



# Idiographic network analysis of discrete mood states prior to treatment

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## Abstract

Idiographic network models based on time-series data have received recent attention for their ability to model relationships among symptoms and behaviours as they unfold in time within a single individual (cf. Epskamp, Borsboom, & Fried, 2018; Fisher, Medaglia, & Jeronimus, 2018). Rather than examine the correlational relationships between variables in a sample of individuals, an idiographic network examines correlations within a single person, averaged over many time points. Because the approach averages over time, the data must be stationary (i.e. relatively consistent over time). If individuals experience varying states over time—different mixtures of symptoms and behaviours in one moment or another—then averaging over categorically different moments may undermine model accuracy. Fisher and Bosley (2019) address these concerns via the application of Gaussian finite mixture modelling to identify latent classes of time points in intraindividual time-series data from a sample of adults with major depressive disorder and/or generalised anxiety disorder ( $n = 45$ ). The present paper outlines an extension of this work, wherein network analysis is used to model within-class covariation of symptoms. To illustrate this approach, network models were constructed for each intraindividual class identified by Fisher and Bosley (137 networks across the 45 participants, mean classes/person =  $\sim 3$ , range = 2–4 classes/person). We examine the relative consistency in symptom organisation between each individual's multiple mood state networks and assess emergent group-level patterns. We highlight opportunities for enhanced treatment personalisation and review nomothetic patterns relevant to transdiagnostic conceptualisations of psychopathology. We address opportunities for integrating this approach into clinical practice and outline potential shortcomings.

## KEYWORDS

ecological momentary assessment, Gaussian finite mixture modelling, idiographic, latent profile analysis, network analysis

## 1 | INTRODUCTION

In the field of clinical psychology, idiographic methods have been recently recognised as central to understanding individual differences in psychopathology and developing personalised interventions (Wright & Woods, 2020). While the importance of carefully evaluating the individual in order to personalise treatment is not new (cf. cognitive-behavioural case formulation; Persons, Curtis, & Silberschatz, 1991), current idiographic methods offer quantitative tools for doing so with improved precision (Fisher, 2015). Modelling phenomena on a person-by-person basis enables the discovery of within-person, clinically relevant patterns that are typically obscured in group-level models (cf. Fisher, Medaglia, & Jeronimus, 2018). Researchers using idiographic methods have demonstrated the myriad ways in which patterns of thoughts, feelings and behaviours are often deeply idiosyncratic and person-specific (Fisher, 2015; Fisher, Reeves, Lawyer, Medaglia, & Rubel, 2017). Modelling idiographic patterns helps not only to characterise the stark differences *between people*, but also to explore the degree to which intraindividual processes fluctuate and vary across time—that is, how people vary *within themselves* across time.

To date, idiographic approaches have primarily employed statistical models that require data exhibiting stationarity—the maintenance of relatively stable statistical properties across time (Fisher & Bosley, 2019; Fisher et al., 2019). Stationarity implies that all data from the observation period may be treated as representative of the individual generally, at all time points. While detrending approaches can correct for nonstationarity resulting from global shifts in the mean (e.g. a recovery trajectory during a treatment study), correcting for local variation from hour to hour or day to day is more difficult (Fisher & Bosley, 2019; Rabinowitz & Fisher, 2020). This is a potentially significant issue as individuals exhibit different emotional ensembles, behavioural repertoires and cognitive schemas at different points in a given day, week or month. Approaches must be developed that can identify distinct patterns of functioning in intraindividual time-series data.

One such approach was recently presented by Fisher and Bosley (2019), who used Gaussian finite mixture modelling—often referred to as latent profile analysis (LPA)—to identify latent classes of time points in intraindividual affect data. LPA is typically used to locate latent *groups* of response profiles in a sample of individuals, but when applied to time-series data, the analysis returns discrete states or moods. Although the LPA reflects profiles of rank-order differences in the levels of affect variables, it does not reflect the interdependence between variables. Given concerns about nonstationarity, and evidence from Fisher and Bosley that individuals can exhibit distinguishably and significantly different states, it stands to reason that differential patterns of covariation may exist in intraindividual time-series data.

### 1.1 | The present approach

This paper represents an extension of Fisher and Bosley's work. The methods outlined in their 2019 paper provide a road map for

identifying latent mood states within intraindividual time-series data. Here, we outline methods for applying network analysis to these mood states in order to model idiosyncratic patterns and connections among the symptoms. First, we provide requisite background on network analysis. Next, to illustrate the clinical utility of this combined LPA/network approach, we take each intraindividual time-series data set collected by Fisher and Bosley, divide it according to the latent mood states identified in their 2019 paper and apply network analysis to each mood state. We highlight how the present approach may (a) enhance treatment personalisation for individuals; and (b) reveal generalisable patterns in the within-person symptom dynamics useful for transdiagnostic categorisation of psychopathology.

### 1.2 | Network analysis

Recently, network analysis has gained considerable traction in psychology. Researchers have suggested a network theory of mental health, conceiving of mental disorders as groups of causally connected symptoms (Borsboom, 2017). In psychological networks, *nodes* represent observed phenomena (thoughts, feelings or behaviours) and *edges* represent covariation between nodes. Mechanisms underlying these relationships may range from the biological to the societal, but once the connections are strong enough, the network may become self-sustaining (Borsboom, 2017). Networks may be directed, with edges that delineate the temporal direction of node relationships, or they may be undirected, with edges that do not specify relationship direction. Undirected networks have been applied to explore a range of psychopathologies using cross-sectional data, including post-traumatic stress disorder (McNally et al., 2015), schizophrenia (Strauss et al., 2019) and major depressive disorder (MDD) (Borkulo et al., 2015). Networks have also been applied to treatment target identification: standardised measures of centrality may enable evaluation of the relative importance of each node to the integrity of the system (Rubel, Fisher, Husen, & Lutz, 2018). The more well-connected a node, the more likely its disruption may destabilise the overall system, which, for psychopathology, is often the goal (Epskamp, Borsboom, & Fried, 2018).

Network density—the ratio of existing edges to total number of possible edges—has emerged as a potential indicator of illness persistence. Borkulo et al. (2015) showed that those with unremitting MDD at a 1-year follow-up visit exhibited higher levels of baseline MDD network density compared to those with remitting MDD, while controlling for mean symptom levels. In PTSD, network density has been associated with the persistence of symptoms well beyond the duration of the initial stressor, a phenomenon termed 'hysteresis' (McNally, 2017). In sum, interconnectedness among symptoms appears linked to their severity and persistence, across multiple syndromes.

Idiographic network models based on time-series data have received attention due to their ability to model the dynamic relationships among symptoms and behaviours as they unfold over

time within a single individual (cf. Epskamp et al., 2018; Fisher et al., 2018). Rather than examine the correlational relationships between variables across a sample of individuals, an idiographic network examines these correlations in a single person, averaged over many time points. As demonstrated by Fisher et al. (2017), idiographic networks permit a level of granularity impossible at the nomothetic level: in the sample under examination in the present study, Fisher et al. found that the most central nodes at the nomothetic level were inconsequential for many participants at the idiographic level. In these cases, treatment designed based on the nomothetic findings would be insufficient for true personalisation.

The present approach offers an application of idiographic network analysis wherein the potential for personalisation is further increased via the preliminary step of dividing each individual's time series into subsets based on the LPA-identified mood states. This step assuages concerns regarding nonstationarity of data and enables each time series to be described with multiple networks, offering a novel and important level of precision to our understanding of an individual's symptom experience.

### 1.3 | Example study

To further explicate our approach, we present an example study below, which is a secondary analysis of Fisher et al. (2017) and Fisher and Bosley (2019).

## 2 | METHOD

### 2.1 | Participants

Participants were 45 individuals with primary diagnoses of generalised anxiety disorder (GAD,  $n = 23$ ), MDD ( $n = 11$ ) or both ( $n = 11$ ) who were enrolled in an open trial of a personalised cognitive-behavioural intervention for mood and anxiety disorders.

Study clinicians conducted structured clinical interviews to assess diagnosis, and eligible participants completed 30 days of self-reported ecological momentary assessment (EMA) surveys four times a day. These intensive repeated-measures pretreatment data sets (one per person) were utilised in the present analyses. Further details of the study are described in detail elsewhere (Fisher et al., 2019).

### 2.2 | Measures

#### 2.2.1 | Hamilton Anxiety Rating Scale (Hamilton, 1959)

This 14-item clinician-administered scale provides a rating of 0 (not present) to 4 (very severe) for each symptom cluster. Internal consistency is excellent (0.92; Kobak, Reynolds, & Greist, 1993).

#### 2.2.2 | Hamilton Rating Scale for Depression (Hamilton, 1960)

This 13-item clinician-administered scale provides a rating of 0 (not present) to 4 (very severe) for each symptom cluster.

#### 2.2.3 | Experience sampling surveys

Once enrolled, participants began receiving 22-item surveys via text message. Items assessed symptoms of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition criteria for GAD and MDD, and additional items intended to measure negative/positive affect and behaviour. Participants rated their current experience of each item using a visual analogue slider ranging from 0 (not at all) to 100 (as much as possible). Survey timing was personalised: The 12 hr period proceeding each participant's self-reported wake-up was divided into four equivalent windows, and one survey was delivered at quasi-random within each window (while guaranteeing each survey was at least 30 min apart), for a total of four time-stamped surveys per day. Incomplete surveys expired at the end of each window (see Fisher & Boswell, 2016, for details).

### 2.3 | Data preparation and analysis

For the LPA, we used a subset of 10 items from the EMA surveys that capture negative mood associated with MDD and GAD, and accompanying avoidance behaviour: irritable, angry, afraid, worried, down and depressed, hopeless, rumination, loss of interest or pleasure (i.e. anhedonia), avoiding activities and avoiding people. For the *network analysis* of LPA-identified mood states, we further reduced our subset to six (angry, worried, down, hopeless, anhedonia and avoiding people) due to concerns about power in analyses with fewer than 30 observations.

#### 2.3.1 | Idiographic Gaussian finite mixture modelling to identify mood states

Gaussian finite mixture model analyses were applied on a person-by-person basis to each participant's time series containing the 10 selected negative affect and avoidance items. Consistent with the field, we refer to these models as LPA (Muthén & Muthén, 2014; Rosenberg, Beymer, Anderson, & Schmidt, 2018). Our approach differs from the traditional application of LPA: when applied to a nomothetic, cross-sectional data set, LPA yields latent groups of individuals. Here, we applied LPA separately to each individual participant's time series, resulting in a set of unique latent classes composed of clusters of time points. We conceptualise these classes as latent *states*, the number and composition of which vary from person to person.

Analyses were conducted using the *mclust* package in R (Scrucca, Fop, Murphy, & Raftery, 2016). The final number of latent mood classes was determined using four criteria: the Bayesian information criterion (BIC; Schwarz, 1978), the integrated

completed likelihood (Biernacki, Celeux, & Govaert, 2000), the bootstrap likelihood ratio test (Nylund, Asparouhov, & Muthén, 2007) and the sample proportion for the smallest class. See Fisher and Bosley (2019) for complete details. Individual time series were populated with dummy codes indicating presence/absence of each mood state at each time point.

### 2.3.2 | Network analysis of individual classes

We created subsets of each participant's time series based on the LPA-identified mood states. For each mood state, we estimated a contemporaneous network model, with nodes representing survey items and edges representing partial correlations between items. We used the package *mgm* in R (Haslbeck, 2019) for network estimation, employing the *mgm* function to estimate a pairwise mixed graphical model with elastic net regularisation. The extended BIC was used to select the lambda parameter. Alpha was specified for each analysis. Each model was first run with  $\alpha = .50$  to specify an equal mix of the two regularisation techniques: the least absolute shrinkage and selection operator (lasso; Tibshirani, 1996) and ridge regression. Both lasso and ridge penalise coefficients but differ in their approach: lasso penalises the sum of the coefficients' absolute values ( $L_1$  penalty), while ridge penalises the sum of squared coefficients ( $L_2$  penalty). In terms of the inclusion versus exclusion of model parameters, lasso is the more conservative approach, given its ability to penalise coefficients to zero, eliminating them from the model and creating relatively sparser networks. Ridge regression is the more liberal approach, leaving relatively more edges in the estimated network. For all classes exhibiting empty networks at  $\alpha = .50$ , we adjusted the lasso-to-ridge ratio by decreasing alpha by increments of .05 to introduce proportionally more ridge until at least one edge was present. For 39 of the 45 participants, all networks were estimated with alpha at .50. Four participants had one network with alpha < .50 (P003: class 3 alpha = .25; P113: class 1 alpha = .05; P127: class 4 alpha = .45; P217: class 2 alpha = .20). One participant had two networks estimated with alpha < .50 (P169: class 3 alpha = .35, class 4 alpha = .15).

After model estimation, we visualised each network with the *qgraph* function from the *qgraph* package in R (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012). With the *centrality* function, also from *qgraph*, we calculated centrality measures for each network. Using the *network.density* function from the *network* package (Butts, 2008), we calculated network density, the ratio of existing edges to total number of possible edges.

## 3 | RESULTS

### 3.1 | Idiographic latent class analysis to identify mood states

The mixture models of the majority ( $n = 23$ , 51%) of participants resulted in three classes. The remainder exhibited two-class ( $n = 10$ ,

22%) or four-class ( $n = 12$ , 27%) profiles. Forty-seven per cent ( $n = 21$ ) of participants exhibited a clearly defined, elevated-symptom-level class—marked by uniform elevations across all symptoms—and an inverse, decreased-symptom-level class. Specifically, these classes were defined as *elevated* when the mean score of each item was higher than in all other classes for a given individual, and *decreased* when the mean score of each item was the lowest across all of the individual's classes.

### 3.2 | Network analysis of individual classes

In total, there were 137 classes across the 45 participants, with an average of nearly three classes per individual and a range of two to four classes. At the within-subject level, network models were heterogeneous and distinct, exhibiting variation in total number, strength and/or edge valences. Across participants, two patterns emerged: (a) the reversal of edge valence from class to class within persons (i.e. an inversion of the direction of relationship between two nodes); and (b) a positive correlation between class severity and network density between persons.

#### 3.2.1 | Inverted edges

Over half of the participants (53%,  $n = 24$ ) had at least one edge that was positive in one class network and negative in another, representing a persistent but class-varying relationship between two symptoms. Of participants with at least one instance of edge inversion, 33% (8) had two, 21% (5) had three, and 4% (1) had four. The most commonly inverted edges, each of which occurred for five different participants, were between 'worried' and 'anhedonia', 'worried' and 'angry', 'worried' and 'hopeless', and 'down' and 'avoiding people'. Thus, worry may be a catalytic or potentiating phenomenon for these individuals, differentially driving symptom expression under different conditions. It is equally possible that these inversions represent the presence of an unmodelled collider (de Ron, Fried, & Epskamp, 2020), one that may have a specific, mechanistic relationship with worry.

#### 3.2.2 | Correlation between severity and network density

Visual inspection of networks revealed a potential pattern, wherein elevated-symptom classes appeared to exhibit greater network density than that of decreased-symptom classes. Thus, we conducted a post hoc analysis to examine whether class severity significantly predicted network density. This analysis was restricted to the 21 individuals with distinct elevated- and decreased-symptom profiles, as defined by the criteria established above. Results revealed a significant difference in network density between the elevated-symptom classes ( $M = .45$ ,  $SD = .17$ )

and decreased-symptom classes ( $M = .39$ ,  $SD = .22$ ),  $t = -2.80$ ,  $p = .01$ .

### 3.3 | Exemplars

We selected the following two exemplars to showcase the clinical utility of the combined LPA and network analysis results. Results for all participants, including original data and code, are available at [https://osf.io/nfqcp/?view\\_only=1db193ef1dc045e48b9edc6aa1b1f9b3](https://osf.io/nfqcp/?view_only=1db193ef1dc045e48b9edc6aa1b1f9b3).

#### 3.3.1 | Participant 074

Participant 074 was a 56-year-old White woman with a primary diagnosis of MDD and no secondary diagnoses. The LPA revealed a three-class profile for this participant, with an elevated-symptom class and a decreased-symptom class (Figure 1). Network analysis (Figure 2) revealed class 3 (decreased-symptom class with low levels of all symptoms; 40% of all observations, or 48/119) shared no edges with class 1 (elevated-symptom class with high levels of all symptoms; 26% of all observations, or 31/119) or with class 2 (a mid-level class with all symptom levels situated between those in the elevated and decreased classes; 34% of all observations, or 40/119). Neither of the two correlations present in class 3 (partial correlations: anhedonia/anger  $r = .33$ ; anhedonia/hopeless  $r = .24$ ) were present in class 1 or 2. Whereas worry was not correlated with any other items in class 3, both classes 1 and 2 exhibited partial correlations between worry and anhedonia, as well as between worry and feeling down (class 1: worry/anhedonia  $r = .45$ , worry/down  $r = .26$ ; class 2: worry/anhedonia  $r = .25$ , worry/down  $r = .55$ ). Consistent with the post hoc  $t$  test reported above, this participant's elevated-symptom network density (.60) was greater than the density of their decreased-symptom network (.33), which may reveal information about the comparative persistence or inertia of these two mood states.

#### 3.3.2 | Participant 007

Participant 007 was a 33-year-old Black woman with comorbid GAD and MDD, and secondary diagnoses of agoraphobia, social anxiety and specific phobia. Contrary to many of the two-class solutions in the present sample, participant 007 did not exhibit a class with a universally low symptom profile. Instead, the two-class profile for this participant was defined by a distinct anger state (52/151 observations; 34%) and a separate state with mixed anxious and depressive symptoms (99/151 observations; 66%). These class distinctions alone suggest meaningful within-person variation in mood across time. Network analysis results add further nuance: in class 1 (elevated anxiety and depression), we see a positive partial correlation between 'worried' and 'angry' ( $r = .21$ ), while in the class 2 'anger state', this edge is inverted ( $r = -.65$ ). In other words, when in a state of elevated depressive negative affect, this individual's worry and

anger have a small, positive relationship, while in a state marked by anger and an *absence* of other negative affect, anger is more strongly and negatively associated with worry.

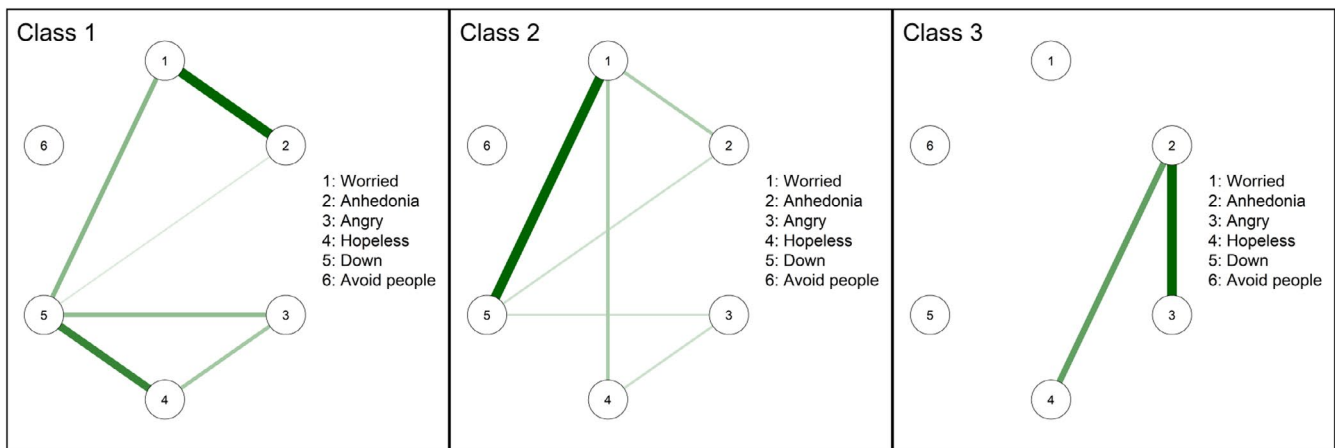
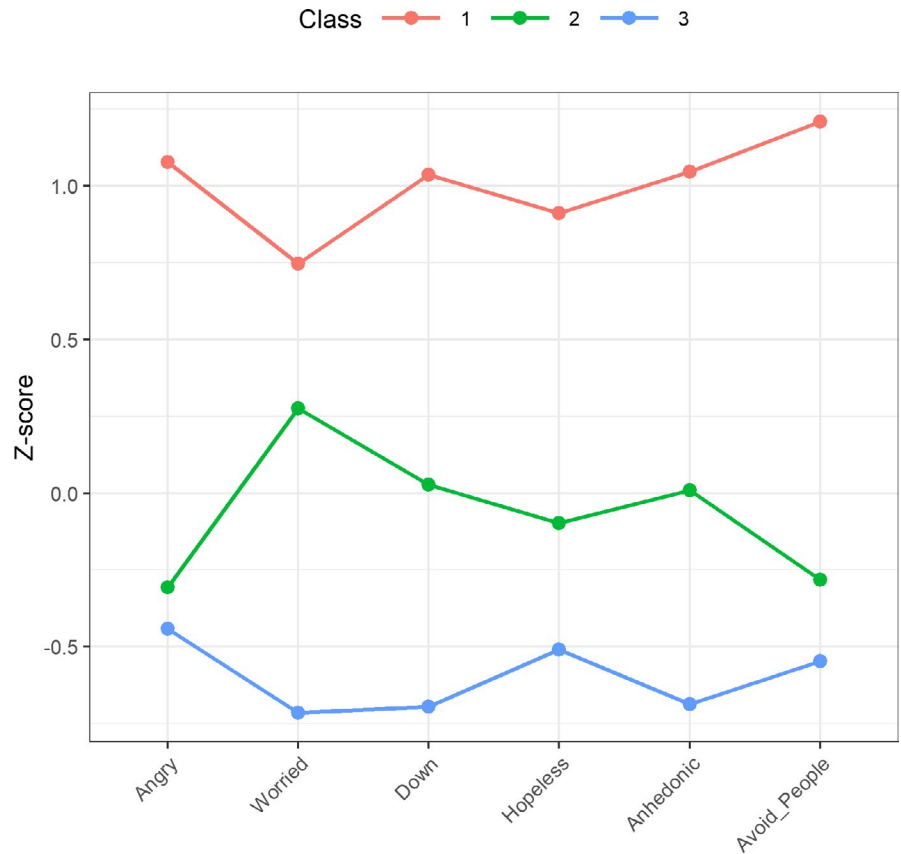
## 4 | DISCUSSION

The current study presents a method for characterising differential patterns of intraindividual symptom covariation via the application of network analysis to LPA-identified within-person mood states. By analysing the network structure of mood states identified in Fisher and Bosley (2019), the present study offers a demonstration of the potential utility of these combined methods. Results from the current example application revealed marked *intraindividual* heterogeneity in network structure from class to class. Network analysis revealed two primary types of within-person, between-class variation in relationships between symptoms within our sample: inverted edges and variation in network density that positively correlated with symptom severity.

### 4.1 | Inverted edges

Over half of the sample had at least one instance of edge inversion between classes, wherein two symptoms that were positively correlated in one class exhibited a negative correlation in another. This finding has a simple yet profound implication: the relationship between any two symptoms is not necessarily stable and equivalently expressed across all time points and contexts. Thus, interventions based on functional analytic interpretations of symptom interrelations may be imprecise or ineffective if they are too general. What may be an effective intervention at one point in time could be wholly ineffective or contraindicated in another. As detailed above, participant 007 exhibited two classes, with worry and anger weakly and positively correlated in one class and strongly, negatively correlated in another. Clinicians might leverage this information to identify antecedents and consequences of the client's anger, with attention to how those factors might differ between the two states. It is important to stress that data analysis does not obviate or replace the necessary work of clinicians. To wit, these results could indicate that anger is functionally adaptive, reducing worry and depressive affect by engaging the individual with environmental frustrations or obstructions. Conversely, anger and its associated arousal could simply preclude anxious and depressive symptoms, creating a predominance of negative affect that suppresses depressive symptoms in the presence of angry symptoms. As the EMA measures capture only the items themselves, in the absence of further interrogation they cannot tell us about contextual factors influencing the client's ratings—but as a clinician, it could be useful to unpack these factors and work with the client to interpret the LPA-derived networks accordingly. Empirically examining how best to leverage these models clinically is likely a worthwhile endeavour for future research.

**FIGURE 1** Latent profile analysis results for participant 074 revealing a three-class profile, with an elevated-symptom class (class 1) and a decreased-symptom class (class 3) [Colour figure can be viewed at wileyonlinelibrary.com]



**FIGURE 2** Contemporaneous networks for participant 074's three classes. (Green edges indicate positive relationships, and red edges indicate negative relationships [this participant has none]. Edge width represents relationship strength: the thicker the edge, the stronger the relationship.) [Colour figure can be viewed at wileyonlinelibrary.com]

### 4.2 | Elevated-/decreased-symptom class density

Results revealed a positive correlation between class severity and network density. Specifically, in an analysis of the 47% ( $n = 21$ ) of cases with distinct elevated-symptom and decreased-symptom classes, a post hoc  $t$  test revealed that network density was significantly higher in elevated-symptom classes. This finding aligns with the literature showing that greater network density at baseline is

associated with illness persistence at follow-up (cf. Borkulo et al., 2015). Insofar as an elevated-symptom class represents the given individual's state of *highest distress*, density may serve as an indicator of global severity and symptom persistence.

Network density may relate to class severity as a function of poor emotion differentiation. Also known as emotion granularity, emotion differentiation is the ability to perceive and distinguish between the nuances of different emotion states (Kashdan, Barrett, & McKnight, 2015). Lesser emotion granularity results in monolithic and

undifferentiated distress. It is plausible that the increased density—itself a reflection of increased covariance among symptoms—betrays an inability to differentiate emotional phenomena, making symptom expression more uniform and, possibly, more intractable. Clients with a wide range of diagnoses may struggle to identify and describe their emotions: poor emotion differentiation has been associated with social anxiety disorder (Kashdan & Farmer, 2014; O'Toole, Jensen, Fentz, Zachariae, & Hougaard, 2014), depression (Starr, Hershenberg, Shaw, Li, & Santee, 2019), eating disorders (Selby et al., 2014) and substance abuse, among many others (Smidt & Suvak, 2015). Clients with low emotion differentiation might describe experiences of distress as feeling *generally bad or unpleasant*, but with limited ability to identify specific emotions (e.g. 'I feel sad'). There is also some evidence (in a sample with social anxiety disorders) that symptoms of anxiety are associated with specific impairment in the differentiation of negative—but not positive—emotions (Kashdan & Farmer, 2014). This is consistent with our finding that *elevated-symptom* classes exhibited higher network density.

If network density indicates poor emotion differentiation, this would be valuable clinical information given that there is abundant empirical evidence that poor emotion differentiation is associated with maladaptive coping when in distress (e.g. binge drinking, aggression or self-harm; Kashdan et al., 2015). Emotion differentiation has been shown to moderate aggressive behaviours in angry individuals, such that those with poorer emotion differentiation report greater provocation in daily life and greater aggressive tendencies (Pond et al., 2012). Further, recent work demonstrates that poor emotion differentiation moderates the relationship between life stress and depression in an adolescent sample (Starr et al., 2019). Crucially, emotion differentiation has been shown to improve with clinical intervention: as one example, a mindfulness-based intervention was recently shown to increase emotion differentiation (Van der Gucht et al., 2019). Thus, network density may be a marker of severity beyond mere symptom level and, conversely, emotion differentiation may represent a novel technique for addressing the symptomatology of *elevated-symptom* classes.

### 4.3 | Feasibility

For clinicians, the current approach offers a data-driven method for exploring clients' lived experience with a high degree of granularity and precision. As with other methods reliant on EMA data collection, execution is contingent upon clients' compliance with frequent smartphone-based surveys, and clinicians' comfort with data analysis. Fortunately, research has shown that compliance with EMA data collection procedures is likely to be high, with studies finding > 80% compliance across a range of clinical and subclinical symptomatic populations (Myin-Germeys, 2018; Soyster, Bosley, Reeves, Altman, & Fisher, 2019). Soyster et al. (2019) demonstrated no significant differences in compliance with EMA sampling across diagnostic groups, indicating that these methods may be utilised with a range of clinical populations.

There is also evidence that clinicians—particularly those with a cognitive-behavioural orientation to treatment—are interested in utilising EMA for the purpose of assessment and treatment planning (Soyster et al., In press). While much future work is needed to make these analyses directly possible for clinicians to run and utilise independently, some progress has been made (Brown, Bosley, Kenyon, Chen, & Levenson, 2019). Future work developing 'black-box' data analytic tools easy to deploy in clinical settings is needed to disseminate methods such as the ones described in the present study.

### 4.4 | Limitations

While the present approach offers many potential advantages, a few limitations merit further consideration. First, the approach employs self-report data collected via EMA. Inherently, these data do not tell us about the nonconscious elements of human functioning. Further, as these data are collected 'in the wild', we know very little about the participant's environmental context at the time of data collection. Future work could address these limitations of EMA by integrating forms of passive data collection that can be used to better assess these domains, such as physiological data and geographical location.

Second, it is unclear how many times per day data should be sampled to optimally capture the constructs of interest. Sampling frequency should be considered carefully as the constructs these items capture are inherently time-varying phenomena. It is possible that different sampling frequencies would yield different latent mood states as a function of a wider or narrower sampling window. If the frequency of measurement does not appropriately match the rate of change in the construct of interest (in this case, emotion), the resulting models will be less accurate.

Although EMA is not a new method for data collection, there has been insufficient empirical investigation into the sampling frequency optimal for phenomena relevant to clinical populations and associated treatment. This may be particularly important when considering models of emotion. While emotions are generally thought to last for seconds or minutes, the literature indicates that their duration is highly variable depending on the emotion category and the context or stimulus that elicited the emotion (Frijda, 2007; Verduyn, Delaveau, Rotgé, Fossati, & Van Mechelen, 2015). Plausibly, emotion duration also varies from person to person. Future research should investigate optimal sampling frequencies for clinically relevant phenomena. In the absence of empirically based sampling rate recommendations, review of literature relevant to specific phenomena of interest as well as discussion with clients is warranted in clinical settings.

Finally, the LPA and network results depend crucially on the items selected a priori. Choosing one additional or one fewer item could meaningfully alter the results of classification and network structure. Therefore, as a clinician, it may be useful to involve the client, the case formulation and the empirical literature in the selection of items to include.

## 5 | CONCLUSION

The current approach represents a promising method for advancing the utility of idiographic methods via (a) offering a solution to the issue of nonstationarity via LPA; and (b) enabling examination of differential patterns of covariation within intraindividual time-series data via network analysis. While the integration of EMA into clinical practice—a necessary prerequisite for the clinical deployment of this approach—has not yet become commonplace, implementation data are promising.

## DISCLOSURES

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit upon the authors or upon any organisation with which the authors are associated.

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