BJPsych | The British Journal of Psychiatry (2026)
1–9. doi: 10.1192/bjp.2026.10650

Original Article

The functional relevance of a short assessment of formal thought disorder in psychosis

Fatme Abboud, Jessica Ahrens, Estèe Ballès, Valentina Bambini, Bill Deakin, Neil A. Harrison, Tilo Kircher, Gina Kuperberg, Peter F. Liddle, Rohit Lodhi, Michael Mackinley, Susan L. Rossell, Krish D. Singh, Iris E. Sommer, Alban Voppel, Farida Zaher, Nadia Zeramdini and Lena Palaniyappan

Background

Formal thought disorder (FTD) is a highly disabling transdiagnostic feature that impedes communication and social ties. Progress in understanding and treating FTD has been hampered by the uncertainties in its assessment.

Aims

We examined if a short 3–5min assessment of transcribed speech can capture the latent dimensions and network structure of FTD and predict functional outcomes.

Method

In a transdiagnostic sample ($N = 666$) with a single longitudinal follow-up over 3–12 months ($n = 244$), we administered the short form of the Thought and Language Index to measure eight individual features of FTD. We determined the baseline factor structure of FTD, its temporal invariance at follow-up, and the predictive validity of FTD dimensions on the global single-item Social and Occupational Functioning Assessment Scale scores at baseline and follow-up. We identified the most influential and putative primary phenomena within the FTD syndrome, using network analysis.

Results

Factor analyses revealed a stable three-factor model of FTD: impoverishment (poverty of speech, weakening of goal), loosening (looseness, illogicality) and peculiarities (peculiar words, peculiar sentences), with excellent fit (Comparative Fit Index: 0.997, root mean square error of approximation: 0.040) and metric invariance over time. Impoverishment and peculiarities predicted functioning at baseline and 3–12 months later (cross-sectional: $\beta = -0.196$, $p < 0.001$ and $\beta = -0.298$, $p = 0.001$, respectively; longitudinal: $\beta = -0.201$, $p = 0.037$ and $\beta = -0.336$, $p = 0.042$, respectively). Looseness and poverty of speech were putative primary features influencing other FTD phenomena. Weakening of goal and peculiar sentences were the most connected phenomena.

Conclusions

By integrating latent variable and network approaches, we provide a unified, empirically grounded framework to interpret FTD assessed using a brief speech task. We report a replicable three-dimensional structure, identify central symptoms that may maintain the FTD syndrome, and the specific dimensions that influence functional disability. These findings clarify the prognostically valuable features of FTD for future mechanistic and interventional research.

Keywords

Disorganisation; language; incoherence; prognosis; transdiagnostic.

Formal thought disorder (FTD) is a transdiagnostic syndrome that severely disrupts communication and social functioning in people with severe mental illnesses.[1,2] Despite its long history in psychiatry,[3] our ability to assess it during routine clinical encounters remains a challenge. Clinicians do not routinely use any of the available but long and arduous instruments to quantify its severity.[4] Short-form of clinically employable instruments that use visual stimuli to evoke disordered thinking, such as the Thought and Language Index (TLI),[5] are a promising approach for large-scale clinical use. In addition, the ubiquitous availability of automatic transcription enables detailed text-based analyses of thought disorder. However, we do not know if such brief procedures capture the various core dimensions of FTD and are relevant to functional outcomes, i.e. predicting social and occupational functions that are affected in the presence of psychosis.

Overcoming methodological limitations with a new approach to uncover FTD dimensions

Development of instruments to assess FTD has faced key methodological limitations, including restricted sample sizes, a narrow focus on schizophrenia and the contamination of ratings by other psychotic symptoms (i.e. *content* contaminating the assessment of *form*, e.g., a person with bizarre delusions is more likely to get rated on the FTD item of illogicality) when the ratings are based on a clinical interview (see McKenna and Oh[6] for a detailed treatment of other challenges). Similarly, on-the-fly rating (i.e. rating thought disturbances without recorded transcripts) carries the risk of over-reliance on momentary clinical impression.[7] As a result, factor analytical reports on the underlying dimensions of FTD have been inconsistent (one to seven factors reported)[8] and fail to provide stable, time-invariant, population-level dimensions. To overcome these issues, we employed a large, transdiagnostic sample and rated FTD exclusively from recorded speech transcripts, thereby minimising bias. Furthermore, we complemented traditional factor analysis with two graphical models: (a) a network approach to reveal direct relationships between FTD phenomena and (b) directed acyclic graphs (DAGs) based on conditional dependence to identify hierarchical relationships. Network analysis can uncover highly connected central phenomena, which, when alleviated, can reduce the overall burden (i.e. identify interventional targets[9]). DAGs can uncover putative causal pathways, providing insight into how clinical phenomena relate to and potentially influence one another, highlighting possible targets for future mechanistic and computational studies. Latent factor models alone cannot disentangle the putative directionality of these relationships.[10]

https://doi.org/10.1192/bjp.2026.10650 Published online by Cambridge University Press

Linking dimensions to functional outcome

Our goal was to integrate latent factor modelling and network analyses to provide a unified framework for describing core elements of FTD from a short, time-efficient instrument. A crucial test for any psychopathological construct is its predictive validity for real-world outcomes.[11] Although FTD is broadly linked to poor functioning,[12–14] studies are divided in linking specific FTD dimensions to this impairment (impoverishment dimension;[15–18] disorganisation dimension[19–21]). There are no FTD-specific interventions available, but remediation appears feasible for several individual phenomena that constitutes FTD (e.g. Bambini et al;[22] see Jimeno[23] for a review). Identifying the functionally relevant aspects of FTD (i.e. those affecting social, occupational and daily functioning) will assist in targeted interventions to improve overall outcomes. Importantly, when FTD is assessed independently of other psychotic symptoms (i.e. without the raters' knowledge of other psychopathology), its severity appears less pronounced,[24] and the relationship with functioning appears to drop by >50%.[13,19] This raises the question of whether FTD as a standalone construct has any value in the prognostic impression that we can make in clinical practice. Using a longitudinal design, we tested the hypothesis that data-driven dimensions from a ring-fenced assessment of FTD (not influenced by a rater's access to the participant's other symptoms) can predict social and occupational functioning both at baseline and 3–12 months later. Our goal was to reliably quantify putative latent dimensions and demonstrate their relevance to real-world functioning, with a view to positioning FTD as a clinically meaningful prognostic specifier for psychotic illnesses.

Method

Participants

Data from eligible participants enrolled in seven separate, geographically diverse studies were pooled for this project: Discourse in Psychosis-University of Western Ontario (DISCOURSE-UWO), IMproved Personalized medicine through deep LEarning in MENTal disorders – Montreal (IMPLEMENT), Tracking Outcomes in Psychosis (TOPSY, London Ontario), Cannabis Effects On White Matter And Outcomes In Early Psychosis (WOW, London Ontario), The Study of Psychosis and the Role of Inflammation and GABA/Glutamate (SPRING, Nottingham, Manchester and Cardiff, UK), Connectivity-Nottingham Study (CONN) and 7-Tesla Schizophrenia Study Nottingham (UK7T). Details on the recruitment and enrolment procedures of participants have been described previously.[25–30] Data-set descriptions can be found in Supplementary Appendix 2 available at https://doi.org/10.1192/bjp.2026.10650. To be eligible for pooled analysis, the participant should have had (a) a clinical diagnosis confirmed by a consensus procedure; (b) the same three-picture version of FTD assessment administered; and (c) FTD ratings completed using recorded and subsequently transcribed speech, using a consensus approach, ensuring a single reliable score for each participant.

At baseline, a total of 666 participants ($n = 472$ with a psychiatric diagnosis, $n = 194$ healthy controls and a clinical high-risk group). Follow-up data were available for 244 participants (177 patients, 67 controls).

Six different diagnostic groups with evidence of psychosis were included (bipolar disorder with psychosis, major depressive disorder with psychosis, psychosis (not otherwise specified), schizoaffective, schizophrenia, schizophreniform disorder, and a clinical high-risk group (from the TOPSY study[31]) with subthreshold psychotic symptoms), in addition to healthy controls with no lifetime diagnosis of a psychiatric disorder. Written informed consent was obtained from all participants, and all study procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2013. All procedures involving human patients were approved by the relevant local ethics boards. Ethics approval codes: DISCOURSE-UWO: 2024-119548-101057; TOPSY: 2025-108268-113605; WOW: 2024-112846-101698; IMPLEMENT: 2022-IUSMD-21-53; SPRING: 14/NW/0298; CONN: 08/H0401/120; UK7T: 10/H0406/49.

Assessments

At baseline, age, gender and diagnosis demographics were collected. Demographic statistics were calculated using the R package *gtsummary* used on the Windows platform.[32]

The TLI was used to assess thought disorder across eight items grouped into two subscales[5]: impoverishment (poverty of speech, weakening of goal and perseveration reflecting negative FTD) and disorganisation (looseness, peculiar word/sentence, illogicality and distractibility reflecting positive FTD). Full description of the TLI items can be found in Supplementary Appendix 1. Each instance is scored from 0.0 to 1.0 in increments of 0.25, based on severity. Participants described three pictures from the Thematic Apperception Test (as described in Sommer et al[33]), which was recorded and manually transcribed and scored by a single assessor blind to functional status and diagnosis at the time of rating (L.P. in consensus with TLI author P.F.L. for UK7T, CONN and SPRING UK; L.P. supervising and achieving consensus with a trained rater for WOW, TOPSY, DISCOURSE-UWO and IMPLEMENT; Note: IMPLEMENT was a schizophrenia-only cohort and diagnostic blinding does not apply). The item scores for the three images were summed to create an overall item score per participant. Total speaking time was restricted to 1 min per picture to control for potential bias owing to verbosity.

To assess social and occupational functioning, the Social and Occupational Functional Assessment Scale (SOFAS) was used in the present study.[34] The SOFAS is a single-item global measure, integrating social/interpersonal and occupational/educational functioning rated on a scale between 1 (lower functioning) and 100 (better functioning), based on a consensus with the clinical team, available medical notes and the information on daily functions provided by the participants. As this scale is susceptible to observer bias, to reduce systematic within-study variations, we only used datasets where the same rater rated all participants in a data-set for the SOFAS analysis (thus, the three-site SPRING study was not included). The functioning of participants from the UK7T study was assessed using the Global Assessment of Functioning (GAF) scale. To harmonise functioning scores across studies, we used the corresponding scores calculated using the equipercentile linking method to transform GAF to SOFAS scores.[35] This method has demonstrated a strong linear linkage ($r = 0.86–0.93$) between GAF and SOFAS in a large, naturalistic data-set, and minimal mean differences between the two scales, proving to be essentially exchangeable with negligible measurement error.[35]

Of those who had baseline (time point 1) TLI data, follow-up (time point 2) data were available for 169 participants for TLI and 216 for SOFAS at 3 months (IMPLEMENT) or 12 months after the first assessment (WOW, DISCOURSE-UWO and TOPSY).

Statistical analysis

Exploratory and confirmatory factor analyses

To identify the underlying latent structure of TLI, we performed exploratory factor analysis (EFA) on TLI scores at baseline after checking assumptions with Bartlett test of sphericity[36] and the

https://doi.org/10.1192/bjp.2026.10650 Published online by Cambridge University Press

Kaiser–Meyer–Olkin (KMO)[37] measure of sampling adequacy. Factors were identified using factor eigenvalues, with an oblique promax rotation method. We performed EFA using maximum likelihood for decomposition. The number of factors was determined using parallel analysis. Variables with communality less than 0.2 were removed.

To confirm the structure of TLI identified using EFA, we performed confirmatory factor analysis (CFA) with structural equation modelling. Multivariate normality was assessed with Mardia's test. Satorra–Bentler correction was applied as it is robust to non-normality.[38] CFA compared the EFA-based data-driven model against a theoretical model that specified two factors, 'disorganisation of thought' (peculiar sentences, illogicality, looseness, peculiar words) and 'impoverishment of thought' (poverty of speech, weakening of goal, perseveration), and compared their fit to the data to identify the best factorisation of TLI. Fit was assessed with Comparative Fit Index (CFI), Tucker–Lewis Index and root mean square error of approximation (RMSEA). EFA and CFA were both performed on JASP version 0.95.4 for Windows.

Network analysis

Network analysis was conducted to investigate the relationship between TLI items. The Extended Bayesian Information Criterion Graphical Lasso (EBICglasso) estimator within JASP version 0.95.4 was used to calculate a sparse Gaussian graphical model.[39,40] A $\gamma = 0.5$ tuning parameter was selected as a regularisation hyperparameter. This value is optimised for prioritising sparsity and high specificity, and minimising the inclusion of spurious edges in the network.[40,41] Network nodes represent a single TLI item, and edges represent partial correlations.[42] Non-parametric bootstrapping was conducted 1000 times to assess edge and centrality stability.

DAGs

To investigate potential causal relationships within TLI items, we computed a Bayesian network using the hill-climbing algorithm available through the R package *bnlearn* used on the Windows platform.[43] Hill-climbing is a greedy search algorithm that begins with a random solution and iteratively identifies more optimal solutions by making incremental changes toward better neighbouring states. In the context of DAGs, hill-climbing adds, deletes or reverses single arcs until an optimum is reached.[44] The algorithm undergoes random restarts to avoid local optima as solutions. To ensure stability, an average network was computed, using networks derived from 1000 bootstrapped samples. The consensus network was built using arcs that appear in 85% of bootstrapped networks.[45] Arc direction is determined by identifying the direction present between TLI subscores in at least 51% of the bootstrapped networks. Network nodes represent a single TLI item, and edges represent direct conditional dependencies.

Path analysis to predict SOFAS at time points 1 and 2

Structural equation modelling was used to examine the relationship between the latent FTD factors and functional outcome (SOFAS score) cross-sectionally (time point 1) and longitudinally (time point 2). Separate models were run with the EFA-derived factor structure and at the individual symptom level. Maximum likelihood estimation with Satorra–Bentler correction was used, and listwise deletion was applied for missing data. Listwise deletion was applied for missing data as the analytic sample was sufficiently large to maintain adequate statistical power, and the lack of two time points of data in most cohorts rendered multiple imputation liable to yield unreliable or biased estimates. Structural equation model analyses predicting SOFAS score were repeated with individual TLI items that constituted the EFA-derived factor structure. Standardised estimates are reported. Structural equation modelling was conducted within JASP version 0.95.4, using the Lavaan mimic.[46]

Time-invariance analysis

A multiple-group CFA was conducted in JASP version 0.95.4, using the Lavaan mimic to test for measurement invariance of the TLI factors across two time points. We tested a series of nested models with increasingly stricter model constraints: configural, weak, strong and strict. Configural invariance assumes an equivalent factor structure across time points (e.g. consistent three-factor solution). Weak or metric invariance introduces factor loading equality constraints, where TLI items contributing to latent variables are the same across time. Strong or scalar invariance introduces equality constraints on the item intercepts; establishing this level of invariance is necessary to allow for meaningful comparisons of the latent factor means across time. Finally, strict invariance requires that the residual variances of TLI scores are equal across time.[47] Model fit was compared with the change in CFI (ΔCFI $<$ 0.010) and RMSEA (ΔRMSEA $<$ 0.015) against successive models, where a deterioration in fit beyond these thresholds would indicate a lack of invariance, and thus lack of stability of dimensions retrieved via factor analysis over time.[48]

For an analysis of between-study variance and the effects of diagnostic heterogeneity of the samples, antipsychotic use or gender ratio on the factor structure on the TLI scores see Supplementary Appendices 3 and 4.

Results

As expected, patients exhibited significantly lower levels of global functioning as measured by the SOFAS at both time point 1 (patients: median, 50 and interquartile range, 20; controls: median, 85 and interquartile range, 6; $p < 0.001$) and time point 2 (Patient: median 56, interquartile range 24; Control: median 85, interquartile range 3; $p < 0.001$). The patient and control groups did not differ significantly in gender distribution at either time point (time point 1: $p = 0.11$; time point 2: $p = 0.3$). Participant demographics can be found in Table 1. The summary of diagnostic information is available in Supplementary Appendix 4; item distribution across data-sets can be found in Supplementary Appendix 5.

Demographics

Factor structure of FTD

The data were suitable for factor analysis (KMO = 0.67; Bartlett's test of sphericity, $\chi^2(21) = 1150.36$, $p < 0.001$). The distractibility item was discarded because of insufficient non-zero values (<1%). The EFA indicated a three-factor solution, which collectively accounted for 51.9% of the variance. Factor 1 (impoverishment) was defined by high loadings from TLI items weakening of goal (0.92) and poverty of speech (0.84). Factor 2 (loosening) was defined by looseness (0.89) and illogicality (0.38) TLI items. Factor 3 (peculiarities) was defined by TLI items peculiar words (0.82) and peculiar sentences (0.58). Perseveration did not load saliently on any factor. The three-factor model showed excellent fit (RMSEA = 0.040, 90% CI 0.000–0.085, CFI = 0.997, Tucker–Lewis Index = 0.980, standardised root mean square residual = 0.009).

In the CFA, the data-driven three-factor model (based on EFA) demonstrated better fit (Satorra–Bentler scaled chi-squared (SBχ^2)(6) = 6.62, $p = 0.358$; CFI = 0.997, Tucker–Lewis Index = 0.994, RMSEA = 0.012) than the theoretical two-factor model (SBχ^2(13) = 57.94, $p < 0.001$; CFI = 0.840, Tucker–Lewis Index = 0.741, RMSEA = 0.072) (ΔSB$\chi^2(7) = 44.27$, $p < 0.001$). The three-factor model was therefore retained for all subsequent analyses (Fig. 1).

https://doi.org/10.1192/bjp.2026.10650 Published online by Cambridge University Press

Table 1 Summary of participant demographics and SOFAS scores

	Time point 1, $n = 666$				Time point 2, $n = 244$			
Demographic characteristic	*n*	Control $n = 194$[a]	Patient $n = 472$[a]	*p*-value[b]	*n*	Control $n = 67$[a]	Patient $n = 177$[a]	*p*-value[b]
Age	666	26 (22, 31)	28 (22, 39)	0.008	244	26 (22, 30)	28 (23, 35)	0.034
Gender, male	666	136 (70%)	359 (76%)	0.11	244	46 (69%)	133 (75%)	0.3
SOFAS	448	85 (81, 87)	50 (40, 60)	<0.001	216	85 (83, 86)	56 (46, 70)	<0.001

SOFAS, Social and Occupational Functional Assessment Scale.
Diagnosis breakdown by cohort is provided in Supplementary Appendix 4.
a. Median (quartile 1, quartile 3); *n* (%).
b. Wilcoxon rank-sum test; Pearson's chi-squared test.

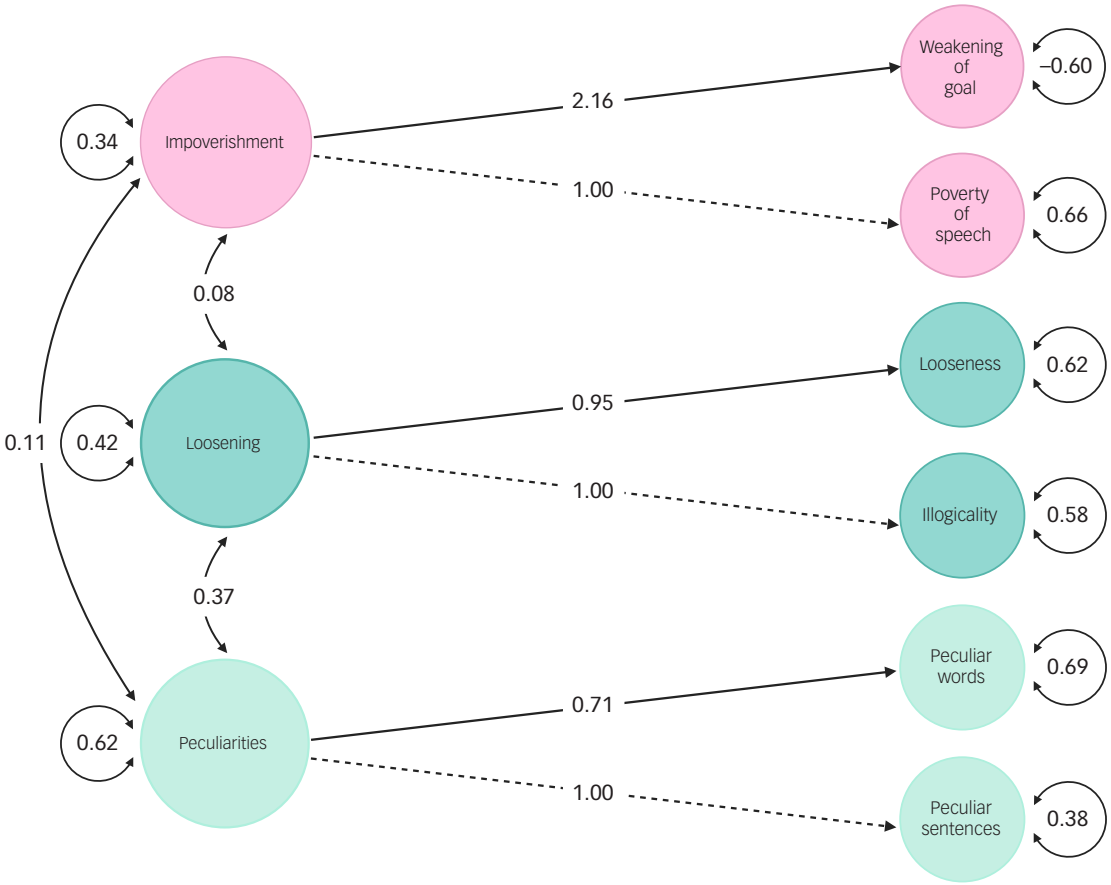

Fig. 1 Path diagram of confirmatory factor analysis of the exploratory factor analysis-derived three-factor model of the Thought and Language Index (TLI). Three dimensions of thought disorder emerge: impoverishment, loosening and peculiarities, each summarising a set of TLI items. Large circles represent latent variables. Small circles represent TLI items. Lines represent the causal effects from the latent factors to the individual items. The correlation of residual errors between variables is indicated by double-headed curved arrows. The circular double-headed arrows represent the variance of error. Unstandardised estimates are presented in this figure.

Network structure of FTD

The network analysis of the seven TLI symptoms at time point 1 revealed a sparse structure (sparsity: 0.24). The strongest edge was observed between weakening of goal and poverty of speech (Fig. 2). Centrality measures indicated that weakening of goal (strength: 1.69, expected influence: 1.75) and peculiar sentences (strength: 0.88, expected influence: 1.00) were the most central symptoms in the network. Bootstrap analyses indicated good stability of both the edge weights and the centrality indices. The inset in Fig. 2 depicts the DAG obtained from the averaged 1000 network structures of the TLI items. Two prominent groupings emerge. First, consistent with the Impoverishment dimension, we found poverty of speech to be strongly predictive of weakening of goal, with both forming a dyad. Second, we found the loosening and peculiarities factors to be forming a hierarchical structure. Looseness item is directly predictive of both illogicality and peculiar sentences; the peculiar sentences item, in turn, predicts peculiar words. This pattern suggests that looseness may be a parent symptom that could have both direct and indirect downstream influences on several TLI items. As in the factor analysis, perseveration remained as an isolated item with no relationships with other TLI items.

Predicting functional outcome

Cross-sectional analysis (time point 1)

A structural equation model with the three latent FTD factors predicting SOFAS score at time point 1 showed acceptable fit ($SB\chi^2(10) = 16.80$, $p = 0.079$; CFI = 0.965, RMSEA = 0.044). Both the impoverishment factor ($\beta = -0.196$, $p < 0.001$) and the peculiarities factor ($\beta = -0.30$, $p = 0.001$) were significant predictors of

https://doi.org/10.1192/bjp.2026.10650 Published online by Cambridge University Press

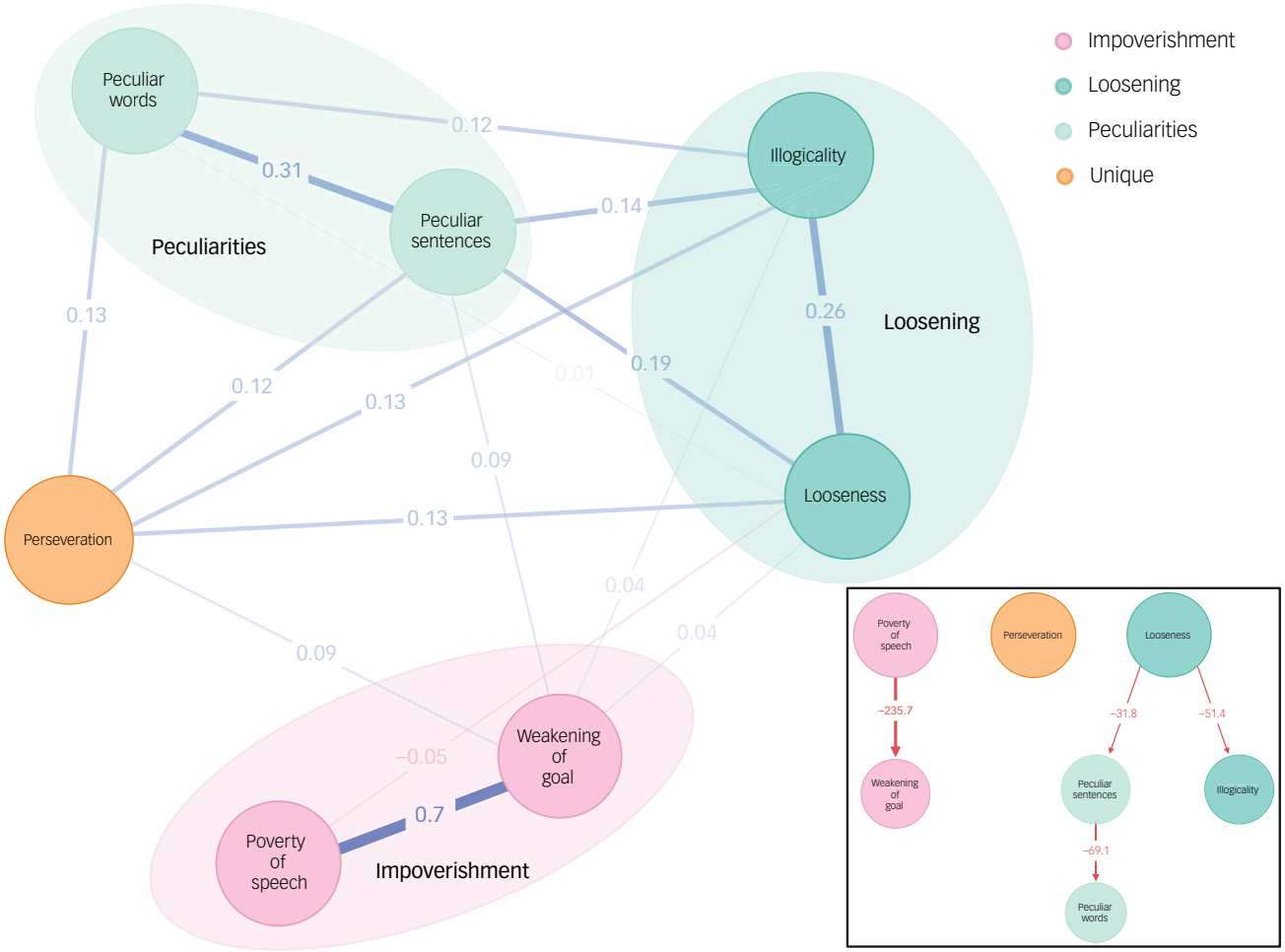

Fig. 2 Network structure and directed acyclic graph of Thought and Language Index items. Nodes are coloured based on their belonging to a specific latent factor. Blue edges (dashed in print version) indicate positive partial correlations or causal effects; red edges (dotted in print version) indicate negative partial correlations or causal effects. Thicker edges indicate a stronger relationship.

low SOFAS score. The loosening factor was not a significant predictor ($\beta = 0.05$, $p = 0.657$). At the symptom level, peculiar sentences ($\beta = -0.17$, $p = 0.001$) and poverty of speech ($\beta = -0.15$, $p = 0.015$) were significant individual predictors of low SOFAS score.

Longitudinal analysis (time point 2)

The structural equation model predicting SOFAS score at time point 2 from time point 1 FTD factors showed excellent fit ($SB\chi^2(9) = 6.78$, $p = 0.660$; CFI = 1.000, RMSEA = 0.000). The impoverishment ($\beta = -0.20$, $p = 0.037$) and peculiarities ($\beta = -0.34$, $p = 0.042$) factors at time point 1 significantly predicted lower SOFAS scores at time point 2, consistent with the pattern seen at time point 1. The loosening factor ($\beta = -0.13$, $p = 0.066$) was not a statistically significant predictor. At the symptom level, no significant predictors were observed. Functional outcome predictions are depicted in Fig. 3.

Item-wise exploratory analysis is available in Supplementary Appendix 6. Of note, SOFAS scores improved on average, whereas weakening of goal and peculiar words reduced in severity across the sample at time point 2; all other TLI items remained stable without notable reductions in patients between time points 1 and 2 (Supplementary Appendix 7).

Time invariance of factor structure

Measurement invariance tests across the two time points showed partial invariance for the TLI factors. Configural (CFI = 0.91, RMSEA = 0.10) and metric invariance (ΔCFI = 0, ΔRMSEA = 0) were supported, indicating an equivalent factor structure and invariant factor loadings. However, scalar (ΔCFI = −0.05, ΔRMSEA = 0.02) and strict invariance (ΔCFI = −0.07, ΔRMSEA = 0.02) models had significantly worse fit. This implies that the constructs have the same structure and meaning over time, allowing for the comparison of factor relationships during illness progression; but absolute changes in the burden of the impoverishment, loosening and peculiarities items over time cannot be estimated using these factors.

Analysis of between-study variance (Supplementary Appendix 3) indicated that two items – weakening of goal and illogicality – are likely to be the most sensitive to inferential clinical judgement, as the sensitivity analysis excluding IMPLEMENT (cohort with unblinded ratings) notably reduced between-study variance for these two items. Nonetheless, relative G coefficients remained ≥0.875 across all items with the full seven-cohort sample, indicating that rank ordering of patients by FTD severity was uncompromised. There were no notable effects from diagnostic heterogeneity, frequency of antipsychotic use or gender ratio on the factor structure on the TLI scores (Supplementary Appendices 3 and 4).

Discussion

By applying both latent factor and network-based methods to a large, geographically diverse and transdiagnostic sample, we report three key findings regarding FTD. First, we showed that short

https://doi.org/10.1192/bjp.2026.10650 Published online by Cambridge University Press

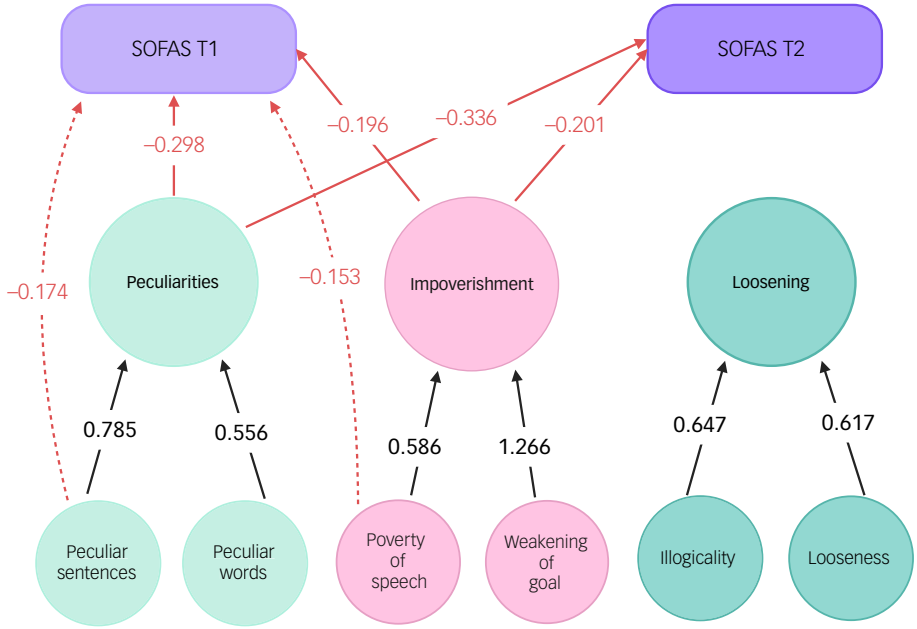

Fig. 3 Functional outcome (SOFAS score) predictions by TLI latent factors and items. Black lines represent loadings of TLI items onto derived latent factors. Standardised loadings are represented in the black text. Red lines (blue in the print version) indicate negative predictions of functional outcomes by latent variables (solid) and TLI items (dashed). Standardised βs are depicted in the red text (blue in the print version). Functional outcomes are depicted in rectangles. Large circles represent TLI latent factors, and small circles represent TLI items. SOFAS, Social and Occupational Functional Assessment Scale; T1, time point 1; T2, time point 2; TLI, Thought and Language Index.

evaluations of FTD generate a reliable factor structure by establishing a longitudinally stable, three-factor model consisting of *peculiarities*, *impoverishment* and *loosening*. Second, we revealed systems-level interactions among FTD items that point to two types of 'active ingredients': we find (a) poverty of speech and looseness to be the putative primary symptoms that are on top of the hierarchy, likely influencing other symptoms downstream and (b) weakening of goal and peculiar sentences to be the most connected central symptoms that sustain overall relationships constituting the FTD syndrome. Third, and most critically for patients, we demonstrated that not all FTD symptoms have equal functional impact; *impoverishment* and *peculiarities* predict real-world functional outcomes up to a year later, whereas *loosening* shows no such impact on functioning. Perseveration was statistically unrelated to the other constituent items, raising the question of whether it is indeed a true FTD phenomenon.

Our factor analytical solution from the short-form TLI identified *impoverishment* (poverty of speech and weakening of goal), *loosening* (looseness and illogicality) and *peculiarities* (peculiar words and sentences) as the key factors, reconciling historical dichotomies. This dimensional three-factor solution is consistent with several studies reported in the systematic review by Zamperoni and colleagues.[8] The separation of *impoverishment* (a 'negative' dimension) from *loosening* and *peculiarities* (both 'positive' dimensions) validates the specific status ascribed to 'negative' FTD by Fish and Hamilton[49] and Andreasen.[50] The further fractionation of the positive FTD into a *loosening* factor and a lexico-syntactic *peculiarities* factor echoes aspects of early proposals of Guislain and Séglas[51,52] that separated surface-level speech disturbances from thought incoherence.

This longitudinal analysis provides some of the strongest evidence to date that FTD has a prospective predictive relationship with functional impairment.[53–56] Prior studies have assessed FTD as a unitary construct, and reported a small-to-medium sized association ($n = 13$ studies).[13] The fact that baseline impoverishment and peculiarities predicted functioning 3–12 months later, suggests that these latent factors are not merely statistical epiphenomena but are drivers of psychosocial disability. We dissected this relationship to uncover the specific influence of impoverishment and peculiarities (which were downstream of looseness in the DAG) on SOFAS scores at two time points, and we considered it in light of the lack of association between SOFAS score and loosening. Indeed, the latter cannot be attributed to an improvement of FTD with time; in our patient sample, both illogicality and looseness that constituted loosening did not improve significantly with time (Supplementary Appendix 7). When we speak less (insufficient) or in a peculiar manner (odd), it may more directly disrupt our everyday language use required for work and relational functions than when we display faulty logic and/or tenuously linked ideas. This aligns with prior studies linking computationally measured reduced fluency and the use of lower frequency words to worse quality of life in schizophrenia,[22] as well as with studies associating poor observer-rated speech/communication and functioning among the unemployed individuals with severe mental illnesses.[57–60] More broadly, this is consistent with pragmatic accounts arguing that deficits in informativity are strongly penalised in communication.[61,62]

The standardised effect sizes observed here ($\beta = 0.20$–0.34) are consistent with a small-to-medium range. This is expected in a complex outcome like SOFAS score, where FTD represents only one of many contributing factors among negative symptoms, cognition, medication and psychosocial context. Clinically, even modest effect sizes of prognostic assessments have utility if they add incremental value to other available information; for TLI, this needs further study. The absence of significant item-level predictors at time point 2 likely reflects reduced statistical power at follow-up.

To our knowledge, the inter-symptom dependency networks in FTD have not been reported before. We observed higher centrality for peculiar sentences and weakening of goal ('connecting' features). Connecting features are active ingredients that maintain the FTD network and likely play a mechanistic role in the persistence of FTD as a syndrome. For instance, a loss of narrative

https://doi.org/10.1192/bjp.2026.10650 Published online by Cambridge University Press

direction (goal) may permit all other peculiarities to be manifested, giving rise to persistent FTD syndrome. Given their high connectivity, we can hypothesise that the connecting features act as potential leverage points, which, when 'treated', can weaken the overall network.

The DAG's hierarchical causal structure, revealing the primacy of poverty of speech and looseness ('cardinal' features), is theoretically compelling. This aligns with computational theories proposing two distinct core deficits:[63–65] a failure of speech initiation (leading to poverty) and a failure of associative constraint (leading to looseness). These 'root-level' deficits could then cascade through the network, giving rise to the secondary symptoms that we observe. These primary symptoms are thus better suited targets for prediction/prevention strategies (e.g.,[66,67]) and to study upstream causal pathways (e.g., via computational modelling[68,69]). Our finding that perseveration was a stand-alone feature requires cautious interpretation. Historically, perseveration has been described as an element of negative FTD,[49,70] and in more severe or chronic samples with sufficiently long examination, it may exhibit greater shared variance with impoverishment. Our findings therefore reflect the structure of FTD within the severity range captured by brief assessment (around 3 min), rather than a definitive claim about perseveration's nosological status.

Our finding of configural and metric, but not scalar, invariance of FTD dimensions with time presents both opportunities and challenges. It confirms that the dimensions of FTD are conceptually stable (configural invariance) with a similar relationship with its constituents (metric invariance) over time. Along with their links to external, clinically meaningful anchors (i.e. social and occupational functioning), TLI-derived latent dimensions allow us to meaningfully use a latent change score model in clinical trials (e.g. if an intervention predicts post-treatment changes). The failure of scalar invariance has a direct implication: latent mean comparisons from short-form TLI across the two time points are not statistically valid (e.g. we cannot meaningfully say that a treatment reduces loosening by 50% from baseline). This limits the direct use of TLI-derived factor scores as end-points in clinical trials, where sensitivity to absolute change is essential. We therefore emphasise that interventional studies should favour other construct-relevant continuous measures (e.g. natural language processing-based scores[71]) with demonstrated scalar invariance, or focus on the cardinal/connecting symptoms identified here.

We acknowledge several limitations of this work. First, although our sample is large, geographically diverse and transdiagnostic, it remains a research cohort with volunteer bias (most patients volunteered for a neuroimaging study); this may affect generalisability to the clinic. We did not assess lifetime exposure to antipsychotics and burden of other symptoms in the present study; these factors may have influenced SOFAS scores in our sample. SOFAS is a single-item, global rating of functioning, it does not allow for examination of independent associations with social, interpersonal or occupational functioning. Future studies should incorporate multi-item, domain-specific measures to enable a more nuanced understanding of how FTD symptoms relate to distinct areas of functioning. Second, our findings are tied to the short version of the TLI instrument, which, despite minimising contamination of other symptoms, may not capture the full spectrum of FTD assessed by other scales such as the Thought and Language Disorder scale (TALD)[72] or the Thought, Language and Communication[50] scale. TLI ratings were based on brief responses to pictures, without demands for turn-taking or self-disclosure processes that may elicit additional FTD phenomena. The brief 1 min limit per picture may also suppress certain symptoms (e.g. perseveration, distractibility), reducing their variance. We also note that some of the items may be more sensitive to lack of diagnostic blinding (weakening of goal and illogicality); although this did not affect our factor solutions, caution is warranted when comparing raw scores across studies with variable diagnostic blinding.

The network models are based on between-individual variance, not within-individual dynamics; thus, they represent plausible rather than proven structures within the studied system. The proposed causal pathways require future replication and longitudinal and experimental validation. The DAGs used here are based on conditional dependence patterns from observational, cross-sectional data and assume causal sufficiency (i.e. no unmeasured common causes). Potential unmeasured confounders of speech production (e.g. language barriers, limited vocabulary) could, in principle, alter the inferred direction of relationships. These causal structures are therefore best regarded as hypothesis-generating and require longitudinal designs for confirmation. Finally, our hypothesis was restricted to the functional relevance of independently assessed FTD; we did not test if other overlapping determinants (negative symptoms, cognitive deficits or intervention) influence the predictive relationship in this sample. Moreover, we did not have access to systematic comorbidity data across cohorts, limiting our ability to determine the contribution of co-occurring depression or anxiety to SOFAS scores. Our results do not imply FTD as being the sole contributor for social dysfunction.

To conclude, by integrating latent variable and network approaches, we provide a unified, empirically grounded framework for measuring FTD with a brief instrument. Integrating this to clinical workflows would require prospective validation in routine clinical settings, demonstrating that the added assessment burden (around 3 min) produces clinically actionable information beyond what is captured by unitary dimensional approaches (e.g. the DSM-5). We call for an evaluation of the utility of prognostic assessments based on short speech-based assessment of impoverishment, loosening and peculiarities in routine clinical practice, within the limitations posed by psychometric properties reported here. We anticipate this line of work to provide psychopathological targets for formal explanatory computational modelling of FTD, as well as potential targets for interventional research. Our findings also underscore the need for the development of next-generation FTD assessment tools, such as those leveraging automated approaches to achieve superior measurement invariance and serve as robust end-points in clinical trials.

Fatme Abboud, Department of Psychiatry, Douglas Mental Health University Institute, McGill University, Montreal, Canada; **Jessica Ahrens**, Department of Psychiatry, Douglas Mental Health University Institute, McGill University, Montreal, Canada; and Integrated Program in Neuroscience, McGill University, Canada; **Estèe Ballès**, Medical Sciences, Western University, Canada; **Valentina Bambini**, Laboratory of Neurolinguistics and Experimental Pragmatics (NEPLab), IUSS, Italy; **Bill Deakin**, Division of Psychology and Mental Health, The University of Manchester, UK; **Neil A. Harrison**, Division of Psychiatry & Clinical Neuroscience, Cardiff University, UK; and Cardiff University Brain Research Imaging Centre, Cardiff, UK; **Tilo Kircher**, Department of Psychiatry, University of Marburg, Germany; **Gina Kuperberg**, Department of Psychiatry, Massachusetts General Hospital, Harvard Medical School, Boston, USA; and Department of Psychology and Center for Cognitive Science, Tufts University, USA; **Peter F. Liddle**, Centre for Translational Neuroimaging for Mental Health, Division of Psychiatry and Applied Psychology, University of Nottingham, UK; **Rohit Lodhi**, Department of Psychiatry, Schulich School of Medicine & Dentistry, Western University, Canada; **Michael Mackinley**, London Health Sciences Research Institute, London, Ontario, Canada; **Susan L. Rossell**, Centre for Mental Health and Brain Sciences, Swinburne University of Technology, Australia; and InsideOut Institute, Australia; **Krish D. Singh**, School of Psychology, Cardiff University Brain Research Imaging Centre, UK; **Iris E. Sommer**, Department of Neuroscience, University Medical Centre Groningen, The Netherlands; **Alban Voppel**, Department of Psychiatry, Douglas Mental Health University Institute, McGill University, Montreal, Canada; and Department of Psychiatry, McGill University, Canada; **Farida Zaher**, Department of Psychiatry, Douglas Mental Health University Institute, McGill University, Montreal, Canada; **Nadia Zeramdini**, Department of Psychiatry, Douglas Mental Health University Institute, McGill University, Montreal, Canada; **Lena Palaniyappan**, Department of Psychology, McGill University, Montreal, Canada; and London Health Sciences Research Institute, London, UK

Correspondence: Lena Palaniyappan. Email: lena.palaniyappan@mcgill.ca

First received 17 Dec 2025, final revision 6 Mar 2026, accepted 28 Mar 2026

https://doi.org/10.1192/bjp.2026.10650 Published online by Cambridge University Press

Supplementary material

The supplementary material is available online at https://doi.org/10.1192/bjp.2026.10650

Data availability

The data that support the findings of this study are available on request from the corresponding author, L.P. For DISCOURSE-UWO and TOPSY, anonymised data are made available to qualified researchers through https://talkbank.org/psychosis/, a collaboration between the DISCOURSE in Psychosis consortium (https://discourseinpsychosis.org/) and TalkBank. Restrictions apply, and conditions are accessible via the TalkBank URL above.

Acknowledgements

We would like to thank the patients for taking the time to participate in this study, as well as the numerous research assistants and clinicians who were involved in this project. The authors wish to thank Paulina Dzialoszynski, Sabrina D. Ford and Betsy Schaefer (London Health Sciences Center) for clinical recruitment and coordination; Julie Richard and Hooman Ganjavi for logistical support; Ridha Joober, David Bloom, Karim Tabbane and Martin Lepage (Douglas University Mental Health Institute) for assistance with recruitment; Emanuel Schwarz (Heidelberg), Alkomiet Hasan and Nikolaos Koutsouleris (Munich) for resources; Zia Katshu and Mohan Rathnaiah (University of Nottingham), Catherine C. Gregory (University of Manchester) and Gemma Williams (Cardiff University) for recruitment to SPRING; all staff members of Prevention and Early Intervention Program for Psychoses-London for recruitment and clinical support; and all participants for their time and effort in participating in the study. We thank the DISCOURSE consortium (https://discourseinpsychosis.org/) steering group for their assistance in developing the speech assessment protocol. We acknowledge Marie-Ange Barthel, Douglas Research Centre, for her assistance in curating the data used in this work.

Author contributions

F.A. contributed to data curation, formal analysis, visualisation, writing the original draft and reviewing and editing the manuscript. J.A., E.B., P.F.L., M.M., A.V., F.Z. and N.Z. contributed to data acquisition, data curation and reviewing and editing the manuscript. V.B., N.A.H., T.K., G.K., S.L.R. and I.E.S. reviewed and edited the manuscript. B.D., R.L. and K.D.S. contributed to study resources and reviewing and editing the manuscript. L.P. contributed to study conceptualisation, resources, data acquisition, supervision, funding acquisition, writing the original draft and reviewing and editing the manuscript.

Funding

DISCOURSE-UWO: Data collected for this study were supported by the Academic Medical Organization of Southwest Ontario (AMOSO Innovation). TOPSY: Data acquisition was supported by the Canadian Institutes of Health Research Foundation Grant (375104/2017), Innovation Fund for Academic Medical Organization of Southwest Ontario, Bucke Family Fund, The Chrysalis Foundation and The Arcangelo Rea Family Foundation (London, Ontario). IMPLEMENT: This work was supported by the Canadian Institutes of Health Research ERA-Net Personalized Medicine Programme (grant number ENP-161423). SPRING: This work was supported by a Medical Research Council (grant number MR/K020803/1; Experimental Medicine Challenge project grant: Defining the disturbance in cortical glutamate and GABA function in psychosis, its origins and consequences). We would also like to acknowledge several other Medical Research Council grants (numbers MR/M006301/1 and MR/K005464/1). WOW: Data acquired for this study were funded by grant 371730 from the Canadian Institutes of Health Research. CONN: This work was funded by Medical Research Council, UK (grant number G0601442). UK7T: This work was supported by the Wellcome Trust (grant number WT096002/Z/11) and an internal grant from the School of Community Health Sciences, University of Nottingham, UK. A.V. is supported by a National Alliance for Research on Schizophrenia and Depression Young Investigator Grant (number 32574) from the Brain & Behaviour Research Foundation. J.A. is supported by the Canadian Institutes of Health Research, Schizophrenia Society of Canada Foundation, the Canadian Consortium for Early Intervention in Psychosis, the Fonds de Recherche du Quebec – Santé and Quebec Bio-Imaging Network. L.P. is supported by the Monique H Bourgeois Chair in Developmental Disorders, and part of the data reported here was collected with supports from the Academic Medical Organization of Southwestern Ontario Opportunities Fund, Tanna Schulich Endowment, Bucke Funds, Chrysalis Foundation (London, Ontario) and the Wellcome Trust (Research Training Fellowship number WT096002/Z/11/Z). L.P. also received a salary award from the Fonds de Recherche du Quebec-Santé (FRQS; number 366934) and supported by the FRQS through a Research Centre Grant to Douglas Research Centre (https://doi.org/10.69777/5230). T.K. receives funding from the German Research Foundation (project grant number 2107, SFB/TRR 393) ('Trajectories of Affective Disorders', project grant number 521379614), and the Germany's Excellence Strategy (EXC 3066/1 'The Adaptive Mind', project number 533717223), as well as the DYNAMIC Center, funded by the LOEWE programme of the Hessian Ministry of Science and Arts (grant number LOEWE1/16/519/03/09.001(0009)/98).

Declaration of interest

L.P. reports personal fees for serving as Chief Editor for the Canadian Medical Association Journals; speaker honorarium from Janssen Canada and Otsuka Canada, SPMM Course Limited, UK; book royalties from Oxford University Press; and investigator-initiated educational grants from Otsuka Canada outside the submitted work, in the past 5 years. K.D.S. is a member of the scientific advisory board for Draig Therapeutics. The other authors have no conflict of interest to disclose.

References

1 Kircher T, Bröhl H, Meier F, Engelen J. Formal thought disorders: from phenomenology to neurobiology. *Lancet Psychiatry* 2018; **5**: 515–26.

2 Roche E, Creed L, MacMahon D, Brennan D, Clarke M. The epidemiology and associated phenomenology of formal thought disorder: a systematic review. *Schizophr Bull* 2015; **41**: 951–62.

3 Jerónimo J, Queirós T, Cheniaux E, Telles-Correia D. Formal thought disorders-historical roots. *Front Psychiatry* 2018; **9**: 572.

4 Zamperoni G, Tan EJ, Sumner PJ, Rossell SL. Exploring the conceptualisation, measurement, clinical utility and treatment of formal thought disorder in psychosis: a Delphi study. *Schizophr Res* 2024; **270**: 486–93.

5 Liddle PF, Ngan ETC, Caissie SL, Anderson CM, Bates AT, Quested DJ, et al. Thought and Language Index: an instrument for assessing thought and language in schizophrenia. *Br J Psychiatry* 2002; **181**: 326–30.

6 McKenna PJ, Oh TM. *Schizophrenic Speech: Making Sense of Bathroots and Ponds that Fall in Doorways*. Cambridge University Press, 2005.

7 de Bruin EI, Verheij F, Wiegman T, Ferdinand RF. Assessment of formal thought disorder: the relation between the Kiddie Formal Thought Disorder Rating Scale and clinical judgment. *Psychiatry Res* 2007; **149**: 239–46.

8 Zamperoni G, Tan EJ, Rossell SL, Meyer D, Sumner PJ. Evidence for the factor structure of formal thought disorder: a systematic review. *Schizophr Res* 2024; **264**: 424–34.

9 Borsboom D, Cramer AOJ, Schmittmann VD, Epskamp S, Waldorp LJ, Tractenberg RE. The small world of psychopathology. *PLOS One* 2011; **6**: e27407.

10 Kuipers J, Moffa G, Kuipers E, Freeman D, Bebbington P. Links between psychotic and neurotic symptoms in the general population: an analysis of longitudinal British National Survey data using Directed Acyclic Graphs. *Psychol Med* 2019; **49**: 388–95.

11 Jablensky A. Psychiatric classifications: validity and utility. *World Psychiatry* 2016; **15**: 26–31.

12 Oeztuerk OF, Pigoni A, Antonucci LA, Koutsouleris N. Association between formal thought disorders, neurocognition and functioning in the early stages of psychosis: a systematic review of the last half-century studies. *Eur Arch Psychiatry Clin Neurosci* 2022; **272**: 381–93.

13 Marggraf MP, Lysaker PH, Salyers MP, Minor KS. The link between formal thought disorder and social functioning in schizophrenia: a meta-analysis. *Eur Psychiatry* 2020; **63**: e34.

14 Norman RMG, Malla AK, Cortese L, Cheng S, Diaz K, McIntosh E, et al. Symptoms and cognition as predictors of community functioning: a prospective analysis. *Am J Psychiatry* 1999; **156**: 400–5.

15 Bowie CR, Harvey PD. Communication abnormalities predict functional outcomes in chronic schizophrenia: differential associations with social and adaptive functions. *Schizophr Res* 2008; **103**: 240–7.

16 Wilcox J, Winokur G, Tsuang M. Predictive value of thought disorder in new-onset psychosis. *Compr Psychiatry* 2012; **53**: 674–8.

17 Ucok A, Karakaş B, Şahin OS. Formal thought disorder in patients with first-episode schizophrenia: results of a one-year follow-up study. *Psychiatry Res* 2021; **301**: 113972.

18 Mackinley M, Limongi R, Silva AM, Richard J, Subramanian P, Ganjavi H, et al. More than words: speech production in first-episode psychosis predicts later social and vocational functioning. *Front Psychiatry* 2023; **14**: 1144281.

19 Roche E, Segurado R, Renwick L, McClenaghan A, Sexton S, Frawley T, et al. Language disturbance and functioning in first episode psychosis. *Psychiatry Res* 2016; **235**: 29–37.

20 Mutlu E, Abaoğlu H, Barışkın E, Gürel ŞC, Ertuğrul A, Yazıcı MK, et al. The cognitive aspect of formal thought disorder and its relationship with global social functioning and the quality of life in schizophrenia. *Soc Psychiatry Psychiatr Epidemiol* 2021; **56**: 1399–410.

21 Comparelli A, Corigliano V, Forcina F, Bargagna P, Montalbani B, Falcone G, et al. The complex relationship among formal thought disorders, neurocognition, and functioning in nonacutely ill schizophrenia patients. *J Nerv Ment Dis* 2020; **208**: 48–55.

22 Bambini V, Agostoni G, Buonocore M, Tonini E, Bechi M, Ferri I, et al. It is time to address language disorders in schizophrenia: a RCT on the efficacy of a novel training targeting the pragmatics of communication (PragmaCom). *J Commun Disord* 2022; **97**: 106196.

23 Jimeno N. Language and communication rehabilitation in patients with schizophrenia: a narrative review. *Heliyon* 2024; **10**: e24897.

24 de Bruin EI, de Nijs PFA, Verhulst FC, Huizink AC. The association between formal thought disorder and finger print asymmetry in children with a psychiatric disorder: an exploratory study. *Eur Child Adolesc Psychiatry* 2012; **21**: 691–8.

25 Ahrens J, Ford SD, Schaefer B, Reese D, Khan AR, Tibbo P, et al. Convergence of cannabis and psychosis on the dopamine system. *JAMA Psychiatry* 2025; **82**: 609–17.

26 Palaniyappan L, Al-Radaideh A, Gowland PA, Liddle PF. Cortical thickness and formal thought disorder in schizophrenia: an ultra high-field network-based

https://doi.org/10.1192/bjp.2026.10650 Published online by Cambridge University Press

morphometry study. *Prog Neuropsychopharmacol Biol Psychiatry* 2020; **101**: 109911.

27 Palaniyappan L, Simmonite M, White TP, Liddle EB, Liddle PF. Neural primacy of the salience processing system in schizophrenia. *Neuron* 2013; **79**: 814–28.

28 Melshin G, DiMaggio A, Zeramdini N, MacKinley M, Palaniyappan L, Voppel A. Taking a look at your speech: identifying diagnostic status and negative symptoms of psychosis using convolutional neural networks. *NPP Digit-Psychiatry Neurosci* 2025; **3**: 19.

29 Gascoyne LE, Brookes MJ, Rathnaiah M, Katshu MZUH, Koelewijn L, Williams G, et al. Motor-related oscillatory activity in schizophrenia according to phase of illness and clinical symptom severity. *NeuroImage Clin* 2021; **29**: 102524.

30 MacKinley M, Ford SD, Jeon P, Théberge J, Palaniyappan L. Central oxidative stress and early vocational outcomes in first episode psychosis: a 7-tesla magnetic resonance spectroscopy study of glutathione. *Schizophr Bull* 2022; **48**: 921–30.

31 Dalal TC, Liang L, Silva AM, Mackinley M, Voppel A, Palaniyappan L. Speech based natural language profile before, during and after the onset of psychosis: a cluster analysis. *Acta Psychiatr Scand* 2025; **151**: 332–47.

32 Sjoberg D,D, Whiting K, Curry M, Lavery J,A, Larmarange J. Reproducible summary tables with the gtsummary package. *R J* 2021; **13**: 570–80.

33 Sommer IE, Derwort AMC, Daalman K, de Weijer AD, Liddle PF, Boks MPM. Formal thought disorder in non-clinical individuals with auditory verbal hallucinations. *Schizophr Res* 2010; **118**: 140–5.

34 Morosini P, Magliano L, Brambilla L, Ugolini S, Pioli R. Development, reliability and acceptability of a new version of the DSM-IV Social and Occupational Functioning Assessment Scale (SOFAS) to assess routine social funtioning. *Acta Psychiatr Scand* 2000; **101**: 323–9.

35 Samara MT, Engel RR, Millier A, Kandenwein J, Toumi M, Leucht S. Equipercentile linking of scales measuring functioning and symptoms: examining the GAF, SOFAS, CGI-S, and PANSS. *Eur Neuropsychopharmacol* 2014; **24**: 1767–72.

36 Tobias S, Carlson JE. Brief report: Bartlett's test of sphericity and chance findings in factor analysis. *Multivar Behav Res* 1969; **4**: 375–7.

37 Kaiser MO. Kaiser–Meyer–Olkin measure for identity correlation matrix. *J R Stat Soc* 1974; **52**: 296–8.

38 Satorra A. Scaled and adjusted restricted tests in multi-sample analysis of moment structures. In *Innovations in Multivariate Statistical Analysis: A Festschrift for Heinz Neudecker* (eds RDH Heijmans, DSG Pollock, A Satorra): 233–47. Springer US, 2000.

39 Friedman J, Hastie T, Tibshirani R. Sparse inverse covariance estimation with the graphical lasso. *Biostatistics* 2008; **9**: 432–41.

40 Foygel R, Drton M. Extended Bayesian information criteria for Gaussian graphical models. *Adv Neural Inf Process Syst* 2010; **23**: 2020–8.

41 Epskamp S, Fried EI. A tutorial on regularized partial correlation networks. *Psychol Methods* 2018; **23**: 617–34.

42 JASP Team. *JASP*. JASP Team, 2025 (https://jasp-stats.org/).

43 Scutari M. Learning Bayesian networks with the bnlearn R Package. *J Stat Softw* 2010; **35**: 1–22.

44 Russell S, Norvig P. *Artificial Intelligence: A Modern Approach*. Pearson, 2016.

45 Sachs K, Perez O, Peer D, Lauffenburger DA, Nolan GP. Causal protein-signaling networks derived from multiparameter single-cell data. *Science* 2005; **308**: 523–9.

46 Rosseel Y. lavaan: an R package for structural equation modeling. *J Stat Softw* 2012; **48**: 1–36.

47 Putnick DL, Bornstein MH. Measurement invariance conventions and reporting: the state of the art and future directions for psychological research. *Dev Rev* 2016; **41**: 71–90.

48 Brown TA. *Confirmatory Factor Analysis for Applied Research* 2nd ed. The Guilford Press, 2015.

49 Fish FJ, Hamilton MJ. *Fish's Schizophrenia* 2nd ed. J. Wright, 1976.

50 Andreasen NC. Scale for the assessment of thought, language, and communication (TLC). *Schizophr Bull* 1986: **12**: 473–82.

51 Séglas J. *Des Troubles du Langage Chez les Aliénés* [*On Language Disorders in the Mentally Ill*]. J. Rueff, 1892.

52 Guislain J. *Leçons orales sur les phrénopathies, ou, Traité théorique et pratique des maladies mentales: cours donné à la Clinique des établissements d'aliénés à Gand* [*Oral Lectures on Phrenopathies, or, A Theoretical and Practical Treatise on Mental Diseases: A Course Given at the Clinical Establishment for the Mentally Ill in Ghent*]. E. Vanderhaeghen, 1880.

53 Mucci A, Galderisi S, Gibertoni D, Rossi A, Rocca P, Bertolino A, et al. Factors associated with real-life functioning in persons with schizophrenia in a 4-year follow-up study of the Italian network for research on psychoses. *JAMA Psychiatry* 2021; **78**: 550–9.

54 Abplanalp SJ, Braff DL, Light GA, Nuechterlein KH, Green MF, et al. Consortium on the genetics of schizophrenia-2. Understanding connections and boundaries between positive symptoms, negative symptoms, and role functioning among individuals with schizophrenia: a network psychometric approach. *JAMA Psychiatry* 2022; **79**: 1014–22.

55 Sigaudo M, Crivelli B, Castagna F, Giugiario M, Mingrone C, Montemagni C, et al. Quality of life in stable schizophrenia: the relative contributions of disorganization and cognitive dysfunction. *Schizophr Res* 2014; **153**: 196–203.

56 Yalınçetin B, Ulaş H, Var L, Binbay T, Akdede BB, Alptekin K. Relation of formal thought disorder to symptomatic remission and social functioning in schizophrenia. *Compr Psychiatry* 2016; **70**: 98–104.

57 Agostoni G, Bambini V, Bechi M, Buonocore M, Spangaro M, Repaci F, et al. Communicative-pragmatic abilities mediate the relationship between cognition and daily functioning in schizophrenia. *Neuropsychology* 2021; **35**: 42–56.

58 Adamczyk P, Daren A, Sułecka A, B⊠dziński P, Cichocki Ł, Kalisz A, et al. Do better communication skills promote sheltered employment in schizophrenia? *Schizophr Res* 2016; **176**: 331–9.

59 Dickinson D, Bellack AS, Gold JM. Social/communication skills, cognition, and vocational functioning in schizophrenia. *Schizophr Bull* 2007; **33**: 1213–20.

60 Lexén A, Bejerholm U. Exploring communication and interaction skills at work among participants in individual placement and support. *Scand J Occup Ther* 2016; **23**: 314–9.

61 Panzeri F, Foppolo F. Children's and adults' sensitivity to gricean maxims and to the maximize presupposition principle. *Front Psychol* 2021; **12**: 624628.

62 Davies C, Katsos N. Are interlocutors as sensitive to over-informativeness as they are to under-informativeness. In *Proceedings of the Workshop on Production of Referring Expressions: Bridging Computational and Psycholinguistic Approaches (PRE-CogSci-09) (Amsterdam, Netherlands, 29 Jul 2009).* Tilburg University: 282–7, 2009.

63 Bora E, Yalincetin B, Akdede BB, Alptekin K. Neurocognitive and linguistic correlates of positive and negative formal thought disorder: a meta-analysis. *Schizophr Res* 2019; **209**: 2–11.

64 Sharpe V, Mackinley M, Nour Eddine S, Wang L, Palaniyappan L, Kuperberg GR. Selective insensitivity to global versus local linguistic context in speech produced by patients with untreated psychosis and positive thought disorder. *Biol Psychiatry* 2026; **99**: 154–64.

65 Spitzer M. A cognitive neuroscience view of schizophrenic thought disorder. *Schizophr Bull* 1997; **23**: 29–50.

66 Zaher F, J Ahrens, Voppel A, Palaniyappan L. A review of speech markers in bipolar disorder mental states. *OSF* [Preprint] 2025. Available from: https://osf.io/qxzfs/overview.

67 Corcoran CM, Cecchi GA. Using language processing and speech analysis for the identification of psychosis and other disorders. *Biol Psychiatry Cogn Neurosci Neuroimaging* 2020; **5**: 770–9.

68 Fradkin I, Adams RA, Siegelman N, Moran R, Dolan RJ. Latent mechanisms of language disorganization relate to specific dimensions of psychopathology. *Nat Ment Health* 2024; **2**: 1486–97.

69 Gutiérrez E, Quesada C, DeFraites E, Harper DJ, Mandavia AD. Interpretable LLM-based detection of loose associations using synthetic speech data in early psychosis. *Schizophr Bull* [Epub ahead of print] 5 Sep 2025. Available from: https://doi.org/10.1093/schbul/sbaf125.

70 Crider A. Perseveration in schizophrenia. *Schizophr Bull* 1997; **23**: 63–74.

71 Corona Hernández H, Corcoran C, Achim AM, de Boer JN, Boerma T, Brederoo SG, et al. Natural language processing markers for psychosis and other psychiatric disorders: emerging themes and research agenda from a cross-linguistic workshop. *Schizophr Bull* 2023; **49**: S86–92.

72 Kircher T, Krug A, Stratmann M, Ghazi S, Schales C, Frauenheim M, et al. A rating scale for the assessment of objective and subjective formal Thought and Language Disorder (TALD). *Schizophr Res* 2014; **160**: 216–21.

https://doi.org/10.1192/bjp.2026.10650 Published online by Cambridge University Press