Supplemental Materials

Supplemental Table S1. Criteria for psychosis

Assessments	Threshold Indicating Presence of Psychosis	Inter-rater Reliability [a] (weighted kappa) [a]
Delusions	≥3	0.829**
Conceptual disorganization	≥4	0.459**
Hallucinatory behavior	≥3	0.737**
Suspiciousness / persecution	≥5	0.469**
Unusual thought content	≥4	0.739**
Presence/absence of any of the above symptoms exceeding threshold score for psychosis	—	0.690** [b]

[a] – using independent interviews of the same participant on the same day by different interviewers (N=26)
[b] – unweighted kappa statistic for psychosis state
**p<0.01

Section S1. Modified Charlson Comorbidity Index

This score was initially developed to assess the impact of medical conditions on 1-year mortality and has since been updated (Quan et al., 2011). For the present study we included the following conditions in the index calculation (1 point each unless otherwise stated): age (for each decade over age 40), diabetes mellitus, congestive heart failure, cerebrovascular disease, dementia, chronic pulmonary disease, peptic ulcer disease, moderate-to-severe renal disease (2 points), lymphoma (2 points), leukemia (2 points), tumor (2 points), moderate-to-severe liver disease (3 points), and AIDS (6 points).

Section S2. Multilevel Network Estimation

Psychotic symptom networks were constructed to estimate the relationships between psychotic symptoms across time. Auto-regressive models allow for the estimation of the stability or change in a variable over time within an individual. For auto-regressive models with a lag of one timepoint, a variable at timepoint *t-1* is used to predict that

same variable at timepoint *t*. Vector autoregressive (VAR) models extend this approach by modeling these lagged effects for multiple variables. In doing so, the VAR models estimate both the auto-regressive effects (how well a variable predicts itself at the next timepoint) as well as the cross-regressive effects (how well one symptom predicts a different symptom at the next timepoint). When a multilevel framework is used, a VAR model is estimated for each individual and the population overall to capture the within-individual dynamic interactions between variables over time (Epskamp, Waldorp, Mõttus, & Borsboom, 2016a).

Recently, theoretical and empirical evidence has shown that the patterns observed in the population may not be generalizable to the processes that occur within the individual. As an example, people who exercise more may have lower risk of heart attack (negative between-person effect), while one is more likely to experience a heart attack while exercising (positive within-person effect) (Curran & Bauer, 2011). This phenomenon is known as Simpson's paradox. The between-person and within-person effects can differ in both magnitude and direction and have different clinical implications. In addition, the relationship between symptoms may differ when examined at one moment versus over time. For example, hunger may predict subsequent eating, but hunger and eating may not co-occur. Thus, it is necessary to disaggregate the between-individual and within-individual effects, as well as the cross-sectional and the temporal effects, which is possible when the outcome and predictors are both tracked repeatedly over time in longitudinal study design.

In order to disentangle the within-person psychotic symptom dynamics from within-person cross-sectional ("state") and stable between-person differences ("trait"), we used a multilevel VAR modeling approach (Bringmann et al., 2013; Epskamp, Waldorp, et al., 2016a). We applied the two-step multilevel VAR strategy outlined in Epskamp et al. (2016) and executed in *mlVAR* package (Epskamp, Deserno, & Bringmann, 2017).

This multilevel VAR modeling approach was selected for several reasons. First, this model allows for the study of multivariate responses, and thus the interrelationships

between several outcomes (i.e., psychotic symptoms), unlike other longitudinal approaches such as mixed effects modeling. Second, the multilevel framework enables the examination of both within-person and between person effects, which may better capture personal change over time and how that may differ from trends seen across the population. This approach has been demonstrated as robust with a similar number of nodes in simulation study (Epskamp, Waldorp, Mõttus, & Borsboom, 2016b). Third, Gaussian Graphical Models have been estimated and found to fit the present scale, PANSS (Kay, Fiszbein, & Opler, 1987) well (Isvoranu et al., 2017). Fourth, this is the first longitudinal study of psychotic symptom networks and thus prior knowledge of expected relationships is limited. Thus, this probabilistic (i.e., frequentist) approach permits the estimation of effects using available data, perhaps to inform priors of future Bayesian inquiry.

We sought to deconstruct the psychotic symptom interrelationships into a multilevel network representing the “trait”, “state”, and “dynamic” components. First, to separate between-person and within-person effects, symptom scores were person-mean centered prior to multilevel VAR analysis (Hamaker & Grasman, 2014; Wang & Maxwell, 2015). The first step of multilevel VAR estimation yields participants’ mean symptom severity across assessments and participants’ time-varying fluctuations around their own mean in symptom severity score. This estimates the stable between-person similarities and differences in the population (Between-Person Network), as well as estimates of how symptoms interact to influence these fluctuations over time (Temporal Network). In the second step, the residuals were used to estimate the Contemporaneous Network, a map of how symptoms correlated within an individual at a given moment (within-individual cross-sectional relationships).

The multilevel VAR model can be understood as:

Level 1 Model:

$$\mathbf{Y}_{it} \mid \mathbf{y}_{it} = N\left(\mu_i + \mathbf{B}_i\left(\mathbf{y}_{i(t-1)} - \mu_i\right), \Theta_i\right)$$

Level 2 Model:

$$\begin{matrix} \mu_i \\ \text{Vec}\left(\mathbf{B}_i\right) \end{matrix} \sim N\left(\mathbf{f}, \Omega\right),$$

where, **y** is a vector of symptoms at time *t* for each individual *i*, μ denotes the stationary mean symptom scores, **B** encodes the matrix of within-person temporal effects (Temporal Network), and Θ denotes the partial correlation matrix of model residuals (i.e., Contemporaneous Network). In the Level 2 model, **f** represents the matrix of fixed effects (Between-Person Network) and Ω denotes the random effects distribution.

The model has several assumptions. First, stationarity over time is assumed. The Kwiatkowski-Phillips-Schmidt-Shin unit root (KPSS) test was used to assess stationarity of each variable over time. The KPSS test suggested a non-stationary trend for delusions, suspiciousness, hallucinatory behaviour, and confirmed stationarity of other variables. We applied a detrending procedure to all time series data with a non-stationary trend (Wang & Maxwell, 2015). To do so, we ascertained the residuals of a mixed effects linear model with random intercept and slope of the non-stationary variable with time as the only independent variable. Non-stationary time series data were replaced with these residuals plus the corresponding within-individual mean value. Second, the model also assumes normality of the joint conditional distribution and the marginal distribution of each variable tested by visual inspection of diagnostic plots. The person-specific random effects are estimated from a multivariate normal distribution. Third, multilevel VAR models assume equal intervals between time points (Bringmann et al., 2016; Epskamp, Waldorp, et al., 2016a).

Recently, there has been increased discussion about the impact of symptom score variance on network estimation. In accordance with Bulteel et al. (2016) and Schuurman et al. (2016), symptom scores were within-person standardized to limit the impact of any

differences in symptom variance. To test whether symptom variance may contribute to observed network differences, unstandardized and within-person standardized estimates were compared (Bulteel, Tuerlinckx, Brose, & Ceulemans, 2016; Schuurman, Ferrer, de Boer-Sonnenschein, & Hamaker, 2016). Of note, only participants that experienced change in their symptom severity could be included in the Temporal Network (n=294, 78.4%). Additionally, while centering the data allows for separation of within-person and between-person effects, this transformation may lead to under-estimation of the autoregressive effects (Hamaker & Grasman, 2014). Additionally, there has been concern that using single item measures may introduce measurement error (McNally, 2016). We address this concern by examining the inter-rater reliability of the symptom scores (**Supplemental Table S1**) and test the stability of network estimates using permutation approaches described below. Previous item response analysis identified that these cardinal symptoms were good to very good for discriminating symptom severity (Santor, Ascher-Svanum, Lindenmayer, & Obenchain, 2007). We also examine for structural equivalence between highly correlated items (Fried & Cramer, 2017; Lorrain & White, 1971), to test if the items are in fact representing the same clinical phenomena (i.e., have equivalent connections to other symptoms and therefore an equivalent role in the network). Last, as with any multiple regression analysis, the edge-weights represent only the independent, unique direct effects and do not include the shared effect of multiple symptoms (Bulteel et al., 2016).

In addition to examining within-person dynamics, between-person similarities and differences were estimated to determine the aggregate tendency for pairs of psychotic symptoms to be associated in the population. In the Between-Person Network, the edges represent the partial correlation matrix between individuals' mean symptom severity scores. In this way, we estimate the tendency of symptoms to co-occur in a population. For example, do individuals with severe delusions, on average, tend to experience severe suspiciousness? The relationships between the person-specific means represent the Level 2 part of the multilevel VAR model and is the vector of the intercepts for each symptom model. These relationships are not ordered over time, and, thus, the partial correlation matrix is symmetric. The partial correlation matrix is the

association between two variables, given all other variables in the network. The partial correlation matrix can be estimated by either standardizing the inverse variance-covariance matrix of the network, or by performing node-wise multiple regression with all other variables as covariates (Epskamp & Fried, 2016). Thus, partial correlations values equaling zero (visualized as an absent edge) mean that the two variables are independent, conditioning on all other variables in the network.

Lastly, the Contemporaneous Network is an undirected network representing the co-occurrence of symptoms within an individual at a given time. Multilevel VAR model residuals were used to estimate how much of the unexplained variance in symptoms at time *t* were explained by another co-occurring symptom, conditioning on other co-occurring symptoms.

Centrality measures were calculated for each symptom within the Between-Person, Contemporaneous, and Temporal Networks. The centrality measures include strength, closeness, and betweenness (Opsahl, Agneessens, & Skvoretz, 2010). Strength is the sum of the edge weights for each node and thus is a measure of local structure. In a directed graph (Temporal Network), the sum of outgoing edges is called out-strength and is a measure of the symptom's influence on other symptoms in the network. The in-strength is the sum of incoming edges and is an indicator of how downstream a symptom is in the activation cascade. Closeness estimates how proximate the symptom is to all other nodes in the network and is the sum of the inverse shortest paths to each other node. Closeness estimates the efficiency by which a symptom may exert its influence. Lastly, betweenness is the number of paths the symptom mediates, and thus represents its role as a gatekeeper, transmitting activation between other pairs of nodes.

Section S3. Missing Data Assessment

Missing data is highly common in longitudinal studies. Thus, data were first assessed for the rate and pattern of missingness, including missing visits or missing variables.

When data are considered missing completely at random, the probability of missingness is independent of both the variable itself and other observed variables (Wu, 2009). In the case of data being missing at random, the probability of a variable being missing is unrelated to the values of the variable itself, but may be dependent on observed variables. To test whether data are missing at random and whether the patterns of missingness alter our findings, we performed several sensitivity and multiple imputation analyses (Bieling et al., 2015). Sensitivity analyses were performed to compare participants with missing versus available data on factors that may be related to the variable in question, including any available observations of the variable itself. Further, groups of participants with differing number of total visits made or differing reasons for discontinuation were compared.

Overall, participants who remained engaged in the study for five years were similar to those who discontinued. Notably, measures related to our primary outcomes of psychosis were not associated with whether a psychotic symptom assessment was missed (**Supplemental Table S2**). Younger age, however, was associated with psychosis missingness over the five years. Discontinuation occurred due to death (41, 11.0% individuals; contributing 1176, 5.1% months during follow-up), lost to follow-up (77, 20.5% individuals; contributing 3671, 16.0% months; e.g., moved away from Vancouver, incarceration, or living in a treatment facility may precede discontinuation), or withdrawal from the study (6, 1.6% individuals; contributing 255, 1.1% months). There were differences between individuals who died versus those who were lost to follow-up for other reasons (LTF): older age (mean, SD: Died 49.4, 8.4 versus LTF 39.7, 10.4, $p<0.001$), and less likely to have methamphetamine dependence (proportion: Died 7.7% versus LTF 32.9%, $p=0.006$), or a history of homelessness (proportion: Died 52.6% versus LTF 79.7%, $p=0.005$). Thus, participants censored in the survival analysis did not seem to discontinue due to greater co-occurring morbidity or illness (i.e., non-informative censoring). Altogether, data appeared to be missing at random and unrelated to psychosis, though differences in reasons for discontinuation (i.e., death) should be considered in multiple imputation analysis.

Multiple imputation analysis was performed using the *mice* package (van Buuren & Groothuis-Oudshoorn, 2011). First, imputed values are generated from regression models with relevant predictors, including covariates from the main model of interest, and other variables that related to a variable's missingness (Bieling et al., 2015). Based in the sensitivity analyses, death (time-varying indicator), Trauma History Questionnaire (THQ) score by age 18, history of homelessness, and methamphetamine, cannabis, and alcohol dependence were included as potentially relevant predictors. Continuous variables were predicted by posterior mean matching and binary variables were predicted by logistic regression. We imputed ten completed datasets with ten iterations for the imputation analysis, as is recommended by standard multiple imputation procedures (Rubin, 1987). Second, the main model of interest was fit to each of the completed datasets. Last, the parameter estimates from the second step were pooled to produce final parameter estimates. Pooled parameter estimates from the imputed datasets were compared to those from the complete-case analysis to test whether our findings and inferences were affected by missing data.

Supplemental Table S2. Factors associated with missingness of PANSS assessments

Factor	β	$SE_β$	p
Age (years)	-0.034	0.013	0.007*
Sex	-0.235	0.290	0.421
Died (time-varying)	-0.671	0.426	0.115
Psychotic Baseline	0.046	0.226	0.838
Completed High School	0.142	0.274	0.603
SOFAS BL	0.007	0.010	0.517
THQ score	0.037	0.047	0.432
Schizophrenia/Schizoaffective Disorder	0.374	0.352	0.288
Methamphetamine Dependence	0.087	0.276	0.753
Cannabis Dependence	-0.255	0.261	0.328
Powder Cocaine Dependence	-0.400	0.278	0.150
Alcohol Dependence	0.086	0.312	0.782
Persistent Sequelae of Past TBI	-0.316	0.374	0.397
Ever homeless	0.300	0.266	0.260

PANSS = Positive and Negative Syndrome Scale; SOFAS = Social and Occupational Functioning Assessment Scale; BL = baseline; THQ = Trauma History Questionnaire; TBI = traumatic brain injury; SE = standard error of effect coefficient.

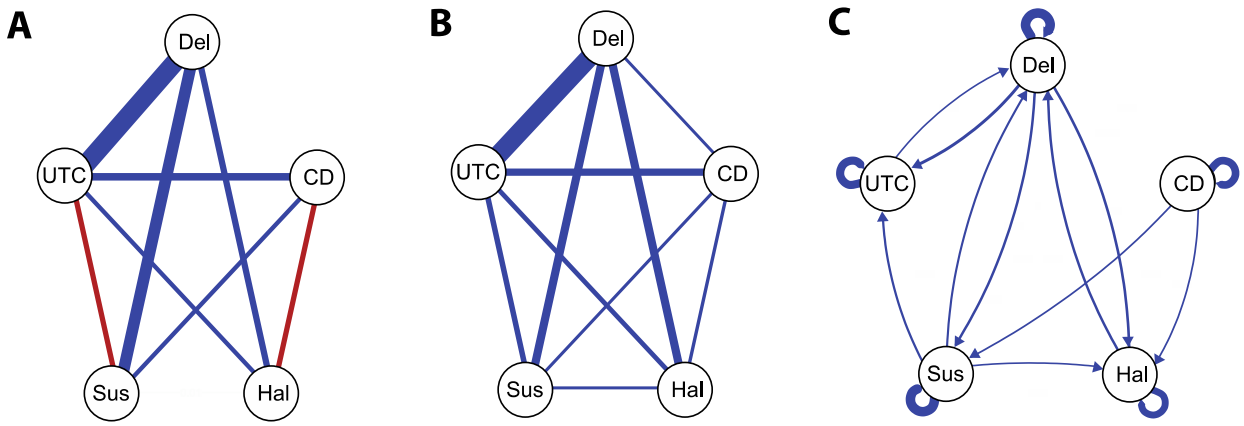
Section S4. Group Comparison of Symptom Network Connectivity and Structure

Lastly, networks were estimated for two groups: participants with and without lifetime psychotic disorder diagnosis. Currently there is no gold standard approach for comparing dynamic networks between groups. However, there are two methods in the literature available for comparing Temporal networks between groups.

First, Bringmann et al. (2013) compared groups of participants by constructing an omnibus model of all participants including an interaction term between group membership and the cross-regressive and auto-regressive effects in the Temporal Network model directly. We applied this approach in our current study to compare the Temporal networks between history-positive and history-negative groups. In brief, each node represents a symptom and each edge represents the lagged effect coefficient between two nodes. Multilevel VAR modeling was used to estimate the directed edge weights between psychosis symptoms over time. Specifically, a multilevel VAR(1) model was fit for each symptom, where a symptom at time point *t* served as a dependent variable and the five time-lagged symptoms at time point *t-1* (past month) served as predictors, with interaction terms between these predictors and group membership (binary variable) estimating the difference in edge-weights between groups.

Second, Klippel et al. (2017) proposed a permutation procedure to estimate group differences in average Temporal network connectivity (density), including both auto-regressive and cross-regressive connectivity, as well as group differences in each edge-weight. In brief, group membership was reshuffled between participants 10,000 times, models were re-fitted, and the permutation distributions of the size of the group differences were obtained under the null hypothesis. The size of the observed group differences for connectivity or edge-weight were compared to the permutation distribution to determine level of significance of group differences in Temporal Network characteristics.

Supplemental Figure S1. Whole sample multilevel network



Multilevel network of psychotic symptoms including A. Between-Person Network, B. Contemporaneous Network, and C. Temporal Network. (n=375, 14,622 observations) Del = delusions; CD = conceptual disorganization; Hal = hallucinatory behavior; Sus = suspiciousness or persecution; UTC = unusual thought content; Blue = positive edges; Red = negative edges. Out-Edges = outgoing edges; In-Edges = incoming edges. Edge thickness indicates edge-weight.

Supplemental Table S3. Psychotic symptom network centrality for whole sample

	Del	CD	Hal	Sus	UTC
Temporal Network					
Out-strength	0.220	0.067	0.081	0.166	0.059
In-strength	0.118	0.093	0.138	0.097	0.148
Closeness	0.012	0.006	0.005	0.010	0.005
Betweenness	5	0	0	2	0
Contemporaneous Network					
Strength	0.895	0.381	0.458	0.454	0.827
Closeness	0.045	0.025	0.028	0.027	0.041
Betweenness	6	0	0	0	2
Between-Person Network					
Strength	1.471	0.649	0.631	0.619	1.201
Closeness	0.078	0.048	0.050	0.056	0.071
Betweenness	6	0	0	0	2

Supplemental Table S4. Permutation analysis results of differences in edge-weights between History-Positive and History-Negative Groups

Lagged *t-1* Symptom	Symptom at Time *t*	Edge-Weight Difference	p-value
P1	P1	-0.0396	0.3026
P2	P1	-0.0224	0.3476
P3	P1	-0.0024	0.9050
P6	P1	-0.0065	0.7718
G9	P1	0.0220	0.4488
P1	P2	-0.0384	0.2592
P2	P2	0.0181	0.5986
P3	P2	0.0111	0.6374
P6	P2	-0.0114	0.6460
G9	P2	0.0246	0.4506
P1	P3	-0.0695	0.0508
P2	P3	-0.0113	0.6974
P3	P3	0.0507	0.1258
P6	P3	0.0026	0.9264
G9	P3	0.0316	0.2254
P1	P6	-0.0883	0.0022
P2	P6	-0.0089	0.7362
P3	P6	-0.0219	0.3614
P6	P6	0.0606	0.0500
G9	P6	0.0596	0.0226
P1	G9	-0.0799	0.0170
P2	G9	0.0087	0.6824
P3	G9	0.0132	0.5926
P6	G9	0.0089	0.7010
G9	G9	0.0430	0.2018

Supplemental Table S5. Risk factors for psychotic symptom network global connectivity

	Global Connectivity					
	Unadjusted			Adjusted‡		
	B	SE	p	B	SE	p
History of psychotic disorder	0.022	0.047	0.644	0.019	0.048	0.696
Age, years	0.004	0.002	0.077	0.006	0.003	0.019
Female sex	-0.060	0.057	0.293	-0.062	0.056	0.271
Past homelessness	-0.026	0.052	0.616	—	—	—
Completed high school or equivalent	-0.008	0.046	0.867	—	—	—
THQ score by age 18	0.013	0.009	0.156	—	—	—
Methamphetamine dependence	0.102	0.051	0.044	0.135	0.052	0.010
Cannabis dependence	0.035	0.048	0.472	—	—	—
Alcohol dependence	-0.013	0.059	0.826	—	—	—
Cocaine dependence	-0.051	0.049	0.300	—	—	—
Heroin dependence	0.009	0.048	0.853	—	—	—
Antipsychotic treatment	-0.048	0.064	0.454	—	—	—

THQ: Trauma History Questionnaire.

‡ n=290

Supplemental Table S6. Risk factors for unique edge-weight Del-UTC

	Del-UTC edge-weight					
	Unadjusted			Adjusted ‡		
	B	SE	p	B	SE	p
History of psychotic disorder	-0.016	0.008	0.042	-0.020	0.008	0.013
Age, years	-0.001	0.001	0.973	0.001	0.001	0.651
Female sex	-0.003	0.010	0.719	-0.006	0.009	0.543
Past homelessness	0.006	0.009	0.528	—	—	—
Completed high school or equivalent	0.012	0.008	0.130	—	—	—
THQ score by age 18	0.002	0.002	0.148	—	—	—
Methamphetamine dependence	0.025	0.009	0.004	0.030	0.009	<0.001
Cannabis dependence	0.006	0.008	0.493	—	—	—
Alcohol dependence	-0.003	0.010	0.736	—	—	—
Cocaine dependence	-0.012	0.008	0.135	—	—	—
Heroin dependence	0.009	0.008	0.287	—	—	—
Antipsychotic treatment	-0.009	0.011	0.409	—	—	—

Del = Delusions. UTC = Unusual Thought Content. The edge-weight is the association of change in Del severity at timepoint *t-1* and the change in UTC severity at timepoint *t*.

‡ n=290

Supplemental Table S7. Risk factors for unique edge-weight

	Del-Sus edge-weight					
	Unadjusted			Adjusted ‡		
	B	SE	p	B	SE	p
History of psychotic disorder	-0.001	0.002	0.903	-0.001	0.002	0.956
Age, years	0.001	0.001	0.499	0.001	0.001	0.576
Female sex	-0.003	0.003	0.320	-0.003	0.003	0.354
Past homelessness	0.002	0.002	0.371	—	—	—
Completed high school or equivalent	-0.001	0.002	0.580	—	—	—
THQ score by age 18	0.003	0.004	0.384	—	—	—
Methamphetamine Dependence	0.001	0.002	0.680	—	—	—
Cannabis Dependence	0.001	0.002	0.542	—	—	—
Alcohol Dependence	0.002	0.003	0.393	—	—	—
Cocaine Dependence	-0.001	0.002	0.953	—	—	—
Heroin Dependence	-0.001	0.002	0.839	—	—	—
Antipsychotic Treatment	0.001	0.003	0.845	—	—	—

Del = Delusions. Sus = Suspiciousness. The edge-weight is the association of change in Del severity at timepoint *t-1* and the change in Sus severity at timepoint *t*.

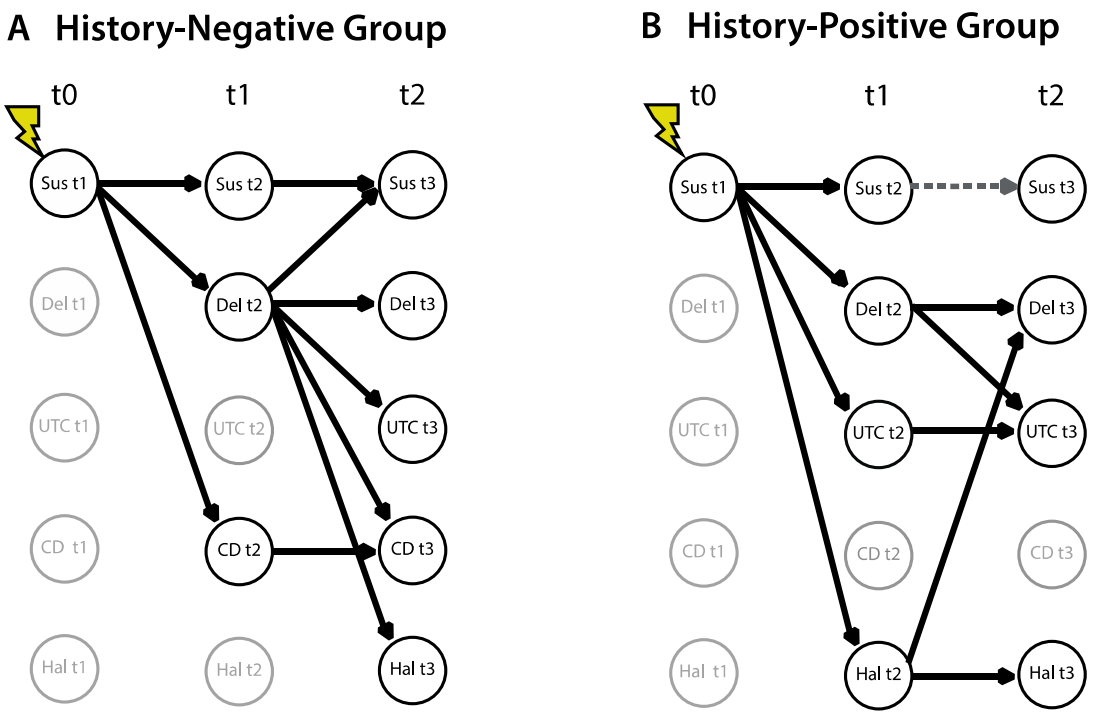
‡ n=290

Supplemental Table S8. Relationships between symptom auto-regression and stationary mean severity

	Delusions		Conceptual Disorganization		Hallucinatory Behavior		Suspiciousness/ Persecution		Unusual Thought Content	
Autoregressive Effects										
Mean, SD	0.21	0.12	0.18	0.09	0.13	0.11	0.19	0.10	0.14	0.09
Min, Max	-0.07	0.47	-0.08	0.46	-0.15	0.49	-0.03	0.45	-0.16	0.33
	β	SE_β	β	SE_β	β	SE_β	β	SE_β	β	SE_β
Age	0.010	0.007	0.008	0.005	-0.007	0.006	0.021***	0.006	-0.002	0.005
Sex	-0.001	0.017	-0.017	0.014	-0.010	0.016	-0.023	0.014	-0.017	0.013
Stationary mean	0.013	0.007	0.021***	0.005	0.033***	0.006	0.015**	0.006	0.015**	0.005
Stationary mean* age interaction	—	—	—	—	0.024***	0.007	—	—	—	—

* p<0.05, ** p<0.01, *** p<0.001

Supplemental Figure S2. Schematic of Temporal Network activation of History-Negative and History-Positive Groups



The symptom activation cascade as estimated by the Temporal Networks of the (A) History-Negative group and (B) History-Positive group. Given a hypothetical event that triggers the activation of suspiciousness, there are different possible downstream consequences – and opportunities for prevention or therapy – between each group. Black arrows represent estimated effects from Temporal Network. Dashed lines represent the unknown persistent effects given that the multilevel VAR(1) model tests only 1-month lag. Del = delusions; CD = conceptual disorganization; Hal = hallucinatory behavior; Sus = suspiciousness/persecution; UTC = unusual thought content; lightning bolt represents a potential triggering event (i.e., stressor).

References

Bates, D., Maechler, M., Bolker, B., & Walker, S. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software*, *67*(1), 1–48.

Bringmann, L. F., Pe, M. L., Vissers, N., Ceulemans, E., Borsboom, D., Vanpaemel, W., … Kuppens, P. (2016). Assessing temporal emotion dynamics using networks. *Assessment*, *23*(4), 425–435. https://doi.org/10.1177/1073191116645909

Bringmann, L. F., Vissers, N., Wichers, M., Geschwind, N., Kuppens, P., Peeters, F., … Tuerlinckx, F. (2013). A network approach to psychopathology: New insights into clinical longitudinal data. *PLoS ONE*, *8*(4). https://doi.org/10.1371/journal.pone.0060188

Bulteel, K., Tuerlinckx, F., Brose, A., & Ceulemans, E. (2016). Using Raw VAR Regression Coefficients to Build Networks can be Misleading. *Multivariate Behavioral Research*, *51*(2–3), 330–344. https://doi.org/10.1080/00273171.2016.1150151

Curran, P. J., & Bauer, D. J. (2011). The disaggregation of within-person and between-person effects in longitudinal models of change. *Annual Review of Psychology*, *62*, 583–619. https://doi.org/10.1146/annurev.psych.093008.100356.The

Epskamp, S., Borsboom, D., & Fried, E. I. (2016). Estimating Psychological Networks and their Accuracy: A Tutorial Paper. https://doi.org/10.3758/s13428-017-0862-1

Epskamp, S., Cramer, A., Waldorp, L., Schmittmann, V., & Borsboom, D. (2012). Qgraph : Network Visualizations of Relationships in. *Journal of Statistical Software*, *48*(4), 1–18.

Epskamp, S., Deserno, M. K., & Bringmann, L. F. (2017). mlVAR: Multi-level Vector Autoregression.

Epskamp, S., & Fried, E. I. (2016). A tutorial on regularized partial correlation networks. *ArXiv Preprint ArXiv*, 1–28. https://doi.org/10.1103/PhysRevB.69.161303

Epskamp, S., Waldorp, L. J., Mõttus, R., & Borsboom, D. (2016a). Discovering psychological dynamics: The Gaussian Graphical Model in cross-sectional and time-series data.

Epskamp, S., Waldorp, L. J., Mõttus, R., & Borsboom, D. (2016b). Discovering Psychological Dynamics: The Gaussian Graphical Model in Cross-sectional and Time-series Data, 1–56.

Fried, E.I & Cramer, A. O. J. (2017). Moving forward: Challenges and directions for psychopathological network theory and methodology. *Perspectives on Psychological Science*. https://doi.org/10.17605/OSF.IO/BNEK

Friedman, J., Hastie, T., & Tibshirani, R. (2007). Sparse inverse covariance estimation with the lasso, 1–14. https://doi.org/10.1093/biostatistics/kxm045

Hamaker, E. L., & Grasman, R. P. P. P. (2014). To center or not to center? Investigating inertia with a multilevel autoregressive model. *Frontiers in Psychology*, *5*(OCT), 1–15. https://doi.org/10.3389/fpsyg.2014.01492

Isvoranu, A.-M., Van Borkulo, C. D., Boyette, L. Lou, Wigman, J. T. W., Vinkers, C. H., Borsboom, D., … Myin-Germeys, I. (2017). A network approach to psychosis: Pathways between childhood trauma and psychotic symptoms. *Schizophrenia Bulletin*, *43*(1), 187–196. https://doi.org/10.1093/schbul/sbw055

Kay, S. R., Fiszbein, A., & Opler, L. A. (1987). The Positive and Negative Syndrome

Scale (PANSS) for schizophrenia. *Schizophrenia Bulletin*, *13*(2), 261–276.
Klippel, A., Viechtbauer, W., Reininghaus, U., Wigman, J., Borkulo, C. Van, Myin-germeys, I., & Wichers, M. (2017). The Cascade of Stress : A Network Approach to Explore Differential Dynamics in Populations Varying in Risk for Psychosis. *Schizophrenia Bulletin*. https://doi.org/10.1093/schbul/sbx037
Lorrain, F., & White, H. (1971). Structural equivalence of individuals in social networks. *The Journal of Mathematical Sociology*, *1*(1), 49–80.
McNally, R. J. (2016). Can network analysis transform psychopathology? *Behaviour Research and Therapy*, *86*, 95–104. https://doi.org/10.1016/j.brat.2016.06.006
Opsahl, T., Agneessens, F., & Skvoretz, J. (2010). Node centrality in weighted networks: Generalizing degree and shortest paths. *Social Networks*, *32*(3), 245–251. https://doi.org/10.1016/j.socnet.2010.03.006
Pe, M. L., & Kuppens, P. (2012). The dynamic interplay between emotions in daily life: Augmentation, blunting, and the role of appraisal overlap. *Emotion*, *12*(6), 1320–1328. https://doi.org/10.1037/a0028262
Quan, H., Li, B., Couris, C. M., Fushimi, K., Graham, P., Hider, P., … Sundararajan, V. (2011). Updating and validating the charlson comorbidity index and score for risk adjustment in hospital discharge abstracts using data from 6 countries. *American Journal of Epidemiology*, *173*(6), 676–682. https://doi.org/10.1093/aje/kwq433
Rubin, D. B. (1987). *Multiple imputation for nonresponse in surveys*. New York: Wiley Online Library.
Santor, D. A., Ascher-Svanum, H., Lindenmayer, J. P., & Obenchain, R. L. (2007). Item response analysis of the Positive and Negative Syndrome Scale. *BMC Psychiatry*, *7*, 1–10. https://doi.org/10.1186/1471-244X-7-66
Schuurman, N. K., Ferrer, E., de Boer-Sonnenschein, M., & Hamaker, E. L. (2016). How to compare cross-lagged associations in a multilevel autoregressive model. *Psychological Methods*, *21*(2), 206–221. https://doi.org/10.1037/met0000062
Singer, J., & Willett, J. (2003). *Applied longitudinal data analysis*. New York: Oxford University Press.
van Borkulo, C., Boschloo, L., Borsboom, D., Penninx, B. W. J. H., Waldorp, L. J., & Schoevers, R. A. (2015). Association of Symptom Network Structure With the Course of Depression. *JAMA Psychiatry*, *72*(12), 1219. https://doi.org/10.1001/jamapsychiatry.2015.2079
van Buuren, S., & Groothuis-Oudshoorn, K. (2011). mice: Multivariate imputation by chained equations in R. *Journal of Statistical Software*, *45*(3), 1–67. https://doi.org/10.18637/jss.v045.i03>.
Wang, L. P., & Maxwell, S. E. (2015). On disaggregating between-person and within-person effects with longitudinal data using multilevel models. *Psychological Methods*, *20*(1), 63–83.
Wu, L. (2009). Mixed Effects Models for Complex Data. *Journal of Chemical Information and Modeling*, *53*(9), 1689–1699. https://doi.org/10.1017/CBO9781107415324.004