Exploring the Idiographic Dynamics of Mood and Anxiety via Network Analysis

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Abstract

Individual variation is increasingly recognized as important to psychopathology research. Concurrently, new methods of analysis based on network models are bringing new perspectives on mental (dys)function. This current work analyzed idiographic multivariate time series data using a novel network methodology that incorporates contemporaneous and lagged associations in mood and anxiety symptomatology. Data were taken from 40 individuals with generalized anxiety disorder (GAD), major depressive disorder (MDD), or comorbid GAD and MDD, who answered questions about 21 descriptors of mood and anxiety symptomatology four times a day over a period of approximately 30 days. The model provided an excellent fit to the intraindividual symptom dynamics of all 40 individuals. The most central symptoms in contemporaneous systems were those related to positive and negative mood. The temporal networks highlighted the importance of anger to symptomatology, while also finding that depressed mood and worry -- the principal diagnostic criteria for GAD and MDD -- were the least influential nodes across the sample. The method's potential for analysis of individual symptom patterns is demonstrated by three exemplar participants. Idiographic network-based analysis may fundamentally alter the way psychopathology is assessed, classified, and treated, allowing researchers and clinicians to better understand individual symptom dynamics.

Keywords: network models, person-specific dynamics, personalized assessment, idiographic research, centrality measures

Summary: This study provides a means by which to model the symptom experiences of individual patients as mathematical networks. Once a network is built, it can be used to understand the experience of each individual.

Network analysis is increasingly popular in psychopathology research (Borsboom, 2017; Borsboom & Cramer, 2013). The network approach conceptualizes psychological syndromes and disorders as sets of associations between constructs that mutually interact with and reinforce each other over time. Network analysis leverages graph theory and graph-theoretic methods to study interrelated phenomena such as psychopathological symptoms as systems of nodes and edges – with each symptom a node and the edges between nodes reflecting the covariation between symptoms. Network models yield information about the relative influence each variable has in the network by the means of centrality measures (e.g. Opsahl, Agneessens, & Skvoretz, 2010). Thus, network methodology holds the potential to allow researchers to articulate and examine complex dynamics among constituent elements of a psychopathological system.

The inferences that can be drawn from these analyses are contingent on the nature of the input data and the particular method of analysis (c.f. Epskamp, Waldorp, Mõttus, & Borsboom, Submitted). To date, network analysis has largely been used to understand psychopathology using data aggregated across individuals (i.e. nomothetic analyses). This approach has already generated important insights. For instance, Cramer, Waldorp, van der Maas, and Borsboom (2010) demonstrated that the symptoms of generalized anxiety disorder (GAD) and major depressive disorder (MDD) – two putatively distinct diagnostic categories – correlated as strongly across disorders as they did within disorders. This finding is consistent with a topological understanding of psychopathology that rejects latent disease entities in favor of complex interactions among system components (Borsboom, 2017). Nevertheless, although the nomothetic approach provides important information about the nature of average associations between symptoms within a group, it necessarily overlooks information about associations between symptoms in any single individual across and within time (Molenaar, 2004). To this

end, Bringmann et al. (2013) showed that associations revealed on an individual level are meaningfully different from the overall average network. Consequently, it has been argued that researchers should employ person-specific methodologies, in order to understand the dynamic interplay of mental states on an individual (i.e. idiographic) level (Fisher, 2015; Molenaar, 2004). For overlapping disorders such as MDD and GAD that share diagnostic criteria or exhibit high rates of co-occurrence (Brown, Campbell, Lehman, Grisham, & Mancill, 2001) a more granular analysis of intra-individual symptom dynamics could help to illuminate the hierarchical predominance of symptoms in individual symptom networks. Understanding the idiographic dynamics of mood and anxiety in this way could help to potentiate more precise assessment and classification of psychopathology by delineating idiographic and nomothetic sources of variance, in order to better-distinguish idiosyncrasy from commonality (Fisher, 2015).

Thus, there is a pressing need to delineate the structure and function of psychopathology at the idiographic level (Fisher, 2015). Molenaar has argued forcefully against assumptions of equivalence between intra- and inter-individual variation (2004), and compelling evidence has shown that widely replicated, empirically-supported nomothetic models may not hold at the individual level (Borkenau & Ostendorf, 1998). That is, statistical relationships derived from nomothetic analyses (i.e. data aggregated across patients) do not necessarily generalize to individuals. It follows that we should be curious at the very least, if not deeply skeptical about our common assumptions about the structure and dynamic organization of psychopathology, and invested in the exploration of idiographic models.

Network models are exceptionally well-suited to model these person-specific associations. Yet, only one study has focused on these intra-individual relationships. Assessing depressive symptoms up to 10 times a day over a period of 239 days, Wichers et al. (2016)

presented changes in the network structure of mental states of a patient with a history of several major depressive episodes who volunteered to discontinue medication treatment during this period. Several days after the double-blind discontinuation of medication, the patient's depressive symptoms shifted from a healthy range of functioning into a symptom range reflecting severe impairment. The authors showed that as this shift in mood approached, the network structure changed including a greater proliferation and increased strength of connections between the measured mental states. Thus, idiographic methodologies likely hold the potential for guiding the data-driven tailoring of treatment to the specific needs of patients.

Network models are also well-suited to describing temporal structure. Since temporal precedence is a minimum requirement for causality (Granger, 1969; Pearl, 2003), models based on temporally-structured data hold the promise of elucidating which variables are the driving force of some putative underlying causal process. Yet, the majority of work in psychopathology to date has focused on cross-sectional networks. Although researchers have conceptualized these networks as causal systems (c.f. Borsboom & Cramer, 2013; McNally et al., 2015), the data underlying cross-sectional models are collected at a single point in time and cannot reveal the direction of influence between two nodes. Moreover, we should not take for granted that cross-sectional networks reflect temporal processes, or vice-versa. Bos and colleagues (2017) recently demonstrated that the two approaches provide different conclusions about the topology and temporal dynamics of depressive networks. In a sample of 104 individuals with major depressive disorder (MDD), contemporaneous and temporal network models produced distinct patterns of association and centrality among depressive symptoms. Moreover, correlations of node centrality between the two approaches revealed only modest relationships between the rank order of symptom influence. For example, whereas anhedonia exhibited the lowest node centrality

contemporaneously, it was the most influential symptom temporally (Bos et al., 2017).

To meet the demands of temporality and idiography, specialized data collection and analysis must be employed. Specifically, time series data and analyses in which the sample size is a function of the number of observations, rather than the number of individuals. Time series data facilitate the exploration of relationships within and across time – the contemporaneous and lagged relationships. Comparable to cross-sectional models, contemporaneous time series models reflect the structural relationships between nodes at any given moment. However, instead of between-subjects correlations at a single point in time, the contemporaneous correlations in time series models reflect the relationships between nodes within individuals, averaged across time. In addition, time series models can examine the temporal relationships between states at one time point (*t)* and states at a later time-point (*t+1).* Time-lagged edges provide temporal precedence, which more closely approximates the potential direction of the causal relationship (e.g., rumination *leads* to difficulties concentrating).

Contemporaneous and temporal representations of human phenomenology and behavior present different vantages of psychopathology. The symptomatology of mood and anxiety disorders is by its nature manifold, composed of multiple co-occurring – and thus, contemporaneous – thoughts, feelings, and actions. In addition, the experience of a symptom in one moment in time likely drives the variation in other symptoms at later points in time. As Beck proposes, negative thoughts about the self, others, and the world generate depressogenic schemas that promote depressed mood (Beck, Rush, Shaw, & Emery, 1987). Such a proposal can, ostensibly, be directly tested by modeling the degree to which negative thoughts covary in time, and the degree to which these thoughts drive depressed mood across time. Thus, both contemporaneous and temporal models can provide critical information about the form and

function of psychopathology. On the one hand, a contemporaneous network may provide important information about the structural organization of the psychopathological system (i.e., which symptoms are experienced simultaneously), but omit directional information about the relationships between symptoms (i.e., which symptoms trigger other symptoms). Conversely, temporal networks provide information about the direction of influence over time while potentially omitting important associations captured by the contemporaneous approach.

The aim of the present paper was to explore the idiographic structure of mood and anxiety symptomatology via contemporaneous and temporal network models, in order to better understand the topology and temporal dynamics of the putative mood and anxiety disorders. Consistent with established procedures (Clasen, Fisher, & Beevers, 2015; Fisher, 2015; Molenaar, 1985), we estimated structural equation models on a person-by-person basis, to retrieve contemporaneous and temporal symptom associations. Parameters from these models were then used to generate contemporaneous concentration networks at times *t* and *t+1*, and temporal networks composed of time-lagged paths between time points. The centrality measures strength, instrength, and outstrength were employed to quantify the relative influence of each node across individual networks. Finally, centrality measures were aggregated across participants in order to examine generalizable nomothetic features. Thus, we endeavored to foment the exploration of person-specific models of psychopathology, as well as to explore the structure of putative psychological syndromes at the nomothetic level.

Method

Participants

The present data were drawn from participants enrolled in an ongoing research study described in full detail elsewhere (Fisher & Boswell, 2016). Briefly, the study is a multiphase

personalized psychotherapy study in which participants with primary generalized anxiety disorder (GAD) or primary major depressive disorder (MDD) complete intensive repeated measures assessments for at least 30 days prior to therapy. Individuals with symptomatic experiences consistent with GAD and MDD were directed by flyers, referrals, and internet advertisements to contact the first author's laboratory at the University of California, Berkeley. Participants (n = 40) in the current study were predominantly female (n = 26, 65%) and White (n = 19, 47%). Of the non-White participants, three (8%) identified as Black, nine (22%) identified as Asian/Asian American, 6 (15%) identified as Latino, and 3 (8%) identified as Other. The mean number of years of education in the sample was 15.5 (*SDI* = 2.29) and the mean household income was $42,973.47 (*SD* = $58,385.49). Table 1 provides the participant characteristics.

After passing a brief telephone screening interview, participants were invited to present for an in-person structured clinical interview. The Anxiety and Related Disorders for *DSM*-5 (Brown & Barlow, 2014) was administered by graduate students in clinical psychology under the supervision of a PhD-level clinical psychologist. Inclusion criteria were primary diagnosis of GAD or MDD, age of 18 to 65 years, and a web-enabled mobile phone. Exclusion criteria were any history of psychosis or mania, concurrent treatment or cognitive behavioral treatment within the past 12 months, and PRN medication. Of 148 potential participants that presented for clinical assessment, 49 (33%)met inclusion criteria for the current study. Of these, one declined to participate, one provided insufficient survey data[1], and six withdrew during the 30-day survey period, leaving 40 viable cases for the present study. Twenty-five participants met for a current primary diagnosis of GAD and 15 participants met for a current primary diagnosis of MDD. Nineteen participants with GAD met for at least one current comorbid disorder: Social anxiety

[1] This individual (P002) did not complete all survey items and exhibited little to no variability in those items they completed. Study protocol was ammended to provide real-time feedback to participants following this outcome.

disorder (SAD), *n* = 14; MDD, *n* = 6; specific phobia, *n* = 3; panic disorder, *n* = 1; agoraphobia, *n* = 3; posttraumatic stress disorder, *n* = 3; persistent depressive disorder, *n* = 2. Sixteen participants with MDD met for at least one comorbid disorder: GAD, *n* = 10; SAD, *n* = 4; specific phobia, *n* = 2; panic disorder, *n* = 1; agoraphobia, *n* = 1; posttraumatic stress disorder, *n* = 1; alcohol use disorder, *n* = 1. The mean overall clinician ratings for the present study sample on the Hamilton Rating Scale for Depression and the Hamilton Rating Scale for Anxiety were 14.10 (*SD* = 4.34) and 16.85 (*SD* = 6.74), respectively.

Interrater reliability was calculated based on video recordings of the structured clinical interviews. Two videos (participants 7 and 37) were lost in the course of the study – one overwritten and one not adequately captured – leaving 38 participants available for reliability ratings. Following the recommendation of McHugh (2012), we calculated both percent agreement and Cohen's *kappa* to rate diagnostic interrater reliability. The inclusion criteria, GAD and MDD, returned *kappa* values of .68 and .84, and percent agreement of 95% and 92%, respectively, with two mismatches for GAD and three mismatches for MDD. Percent agreement and *kappa* values for secondary diagnoses were: SAD (92%, κ = .79); specific phobia (92%, κ = .63); panic disorder (95%, κ = .64); agoraphobia (97%, κ = .79); posttraumatic stress disorder (97%, κ = .84); alcohol use disorder (100%, κ = 1).

Measures

Hamilton Anxiety Rating Scale (HARS; Hamilton, 1959). The HARS assesses severity of anxious symptomatology. This 14-item clinician administered scale provides a severity rating of each overarching symptom cluster on a scale from 0 (not present) to 4 (very severe). Internal consistency is excellent (α =.92) (Kobak, Reynolds, & Greist, 1993). Retest reliability for the HARS was very good (ICC = .86) across 2 days and inter-rater reliability ranged from an ICC of

.74 -.96 (Bruss, Gruenberg, Goldstein, & Barber, 1994). Construct validity has also been demonstrated in clinical samples (Beck & Steer, 1991).

Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960). The HRSD was developed to assess the severity of depressive symptomatology. This 13-item clinician administered scale provides a rating of severity of each overarching symptom cluster on a scale from 0 (not present) to 4 (very severe/incapacitating). Internal consistency of the HRSD ranges from adequate to good (α=.73 to .81) (Moras, di Nardo, & Barlow, 1992; Riskind et al., 1987). Interrater reliabilities of the HRSD total score range from .78 - .82 (Moras et al., 1992; Riskind et al., 1987). HRSD scores correlate significantly with self-report measures of depression in clinical samples (Steer, McElroy, & Beck, 1983).

Experience Sampling Survey. Following study enrollment, participants' personal mobile phone numbers were entered into a secure web-based survey system. This system sent pings to participant mobile phones with survey prompts four times per day. Each prompt was received by the participant as a text message containing a hyperlink to a web-based survey. Each ping populated the back-end system with a time stamp, whether the participant completed the survey or not. Participants completed surveys for a minimum of 30 days. For each survey, participants rated their experience of each item over the preceding hours using a 0-100 visual analog slider with the anchors *not at all* and *as much as possible* for the 0 and 100 positions, respectively. Surveys contained the extant symptoms of the *DSM-5* criteria for GAD and MDD (*down and depressed, hopeless, loss of interest or pleasure, worthless or guilty, worried, restless, irritable, difficulty concentrating, muscle tension, fatigued*), as well as an additional 11 items gauging positive affect (*positive, energetic, enthusiastic, and content*), negative affect (*angry* and *afraid*), rumination (*dwelled on the past*), behavioral avoidance *(avoided people,*

avoided activities, and procrastinated), and reassurance seeking (*sought reassurance*). Readers should not that, for the sake of brevity, *loss or interest or pleasure* is referred to as *anhedonic,* and *worthless or guilty* is referred to simply as *guilty* from here on. Supplementary materials S1 provides a complete list of survey items.

The mean number of observations across the 40 participants was 130.43 (SD = 19.27), with a minimum of 87 and a maximum of 212.

Data Preparation and Analysis

Cubic spline interpolation. For each individual's data, the time stamps accompanying each multivariate time series were used to calculate the cumulative time. This time variable was then used to apply linear detrending to each variable, for each person. Ordinary least squares regression models were used to regress each variable on the cumulative time. The residuals from these models were retained as detrended, mean stationary time series. Given that data were unevenly sampled in time (with sampling intervals of 4, 4, 4, and 12 hours) a cubic spline interpolation was applied to detrended time series in order to resample the data to an even, six-hour sampling interval. In order to examine the degree to which the interpolation introduced bias to the analyses, a simulation study was conducted. Results of this simulation are provided in Supplemental materials S2. Across a number of simulated conditions, the cubic spline interpolation recovered greater than 99% of the known autocorrelation.

Structural equation modeling. Structural equation models were then conducted separately for each individual in LISREL (version 9.2). The structure of these models was similar to methods previously applied to intensive repeated measures data (Clasen et al., 2015; Fisher, 2015; Fisher & Boswell, 2016; Fisher & Woodward, 2014). Models were run as single-indicator dynamic factor models (Molenaar, 1985), with a zero matrix for the observed errors,

identity matrix (diagonal matrix of 1's) for the factor loadings, and the variance for each variable expressed in the latent disturbances. All contemporaneous correlations between variables at times *t* and *t+1* were freely estimated.

Automatic search procedure. We employed an automatic search procedure, utilizing the Lagrange multiplier test, to determine the lagged regression structure for each individual. An input syntax was created in which only the autoregressions for each variable from time *t* to *t+1* were estimated. The automatic search paradigm was constrained to search only cross-lagged regression paths from time *t* to *t+1*. Thus, all contemporaneous regressions and all regressions from *t+1* to *t* (backward in time) were suppressed. Initial models were run without any cross-lagged paths from t to t+1. After each run, the regression path from time *t* to *t+1* with the largest associated chi-square change was added to the model and the model was rerun. Lagged regression paths were added to each model until no remaining paths were associated with a chi-square change of at least 5.00.

This automatic search procedure is consistent with previous work in brain connectivity mapping (Gates, Molenaar, Hillary, Ram, & Rovine, 2010) and psychopathology (Clasen et al., 2015). Nevertheless, we conducted a simulation study to assess the degree to which the automatic search procedure could accurately recover known covariance structures. The R package simsem (Pornprasertmanit, Miller, & Schoemann, 2016) was used to generate simulated data from each of the 40 final models. Five simulated multivariate time series were generated for each participant. Each simulated data set was then fit to the putative final model and examined for model fit. All 200 models returned excellent fit as measured by the root mean square error of approximation (RMSEA; $< .06$) and the confirmatory fit index (CFI; $\geq .95$). Chi-square tests

returned a 95% recovery rate, with 10/200 models reflecting significant misfit (i.e. $p < .05$). Complete results of this simulation are provided in Supplemental materials S3.

Extraction of matrices for network analysis. Following completion of the automated search procedure for each individual, the resulting model parameters were used as inputs for three separate network models. That is, the standardized values provided in the LISREL output were extracted and used to build input matrices for network analyses. The matrices of interest were LISREL's psi matrix and beta matrix. The psi matrix contains the variances and covariances for the variables at time *t* and time *t+1*, respectively. When standardized, these are scaled as correlations. The beta matrix contains the directional (lagged) regression relationships between time *t* and time *t+1*. Because LISREL syntax and output is based upon matrix algebra, the output is amenable to use as inputs for network analysis.

Contemporaneous correlation matrices. The standardized coefficients for the contemporaneous relationships at time *t* and time *t+1* were reformatted from the LISREL output into two, separate, symmetric matrices of correlation coefficients. The contemporaneous correlations between variables at time *t* were exogenous and thus not conditioned on any predictors. We will refer to these as the lag-0 correlations. The lag-0 correlations allow the construction of concentration networks similar to recent work in PTSD (McNally et al., 2015), MDD (van Borkulo et al., 2015), and other disorders (Wigman et al., 2015), albeit at an idiographic, rather than nomothetic level. The residual correlations between variables at time *t+1* represent the variance-covariance not explained by autoregressions or cross-lag regression paths. The correlations among the residuals could represent causal relationships occurring at time intervals faster than the lag length, residual partial correlations, exogenous (unmeasured) influences on the system, noise, or some combination thereof. For each contemporaneous matrix,

we then estimated a sparse partial correlation network using a LASSO regularization method implemented in *R* (version 3.3.1; R Core Team, 2016) with package *qgraph* (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012).

Lagged regression matrix. In order to generate the temporal network model, the matrix of standardized lagged regression coefficients (beta) was similarly extracted from the LISREL output. However, it is important to note that it in LISREL convention, columns predict rows, whereas it is the convention in network analysis for rows to predict columns. Thus, the beta matrix was transposed before being used to generate a temporal network model in *qgraph*.

Centrality. The contemporaneous networks at time *t* and time *t+1* were used to calculate strength centrality – the sum of the edge weights associated with a given node. Finally, the temporal model reflects the time-lagged associations between variables, and allowed us to calculate instrength (the degree to which a given node was predicted by other nodes) and outstrength (the degree to which a given node predicted other nodes).

Results

Model fit

Model fit was evaluated with the root mean square error of approximation (RMSEA), Brown's chi-square goodness-of-fit test (Brown & Cudeck, 1993), and the confirmatory fit index (CFI). Non-significant chi-square tests, RMSEA values less than .060, and CFI values greater than or equal to .95 reflect excellent fit (see Hu & Bentler, 1999). All 40 models exhibited excellent fit, as reflected by CFI and chi-square. Participant 19 exhibited only an acceptable RMSEA value of .062. Table 2 provides the fit statistics, degrees of freedom, and number of off-diagonal (i.e. non-autoregressive) regression paths for all 40 models. For each model, the number of off-diagonal regression paths is equivalent to the number of times the model was rerun by the

automatic search procedure. The input syntax for the automatic search procedure and all final models have been provided on the Open Science Framework at https://osf.io/zefbc/.

Nomothetic Results

As noted above, concentration models derived from the correlations at time *t* and time *t+1* were used to calculate the contemporaneous strength across the 21 variables. The temporal models were used to calculate instrength, and outstrength across time. Thus, the contemporaneous models reflected the centrality of each node as a function of its connectivity to the overall network and the temporal models captured the degree to which (a) variation in a given node was transmitted across time and affected related nodes forward in time (outstrength), or (b) variation in any number of other nodes affected a single node forward in time (instrength). For each result, values were normalized within participants (divided by the maximum value), in order to make them comparable across participants.

Contemporaneous concentration networks. Figure 1 presents the results for the average normalized contemporaneous strength across the 40 individual lag-0 and residual concentration networks. Values were normalized such that the maximum possible strength was 1 and the minimum 0. Results were largely consistent across the lag-0 and residual models. *Down* exhibited the greatest strength in lag-0 models and the second-highest strength in residual models. Meanwhile, the item *positive*, exhibited the inverse ranking. Conversely, *restlessness, procrastination, tension,* and *reassurance seeking* exhibited the lowest strength in both models. Table 4 presents the item labels in rank order.

Temporal networks. Because the temporal models in the present study were derived from models with a lag-1 regression structure, it is important to note that interpretations should not be extended beyond a single lag. That is, whereas the graphical representation of the directed

network model gives the appearance that information may flow from node A to node B, and then on to nodes C and D, each of these directed edges are estimated from a single lag only. Thus, a formal test of the implied structure – from A to B to C to D – would require additional lags. Converse to this, instrength and outstrength represent the total incoming and outgoing information across a single lag – the number and degree of directed paths predicting a given node from moment to moment or being predicted by a given node from moment to moment.

As can be seen in Figure 1, *anger* exhibited the greatest average instrength, followed closely by reassurance seeking and the depressive symptoms, *guilty*, *anhedonic*, *hopeless*, *content,* and *down*. These results reflect that the most prominent downstream experiences for these individuals were largely mood-related. Regarding outstrength, the depression-related items *positive* and *hopeless* were the two most influential predictive nodes, followed by *angry* and *irritable*. However, the principal diagnostic criteria for GAD and MDD – depressed mood and worry – were the least influential nodes across the sample. Table 4 presents the item labels in rank order.

Exemplar participants[2]

Whereas nomothetic analyses help to delineate generalizable features of psychopathology across the population, the strength of the idiographic approach is to generate more granular results that provide rich detail at the level of the individual. To this end, three exemplar participants are described to highlight key features of the present analytic approach. Space limitations preclude a detailed description and interpretation of all the nuanced relationships each network comprises. Thus, our description is reduced to the most important variables in each of the networks, where importance is defined by strength, instrength, and outstrength. Participants

[2] Complete input data, R and LISREL syntax, and model outputs for the complete sample (n=40) available at https://osf.io/zefbc/.

P25 and P145 both presented with primary GAD and secondary SAD. Participant P72 presented with primary MDD and secondary GAD.

Figures 2, 3, and 4 display the network models for P25, P145, and P72, respectively. For each figure, Panel A represents the lasso regularized contemporaneous relationships at time point *t* – what we have referred to as the contemporaneous concentration network; Panel B represents the lagged regression paths indicated by the automatic search procedure; and Panel C represents the lasso regularized contemporaneous residual relationships at time point *t+1*. In each figure the orange nodes represent variables at time *t* and the blue nodes represent variables at time *t+1*.

Example 1 – Participant 25 (P25). Participant 25 was a 28-year-old White male with primary GAD and secondary SAD. Table 3 presents the strength, instrength, and outstrength values and Figure 2 presents the network models for P25. The most influential node in both contemporaneous networks for this individual was *avoiding activities*, however, this node exhibited no directional influence and thus returned an outstrength value of zero. From this result we can infer that, whereas avoidance is densely connected to P25's overall symptomatology *within* time, it does not appear to be driving variation in that symptomatology over time. In fact, only nine of the 21 variables exhibited predictive influence over time: *energetic, content, down and depressed, anhedonic, worried, tense, fatigued, difficulty concentrating,* and *procrastinating*. Although considered to be a secondary diagnostic symptom, *difficulty concentrating* exhibited the greatest outstrength, and thus the greatest influence over time. *Difficulty concentrating* exhibited predictive influence over three symptoms: *fatigue, avoiding activities,* and *avoiding people*. The items with the greatest instrength were restless and fatigued, indicating that for P25, the strongest downstream effects were somatic in nature.

Example 2 – Participant 145 (P145). Participant 145 was a 45-year-old female with

primary GAD and secondary SAD who designated her racial/ethnic identity as "other." Table 3 presents the strength, instrength, and outstrength values and Figure 2 presents the network models for P145. The outstrength values for P145 revealed that *positive* was the most influential node in this patient's symptom network from moment to moment. Consistent with this, *positive* exhibited the second-highest lag-0 strength and the highest residual strength within the contemporaneous models, reflecting the structural and dynamic predominance of positivity in P145's symptomatology. Across time, positivity exhibited directed paths to four nodes at *t+1*. Increases in positive feelings at *t* preceded decreases in *anger, reassurance seeking, worry*, and an increase in *procrastination* at *t+1*.

Thus, it appears that increased positive feelings paradoxically drove both positive and negative association circuits – decreasing negative affect while increasing avoidance behavior. Examination of the residual model (time *t+1*), revealed *positive* at time *t+1* exhibited a number of additional negative contemporaneous relationships with negative affect (*fear, anger, depressed mood, anhedonia, restlessness,* and *fatigue*) and positive contemporaneous relationships with other facets of positive affect (*enthusiasm* and *energetic*). Thus, positivity appears to have played markedly different functional roles in the regulation of affect versus behavior. Given P145's comorbid GAD and SAD, exploring the role of positivity in the promotion of avoidance behavior would plausibly be a promising treatment strategy, given the attenuating role of positivity in decreasing a broad range of negative affect.

Example 3 – Participant 72 (P72). Participant 72 was a 35-year-old, Asian-American woman with primary MDD and secondary GAD. Table 3 presents the strength, instrength, and outstrength values and Figure 2 presents the network models for P72. Consistent with P145, *positive* exhibited the greatest outstrength for this participant. Across time, lower positivity led to

increased *guilt, anhedonia,* and *hopelessness*. In turn, hopelessness – the node with the greatest strength in both lag-0 and residual models – was contemporaneously connected to rumination and procrastination. This subnetwork demonstrates how impairments in positive affect could be transmitted to increased depressogenic cognition, negative affect, and avoidance behavior over time. Conversely, for this individual, increases in positive affect mitigated these depressogenic factors.

The directed effects in P72's model provide a template for drawing functional analytic conclusions from network model methodologies on a single case basis. Avoiding people exhibited the second-greatest outstrength, with negative lagged effects on *enthusiasm* and *energetic* and positive lagged effects on *avoiding activities* and *content*. Thus, greater avoidance of other people led to decreased energy and less enthusiasm, but also increased endorsements of feeling content. The latter relationship plausibly reflects the negative reinforcement of avoidance behavior through the removal of distress. However, avoiding people also exerted a positive lagged effect on avoiding activities which, in turn, was correlated with increases in depressed mood at time *t+1*. Thus, for P72, attempts to reduce distress through avoidance, while proximally reinforcing, may have led to indirect increases in negative affect.

Discussion

The aim of the current paper was to explore the idiographic topology and temporal dynamics of mood and anxiety symptoms via concurrently-estimated contemporaneous and temporal network models. We utilized intensive repeated measures data to generate time series models of psychopathology on a person-by-person basis. Our approach comprised three steps: In the first step, we used structural equation modeling to simultaneously estimate the contemporaneous and lagged relationships among mood and anxiety variables in person-specific

time series data (Clasen et al., 2015), yielding lag-0 correlations at time *t*, lagged regression effects between times *t* and *t+1*, and residual correlations at time *t+1*. This approach enabled the assessment of measurement imprecision and provided indices of model fit. In the second step, the model parameters were extracted from the structural equation models and used to create input matrices for network analysis. Finally, network models were generated with established procedures. For the contemporaneous correlations, a regularization procedure was applied to the lag-0 correlations at *t* and residual correlations at *t+1* in order to arrive at parsimonious, partial-correlation networks (Epskamp, 2016; Epskamp & Fried, 2016; Friedman, Hastie, & Tibshirani, 2014). The lagged regression structure was visualized via network analysis to model the predictive effects from time *t* to time *t+1*. Strength was used to measure the centrality of contemporaneous nodes and instrength and outstrength were used to measure the centrality of temporal nodes. Data from three exemplar participants were presented in order to demonstrate the utility and granularity of person-specific models of psychopathology.

The mean scores across the sample were used to draw nomothetic inferences about contemporaneous and temporal node centrality. Lag-0 and residual contemporaneous models produced relatively consistent results. The most densely interconnected contemporaneous symptoms were *down, positive, content, enthusiastic,* and *energetic.* Thus, the most central symptoms in contemporaneous systems were those related to positive and negative mood. The temporal models reflected the degree to which a given variable was predicted by other variables (instrength) and the degree to which a given variable predicted other variables (outstrength) from time *t* to time *t+1*. Consistent with contemporaneous models, *positive* exhibited the greatest outstrength, followed by *hopeless, angry* and *irritable.* Thus, the most potent drivers of symptomatology from moment to moment were positive mood, hopelessness, anger, and

irritability, but *not* depressed mood, anhedonia, or worry (the putative cardinal symptoms of MDD and GAD). In fact, *worried* and *down* were the two least influential temporal nodes. These results seem to underline the importance of positive affect in distress syndromes, while also placing a spotlight on anger as a potentially primary experience in mood and anxiety. In fact, anger also exhibited the greatest instrength, making it the most prominent downstream experience across the sample, in addition to its outstrength centrality.

There is some existing evidence to suggest that anger may be a common feature of mood and anxiety disorders such as GAD and MDD (Deschênes, Dugas, Fracalanza, & Koerner, 2012; Fisher et al., 2015). Fava and Rosenbaum (1998) found that one third of outpatients with MDD experience attacks of anger that include autonomic symptoms, such as elevated heart rate and sweating. Meanwhile, GAD is associated with anger episodes (Hawkins & Cougle, 2011) and aggressive behavior, even when controlling for Axis I and Axis II comorbidities (Posternak & Zimmerman, 2002). Although it is common in the literature to conflate or combine anger and irritability (c.f. Ellis, Shumake, & Beevers, 2016), results of the current study provided some evidence for the dissociation of these constructs. Whereas, anger exhibited the greatest instrength, irritability exhibited only the 17th-greatest instrength, with the two constructs separated by 27% of the total normalized scale. Nevertheless, anger and irritability showed only modest separation in contemporaneous models (8% and 10% of normalized scale for lag-0 and residual models), and the two constructs exhibited the third and fourth greatest outstrength. Thus, in the present study anger and irritability presented as relatively equal predictors of mood and anxiety symptoms, though anger was the far more common reaction to other negative mood states.

Despite its prominent role in the temporal dynamics of the current sample, anger is not a

clinical criterion for either GAD or MDD. Worry and depressed mood, however, are inclusion criteria for the diagnosis of GAD and MDD, respectively. Moreover, these dimensions are often treated as proxies for their constituent clinical syndromes (c.f. Brown & Barlow, 2009; Brown, Chorpita, & Barlow, 1998; Naragon-Gainey, Prenoveau, Brown, & Zinbarg, 2016). Despite this, the present study found weak support for the primacy of worry in the current sample and mixed results regarding the importance and influence of depressed mood. Worry was the 10th most central item in lag-0 contemporaneous models, the 12th most central in residual contemporaneous models, and exhibited the 13th and 20th greatest instrength and outstrength, respectively. Thus, in the present sample, (a) worry was not a densely connected node contemporaneously, (b) was not a common downstream endpoint, and (c) exhibited little influence over other nodes from moment to moment. Depressed mood was the most densely interconnected node and the 2nd most densely interconnected node in lag-0 and residual contemporaneous models, indicating that responses to the item *down and depressed* correlated strongly with a number of other symptoms and behaviors at each survey ping. Additionally, depressed mood exhibited the 7th–highest instrength, making it a relatively common downstream effect. However, depressed mood exhibited the lowest outstrength.

Participants' ratings of the degree to which they felt positive exhibited the highest centrality across strength and outstrength measures, despite exhibiting the lowest instrength. This indicates that positive mood was the most densely connected and outwardly influential node, but the least common downstream experience across individuals. Thus, whereas positivity might not manifest as a presenting complaint for participants, it appears to hold important structural and dynamic roles in the symptomatology of mood and anxiety pathology. Evidence linking deficits in positive affect to MDD and SAD has been well-documented and widely-disseminated (e.g.

Brown et al., 1998), and some have argued that low positive affect demonstrates greater specificity for identifying clinical depression than the putative diagnostic criteria (Watson & Naragon-Gainey, 2010). The current findings therefore underscore the potential structural importance of positive affect to mood and anxiety pathology. Additionally, the current findings provide some initial evidence for a causal or, at least, predictive role of positive affect, highlighting the possible utility of positive mood as an influential treatment target. In fact, Bosley, Fisher, and Taylor (2016) recently found that treatment gains in worry and negative affect did not generalize to improvements in positive affect in cognitive behavioral therapy for GAD. Future research in psychopathology should continue to examine the primacy, malleability, and influence of positive affect in mood and anxiety syndromes.

The proposed network methodology presents a unique opportunity to explore the points of intersection and diversion among idiographic models of psychopathology. That is, similarities among centrality measures may point to common processes, whereas structural and dynamic idiosyncrasies may illuminate areas of greater individual diversity in mood and anxiety pathology. To this end, it is interesting to note that whereas positive mood was the most influential node for two of the three exemplar participants (P72, and P145), it exhibited an outstrength value of zero for P25. Thus, the proposed model can help to differentiate idiographic symptom dynamics at a fairly granular level. That is, although the aggregated data pointed to *positive* as a potential causal influence *generally*, this item had no predictive influence over other symptoms and behaviors for participant 25. Other points of convergence and divergence can be readily divined from the presence or absence of influence among certain nodes. For instance, whereas fear demonstrated some degree of outstrength influence for 31 of the 40 participants (78%), and was the *most* influential node for one participant (P010), it exhibited no temporal

influence for nine participants, including the three exemplar participants. Therefore, the three selected exemplars may belong to a minority subset of individuals with mood and anxiety pathology for whom fear is not a principal, potentially causal factor.

Idiographic network models hold the potential to revolutionize the classification and assessment of mood and anxiety psychopathology. For instance, P25 presented with primary GAD and secondary SAD. However, difficulty concentrating – considered a secondary symptom of GAD – exhibited the greatest outstrength for this individual. Moreover, whereas worry (the cardinal symptom of GAD) exhibited moderate strength and outstrength centrality, fear and avoidance, the principal features of SAD, exhibited no temporal influence for this individual. Perhaps even more striking is P72, who presented with primary MDD and comorbid GAD. For this individual, anhedonia and depressed mood, which are required for a diagnosis of MDD, exhibited the 4^{th} and 9^{th} greatest outstrength, and worry only the 12^{th} greatest outstrength. Quite simply, these data point to an emerging truth: that the heterogeneity of these disorders may be too great to restrict within a nomothetic nosology. Pursuing an idiographic science may not only facilitate more accurate and granular models of psychopathology, it may yield mechanisms for the realization of precision interventions.

Despite the general efficacy of psychological interventions like cognitive behavioral therapy, a considerable number of patients still do not benefit from these well-established and effective treatment packages (e.g. Lambert, 2013). Tailoring psychological treatments to the specific characteristics of individual patients is currently discussed as a promising strategy for enhancing the effects of psychotherapy (e.g. Ng & Weisz, 2015; Rubel & Lutz, 2017); however, two crucial questions remain: What specific characteristics should be targeted? And what is the framework for matching patient characteristics to targeted interventions? The application of

idiographic network models may provide a method by which researchers and clinicians can leverage the complex and multifarious pieces of information within individual systems in order to identify the most influential variables in a network. Future psychopathology research should look to use these methods to develop and test algorithms that match person-specific dynamics to putative mechanisms of action (Borsboom, 2017; Fisher, 2015; Fried et al., 2017), which may help to innovate and accelerate precision mental health care (Insel, 2009).

The following limitations of the presented approach are noteworthy. First, although the temporal models comprised directed effects from one time point to another, these should not be interpreted as causal. Since no experimental manipulation was conducted these temporal associations could potentially be the result of unmeasured time-varying confounding variables (Fried & Cramer, In Press). Furthermore, the contemporaneous effects should likewise be interpreted with caution. Within the current study's data collection paradigm, patients assessed symptom levels for intervals of approximately four hours. As a consequence, symptoms which affect other symptoms relatively rapidly (i.e. in a shorter time span) would be conceptualized as contemporaneous, despite a true statistical relationship in which one predicts the other in time. Thus, whether an association is modeled as contemporaneous or temporal is partly dependent on the assessment design of a given experience sampling study, which should be kept in mind when interpreting these kind of relations.

Finally, it is important to be aware that our proposed methodology is designed for the analysis of person-specific relationships and not suitable for aggregated data, as nomothetic data cannot capture or reflect the dynamic interplay among symptoms and behaviors within individuals (Molenaar, 2004). However, as has been shown for the centrality measures, general principles can be derived by aggregating results of the individual models across patients. Despite

these limitations and cautionary remarks, we believe that the idiographic network models presented here represent a promising approach for quantifying the structure and dynamics of psychological systems within individuals. These models may help researchers and clinicians to better-understand the individual symptom dynamics of each individual on a person by person basis.

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Table 1: Participant characteristics.

ID	Sex	Age (years)	Ethnicity	Primary Diagnosis	Comorbidities	HAM-D	HAM-A
1	Female	18-24	Latino	MDD	GAD, Panic	23	27
3	Male	25-30	White	MDD	GAD	16	15
4	Female	25-30	Latino	GAD	N/A	16	33
6	Male	18-24	White	MDD	GAD, SAD	13	13
7	Female	25-30	Black	MDD	GAD, Agor., SAD, Spec. Phob.	11	17
8	Female	18-24	Asian American	MDD	GAD, PTSD, Body Dys.	19	15
9	Female	28-24	Other	GAD	SAD, Specific Phobia	17	9
10	Male	25-30	Asian American	GAD	MDD, SAD	22	22
12	Female	31-40	Latino	GAD	Agoraphobia	9	13
13	Male	18-24	White	GAD	MDD, SAD	14	19
14	Male	18-24	Latino	MDD	N/A	10	12
19	Female	25-30	Asian American	MDD	SAD	10	10
21	Male	51-60	Other	GAD	SAD	15	16
23	Female	51-60	White	GAD	N/A	8	7
25	Male	25-30	White	GAD	SAD	15	14
33	Female	18-24	White	GAD	Agoraphobia, SAD, OCD	8	14
37	Female	18-24	Latino	GAD	SAD, Specific Phobia	12	23
40	Female	25-30	White	GAD	Agoraphobia, SAD, MDD	21	41
48	Male	51-60	Asian American	MDD	GAD, SAD, Specific Phobia	14	17
68	Female	31-40	White	GAD	N/A	11	14
72	Female	31-40	Asian American	MDD	GAD	15	13
74	Female	51-60	White	MDD	N/A	12	10
75	Female	18-24	White	GAD	N/A	18	23
100	Male	25-30	White	GAD	PTSD	7	14
111	Female	18-24	Asian American	GAD	Panic, SAD, PTSD	18	15
113	Female	41-50	Black	GAD	SAD, Specific Phobia	4	15
115	Female	31-40	White	GAD	MDD, SAD	18	19
117	Male	51-60	White	MDD	GAD, AUD	12	18
127	Male	25-30	Latino	GAD	SAD	9	13
137	Male	41-50	Asian American	MDD	N/A	16	15
139	Female	51-60	White	MDD	GAD	14	12
145	Female	41-50	Other	GAD	SAD, PTSD	21	30
160	Male	41-50	White	GAD	PDD	13	11

163	Female	51-60	Asian American	MDD	GAD	16	16
169	Male	25-30	White	MDD	N/A	13	15
202	Female	31-40	White	GAD	PDD	10	11
203	Female	18-24	Asian American	GAD	MDD, SAD	18	20
204	Female	51-60	White	GAD	N/A	12	16
215	Female	31-40	Black	GAD	N/A	17	23
217	Female	25-30	White	GAD	MDD	17	14

Note. HAM-D = Hamilton Rating Scale for Depression score; HAM-A = Hamilton Rating Scale for Anxiety score; MDD = major depressive disorder; GAD = generalized anxiety disorder; Panic = panic disorder; Spec Phob = specific phobia; Agor = agoraphobia; PTSD = posttraumatic stress disorder; Body = body dysmorphia; AUD = alcohol use disorder; PDD = persistent depressive disorder.

Table 2: Fit statistics for idiographic structural equation models.

ID	RMSEA	CFI	χ^2	p	DF	# Off-Diagonal
1	< .001	1.00	350.23	0.97	401	19
3	0.021	0.99	356.89	0.94	400	20
4	0.015	0.99	356.80	0.96	404	16
6	0.030	0.98	376.59	0.79	400	20
7	0.043	0.95	366.12	0.92	405	15
8	0.032	0.98	387.44	0.64	398	22
9	0.014	1.00	359.58	0.92	398	22
10	0.011	1.00	348.60	0.96	397	23
12	0.021	0.98	368.33	0.88	402	18
13	0.021	0.99	348.68	0.97	400	20
14	0.026	0.99	361.27	0.82	387	33
19	0.062	0.95	446.88	0.05	398	22
21	0.056	0.97	416.02	0.30	402	18
23	0.030	0.97	351.59	0.92	391	29
25	0.050	0.97	403.12	0.48	402	18
33	0.035	0.98	392.11	0.63	402	18
37	0.040	0.98	393.43	0.41	388	32
40	< .001	1.00	315.22	0.99	398	22
48	0.024	0.99	366.85	0.87	399	21
68	0.033	0.99	368.70	0.82	395	25
72	0.037	0.97	379.81	0.70	395	25
74	0.019	1.00	335.68	0.96	382	38
75	0.020	0.99	365.90	0.87	397	23
100	0.036	0.99	399.90	0.52	402	18
111	0.011	1.00	346.80	0.95	393	27
113	0.026	1.00	391.84	0.30	378	42
115	< .001	1.00	308.99	0.99	391	29
117	0.032	0.99	380.07	0.49	380	40
127	0.031	0.99	374.81	0.79	398	22
137	0.040	0.98	389.95	0.53	393	27
139	0.019	0.99	341.68	0.98	396	24
145	0.013	1.00	357.67	0.95	403	17
160	< .001	1.00	325.44	0.99	404	16
163	0.029	0.98	380.44	0.75	400	20
169	0.045	0.97	390.07	0.50	391	29
202	0.051	0.97	419.76	0.12	387	33
203	0.046	0.98	397.63	0.41	392	28
204	0.055	0.97	416.42	0.10	381	39
215	0.005	1.00	334.64	0.99	393	27
217	0.037	0.99	361.24	0.37	353	67
Average	0.028	0.98	370.83	0.71	394.4	25.6

Note: #Off-diagonal = number of non-autoregressive lagged directional paths; χ^2= Brown's χ^2 (1984).

Table 3: Within-individual normalized values for strength at time t (lag-0) and t+1 (residual), and instrength and outstrength derived from temporal models, for participants 25, 72, and 145.

	Lag-0 Strength			Residual Strength			InStrength			OutStrength		
	P25	**P72**	**P145**	**P25**	**P72**	**P145**	**P25**	**P72**	**P145**	**P25**	**P72**	**P145**
Energetic	.53	.83	.91	.59	.72	.91	.21	.63	.32	.44	.46	0
Enthusiastic	.80	.65	.85	.87	.55	.86	.18	.34	1	0	.36	.54
Content	.73	.62	.55	.77	.61	.57	.22	1	0	.57	0	.44
Positive	.75	.83	.95	.72	.76	1	0	.24	0	0	1	1
Down	.73	.72	.81	.71	.56	.74	0	.76	.64	.56	.30	0
Anhedonic	.65	.63	.72	.68	.60	.62	0	.40	0	.21	.45	0
Hopeless	.91	1	.89	.97	1	.75	0	.84	.61	0	.39	.20
Guilty	.83	.92	.51	.82	.84	.58	0	.72	0	0	0	0
Ruminating	.50	.46	.64	.80	.51	.67	0	0	0	0	.23	0
Worried	.89	.61	.85	.74	.50	.88	0	0	.41	.55	.21	.23
Afraid	.44	.42	1	.63	.37	.99	0	0	.66	0	0	0
Restless	.61	.62	.91	.88	.66	.98	1	.22	0	0	0	.21
Irritable	.67	.28	.78	.86	.26	.88	.19	0	0	0	0	0
Angry	.69	.50	.79	.82	.45	.85	0	0	.44	0	.16	.24
Tense	.41	.35	.46	.44	.42	.55	0	.37	0	.47	.35	0
Fatigued	.69	.52	.61	.72	.52	.57	.93	.90	.33	.51	.28	.21
Diff. Concentrating	.87	.63	.50	.83	.69	.49	.55	.62	.33	1	0	.20
Reassurance Seeking	.43	.22	.56	.63	.21	.51	.44	0	.76	0	.17	.16
Avoiding Activities	1	.51	.86	1	.47	.92	.26	.55	0	0	.40	.40
Avoiding People	.65	.57	.77	.73	.48	.60	.51	0	.40	0	.84	0
Procrastinating	.49	.67	.76	.55	.77	.86	0	0	.37	.32	.15	0

Table 4: Survey items, ranked by strength (lag-0 and residual), instrength, and outstrength, for the complete sample.

Lag-0	**Residual**	**InStrength**	**OutStrength**
Down	Positive	Angry	Positive
Positive	Down	Reassurance Seeking	Hopeless
Content	Enthusiastic	Guilty	Angry
Enthusiastic	Energetic	Anhedonic	Irritable
Energetic	Content	Hopeless	Avoiding Activities
Anhedonic	Irritable	Content	Procrastinating
Avoiding Activities	Anhedonic	Down	Anhedonic
Hopeless	Hopeless	Fatigued	Restless
Irritable	Avoiding Activities	Energetic	Afraid
Worried	Guilty	Diff. Concentrating	Content
Guilty	Avoiding People	Avoiding People	Ruminating
Avoiding People	Worried	Tense	Tense
Afraid	Angry	Worried	Avoiding People
Angry	Diff. Concentrating	Restless	Fatigued
Diff. Concentrating	Afraid	Enthusiastic	Guilty
Fatigued	Ruminating	Afraid	Enthusiastic
Ruminating	Fatigued	Irritable	Diff. Concentrating
Restless	Restless	Procrastinating	Energetic
Procrastinating	Procrastinating	Ruminating	Reassurance Seeking
Tense	Tense	Positive	Worried
Reassurance Seeking	Reassurance Seeking	Avoiding Activities	Down

Figure 1: Normalized values for aggregated intraindividual strength at times t and t+1, instrength, and outstrength.

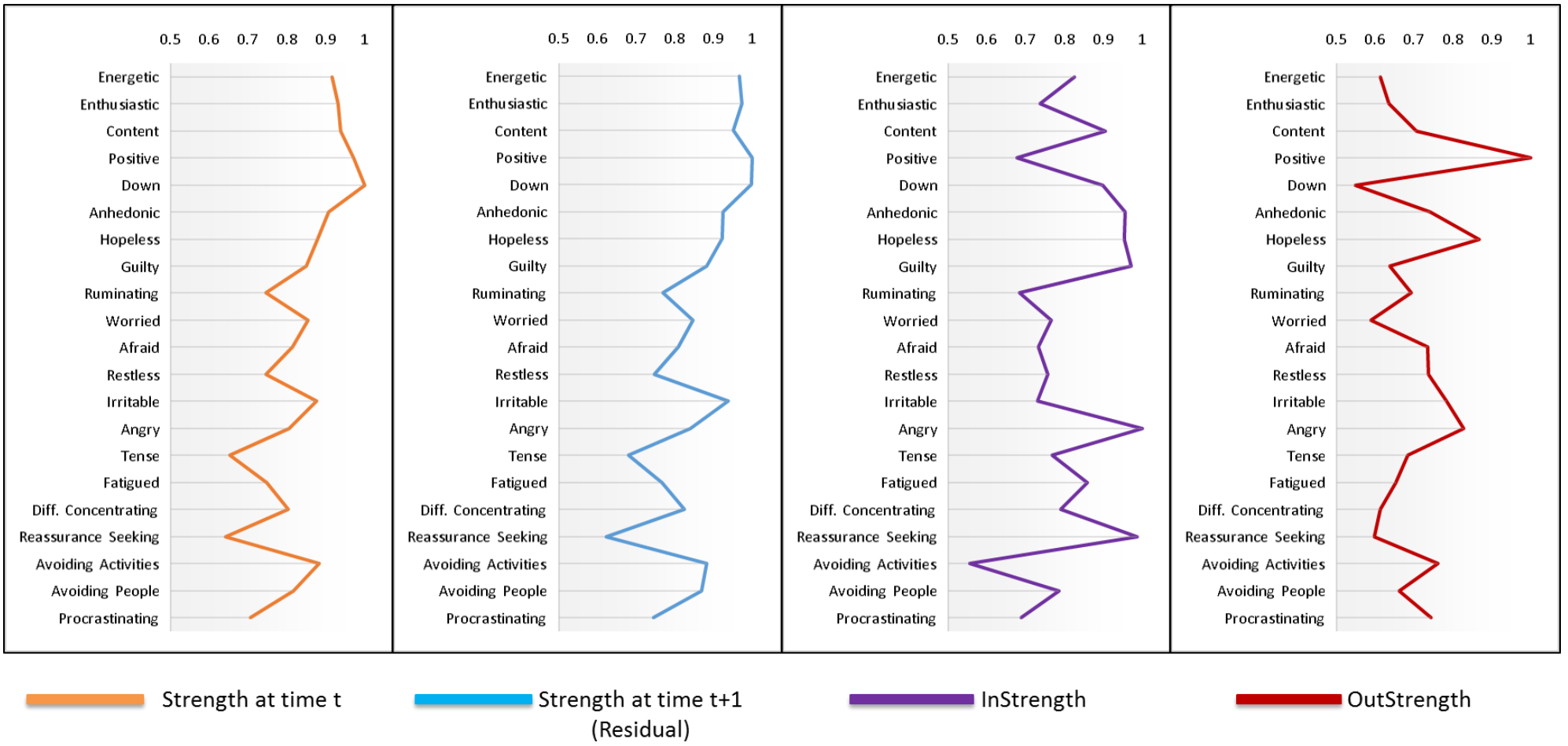

Figure 2: Contemporaneous concentration network at time *t* (A), directed network for lagged relationships between *t* and *t+1* (B), and residual concentration network at time *t+1* (C) for P25.

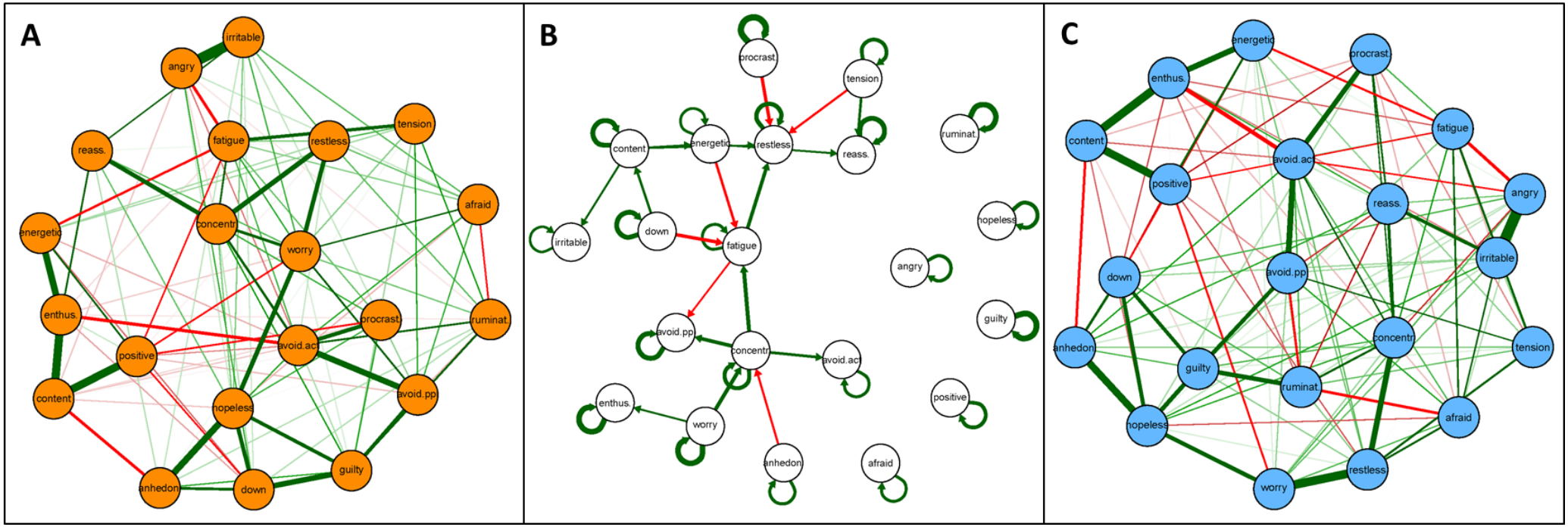

Figure 3: Contemporaneous concentration network at time *t* (A), directed network for lagged relationships between *t* and *t+1* (B), and residual concentration network at time *t+1* (C) for P145.

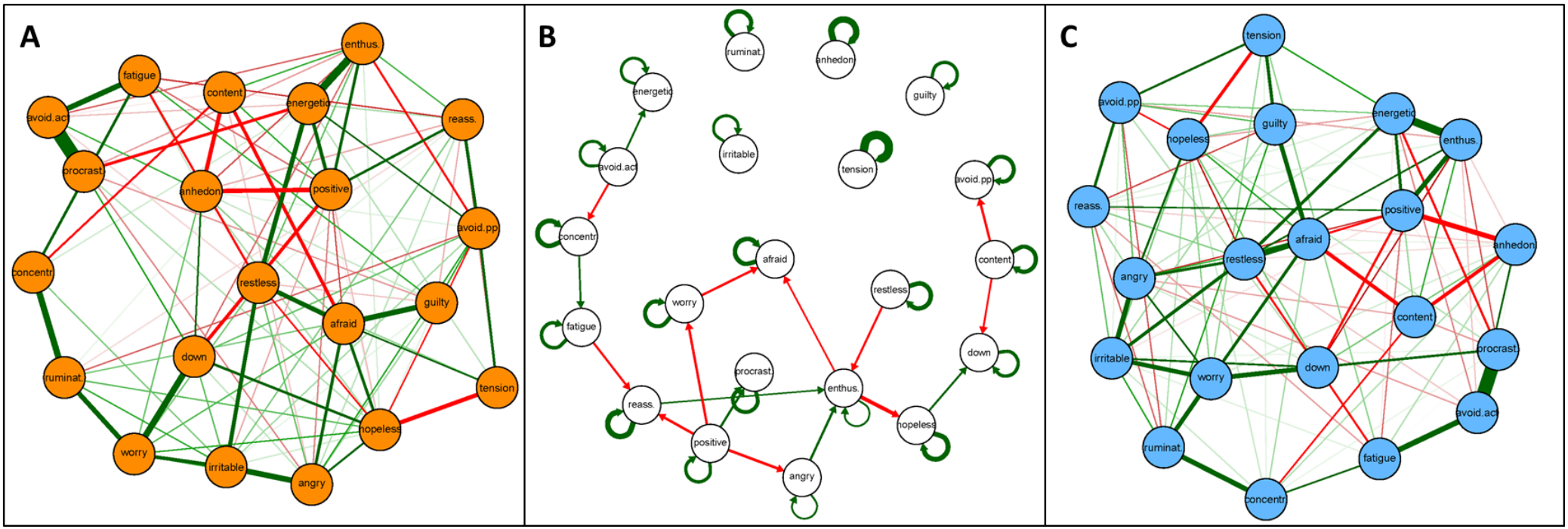

Figure 4: Contemporaneous concentration network at time *t* (A), directed network for lagged relationships between *t* and *t+1* (B), and residual concentration network at time *t+1* (C) for P72.