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# Towards an Integrated Staging Model for Bipolar Disorders: An International Society for Bipolar Disorders (ISBD) Staging Task Force Consensus Report

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## ABSTRACT

**Objectives:** Three clinical staging models for bipolar disorder (BD) have been published, each with a different but complementary approach and focus. The International Society for Bipolar Disorders Staging Task Force aimed to integrate these models into one comprehensive three-dimensional staging model, separately rating bipolar spectrum, nonmood psychopathology, and (inter-episodic) functional impairment.

**Method:** Via a series of in-person and online meetings, Delphi-surveys, and email discussions, the task force members step-by-step reached consensus on the overall structure of the model and the definition of the various stages.

**Results:** The resulting BD staging model describes the progression of mood psychopathology, nonmood psychopathology, and functional impairment in three independently rated dimensions. The stages of the mood psychopathology are M0 (at risk for BD having a 1st or 2nd degree family member with BD); M1 (prodromal); M2 (first episode classifying for BD-I or BD-II); M3 (recurrent BD); and M4 (chronic unremitting BD), with various substages. Single or recurrent major depression in persons at familial risk for BD is positioned in stage M1. The stages for nonmood psychopathology are: N0 (no symptoms); N1 (subsyndromal symptoms); N2 (one nonmood disorder); N3 (two nonmood disorders); N4 (three or more nonmood disorders). The stages for functional impairment are: F0 (no); F1 (mild); F2 (moderate); F3 (marked); and F4 (severe impairment). Fictitious case vignettes are presented to demonstrate how to apply the model. Limitations of clinical staging are discussed.

**Conclusion:** The proposed three-dimensional clinical staging model for illness progression comprehensively captures the complexity of illness presentation and heterogeneous illness progression in BD, including psychiatric comorbidities and degree of functional impairment. The model provides a framework that requires validation in research and clinical practice.

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## 1 | Introduction

Clinical staging as applied to Psychiatry entails classifying mental disorders based on the natural history or evolution of illness onset and progression [1]. The underlying goals, much like staging used in other medical disciplines such as oncology, are to guide prognosis, treatment, care pathways and discovery research. A clinical staging framework is important for clinical practice and can complement traditional categorical taxonomy, leading to more accurate, fine-grained, developmentally informed assessment and individualized risk prediction. Staging could also help clinicians determine the most appropriate and personalized treatment approach. For example, high-risk and early-stage illness may require less invasive treatment focused on prevention and early intervention, respectively [2]; while advanced stages may require more intensive intervention [3]. Mapping of clinical stages with the natural history of a disorder provides a refined framework to map more directly to biomarkers and underlying pathogenic processes that in turn inform the development of stage-specific interventions to improve quality of remission and prevent progression and the development of more severe and refractory illness [4]. Other important benefits include personalizing risk and prognostic prediction and individualizing the treatment approach [5]. Notwithstanding extensive work in precision psychiatry, we currently do not have frameworks ready for clinical primetime, and staging offers a pragmatic step towards personalized clinical practice.

Current diagnostic classification systems yield broad heterogeneous diagnostic constructs which present an obstacle for advances in both clinical practice and research and do not support clinical staging. The Diagnostic and Statistical Manual of Mental Disorders (DSM-5-TR) and the International Classification of Diseases (ICD-11) rely on a checklist of often nonspecific symptoms, which are open to interpretation and overlap across different disorders. The diagnostic rules allow for many ways that individual patients can meet the same diagnosis. This means that patients who share a diagnosis such as bipolar disorder (BD) may have a different onset [6–8], clinical course [9, 10], quality of remission [11], treatment response [12], and genetic and neurobiological correlates [13, 14]. To clinically stage an illness that will inform prognosis, individual treatment selection, and discovery research, it is necessary to incorporate a range of information such as familial risk, developmental stage, predictive antecedent conditions, and quality of remission or functioning, as well as comorbid conditions into the equation. Otherwise, we are limited to generating broad staging templates across different disorders and heterogeneous subtypes that describe a generic progression of illness (summing up very different illness trajectories) that do not necessarily inform treatment or understanding of the pathogenesis. To develop more refined clinical staging models that map homogenous subtypes with characteristic trajectories and reliable genetic and neurobiological correlates, a much more nuanced, longitudinal, and prospective characterization and assessment of high-risk and patient samples will be required.

Recently, transdiagnostic staging models have been described especially for application in youth mental health, where the evolution of psychopathology and illness trajectories are variable and clinical endpoints are hard to predict [15–17]. Still, despite

overlapping clinical features and neurobiological mechanisms among currently defined major psychiatric disorders such as schizophrenia spectrum-, bipolar-, and recurrent-depressive disorders, adopting a transdiagnostic approach to staging across different heterogeneous psychiatric disorders at this point has significant disadvantages. First, it increases the complexity and heterogeneity of a staging model that already has multiple components. Second, given different antecedent and early symptoms across disorders, applying a generic staging framework may compromise our ability to advance understanding of individual illness pathogenesis or to map specific treatment meaningfully to clinical stages. For example, schizophrenia and related psychotic disorders have neurodevelopmental antecedents and cognitive decline prior to the onset of full-threshold disorder [18]. In contrast, prominent symptoms prior to the onset of BD include anxiety, mood lability, subthreshold manic symptoms, and sleep disturbances [19–22]. In addition, while some early stage phenotypes like depression and anxiety are diagnostically poorly specific, hypomania or mania have tight diagnostic implications even if the earliest presentation. Therefore, we have opted from the start for a BD-specific approach in the current model.

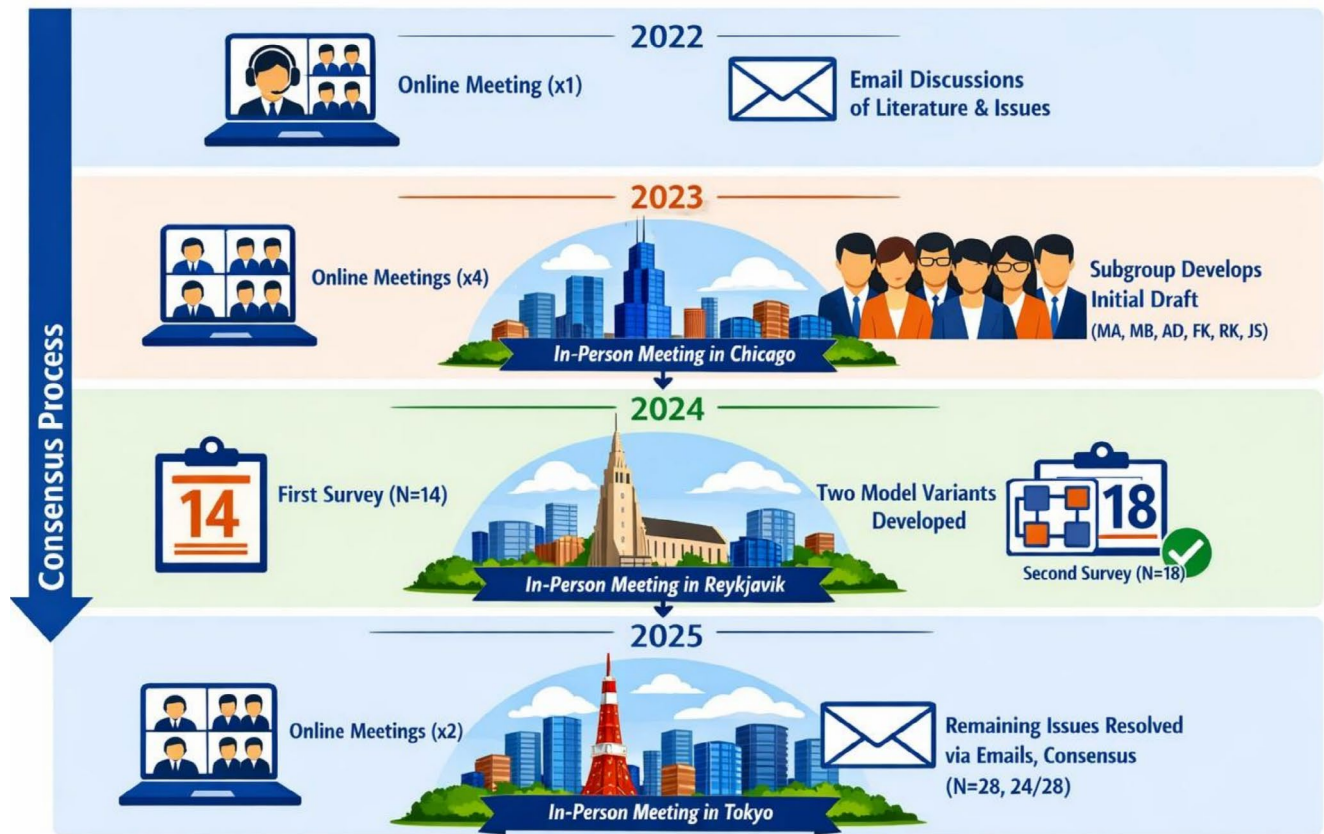
Over the past two decades, three major staging models for BD have been proposed, as described in detail in previous task force papers [23, 24]. These models approach illness progression, and hence staging, from different and complementary perspectives. To summarize, the model by Berk et al. is mainly defined by manic episode onset and mood recurrence [25], the model by Kapczinski et al. by inter-episodic functional impairment [26], while the model by Duffy et al. describes the early developmental trajectories into classical BD or psychotic bipolar spectrum disorder [22, 27]. All three models distinguish between at risk (or latent), prodromal, and clinically manifest BD; but with different emphasis on emerging, established, and chronic, refractory illness.

In our previous task force paper, we have reviewed the evidence for these existing models, proposed a nomenclature for the determinants of illness progression and clinical staging, and anticipated the development of a multidimensional illness-specific clinical staging model for BD [23]. In the current paper, we describe the process to reach consensus among the ISBD Staging Task Force members, and present the resulting clinical staging model for BD, which may serve as a framework for future research. The proposed model combines major ingredients of the existing BD models, and incorporates elements beyond the strict domain of mood psychopathology. Details of how the Task force step by step reached consensus are presented in Tables S1–S6.

## 2 | Methods

The overall consensus process is visualized in Figure 1.

Following the publication of the ISBD Staging Task Force paper addressing a proposed nomenclature for staging [23], members of the task force met online in 2022, 2023, 2024, and 2025, and in person during ISBD conferences in June 2023 (Chicago), October 2024 (Reykjavik), and September 2025 (Tokyo). After discussing various aspects of staging as summarized in the Introduction, during the 2023 in-person



**FIGURE 1** | AI-generated picture of the overall consensus process towards an integrated staging model for bipolar disorder.

meeting the task force decided to develop a BD-specific, multi-dimensional clinical staging model. The model would include (1) “at familial risk but asymptomatic,” “prodromal,” and clinically manifest stages from the perspective of mood disorder psychopathology (originally named “episodes”); (2) nonmood disorder psychopathology (originally named “comorbidity”); and (3) overall functioning between mood episodes, including cognitive functional impairment. These three dimensions are each rated independently. A subgroup of the task force (M.A.; M.B.; A.D.; F.K.; R.K.) discussed some basic issues leading to an initial rough draft of the model, which was further discussed during online meetings. In the preparation of the 2024 in-person meeting, the overall results of these discussions were sent to the entire task force in a first survey including 11 questions about the design of the model to be discussed at that meeting (Table S1). Task force members who would not attend the in-person meeting could give their comments by email (summarized in Table S2). Based on the main points of the discussion at the meeting, and the comments by email, two variants of a three-dimensional staging model were constructed and sent to all task force members (Table S3). This was followed by a second online survey with seven questions addressing the main issues of these alternative models to reach consensus (Table S4). Task force members could add comments to any of these questions (summarized in Table S5). The results of this second survey, and areas of disagreement were further discussed in online meetings in January and April 2025. In these meetings, a considerable degree of consensus was reached for the main structure of the model and the definition of the various stages (summarized in Table S6). Based

on the discussions during and briefly after the September 2025 in-person meeting, consensus was reached on the proposed model, and the results are presented in this paper. Consensus was reached when a majority of task force members agreed with the original statements in the surveys, adapted statements, and subsequent points raised throughout meetings and email correspondence.

### 3 | Results

The 11 questions in the first survey regarding the overall design of the staging model (Table S1) were sent to all  $n=27$  task force members, of whom  $n=14$  gave a response by email (summarized in Table S2). The main comments that were the subject of subsequent discussions concerned: whether a family history of BD is sufficient to define “at risk”; which subsyndromal symptoms can be considered part of a prodromal bipolar spectrum; whether major depression should be regarded as a prodromal stage of BD in individuals at risk, or alternatively as a separate comorbid disorder; whether cognitive impairment should be separated from overall functional impairment; and whether (some) somatic illnesses should be included as comorbid conditions. The in-person meeting in October 2024 was attended by a subgroup of the task force. In this meeting, these questions (Table S1) and responses (Table S2) were discussed.

Subsequently, two alternative staging models with a different position of major depressive episodes without a history of

(hypo)mania in persons at risk for BD (Table S3), together with a second survey, were sent to all  $n = 27$  Task Force members, of whom  $n = 18$  responded (Table S4). Some members provided comments contextualizing their responses (summarized in Table S5). There was substantive consensus on four of the seven questions, but not yet on three issues: the position of major depression without a history of (hypo)mania in persons at risk for BD; separating cognitive from overall functional impairment; and including somatic illnesses as comorbid conditions.

The results of the second survey were discussed in subsequent online meetings to reach optimal consensus, leaving some points of discussion and areas to be further worked out (summarized in Table S6). The main issues that were discussed addressed the early stages of BD, especially the inclusion of subsyndromal symptomatology and its specificity to predict BD in children and adolescents at familial risk. Further differentiating the model based on a life course perspective (children/adolescents versus adults) was considered, but it was agreed that this would make the model too complex for practical clinical applications.

The second dimension was originally named “comorbidity,” and there was extensive discussion whether, next to psychiatric disorders, somatic diseases should be included here. Eventually, the task force majority agreed that this would make the model too complex, apart from the question of which somatic condition would have an impact on the progression of BD.

A similar recurrent issue concerned whether to separate overall functional impairment from cognitive impairment. In general, the task force acknowledged that the model should be precise enough to capture BD illness progression, but not so complex that it cannot be used in clinical practice or even in research. A semi-final draft of the proposed consensus staging model was then presented to all task force members, discussed online and subsequently during an in-person meeting in September 2025. After that meeting it was finalized, resolving remaining issues via email including all task force members. The final model is presented in Table 1.

The proposed three-dimensional BD-specific clinical staging model includes mood psychopathology (M), nonmood psychopathology (N), and the level of functioning when not in an acute mood episode (F). Psychiatric disorders are classified according to DSM-5-TR or ICD-11. The stages 0, 1, 2, 3, and 4 are rated independently for M, N, and F, making any combination possible and thereby reflecting the heterogeneous nature and course of BD.

Stages M0 and M1a/b/c are only applicable for individuals at risk for BD, determined by having a 1st or 2nd degree family member with BD. After some discussion, 2nd degree family members were included, reflecting that familial risk is not just dichotomous but on a continuum. For individuals at risk, having a single or recurrent major depressive episode is considered a potential antecedent for BD, reflected in substage M1b and M1c, respectively. Subsyndromal symptoms within the mood disorder spectrum (homotypic symptoms) are also included in stage M1. Subsyndromal symptoms that do not fall in the mood disorder spectrum (heterotypic symptoms) are positioned in stage N1. The previously proposed definition of symptoms as homotypic or heterotypic [23] was again agreed upon during the consensus process.

Stage M1d includes all DSM-5-TR (and corresponding ICD-11) subthreshold bipolar spectrum disorders, that is, those disorders that have manic symptoms but do not meet the criteria for Bipolar-I-Disorder (BD-I) or Bipolar-II-Disorder (BD-II): Cyclothymic Disorder; Other Specified Bipolar and Related Disorder; Unspecified Bipolar and Related Disorder; Unspecified Mood Disorder; Substance/Medication-Induced Bipolar and Related Disorder; Bipolar and Related Disorder Due to Another Medical Condition. These conditions could be seen as Ultra-High Risk (UHR) for BD-I and BD-II, and thus substage M1d applies beyond individuals at risk as defined above, to anyone having experienced manic symptomatology as defined by these conditions. Note that the broad category of depressive disorders in persons not at risk for BD is not part of this BD-specific staging model.

The consensus of the task force was that BD-I and BD-II should be seen as definite bipolar disorders, thus defining the threshold of stage M2: clinically manifest BD. Stages M2, M3, and M4 thus are applicable for all individuals with an established diagnosis of BD-I or BD-II, regardless of whether they had previously been defined as at risk for BD. By definition, those with major depressive disorder (MDD) or other depressive disorder but not defined as at risk for BD are not classified in stage M1, although if reaching stage M2 they could have had such a diagnosis before their first (hypo)mania. Thus, progression from MDD to BD in individuals not at familial risk for BD falls out of the scope of this BD-specific staging model. Stage M2 includes persons with a first manic episode (BD-I) and those with a first hypomanic episode with previous depression (BD-II). Individuals who only had hypomanic episodes (stage M1d) and then have a first major depressive episode and thus classify as BD-II also enter stage M2. Stage M3 refers to recurrent BD-I or BD-II, without (M3a) or with (M3b) subsyndromal mood symptoms between (hypo)manic and depressive episodes. Note that in stages M3 or M4, a diagnosis of BD-II may change to BD-I, when a first manic episode occurs after a previous hypomania. Stage M4 refers to chronic, unremitting BD, including a rapid cycling course of illness for the duration of at least 2 years.

Nonmood psychopathology may be absent (stage N0), restricted to subsyndromal heterotypic symptoms (N1), or include one (N2), two (N3), or three or more (N4) nonmood psychiatric disorders as defined by DSM-5-TR or ICD-11.

Functional impairment, including cognitive functioning, is rated as absent (F0), mild (F1), moderate (F2), marked (F3), or severe (F4) impairment. In connection to stages M1b/c/d, M2, and M3, functional impairment strictly refers to overall functioning during the interval between (hypo)manic and depressive episodes, which is by definition absent in stage M4.

### 3.1 | Case Vignettes

In Table 2, we present some fictitious case vignettes to illustrate the independent rating of M, N, and F of the staging model. Table 2 also demonstrates how stages can evolve over time within a given individual.

**TABLE 1** | Three-dimensional clinical staging model for Bipolar Disorders, as proposed by the ISBD Staging Task Force.

Stage <sup>a</sup>	M: Mood psychopathology <sup>b</sup>	Stage <sup>a</sup>	N: Nonmood psychopathology <sup>c</sup>	Stage <sup>a</sup>	F: Functional impairment <sup>d</sup>
M0 <sup>e</sup>	Increased risk: 1st or 2nd degree family history of BD and no mood symptoms	N0 <sup>f</sup>	No nonmood symptoms	F0 <sup>f</sup>	No functional impairment
M1 <sup>e</sup>	1st or 2nd degree FH of BD, and: M1a Homotypic subsyndromal symptoms <sup>g</sup> M1b First Major Depressive Episode (DSM-5-TR) <sup>h</sup> M1c Recurrent Major Depression (DSM-5-TR) [9] M1d Subthreshold Bipolar Spectrum <sup>i</sup>	N1 <sup>f</sup>	Subsyndromal nonmood symptoms (heterotypic subsyndromal symptoms <sup>j</sup> )	F1 <sup>f</sup>	Only mild impairment
M2 <sup>e</sup>	First (hypo) manic episode leading to a diagnosis Bipolar Disorder I or II (DSM-5-TR) [9]	N2 <sup>f</sup>	One nonmood DSM-5-TR disorder <sup>h</sup> (specify)	F2 <sup>f</sup>	Moderate impairment
M3 <sup>e</sup>	M3a Recurrence(s) without interepisodic subsyndromal mood symptoms M3b Recurrence(s) with interepisodic subsyndromal mood symptoms	N3 <sup>f</sup>	Two nonmood DSM-5-TR disorders <sup>h</sup> (specify both)	F3 <sup>f</sup>	Marked impairment
M4 <sup>e</sup>	Chronic, nonremitting Bipolar Disorder	N4 <sup>f</sup>	Three or more nonmood DSM-5-TR disorders <sup>h</sup> (specify all)	F4 <sup>f</sup>	Severe impairment

<sup>a</sup>All stages are rated independently for M, N, and F. This staging model is only relevant for persons at risk for bipolar disorder or with established bipolar disorder.

<sup>b</sup>M refers to the symptomatology of bipolar spectrum disorders in the broadest sense.

<sup>c</sup>N refers to any psychiatric disorder apart from depressive and bipolar disorders.

<sup>d</sup>F in case of stage M2 and M3: functioning after symptomatic recovery of most recent mood episodes.

<sup>e</sup>Stages M0 and M1a,b,c are only relevant for persons at risk for bipolar disorder, while stages M1d, M2, M3, and M4 are relevant for persons with established bipolar (spectrum) disorder.

<sup>f</sup>All stages in N and F are relevant for both persons at risk for bipolar disorder and persons with established bipolar disorder.

<sup>g</sup>Homotypic: mood lability, mood elation, irritability, mood swings, subsyndromal depressive or hypomanic symptoms.

<sup>h</sup>Disorders may be correspondingly classified according to ICD-11.

<sup>i</sup>Stage M1d (Subthreshold Bipolar Spectrum) includes DSM-5-TR (and similar ICD-11) bipolar spectrum disorders that do not meet the criteria for BD-I or BD-II: Cyclothymic Disorder; Other Specified Bipolar and Related Disorder; Unspecified Bipolar and Related Disorder; Unspecified Mood Disorder; Substance/Medication-Induced Bipolar and Related Disorder; Bipolar and Related Disorder Due to Another medical Condition.

<sup>j</sup>Heterotypic (nonspecific for mood disorders): for example, anxiety, sleep disturbance, behavior disturbance, substance use.

## 4 | Discussion

The staging model for BD presented in this paper, developed by the ISBD Staging Task Force, aims to integrate three existing staging models for BD that took different but complimentary approaches to mapping the developmental trajectory and progression of BD, as we anticipated in a previous paper of the task force [23]. Moreover, given the heterogeneous psychopathological contexts in which BD often manifests, either seen as “nonmood antecedents” [22, 27, 28], “comorbidities” in DSM-5-TR [29], or “illness extension” [17], we added the dimension of “nonmood psychopathology.” Functional impairment that patients may experience in the interval after symptomatic recovery from (hypo)manic or depressive episodes, which was already part of the staging model by Kapczinski et al. [26], and may result not only from BD itself but also from comorbid psychiatric conditions, is separately addressed in the third dimension of the

proposed model. Taken together, this resulted in a multidimensional clinical staging model of neuroprogression [30] like staging models in somatic medicine, for example, the TNM model in oncology that also reflects various aspects of illness progression and is regularly updated when new evidence is available [31, 32].

During the consensus process that took some 3 years and was the result of in-person meetings at yearly ISBD conferences in 2023, 2024, and 2025, online meetings in-between, two surveys, and numerous discussions by email (see the [Supporting Information](#) for details), the task force reached step-by-step consensus on the most important features of the model: the choice for a BD-specific model rather than a transdiagnostic model that was already decided from the start was confirmed by the task force; the overall structure in three dimensions; rating these three dimensions independently; the definition of being “at risk” defined by family history; the position of major

**TABLE 2** | Fictitious case vignettes to illustrate the independent rating of the three dimensions of the proposed MNF staging model for bipolar disorders.

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Maria, 21, father with bipolar-I disorder; without mental health problems; and functioning well:

M0–N0–F0

Jennifer, 16, grandmother with bipolar-I disorder; suffering from minor depressive symptoms and eating difficulties; causing some problems at school:

M1a–N1–F1

David, 32, brother with bipolar-I disorder; suffering from his second major depression; but functioning well between episodes:

M1c–N0–F0

Lara, 28, no family history of BD; now recovered from her first manic episode; but at the time still unable to return to her work, mainly because of her long-standing alcohol use disorder:

M2–N2–F2

Ella, 46, father with BD-I; having had two depressive and one manic episode; and now functioning well with lithium:

M3a–N0–F0

Henry, 51, no family history of BD, having BD-I since age 21, with rapid cycling for four years despite treatment; comorbid alcohol use disorder and panic disorder; since one year living in a sheltered care home:

M4–N3–F4

*Example of illness progression*

Joe, at 14, mother with bipolar-II disorder; no mood symptoms; diagnosed with ADHD; and functioning well with methylphenidate:

M0–N2–F0

Joe, at 21, previously diagnosed with ADHD; now had a first major depression, with full recovery; functioning well as before:

M1b–N2–F0

Joe, at 24, previously diagnosed with ADHD and major depression; now had a first manic episode; with mild cognitive problems some months after full recovery:

M2–N2–F1

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depression as part of BD evolution in individuals at risk; and either BD-I or BD-II as the threshold for the diagnosis of BD and thus stage M2.

We chose to focus on a BD-specific staging model due to the complex episodic nature of the disorder including two distinct although overlapping clinical syndromes (mania and depression) and thereby making it less comparable to other psychiatric disorders. The resulting three-dimensional model (mood psychopathology, nonmood psychopathology, and overall functioning between episodes) bears similarity to the transdiagnostic staging framework for youth mental health as described by Scott et al. [16] that has separate dimensions for symptoms, functioning, and neurocognition. That framework obviously does not differentiate between mood and nonmood psychopathology, but does differentiate between functioning and neurocognition. For reasons explained, our task force decided to include cognition into overall functioning. While from the start we have opted for a BD-specific approach in the current model, there may be opportunities for expanding this work in the future to predict transdiagnostic risk through collaborations with experts in psychosis and depression.

Apart from a positive 1st or 2nd degree family history of BD, being “at risk” in stage M0 and M1 may be further specified, for example, by using a risk calculator [33–36].

There was extensive discussion about the positioning of major depression in individuals at risk for BD, whether this should be rated as a comorbid condition (stage N2, N3, or N4) or as an early stage of BD (stage M1). Based on extensive research evidence among offspring of parents with BD [22, 27, 28, 37–40] and consensus discussion, the task force opted for the latter and included stages M1b and M1c.

Regarding nonmood psychopathology, only the total number of disorders was considered, although it must be considered that one single disorder such as severe substance abuse may have more impact than two relatively mild disorders such as ADHD and specific phobia. There was discussion among the task force members with regard to somatic conditions that may impact on the course of BD and whether these should be included as comorbidities [41]. However, given the uncertainty surrounding the clinical relevance of somatic conditions on the course and outcome of BD, and that their inclusion would increase

the complexity of the model, the consensus was to include only DSM-5-TR (or corresponding ICD-11) nonmood psychiatric disorders and not somatic conditions. Similarly, after in-depth discussions, cognitive functional impairment was not separately defined from overall functional impairment. The main argument was that the model must be feasible for implementation in daily clinical practice, where specific cognitive dysfunctions may not be easily distinguishable from overall functioning and neuropsychological testing may not be available. Given the previous point, functional impairment clearly caused by a somatic illness is not taken into account. Although there is substantial evidence now that the risk factors and operative biological pathways driving neuroprogression overlap with those for somato-progression [42], the progression of comorbid physical disorders, the pragmatic compromise was made not to include physical probability in this model.

Developing a staging model for any psychiatric disorder is a complex process involving several compromises and the outcome therefore has several limitations, apart from discussions about transdiagnostic versus disorder-specific models. First, given the still limited understanding of the etiopathogenesis and biological mechanisms for psychiatric disorders including BD, and therefore the lack of well-defined biomarkers for any of the stages, such models are limited to clinical staging models, dependent on clinical presentation and longitudinal course, rather than pathological staging models such as the TNM model in oncology. Second, the model presented here is primarily based on expert consensus and only in part on research evidence. Consensus was reached when a majority of task force members agreed with the original statements in the surveys, adapted statements, and subsequent points raised throughout meetings and email correspondence. Major areas of dissenting opinions were whether major depression without a history of (hypo)mania should be seen as an early stage of bipolar disorder in individuals at familial risk, including somatic diseases in the model, and differentiating between cognitive and overall functional impairment. Although parts of the model are well-informed by extensive research (often performed by members of the Task force), as summarized elsewhere [23], the feasibility and validity of the model must be tested in future studies before it can be applied as a tool that helps to guide treatment in daily clinical practice.

The section on functional impairment is only defined globally as absent, mild, moderate, marked, or severe. How these stages should be defined needs further exploration depending on the context in which the model is used. There are many transdiagnostic and disorder-specific scales available to assess functioning in mental illness [43, 44]. In more general clinical settings for patients with BD, functioning may be based on an easy-to-use rating scale such as the FAST [45] or the WHODAS [46], while in more specialized clinical or research settings this may be complemented by the results of neuropsychological testing. Another aspect to be considered for further developments of this model would be the inclusion of resilience and cognitive reserve measures [47] that may be particularly helpful at early stages [48].

Although ideally staging would be based on the natural evolution and course of illness, when applied to individuals in clinical practice or research settings it reflects the observed

(“naturalistic”) rather than the natural unmodified illness progression. In the model proposed here, the impact of any current or past treatment is not taken into account given the large variation within and among patients in any stage. When introducing and applying staging models in clinical practice, to prevent therapeutic pessimism it is important to emphasize that not everyone with the disorder will inevitably proceed through all stages. Moreover, the natural course of BD illness, or its modification by treatment, may result in reversing from stage M4 (chronic) to stage M3 (recurrent) [49]. Likewise, over time there may be recovery from comorbid disorders, as well as improvement in overall functioning, leading to a lower N and M stage respectively.

## 5 | Conclusion

The presented BD-specific clinical staging model for illness progression more comprehensively captures the complexity of the clinical presentation and heterogeneous illness progression in BD. It includes the development of core bipolar symptomatology, psychiatric comorbidities, and degree of functional impairment between mood episodes. The model needs to be tested and validated in research before it can be applied in clinical practice. As a result of future research, including clinical trials that systematically include staging as one of the parameters of illness course, these models may be complemented by findings on stage-specific biomarkers and recommendations for personalized, stage-specific treatment or intervention.

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### Affiliations

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## Data Availability Statement

Data sharing is not applicable to this article as no new data was created or analyzed in this review paper.

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### Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Table S1:** First round of open questions to the ISBD Staging Taskforce members to prepare an in-person meeting in October 2024, requesting a response by email to the Taskforce chair (RK). **Table S2:** Summary of main comments by ISBD Staging Taskforce members to the questions in Table 1 (some questions raised no or only minor comments, which are not included in this table). **Table S3:** Two variants of a 3-axial staging model for bipolar disorder, constructed after the in-person Taskforce meeting in October 2024, with different positioning of depressive episodes in the absence of a history of (hypo)mania, in persons at risk for bipolar disorder. Each axis is rated independently. **Table S4:** Results of the second survey held among  $n=28$  ISBD Staging Taskforce members in November 2024, of whom  $n=18$  responded. **Table S5:** Summary of comments by ISBD Staging Taskforce members to the questions raised in the second survey presented in Table 4. **Table S6:** Areas of agreement and further discussion among Taskforce members and for future development.