly, in the staging model by Ramos et al. [54], clinical impairment levels were assessed through the CIA [55] as a possible adjunctive criterion to allocate AN patients into stages (together with illness duration), even though in an exploratory way [54]. By combining illness duration and clinical impairment as criteria for staging classification compared to illness duration only, significant differences between patients in the two different stages emerged, highlighting that clinical impairment might represent an important severity marker in AN [54]. The presence of medical and psychiatric comorbidities might indeed represent an important severity indicator in patients with ED, as it has been found to partially explain mortality and to be associated with greater psychopathology and illness duration [82,83].

However, more explicit operationalizations of criteria for each stage of progression of ED illness is needed in future staging proposals. Moreover, despite the attempt to apply a biopsychological perspective to staging models in ED, in the reviewed staging models of ED there is a scarcity of biological, medical, metabolic and neurocognitive markers used as criteria to describe and differentiate between stages. A better definition of the clinical psychological features not limited to eating disorders related symptomatology is also warranted.

Furthermore, despite the utility that the staging approach might have for treatment planning and the existence of stage-oriented treatments in other clinical populations as well [29,84], only one of the included proposed staging models [47,48] took specific stage-oriented interventions into account for all the phases of ED patients, whereas in another study treatment recommendations were proposed for SE-AN stage only [49]. Despite the lack of empirical data, the proposed treatment recommendations for the most advanced stages matched the clinical guidelines for the treatment of AN, BN and BED [85,86] quite well.

5. Limitations and future directions

The results of the present review must be considered in light of its limitations. Only three databases were used for the selection of the studies, combining a limited number of keywords as per systematic search guidelines. Studies were selected combining “anorexia nervosa, or bulimia nervosa, or binge eating disorder, or eating disorders” AND “staging” and as a result numerous longitudinal studies that provide important information about the possible different stages of the main ED without using “staging” as a keyword were excluded, thus introducing a possible selection bias in the current work. The purpose of the work, however, was not to review the empirical evidence regarding specific stages in ED, such as recovery, relapse, and chronicity, but rather to provide an overview of proposed models of staging in EDs. Furthermore, considering the recent attention given to the addressed topic, a time criterion was used and might have led to the exclusion of other relevant, but less recent works. Despite these considerations, important recommendations can be made.

The current scoping review underscores the need for further empirical research to operationalize staging in AN and to extend staging models to BN and BED populations, since staging might represent a useful clinical tool to place patients with ED at a specific point of the illness and to guide clinicians in the selection of the right treatment at the right time based on evidence-based data.

A comprehensive definition of staging based on the existing multicomponent data and on a biopsychosocial perspective is warranted in future staging conceptualizations and operationalizations of ED. A major focus on medical features, metabolic and neurological modifications, and biomarkers such as those mentioned in clinical guidelines [85,86] that may characterize ED progression [17,87-89] is warranted, as well as the consideration of common psychological symptoms not limited to eating disorder-related ones [54,90]. Alternatively, in line with the most empirically validated model of EDs [12], operationalizing a comprehensive transdiagnostic staging model of ED should be encouraged as

particularly useful for clinical practice. Conducting further systematic reviews in support of specific characteristics and features of each ED stage (high-risk/prodromal/early/incipient, acute/full/moderate, partial or complete remission, severe and enduring/persistent/chronic) is also warranted.

In particular, a better understanding of the prodromal symptoms that usually precede eating disorders and that have been acknowledged in some of the models [52,58,46,50,47] is needed. Common prodromal symptoms have been identified, including eating difficulties, dietary restrictions, fasting, and weight/shape concerns and other psychiatric disorder-related symptoms, such as anxiety, depression, alcohol use, non-suicidal self-injury and childhood trauma [91-93]. However, recent evidence has underlined the need to further explore the role of premorbid psychiatric diagnoses as possible risk factors of longer ED duration or as indicative of a distinct premorbid phase. Despite longitudinal studies not always allowing the differentiation between risk-factors and premorbid signs of the illness [94] research studies should be attempted. Another aspect that should be further explored concerns the description of the phases that might follow the acute phase, namely residual, remission, relapse and recovery stages, while taking into account the literature of reviews, meta-analyses and longitudinal studies on the residual [15], relapse [17,95], remission [96] and recovery [97] phases.

Considering that only three models out of nine provided empirical data, future empirical research is also warranted to test such staging models, especially to better identify stage-oriented treatments and interventions. To implement the conceptualization of staging models in ED, longitudinal studies as well as novel statistical methodologies, such as the network approach which defines mental disorders as a dynamic interplay of symptoms instead of a consequence of a common cause [98-100], might be useful, as has been found in other clinical populations [101]. Temporal networks, a specific class of networks [102,103], provides information about which symptoms predict an increase in other symptoms at a future time point, as well as the strength of these predictive relationships. We recommend future studies to use network models from longitudinal data, both cross-sectional and temporal networks [104,105], to explore the progression of ED illnesses and the related possible staging models. Network models from longitudinal data inform both individual and inter-individual changes over time through the comparison of repeated measurement, with different objectives and results depending on whether cross-sectional or temporal networks are modeled. More in detail, modeling temporal networks from longitudinal data allows retrieving directional network models, in which the connections between nodes show the directionality of the effect, thus informing on the causal relationship between variables [106,107]. On the other hand, cross-sectional networks allow the comparison of network structures obtained from populations with different levels of ED symptom severity or with different illness duration, as has been done in other clinical populations [108-110]. This would provide insight into the temporal stability or changes of a particular configuration of symptoms, providing the groundwork to identify markers for specific stages of the disorder, from prodromal and early stages to severe and enduring. Similarly, sequence analyses and hierarchical clustering might be used to respectively detect underlying patterns in temporally ordered data and characterize patterns of development of the ED illness [83].

Funding

No external funding was provided for the study.

CRediT authorship contribution statement

E. Tomba: Writing – review & editing, Visualization, Supervision, Project administration, Conceptualization. **L. Tecuta:** Writing – review & editing, Writing – original draft, Validation, Resources, Methodology. **V. Gardini:** Writing – review & editing, Visualization. **G. Tomei:** Writing – review & editing, Visualization. **E. Lo Dato:** Writing – review & editing, Writing – original draft, Methodology, Investigation.

Declaration of competing interest

The authors report that there are no competing interests to declare.

Data availability statement

Due to the non-empirical nature of the work (systematic review) no original data were collected for this article.

References

[1] Treasure J, Duarte TA, Schmidt U. Eating disorders. Lancet 2020;395(10227):899–911. https://doi.org/10.1016/S0140-6736(20)30059-3.
[2] Fairburn CG, Harrison PJ. Eating disorders. Lancet 2003;361(9355):407–16.
[3] Diagnostic and statistical manual of mental disorders. 5th ed., text rev. American Psychiatric Association; 2022. https://doi.org/10.1176/appi.books.9780890425787.
[4] World Health Organization. International statistical classification of diseases and related health problems. 11th ed. 2021. https://icd.who.int/.
[5] Tecuta L, Fava GA, Tomba E. An innovative approach for the assessment of mood disturbances in patients with eating disorders. CNS Spectr 2020;25(1):71–8. https://doi.org/10.1017/S1092852919000798.
[6] Mountain D, Shah PJ. Recovery and the medical model. Adv Psychiatr Treat 2008;14:241–4.
[7] Roberts G, Wolfson P. The rediscovery of recovery: open to all. Adv Psychiatr Treat 2004;10(1):37–48. https://doi.org/10.1192/apt.10.1.37.
[8] Schnyder U. Longitudinal development of symptoms and staging in psychiatry and clinical psychology: a tribute to Giovanni Fava. Psychother Psychosom 2022:1–5. Advance online publication. https://doi.org/10.1159/000527462.
[9] Castellini G, et al. Diagnostic crossover and outcome predictors in eating disorders according to DSM-IV and DSM-V proposed criteria: a 6-year follow-up study. Psychosom Med 2011;73(3):270–9.
[10] Keski-Rahkonen A, Mustelin L. Epidemiology of eating disorders in Europe: prevalence, incidence, comorbidity, course, consequences, and risk factors. Curr Opin Psychiatry 2016;29(6):340–5. https://doi.org/10.1097/YCO.0000000000000278.
[11] Udo T, Grilo CM. Psychiatric and medical correlates of DSM-5 eating disorders in a nationally representative sample of adults in the United States. Int J Eat Disord 2019;52(1):42–50. https://doi.org/10.1002/eat.23004.
[12] Fairburn CG, Cooper Z, Shafran R. Cognitive behaviour therapy for eating disorders: a "transdiagnostic" theory and treatment. Behav Res Ther 2003;41(5):509–28. https://doi.org/10.1016/s0005-7967(02)00088-8.
[13] Linardon J, Hindle A, Brennan L. Dropout from cognitive-behavioral therapy for eating disorders: a meta-analysis of randomized, controlled trials. Int J Eat Disord 2018;51(5):381–91. https://doi.org/10.1002/eat.22850.
[14] Cooper Z, Dalle Grave R. Eating disorders: Transdiagnostic theory and treatment. In: The science of cognitive behavioral therapy. Academic Press; 2017. p. 337–57. https://doi.org/10.1016/B978-0-12-803457-6.00014-3.
[15] Tomba E, Tecuta L, Crocetti E, Squarcio F, Tomei G. Residual eating disorder symptoms and clinical features in remitted and recovered eating disorder patients: a systematic review with meta-analysis. Int J Eat Disord 2019;52(7):759–76. https://doi.org/10.1002/eat.23095.
[16] Tomba E, Tecuta L. The sequential approach in eating disorders: A scoping systematic review [published online ahead of print, 2023 Jul 19]. Eur Eat Disord Rev 2023. https://doi.org/10.1002/erv.3013.
[17] Fichter MM, Quadflieg N, Crosby RD, Koch S. Long-term outcome of anorexia nervosa: results from a large clinical longitudinal study. Int J Eat Disord 2017;50(9):1018–30. https://doi.org/10.1002/eat.22736.
[18] Quadflieg N, Fichter MM. Long-term outcome of inpatients with bulimia nervosa-results from the Christina Barz study. Int J Eat Disord 2019;52(7):834–45. https://doi.org/10.1002/eat.23084.
[19] Fava GA, Kellner R. Staging: a neglected dimension in psychiatric classification. Acta Psychiatr Scand 1993;87(4):225–30. https://doi.org/10.1111/j.1600-0447.1993.tb03362.x.
[20] Hetrick SE, Parker AG, Hickie IB, Purcell R, Yung AR, McGorry PD. Early identification and intervention in depressive disorders: towards a clinical staging model. Psychother Psychosom 2008;77(5):263–70. https://doi.org/10.1159/000140085.
[21] Archer T, Kostrzewa RM, Palomo T, Beninger RJ. Clinical staging in the pathophysiology of psychotic and affective disorders: facilitation of prognosis and treatment. Neurotox Res 2010;18(3–4):211–28. https://doi.org/10.1007/s12640-010-9161-7.
[22] Berk M, Post R, Ratheesh A, et al. Staging in bipolar disorder: from theoretical framework to clinical utility. World Psychiatry 2017;16(3):236–44. https://doi.org/10.1002/wps.20441.
[23] Chung N, Langenbucher J, McCrady B, Epstein E, Cook S. Use of survival analyses to examine onset and staging of DSM-IV alcohol symptoms in women. Psychol Addict Behav 2002;16(3):236–42.

[24] McGorry P. A treatment-relevant classification of psychotic disorders. Aust N Z J Psychiatry 1995;29(4):555–8. https://doi.org/10.3109/00048679509064966.
[25] McGorry PD, Nelson B, Goldstone S, Yung AR. Clinical staging: a heuristic and practical strategy for new research and better health and social outcomes for psychotic and related mood disorders. Can J Psychiatr 2010;55(8):486–97. https://doi.org/10.1177/070674371005500803.
[26] Vieta E, Reinares M, Rosa AR. Staging bipolar disorder. Neurotox Res 2011;19(2):279–85. https://doi.org/10.1007/s12640-010-9197-8.
[27] Yung AR, McGorry PD. The prodromal phase of first-episode psychosis: past and current conceptualizations. Schizophr Bull 1996;22(2):353–70. https://doi.org/10.1093/schbul/22.2.353.
[28] Cross SP, Hermens DF, Scott EM, Ottavio A, McGorry PD, Hickie IB. A clinical staging model for early intervention youth mental health services. Psychiatr Serv 2014;65(7):939–43. https://doi.org/10.1176/appi.ps.201300221.
[29] 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. https://doi.org/10.1080/j.1440-1614.2006.01860.x.
[30] Guidi J, Tomba E, Cosci F, Park SK, Fava GA. The role of staging in planning psychotherapeutic interventions in depression. J Clin Psychiatry 2017;78(4):456–63. https://doi.org/10.4088/JCP.16r10736.
[31] McGorry PD, Killackey E, Yung AR. Early intervention in psychotic disorders: detection and treatment of the first episode and the critical early stages. Med J Aust 2007;187(S7):S8–10. https://doi.org/10.5694/j.1326-5377.2007.tb01327.x.
[32] Nelson B, Amminger GP, Yuen HP, et al. Staged treatment in early psychosis: a sequential multiple assignment randomised trial of interventions for ultra high risk of psychosis patients. Early Interv Psychiatry 2018;12(3):292–306. https://doi.org/10.1111/eip.12459.
[33] Linardon J. Predictors, moderators, and mediators of treatment outcome following manualised cognitive-Behavioural therapy for eating disorders: a systematic review. Eur Eat Disord Rev 2017;25(1):3–12.
[34] Södersten P. Treatment outcomes for eating disorders in Sweden: data from the national quality registry. BMJ Open 2019;9(1):e024179. https://doi.org/10.1136/bmjopen-2018-024179.
[35] Strobel C, et al. Long-term outcomes in treated males with anorexia nervosa and bulimia nervosa-a prospective, gender-matched study. Int J Eat Disord 2019;52(12):1353–64.
[36] Berends T, van Meijel B, Nugteren W, et al. Rate, timing and predictors of relapse in patients with anorexia nervosa following a relapse prevention program: a cohort study. BMC Psychiatry 2016;16(1):316. Published 2016 Sep 8, https://doi.org/10.1186/s12888-016-1019-y.
[37] Herzog DB, Dorer DJ, Keel PK, et al. Recovery and relapse in anorexia and bulimia nervosa: a 7.5-year follow-up study. J Am Acad Child Adolesc Psychiatry 1999;38(7):829–37. https://doi.org/10.1097/00004583-199907000-00012.
[38] Strober M, Freeman R, Morrell W. The long-term course of severe anorexia nervosa in adolescents: survival analysis of recovery, relapse, and outcome predictors over 10-15 years in a prospective study. Int J Eat Disord 1997;22(4):339–60. https://doi.org/10.1002/(sici)1098-108x(199712)22:4<339::aid-eat1>3.0.co;2-n.
[39] Fichter MM, Quadflieg N, Hedlund S. Twelve-year course and outcome predictors of anorexia nervosa. Int J Eat Disord 2006;39(2):87–100. https://doi.org/10.1002/eat.20215.
[40] Kazdin AE, Fitzsimmons-Craft EE, Wilfley DE. Addressing critical gaps in the treatment of eating disorders. Int J Eat Disord 2017;50(3):170–89. https://doi.org/10.1002/eat.22670.
[41] Waller G. Treatment protocols for eating disorders: Clinicians' attitudes, concerns, adherence and difficulties delivering evidence-based psychological interventions. Curr Psychiatry Rep 2016;18(4):36. https://doi.org/10.1007/s11920-016-0679-0.
[42] Peters MDJ, Godfrey C, McInerney P, Munn Z, Tricco AC, Khalil H. Chapter 11: Scoping reviews (2020 version). In: Aromataris E, Munn Z, editors. JBI manual for evidence synthesis, JBI; 2020. Available from, https://synthesismanual.jbi.global. 10.46658/JBIMES-20-12.
[43] Tricco AC, Lillie E, Zarin W, et al. PRISMA extension for scoping reviews (PRISMA-ScR): checklist and explanation. Ann Intern Med 2018;169(7):467–73. https://doi.org/10.7326/M18-0850.
[44] Maguire S, Le Grange D, Surgenor L, Marks P, Lacey H, Touyz S. Staging anorexia nervosa: conceptualizing illness severity. Early Interv Psychiatry 2008;2(1):3–10. https://doi.org/10.1111/j.1751-7893.2007.00049.x.
[45] Centre for Reviews and Dissemination. Systematic reviews: CRD's guidance for undertaking reviews in health care. York, UK: University of York; 2006.
[46] Cosci F, Fava GA. Staging of mental disorders: systematic review. Psychother Psychosom 2013;82(1):20–34. https://doi.org/10.1159/000342243.
[47] 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. https://doi.org/10.1111/eip.12170.
[48] Treasure J, Cardi V, Leppanen J, Turton R. New treatment approaches for severe and enduring eating disorders. Physiol Behav 2015;152(Pt B):456–65. https://doi.org/10.1016/j.physbeh.2015.06.007.
[49] Hay P, Touyz S. Treatment of patients with severe and enduring eating disorders. Curr Opin Psychiatry 2015;28(6):473–7. https://doi.org/10.1097/YCO.0000000000000191.
[50] Maguire S, Surgenor LJ, Le Grange D, et al. Examining a staging model for anorexia nervosa: empirical exploration of a four stage model of severity. J Eat Disord 2017;5:41. Published 2017 Nov 27, https://doi.org/10.1186/s40337-017-0155-1.
[51] Maguire S, Touyz S, Surgenor L, et al. The clinician administered staging instrument for anorexia nervosa: development and psychometric properties. Int J Eat Disord 2012;45(3):390–9. https://doi.org/10.1002/eat.20951.
[52] Ambwani S, Cardi V, Albano G, et al. A multicenter audit of outpatient care for adult anorexia nervosa: symptom trajectory, service use, and evidence in support of "early stage" versus "severe and enduring" classification. Int J Eat Disord 2020;53(8):1337–48. https://doi.org/10.1002/eat.23246.
[53] Lovibond PF, Lovibond SH. The structure of negative emotional states: comparison of the depression anxiety stress scales (DASS) with the Beck depression and anxiety inventories. Behav Res Ther 1995;33(3):335–43. https://doi.org/10.1016/0005-7967(94)00075-u.
[54] Ramos R, Vaz A, Rodrigues TF, et al. Severe and Enduring' stage in anorexia nervosa: comparing eating attitudes, impairment and associated psychopathology. Front Nutr 2022;9:867401. Published 2022 Mar 28, https://doi.org/10.3389/fnut.2022.867401.
[55] Bohn K, Fairburn CG. The clinical impairment assessment questionnaire (CIA 3.0). In: Fairburn CG, editor. Cognitive behavior therapy and eating disorders. New York, NY: Guilford Press; 2008. p. 315–8.
[56] Beumont PJ, Touyz SW. What kind of illness is anorexia nervosa? Eur Child Adolesc Psychiatry 2003;12(Suppl. 1):I20–4. https://doi.org/10.1007/s00787-003-1103-y.
[57] Steinglass JE, Glasofer DR, Dalack M, Attia E. Between wellness, relapse, and remission: stages of illness in anorexia nervosa. Int J Eat Disord 2020;53(7):1088–96. https://doi.org/10.1002/eat.23237.
[58] Bodell LP, Racine SE. A mechanistic staging model of reward processing alterations in individuals with binge-type eating disorders. Int J Eat Disord 2023;56(3):516–22. https://doi.org/10.1002/eat.23875.
[59] Higgins JPT, Thomas J, Chandler J, Cumpston M, Li T, Page MJ, Welch VA, editors. Cochrane handbook for systematic reviews of interventions. 2nd ed. Chichester (UK): John Wiley & Sons; 2019.
[60] Moher D, Liberati A, Tetzlaff J. Altman DG; PRISMA group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. Ann Intern Med 2009 Aug 18;151(4):264–9. W64, https://doi.org/10.7326/0003-4819-151-4-200908180-00135. Epub 2009 Jul 20, 19622511.
[61] Bruch H. Perceptual and conceptual disturbances in anorexia nervosa. Psychosom Med 1962;24:187–94. https://doi.org/10.1097/00006842-196203000-00009.
[62] Mitan LA. Menstrual dysfunction in anorexia nervosa. J Pediatr Adolesc Gynecol 2004;17(2):81–5. https://doi.org/10.1016/j.jpag.2004.01.003.
[63] Mitchell JE, Pyle RL, Eckert ED, Hatsukami D, Lentz R. Electrolyte and other physiological abnormalities in patients with bulimia. Psychol Med 1983;13(2):273–8. https://doi.org/10.1017/s0033291700050881.
[64] Pomeroy C, Mitchell JE. Medical complications of anorexia nervosa and bulimia nervosa. In: Fairburn CG, Brownell KD, editors. Eating disorders and obesity. 2nd ed. New York: Guilford Press; 2002. p. 278–85.
[65] Russell G. The present status of anorexia nervosa. Psychol Med 1977;7(3):363–7. https://doi.org/10.1017/s0033291700004323.
[66] Halmi KA. Anorexia nervosa and bulimia. Annu Rev Med 1987;38:373–80. https://doi.org/10.1146/annurev.me.38.020187.002105.
[67] Klein DA, Walsh BT. Eating disorders: clinical features and pathophysiology. Physiol Behav 2004;81(2):359–74. https://doi.org/10.1016/j.physbeh.2004.02.009.
[68] Laségue.. On hysterical anorexia (a). 1873. Obes Res 1997;5(5):492–7. https://doi.org/10.1002/j.1550-8528.1997.tb00676.x.
[69] Brown A, McClelland J, Boysen E, Mountford V, Glennon D, Schmidt U. The FREED project (first episode and rapid early intervention in eating disorders): service model, feasibility and acceptability. Early Interv Psychiatry 2018;12(2):250–7. https://doi.org/10.1111/eip.12382.
[70] Robinson P. Severe and enduring eating disorder (SEED): Management of complex presentations of anorexia and bulimia nervosa. West Sussex, UK: John Wiley & Sons; 2009.
[71] Russell GF, 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. https://doi.org/10.1001/archpsyc.1987.01800240021004.
[72] Touyz S, Le Grange D, Lacey H, et al. Treating severe and enduring anorexia nervosa: a randomized controlled trial [published correction appears in Psychol med. 2013 Dec;43(12):2512]. Psychol Med 2013;43(12):2501–11. https://doi.org/10.1017/S0033291713000949.
[73] Beaufort IN, De Weert-Van Oene GH, Buwalda VAJ, de Leeuw JRJ, Goudriaan AE. The depression, anxiety and stress scale (DASS-21) as a screener for depression in substance use disorder inpatients: a pilot study. Eur Addict Res 2017;23(5):260–8. https://doi.org/10.1159/000485182.
[74] Blake W, Turnbull S, Treasure J. Stages and processes of change in eating disorders: im- plications for therapy. Clin Psychol Psycho- ther 1997;4(186):191.
[75] Jordan PJ, Redding CA, Troop NA, Treasure J, Serpell L. Developing a stage of change measure for assessing recovery from anorexia nervosa. Eat Behav 2003;3:365–85.
[76] Raffi AR, Rondini M, Grandi S, Fava GA. Life events and prodromal symptoms in bulimia nervosa. Psychol Med 2000;30:727–31.
[77] Berridge KC, Robinson TE. Liking, wanting, and the incentive-sensitization theory of addiction. Am Psychol 2016;71(8):670–9. https://doi.org/10.1037/amp0000059.
[78] Bodell LP, Wildes JE, Goldschmidt AB, et al. Associations between neural reward processing and binge eating among adolescent girls. J Adolesc Health 2018;62(1):107–13. https://doi.org/10.1016/j.jadohealth.2017.08.006.

[79] Treasure J, Leslie M, Chami R, Fernández-Aranda F. Are trans diagnostic models of eating disorders fit for purpose? A consideration of the evidence for food addiction. Eur Eat Disord Rev 2018;26(2):83–91. https://doi.org/10.1002/erv.2578.
[80] Munro C, Thomson V, Corr J, et al. A new service model for the treatment of severe anorexia nervosa in the community: the anorexia nervosa intensive treatment team. Psychiatr Bull 2014;38(5):220–5. https://doi.org/10.1192/pb.bp.113.044818.
[81] de la Fuente-Tomas L, Sánchez-Autet M, García-Álvarez L, et al. Clinical staging in severe mental disorders; bipolar disorder, depression and schizophrenia. Estadificación clínica en los trastornos mentales graves: trastorno bipolar, depresión y esquizofrenia. Rev Psiquiatr Salud Ment (Engl Ed) 2019;12(2):106–15. https://doi.org/10.1016/j.rpsm.2018.08.002.
[82] Himmerich H, Hotopf M, Shetty H, et al. Psychiatric comorbidity as a risk factor for mortality in people with anorexia nervosa. Eur Arch Psychiatry Clin Neurosci 2019;269(3):351–9. https://doi.org/10.1007/s00406-018-0937-8.
[83] Van Alsten SC, Duncan AE. Lifetime patterns of comorbidity in eating disorders: an approach using sequence analysis. Eur Eat Disord Rev 2020;28(6):709–23. https://doi.org/10.1002/erv.2767.
[84] Otto MW, Birk JL, Fitzgerald HE, Chauvin GV, Gold AK, Carl JR. Stage models for major depression: cognitive behavior therapy, mechanistic treatment targets, and the prevention of stage transition. Clin Psychol Rev 2022;95:102172. https://doi.org/10.1016/j.cpr.2022.102172.
[85] American Psychiatric Association, editor. Practice guideline for the treatment of patients with eating disorders. 3th ed. Washington, DC, USA: American Psychiatric Association; 2006. https://psychiatryonline.org/pb/assets/raw/sitewide/practice_guidelines/guidelines/eatingdisorders.pdf.
[86] National Institute for Health and Care Excellence. Eating disorders: recognition and treatment. London, UK: NICE Clinical Guideline (NG69); 2017. https://www.nice.org.uk/guidance/ng69/resources/eating-disorders-recognition-and-treatment-pdf-1837582159813.
[87] Bartholdy S, O'Daly OG, Campbell IC, et al. Neural correlates of failed inhibitory control as an early marker of disordered eating in adolescents. Biol Psychiatry 2019;85(11):956–65. https://doi.org/10.1016/j.biopsych.2019.01.027.
[88] Cowdrey FA, Park RJ, Harmer CJ, McCabe C. Increased neural processing of rewarding and aversive food stimuli in recovered anorexia nervosa. Biol Psychiatry 2011;70(8):736–43. https://doi.org/10.1016/j.biopsych.2011.05.028.
[89] Mestre-Bach G, Potenza MN. Potential biological markers and treatment implications for binge eating disorder and behavioral addictions. Nutrients 2023;15(4):827. https://doi.org/10.3390/nu15040827. Published 2023 Feb 6.
[90] Monteleone AM, Cascino G. A systematic review of network analysis studies in eating disorders: Is time to broaden the core psychopathology to non specific symptoms. Eur Eat Disord Rev 2021 Jul;29(4):531–47. https://doi.org/10.1002/erv.2834. Epub 2021 May 3. PMID: 33942439; PMCID: PMC8251923.
[91] Kong S, Bernstein K. Childhood trauma as a predictor of eating psychopathology and its mediating variables in patients with eating disorders. J Clin Nurs 2009;18(13):1897–907. https://doi.org/10.1111/j.1365-2702.2008.02740.x.
[92] McClelland J, Robinson L, Potterton R, Mountford V, Schmidt U. Symptom trajectories into eating disorders: A systematic review of longitudinal, nonclinical studies in children/adolescents. Eur Psychiatry 2020;63(1):e60. Published 2020 May 26, https://doi.org/10.1192/j.eurpsy.2020.55.
[93] Solmi M, Radua J, Stubbs B, et al. Risk factors for eating disorders: an umbrella review of published meta-analyses. Braz J Psychiatry 2021;43(3):314–23. https://doi.org/10.1590/1516-4446-2020-1099.
[94] Jacobi C. Psychosocial risk factors for eating disorders. Eat Disord Rev 2005;1:59–84.
[95] Berends T, Boonstra N, van Elburg A. Relapse in anorexia nervosa: a systematic review and meta-analysis. Curr Opin Psychiatry 2018;31(6):445–55. https://doi.org/10.1097/YCO.0000000000000453.
[96] Khalsa SS, Portnoff LC, McCurdy-McKinnon D, Feusner JD. What happens after treatment? A systematic review of relapse, remission, and recovery in anorexia nervosa. J Eat Disord 2017;5:20. Published 2017 Jun 14, https://doi.org/10.1186/s40337-017-0145-3.
[97] Zerwas S, Lund BC, Von Holle A, et al. Factors associated with recovery from anorexia nervosa. J Psychiatr Res 2013;47(7):972–9. https://doi.org/10.1016/j.jpsychires.2013.02.011.
[98] van Borkulo C, Boschloo L, Borsboom D, Penninx BW, Waldorp LJ, Schoevers RA. Association of Symptom Network Structure with the course of [corrected] depression [published correction appears in JAMA Psychiatry. 2016 Apr;73(4):412]. JAMA Psychiatry 2015;72(12):1219–26. https://doi.org/10.1001/jamapsychiatry.2015.2079.
[99] Borsboom D, Cramer AO. Network analysis: an integrative approach to the structure of psychopathology. Annu Rev Clin Psychol 2013;9:91–121. https://doi.org/10.1146/annurev-clinpsy-050212-185608.
[100] Isvoranu AM, Borsboom D, van Os J, Guloksuz S. A network approach to environmental impact in psychotic disorder: brief theoretical framework. Schizophr Bull 2016;42(4):870–3. https://doi.org/10.1093/schbul/sbw049.
[101] Wigman JT, van Os J, Thiery E, et al. Psychiatric diagnosis revisited: towards a system of staging and profiling combining nomothetic and idiographic parameters of momentary mental states. PLoS One 2013;8(3):e59559. https://doi.org/10.1371/journal.pone.0059559.
[102] Epskamp S, van Borkulo CD, van der Veen DC, et al. Personalized network modeling in psychopathology: the importance of contemporaneous and temporal connections. Clin Psychol Sci 2018;6(3):416–27. https://doi.org/10.1177/2167702617744325.
[103] Jordan DG, Winer ES, Salem T. The current status of temporal network analysis for clinical science: considerations as the paradigm shifts? J Clin Psychol 2020;76(9):1591–612. https://doi.org/10.1002/jclp.22957.
[104] Blanchard MA, Contreras A, Kalkan RB, Heeren A. Auditing the research practices and statistical analyses of the group-level temporal network approach to psychological constructs: a systematic scoping review. Behav Res Methods 2023;55(2):767–87. https://doi.org/10.3758/s13428-022-01839-y.
[105] Burger J, Isvoranu AM, Lunansky G, et al. Reporting standards for psychological network analyses in cross-sectional data. Psychol Methods 2023;28(4):806–24. https://doi.org/10.1037/met0000471.
[106] Bringmann LF, Vissers N, Wichers M, Geschwind N, Kuppens P, Peeters F, et al. A network approach to psychopathology: new insights into clinical longitudinal data. PLoS One 2013;8(4):e60188. https://doi.org/10.1371/journal.pone.0060188. Erratum in: PloS One 2014;9(4):e96588. PMID: 23593171; PMCID: PMC3617177.
[107] Bringmann LF, Pe ML, Vissers N, Ceulemans E, Borsboom D, Vanpaemel W, et al. Assessing temporal emotion dynamics using networks. Assessment 2016;23(4):425–35. https://doi.org/10.1177/1073191116645909 [PMID: 27141038].
[108] Ashaie SA, Hung J, Funkhouser CJ, Shankman SA, Cherney LR. Depression over time in persons with stroke: a network analysis approach. J Affect Disord Rep 2021;4. https://doi.org/10.1016/j.jadr.2021.100131. PMID: 34528021; PMCID: PMC8438599.
[109] McElroy E, Fearon P, Belsky J, Fonagy P, Patalay P. Networks of depression and anxiety symptoms across development. J Am Acad Child Adolesc Psychiatry 2018 Dec;57(12):964–73. https://doi.org/10.1016/j.jaac.2018.05.027. Epub 2018 Sep 26. PMID: 30522742; PMCID: PMC6290121.
[110] Santos Jr HP, Kossakowski JJ, Schwartz TA, Beeber L, Fried EI. Longitudinal network structure of depression symptoms and self-efficacy in low-income mothers. PLoS One 2018;13(1):e0191675. https://doi.org/10.1371/journal.pone.0191675. PMID: 29360876; PMCID: PMC5779701.