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ORIGINAL PAPER

Dynamic reconfigurations of brain subnetworks in female patients with alcohol use disorder or bulimia nervosa: a resting-state functional magnetic resonance imaging study

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Abstract

Introduction The brain forms a complex functional brain network. This network is dynamically reconfigured to support various cognitive processes. Research on brain network dynamics in patients with alcohol use disorder (AUD) or bulimia nervosa (BN), two highly comorbid psychiatric disorders, remains limited. Previous studies showed altered static network patterns, highlighting that the network is disturbed, but implicitly ignoring network dynamics. This study investigates dynamic network reconfigurations in female patients with AUD or BN and healthy controls (HC).

Methods Resting-state functional MRI data were acquired of 102 female participants (AUD:27, BN:24, HC:51). A sliding-window approach assigned brain regions iteratively to one of seven literature-based subnetworks for each window. Then, previously validated parameters of network reconfiguration were assessed: promiscuity (number of subnetworks switched to) and flexibility (number of switches). These measures were compared between groups and related to behavioral and clinical measures.

Results Compared to HC, patients with BN displayed a higher promiscuity of all brain subnetworks combined, and regionally for the dorsal attention network, with no change in flexibility. Patients with AUD showed no difference in either promiscuity or flexibility. Global and dorsal attention network promiscuity were negatively correlated with subjective stress.

Conclusion Regions typically assigned to the dorsal attention network changed their association with a higher number of other subnetworks in BN compared to HC, which was not seen in AUD. This suggests a less focused dynamic integration of information in patients with BN, which could play a role in their vulnerability to stress, attentional biases and impulsivity.

Keywords Bulimia nervosa · Alcohol use disorder · fMRI · Dynamic functional connectivity · Networks

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Introduction

Alcohol Use Disorder (AUD) and Bulimia Nervosa (BN) are two psychiatric disorders featuring several similarities [1]. For instance, they can each be characterized by binge behavior where patients lose control over their eating or drinking behavior and consume large amounts of food (i.e., binge eating) or alcohol (i.e., binge drinking) within a short period of time [1]. Additionally, AUD and BN are thought to have overlapping etiologies, with factors such as stress, craving, and negative urgency playing a key role in both disorders [2]. Studies specifically looking into the neurobiological underpinnings of AUD and BN show similarities between both disorders when it comes to a reduction in dopamine transmission, a lower integrity of white matter tracts, and a disrupted coordination between brain regions such as the prefrontal cortex and striatum [3–6]. Nevertheless, studies also report differences, as patients with AUD show a more widespread reduction in grey matter volume, while patients with BN are thought to also display a higher grey mater volume in regions such as the prefrontal cortex and insula [6–9]. However, findings are often inconsistent, causing the exact nature of the underlying neurobiological disturbances in both disorders to remain unclear. A better understanding of these disturbances as well as their similarities and differences between both disorders is necessary, as this could facilitate the development of new treatments. Namely, understanding the unique disturbances in each disorder could help to provide disorder-specific interventions for patients. The necessity is underscored further by the finding that up to 60% of patients with AUD and BN who receive treatment do not achieve remission [10, 11].

Functional connectivity, representing an interdependence in the activity of distinct brain areas as measured with functional magnetic resonance imaging (fMRI) [12], is of particular interest since binge behavior could be driven by disturbed functional interactions between brain regions involved in executive functioning and regions promoting behavioral engagement [13]. Importantly, connections between individual brain regions can be combined into larger subnetworks, which together make up the functional brain network [14]. The subnetworks typically consist of strongly connected brain regions that are involved in overlapping cognitive processes [15, 16]. Three subnetworks are particularly important for cognitive functions and their altered connectivity has been linked to many psychiatric disorders: the frontoparietal network (FPN; lateral prefrontal cortex and anterior inferior parietal lobule), the default-mode network (DMN; medial prefrontal cortex, posterior cingulate cortex, posterior inferior parietal lobule) and the ventral attention network (VAN; anterior insula and anterior midcingulate cortex) which is sometimes referred to as the salience network [17–21].

Disturbances in the connectivity between these subnetworks are important for AUD and BN. In people with AUD, studies observe a disrupted synchronicity in the brain activity of regions involved in the FPN, DMN, VAN in rest or while performing a task [22–24]. For BN, a lower connectivity within the VAN and a higher connectivity within the DMN are observed in rest [25, 26]. Additionally, in BN, connectivity between the VAN and the DMN in rest is related to illness severity and body concerns [6, 27]. These studies show that the integration of information across subnetworks such as the DMN and VAN is disturbed in people with AUD or BN. However, most prior studies have only looked at time-averaged (i.e., "static") functional connectivity, whereas recent evidence supports that information is often integrated across subnetworks in a time-variable (i.e., "dynamic") fashion. Indeed, many cognitive processes involve dynamic large-scale changes to how these subnetworks interact. For example, acute stressors have been shown to increase activity in the salience and DMN network [28–30]. Also, more flexibility in the dynamic organization of subnetworks in rest or while performing a task has been related to improved learning, likely by playing an important role in integrating information [31, 32]. Additionally, experiencing more positive emotions has been associated with a higher frequency of reconfigurations of subnetworks (i.e., flexibility) during resting-state, which could explain why patients with a psychiatric disorder who experience fewer positive emotions also have learning difficulties [33]. Indeed, these findings concerning dynamic connectivity could be particularly relevant to AUD and BN, as stress and positive emotions are known triggers of binge behavior [34, 35]. Therefore, research on the dynamic properties of brain functioning in AUD and BN and their association with known important factors may provide novel insights.

Indeed, a previous study on patients with AUD included exploratory analyses on the dynamic reconfiguration of cortical brain regions and showed a reduced flexibility in the anterior cingulate cortex, anterior prefrontal cortex and the anterior insula, which largely overlap with the VAN [15, 36]. Importantly, a disturbance in the dynamics of these regions has been linked to a lower capability of resisting temptations, which could therefore be important in the loss of control that patients experience over their alcohol or food intake [37]. However, the dispersion of reconfigurations across different subnetworks (i.e., promiscuity) has not been evaluated, which describes how spatially focused the dynamic integration of information is and which was associated with stress recovery in individuals with an obsessive-compulsive disorder [38, 39]. Crucially, brain connectivity dynamics have not been explored at all in BN.

Therefore, the present study aims to fill these gaps in the literature by investigating dynamic properties of brain functioning in AUD and BN. It investigates how brain regions are dynamically reconfigured across subnetworks based on resting-state fMRI data from patients with AUD and BN in comparison with controls. More specifically, it explores how the promiscuity and flexibility of brain regions is altered in individuals with a diagnosis of AUD or BN. Although this study investigates the promiscuity and flexibility across all brain subnetworks due to limited prior research, we still expect that some subnetworks are more likely to display differences between patients and controls than others. Namely, based on the previously observed reduced flexibility of VAN regions in AUD and the high overlap between both disorders, we expect a reduction of VAN flexibility and promiscuity in patients with AUD as well as BN compared to controls. Furthermore, given that stress affects functional connectivity and plays a key role in AUD and BN, this study will examine how current stress levels relate to dynamic reconfigurations of brain regions across subnetworks in patients and controls.

Methods

Study participants

In total, 102 female right-handed participants were included in the study (AUD: 27, BN:24, HC:51) after removing 5 participants (BN:4, HC:1) due to artefacts, incidental findings, and corrupted data. Participants were recruited between September 2019 and February 2022 (eMethods 1). The complete in- and exclusion criteria are available in the supplement (eMethods 2). We decided to only include women to limit the influence of sex-based differences concerning functional connectivity [40, 41]. Importantly, the patients needed to meet the criteria for BN or AUD of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [1]. A maximum illness duration of 5 years was set as the role of several psychological, social, and biological factors is thought to change over the course of BN and AUD, and because prolonged alcohol use impacts the brain integrity [42, 43]. Additionally, the patients with AUD needed to drink following a pattern of binge drinking according to the criteria of the National Institute on Alcohol Abuse and Alcoholism (i.e., drinking 4 units of alcohol within 2 h for women), and not following a pattern of episodic, sporadic, or steady drinking [44, 45]. Patients could *not* meet the criteria for both AUD and BN. All participants gave their written informed consent, and the study was approved by the local ethical committee.

General measures

At an in-person assessment, a resident of psychiatry confirmed an individual's eligibility to participate based on the in- and exclusion criteria. The Structured Clinical Interview for DSM-5 (SCID-5-S) was used to confirm the diagnosis of BN or AUD and to screen for other psychiatric disorders [46]. Afterwards, the participants had their weight and height measured with a calibrated scale and stadiometer, to determine the body mass index (BMI), and completed clinical interviews and questionnaires. BN and AUD severity were assessed with the Eating Disorder Examination Questionnaire (EDE-Q) and the Alcohol Use Disorders Identification Test (AUDIT) respectively [47, 48]. The EDE-Q had an excellent internal consistency with a Cronbach's alpha of 0.95 and the AUDIT had a good internal consistency with a Cronbach's alpha of 0.89. Of the patients with BN, 18 (75%) had an EDE-Q score over the clinical cut-off (i.e., 2.8). For the patients with AUD, 26 (96%) had an AUDIT score above the threshold of medium-level problems (i.e., 8), and 13 (48%) had an AUDIT score over the threshold of high-level problems.

MRI data

The resting-state fMRI data were acquired during an MRI scan session which was scheduled at approximately the same time-of-day (median 17:23), the course of which can be found in the supplement (eMethods 3 and eFigure 1). The participants needed to refrain from eating or drinking in the six hours before the session and were not allowed to use substances in the 24 h leading up to the scan session. Participants were asked if they adhered to these instructions, and the session was rescheduled if this was not the case ($n=1$). At the beginning of the scan session, the participants rated their stress levels with a visual analogue scale (VAS). The VAS had ten levels ranging from 0 (not stressed at all) to 10 (never experienced such stress before). During the resting-state fMRI scan, the participants were instructed to close their eyes and stay awake. Scanning was performed on a 3T Achieva dStream Philips MRI scanner with a 32-channel receiver head coil. T2*-weighted echo-planar images were acquired during the resting-state fMRI scan (duration = 450s, 500 volumes, 66 slices, TR = 0.9s, TE = 33ms, flip angle = 65°, voxel size = 2 × 2 × 2.2 mm, MB = 3). Afterwards, a high-resolution T1-weighted image was acquired using a 3D turbo field echo sequence (208 slices, TR = 5.9ms, TE = 2.7ms, flip angle = 8°, voxel size = 0.8 × 0.8 × 0.8 mm).

Data analysis

The data are available upon request.

Data preprocessing

The resting-state fMRI data were preprocessed with fmriprep, version 21.0.1 [49]. The complete information on the preprocessing procedure can be found in the supplement (eMethods 4). Briefly, slice-timing, motion correction, and co-registration to the T1 reference were performed, after which physiological regressors were extracted. Then, fMRI data was resampled to standard space. Importantly, ICA-AROMA was performed on the fMRI data in MNI space after spatial smoothing with an isotropic Gaussian kernel of 6 mm FWHM. Subsequently, the preprocessed data underwent denoising, involving the removal of the mean signals from cerebrospinal fluid, white matter, and the global signal. Additionally, a high-pass filter with a frequency cutoff of 0.02 Hz was applied.

Atlas of regions and subnetworks

An atlas was constructed by combining the 210 cortical regions of the Brainnetome atlas [50, 51]. Afterwards, individual masks were generated to exclude distorted fMRI data if signal intensity was in the lowest quartile of the robust intensity range [52]. Next, these masks were applied to the atlas, and regions with less than 30% coverage across all participants were excluded. Subsequently, the average signal intensity within each remaining anatomical region was computed, forming the time series for that region in the final atlas. Finally, all cortical regions were assigned to one of seven cortical subnetworks based on maximum overlap: the default-mode (DMN), fronto-parietal (FPN), dorsal attention (DAN), ventral attention (VAN), visual, sensorimotor (SMN), and limbic [15]. A deep grey matter network (DGM) was constructed by combining the FIRST-segmented regions. With only 4 regions of the 'limbic network' showing sufficient signal, this subnetwork was removed from the analyses. In the end, there were 194 brain regions assigned to one of 7 subnetworks per participant.

Dynamic subnetwork assignment

Dynamic functional connectivity was calculated using a sliding-window approach where the time series of an individual participant was split into overlapping windows of 60 s (66 volumes) with a step-size of 10s (11 volumes) [53]. Previous research has illustrated that this window length and step-size can adequately capture changes in functional connectivity [54]. Using Matlab 2019a (Natick, MA, USA), windowed connectivity was calculated between using Fisher r-to-z transformed Pearson correlations. Negative values were made positive to capture the strength of the functional link between brain regions. Afterwards, the window-wise assignment of each region to a subnetwork was iteratively re-calculated using a previously established approach [53]. In short, a brain region with the worst assignment to its subnetwork was identified and reassigned to the subnetwork it connects to most strongly. This calculation was reiterated until two successive selections of the same brain region, hence signifying convergence.

Dynamic network reconfiguration metrics

Using the information from the dynamic subnetwork assignment, the different patterns of region reassignment were described with the following measures: (1) promiscuity, which represents the number of subnetworks that a region is part of across all windows; (2) flexibility, which stands for the number of times a region was reassigned, independent of the subnetwork it belongs to. The measures were based on the Dynamic Graph Metrics toolbox [38].

Statistical analyses

Robust linear regression models were fitted to the data as Kolmogorov–Smirnov and Breusch-Pagan tests showed that the normality and homoscedasticity assumptions would be violated if ordinary least squares regression would be used. In a first step, two separate robust linear regression models were constructed where either promiscuity or flexibility across subnetworks was the outcome, and group was a main effect. These models compared whether there was a difference in general promiscuity or flexibility between the groups (i.e., BN vs. HC; AUD vs. HC; AUD vs. BN). In a second step, when a difference in the global network reconfiguration metrics was found, subnetwork-specific robust linear regression models were built with the reconfiguration metric of an individual subnetwork (e.g., DMN) as the outcome, and group as a main factor. The results from these models were corrected for multiple testing using a Benjamini-Hochberg correction. In a third step, the impact of age, BMI, stress, AUDIT and EDE-Q scores, binge eating and binge drinking frequency, illness duration, use of SSRIs, and use of contraceptives on the results were explored by entering them separately as covariates in the robust regression models. The analyses were performed in R (version 4.1.1).

Validation

A sliding-window approach can lead to spurious fluctuations in functional connectivity [55, 56]. To explore whether this was the case in the current dataset, 100 randomized time-series were created by applying a Fourier-transformation to the original time-series before phase-randomisation, and

then applying an inverse Fourier-transformation, as reported previously [53]. These randomized time-series were subjected to the same analyses as the original time-series to calculate promiscuity and flexibility of the subnetworks. Then, to investigate whether there is nonspurious functional connectivity in the data, these measures were compared to the promiscuity and flexibility of the original time-series using a paired sample t-tests. Additionally, to explore whether the results of the current study are actually due to dynamic reconfigurations of the subnetworks, the measures of the randomized time-series were entered as a covariate in the previously described robust linear regression models.

Results

Sample characteristics

The characteristics of the patients with BN ($n=24$), the patients with AUD ($n=27$), and the HC ($n=51$) can be seen in Table 1. There was a significant difference in BMI between the patients with BN (mean=25.7; SD=5.8; CI=23.3–28.3) and the HC (mean=22.4, SD=2.1; CI=21.6–23.3).

Network reconfiguration

A comprehensive overview of the full results can be seen in Table 2; Figs. 1 and 2.

Global network reconfiguration

Promiscuity: Global promiscuity was significantly higher in patients with BN compared to HC (β=0.025, SE=0.012, $p=0.033$, 95% CI=0.003, 0.048). In other words, brain regions of patients with BN were part of more different subnetworks than those of the HC. However, there was no significant difference between patients with AUD and HC (β=0.012, SE=0.011, $p=0.270$, 95% CI=-0.010, 0.034), or patients with AUD and BN (β=-0.013, SE=0.013, $p=0.270$, 95% CI=-0.039, 0.013).

Flexibility: There was no significant difference between the groups when it comes to global flexibility (AUD>HC: β=0.005, SE=0.006, $p=0.345$, 95% CI=-0.006, 0.017; BN>HC: β=0.011, SE=0.006, $p=0.087$, 95% CI=-0.001, 0.022; AUD>BN: β=-0.005, SE=0.007, $p=0.457$, 95% CI=-0.018, 0.008).

Subnetwork-specific reconfiguration

Promiscuity: Patients with BN displayed a significantly higher promiscuity of the DAN than HC (β=0.066, SE=0.021, $p=0.018$, 95% CI=-0.024, 0.107) after correcting for multiple comparisons. There was no significant difference in the promiscuity of the other subnetworks between patients with BN and HC. As no difference concerning global promiscuity was found between patients with AUD and HC, no follow-up analyses were performed to compare the promiscuity of the specific subnetworks between these groups.

Flexibility: No difference concerning global flexibility were observed between the different groups, so no follow-up analyses were performed to compare the flexibility of the specific subnetworks.

Exploratory analyses

Across all participants, higher stress levels during the scan were related to a lower global promiscuity (β=-0.007, SE=0.003, $p=0.011$, 95% CI=-0.012,-0.002) and a lower promiscuity of the DAN (β=-0.013, SE=0.004, $p=0.001$, 95% CI=-0.021,-0.006). In a post-hoc analysis, an ANOVA was performed to explore whether there was a difference in subjective stress between the participant groups (mean[sd]: AUD=3.04[1.95]; BN=2.92[2.26]; HC=2.76[1.62]) as this could have influenced the previously described results. However, there was no significant difference between the groups (F(2, 99) = [1.370], $p=0.823$). Furthermore, entering stress, age, BMI, AUDIT scores, EDE-Q scores, binge eating frequency, binge drinking frequency, illness duration, SSRI use, or contraceptive use as a covariate did not change the significance of the previously described results. The relation between these covariates and the outcomes can be seen in the supplement (eTable1).

Validation

The global promiscuity (t=10.072, df=101, $p<0.001$) and flexibility (t=4.665, df=101, $p<0.001$) of the original time-series were higher than those of the randomized time-series, showing that there is nonspurious functional connectivity in the current dataset. The promiscuity of the whole brain did no longer differ between patients with BN and controls after the promiscuity of the randomized time-series was entered as a covariate (β=-0.006, SE=0.006, $p=0.271$, 95% CI=-0.005,0.018), suggesting that the difference could be actually due to changes in static functional connectivity. However, the promiscuity of the DAN was still higher in patients with BN than controls (β=0.027, SE=0.011, $p=0.016$, 95% CI=-0.005,0.048), which emphasizes that this difference is actually due to changes in the dynamic reconfiguration of the subnetwork.

Table 1 Sample characteristics

	AUD ($n=27$)		BN ($n=24$)		HC ($n=51$)	
	Mean (SD)	95% CI	Mean (SD)	95% CI	Mean (SD)	95% CI
Age	21.7 (4.6)	19.9–23.5	22.3 (3.3)	20.9–23.7	21.7 (2.6)	20.9–22.4
BMI	22.4 (2.1)	21.6–23.3	25.7 (5.8)	23.3–28.3	22.4 (2.2)	21.7–23.0
Illness duration (years)	3.0 (1.2)	2.5–3.4	2.3 (1.5)	1.7–2.9	0 (0)	0–0
Education (years)	14.6 (1.8)	13.9–15.3	15.0 (2.0)	14.2–15.9	15.2 (1.6)	14.2–15.8
AUDIT	13.9 (4.4)	12.2–15.7	4.3 (3.6)	2.7–5.8	3.6 (2.2)	3.0-4.3
EDE-Q						
Restraint	0.8 (1.0)	0.4–1.2	2.8 (1.5)	2.2–3.5	0.4 (0.7)	0.2–0.6
Shape concern	1.7 (1.5)	1.1–2.3	4.3 (1.4)	3.7–4.8	0.9 (1.0)	0.5-1.0
Weight concern	1.3 (1.4)	0.7–1.8	4.1 (1.3)	3.5–4.6	0.7 (1.0)	0.5-1.0
Eating concern	0.5 (0.9)	0.2–0.8	2.8 (1.5)	2.2–3.5	0.2 (0.4)	0.1-04
Total	1.2 (1.1)	0.7–1.6	3.6 (1.2)	3.1–4.1	0.6 (0.7)	0.4–0.8
Eating disorder symptoms (days/4 weeks)						
Binge eating	0 (0)	0–0	10.3 (8.7)	6.6–14.0	0 (0)	0–0
Fasting	0 (0)	0–0	5.7 (6.5)	2.9–8.4	0 (0)	0–0
Vomiting	0 (0)	0–0	4.2 (9.1)	0.4–8.1	0 (0)	0–0
Laxative use	0 (0)	0–0	0.7 (3.3)	0–2.0	0 (0)	0–0
Diuretic use	0 (0)	0–0	0 (0)	0–0	0 (0)	0–0
Compensatory exercise	0 (0)	0–0	6.3 (6.4)	3.6-9.0	0 (0)	0–0
	n (%)	95% CI	n (%)	95% CI	n (%)	95% CI
Binge drinking frequency						
Never	0 (0%)	0–0%	12 (50%)	33–71%	26 (51%)	39–66%
Annually	0 (0%)	0–0%	4 (17%)	0–38%	3 (6%)	0–20%
Semi-annually	0 (0%)	0–0%	1 (4%)	0–25%	6 (12%)	0–26%
Three-monthly	3 (11%)	0–32%	4 (17%)	0–38%	9 (18%)	6–32%
Monthly	6 (22%)	7–43%	2 (8%)	0–29%	5 (10%)	0–24%
Biweekly	12 (44%)	30–66%	0 (0%)	0–0%	2 (4%)	0–18%
Weekly	3 (11%)	0–32%	1 (4%)	0–25%	0 (0%)	0–0%
> Weekly	3 (11%)	0–32%	0 (0%)	0–0%	0 (0%)	0–0%
Therapy (BN/AUD)						
Past	0 (0%)	0–0%	9 (38%)	17–58%	0 (0%)	0–0%
Present[a]	0 (0%)	0–0%	3 (13%)	0–26%	0 (0%)	0–0%
Previous AN	0 (0%)	0–0%	5 (21%)	4–38%	0 (0%)	0–0%
Race and ethnicity						
Caucasian	26 (96%)	93–100%	23 (96%)	92–100%	49 (96%)	95–100%
Latina	1 (4%)	0–10%	0 (0%)	0–0%	0 (0%)	0–0%
Asian	0 (0%)	0–0%	0 (0%)	0–0%	0 (0%)	0–0%
Multi-Racial	0 (0%)	0–0%	0 (0%)	0–0%	1 (2%)	0–8%
Middle-Eastern	0 (0%)	0–0%	1 (4%)	0–11%	0 (0%)	0–0%
Contraceptive use	21 (78%)	61–94%	18 (75%)	57–93%	47 (92%)	85–100%
Amenorrhea	0 (0%)	0–0%	1 (4%)	0–13%	0 (0%)	0–0%
SSRI	3 (11%)	0–24%	3 (13%)	0–26%	0 (0%)	0–0
Comorbidities						
MDD	1 (4%)	0–18%	1 (4%)	0–17%	0 (0%)	0–0%
PD	1 (4%)	0–18%	1 (4%)	0–17%	0 (0%)	0–0%
SAD	1 (4%)	0–18%	1 (4%)	0–17%	0 (0%)	0–0%
PTSD	1 (4%)	0–18%	0 (0%)	0–0%	0 (0%)	0–0%

AN anorexia nervosa; *AUD* alcohol use disorder; *AUDIT* alcohol use disorders identification test; *BMI* body mass index; *BN* bulimia nervosa; *CI* confidence interval; *EDE-Q* Eating Disorder Examination Questionnaire; *MDD* major depressive disorder; *n* number; *PD* panic disorder; *PTSD* post-traumatic stress disorder; *SAD* social anxiety disorder; *SD* standard deviation; *SSRI* Selective serotonin reuptake inhibitors

[a]Patients were in different treatment modalities (i.e., ambulatory psychologist, psychiatrist, dietician or outpatient treatment program)

Table 2 Network reconfiguration results

Measure	Comparison	Network	β	SE	*p*	95% CI
Promiscuity	AUD > HC	Global	0.012	0.011	0.270	– 0.010, 0.034
	BN > HC	**Global**	**0.025**	**0.012**	**0.032**	**0.003**, **0.048**
		DMN	0.015	0.023	0.759[a]	– 0.029, 0.060
		FPN	– 0.010	0.024	0.780[a]	– 0.058, 0.037
		DAN	**0.066**	**0.021**	**0.018**[a]	**0.024**, **0.107**
		VAN	0.012	0.020	0.759[a]	– 0.027, 0.051
		Visual	0.002	0.019	0.900[a]	– 0.035, 0.039
		SMN	0.035	0.024	0.510[a]	– 0.011, 0.081
		DGM	0.027	0.026	0.728[a]	– 0.024, 0.077
	AUD > BN	Global	– 0.013	0.013	0.270	– 0.039, 0.013
Flexibility	AUD > HC	Global	0.005	0.006	0.345	– 0.006, 0.017
	BN > HC	Global	0.011	0.006	0.087	– 0.001, 0.022
	AUD > BN	Global	– 0.005	0.007	0.457	– 0.018, 0.008

Statistically significant results are indicated in bold

AUD alcohol use disorder; *BN* bulimia nervosa; *β* estimate; *CI* confidence interval; *DAN* dorsal attention network; *DGM* deep gray matter; *DMN* default mode network; *FPN* frontoparietal network; *HC* healthy controls; *p* p-value; *SE* standard error; *SMN* sensorimotor network; *VAN* ventral attention network

[a]Corrected for multiple testing with the Benjamini-Hochberg method

Discussion

This study explored how brain regions are dynamically reconfigured across subnetworks during a resting-state scan in patients with AUD or BN, in comparison with healthy controls. The results showed a higher distribution of reconfigurations on average across the whole brain in patients with BN. This was mostly attributable to regions from the DAN being reconfigured to a wider range of other subnetworks. However, no significant difference was observed in the frequency of reconfigurations in patients with BN, nor in either the distribution or frequency of dynamic reconfigurations in patients with AUD. Additionally, higher subjective stress levels were related to a lower distribution of reconfigurations of the regions in the DAN across all participant groups.

This is the first study to investigate network dynamics in patients with BN. The current study finds an increase in the distribution of reconfigurations was observed in these patients, particularly for regions of the DAN. This suggests that the dynamic integration of information from the DAN to other subnetworks became more dispersed, hence seemingly less focused. This was surprising, as in contrast to the VAN [25, 26], no previous observations of disturbed static functional connectivity have been reported for the DAN in BN. Regions of the DAN are primarily located in the intraparietal sulcus and frontal eye fields [57] and play a key role in top-down biasing of sensory areas (e.g., cue-induced expectations of stimuli in a visual area) and working memory [57, 58]. Doing so, the DAN is thought regulate our attention and behavior in response to stimuli, making it possible to control impulsive actions [59]. Indeed, studies suggest that disturbances in the functioning of the DAN play a role in the impulsivity of patients with attention-deficit/hyperactivity disorder [59]. Interestingly, patients with BN show an attentional bias for food and weight stimuli, and display higher levels of impulsivity [60, 61]. Thus, a less focused reconfiguration pattern of regions in the DAN could play a role in the attentional biases and higher impulsivity that patients with BN display.

In contrast to the observations for BN, no altered brain dynamics were observed for AUD. The lack of a difference in reconfiguration frequency or dispersion between patients with AUD and controls stands in contrast to the findings from a previous study, where a lower frequency of dynamic reconfigurations was seen in regions of the VAN as part of an add-on exploratory analysis [36]. One reason for these contrasting findings could be the differences between the study populations, as the previous study included a sample of patients with a substantially longer illness duration [36]. The neurotoxic effects of repeated alcohol misuse could have had a more significant impact on brain structure and function after a longer disease course [62], eventually leading to a reduced flexibility of the VAN. Future studies should, therefore, explore the impact of illness duration on the functional connectivity of brain subnetworks, preferably employing a longitudinal study design. In addition, as patients with AUD display several behavioral similarities with patients with BN, it could be expected that similar changes in DAN dynamics would also be seen in patients with AUD [63, 64]. The reason no alterations in DAN dynamics were observed could be explained by the behavioral similarities having different neurobiological correlates, though the absence of a difference between patients with

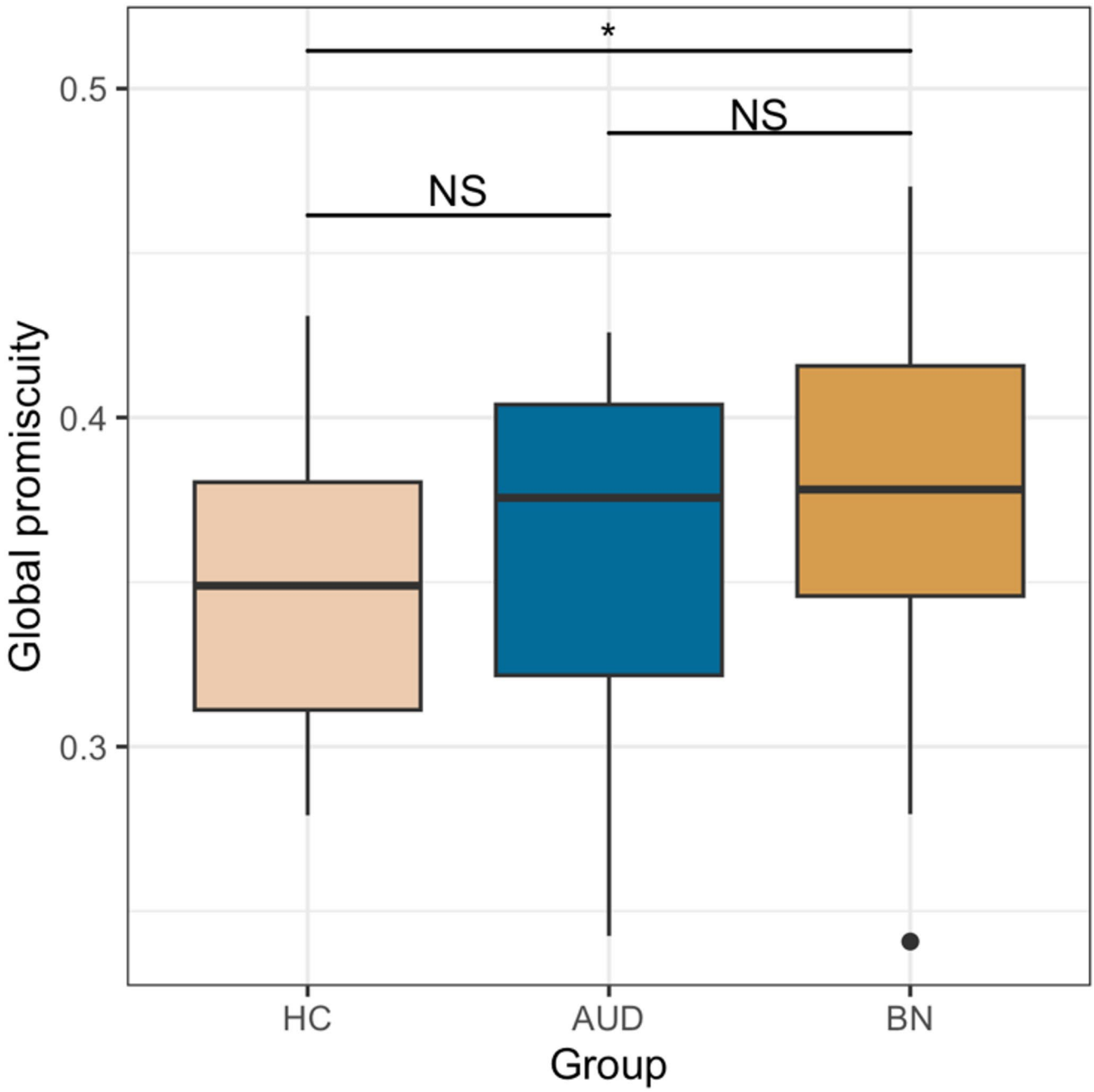

Fig. 1 Global promiscuity. Global promiscuity for the HC, the patients with AUD, and the patients with BN. There was no significant difference between patients with AUD and HC, but patients with BN had a significantly higher global promiscuity compared to HC. Symbols: *, significant. *AUD* alcohol use disorder; *BN* bulimia nervosa; *HC* healthy controls; *NS* not significant

AUD and controls could also have been influenced by the limited sample size of the study and more large-scale studies are needed to draw definitive conclusions, and this could be also be the reason why no significant difference was seen in global promiscuity between patients with AUD and BN.

In addition to the findings concerning the different groups, the results of the current study showed that global and dorsal network promiscuity were negatively associated with subjective stress across all participants. In theory, a higher promiscuity of the DAN could be related to experiencing less stress, as this might allow the DAN exert more control the rest of the brain and hence support top-down attentional processes [57]. However, this seemingly contradicts the finding that patients with BN have a higher promiscuity of the DAN, as they are typically more vulnerable to stress [65]. In turn, a lower promiscuity of the DAN during

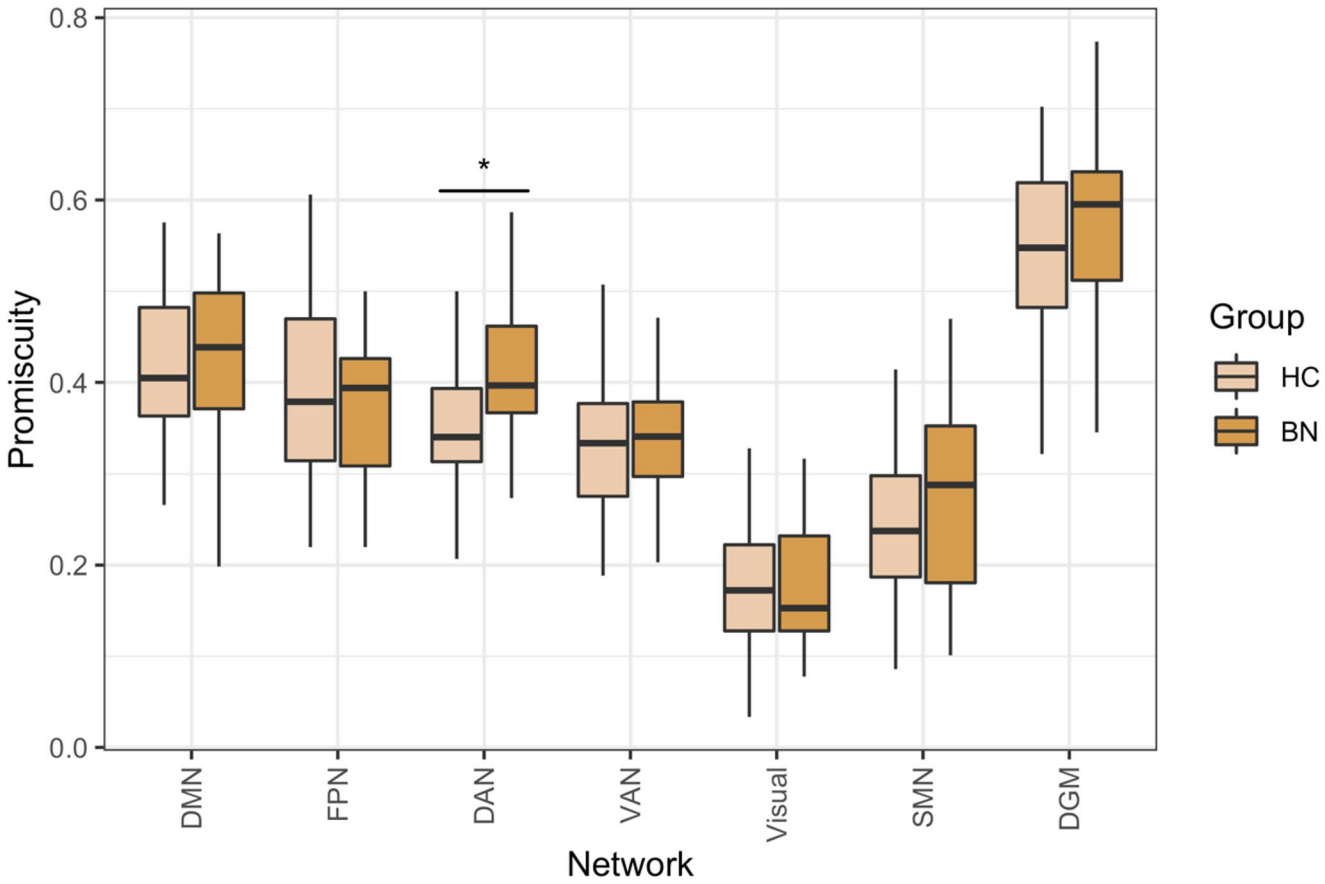

Fig. 2 Subnetwork promiscuity. Subnetwork promiscuity for the HC and the patients with BN. Patients with BN had a signifciantly higher promiscuity of the DAN than HC. There was no significant difference between the groups for the other subnetworks. Symbols: *, significant. *BN* bulimia nervosa; *DAN* dorsal attention network; *DGM* deep gray matter; *DMN* default mode network; *FPN* frontoparietal network; *HC* healthy controls; *SMN* sensorimotor network; *VAN* ventral attention network

moments of subjective stress could be more adaptive, as this could indicate that the DAN limits its connections to regions that are important in the stress response [57]. Regardless, the particular relevance of the DAN was unexpected, as most models of stress processing highlight the prominent role of the VAN and not the DAN [28, 29]. Other studies did observe that prolonged exposure to a stressor was associated with increased DAN functional connectivity [66, 67]. In addition, another recent study showed that the DAN became more strongly connected while recovering from an acute stressor in controls, which was not observed in individuals with bipolar disorder [68]. This further emphasizes the importance of DAN dynamics for adaptive stress processing, and showcases how disturbances in DAN dynamics might be involved in vulnerability and resilience to stress. Interestingly, as patients with BN display a higher promiscuity of the DAN in the current study, it could be suggested that this finding plays a role in the higher stress-vulnerability that is reported in patients with BN [65].

The finding that patients with BN display a higher dispersion of reconfigurations for the DAN could have important implications for possible interventions. Previous studies showed that mindfulness training can increase the functional connectivity within the DAN and improve attention, and that being less mindful was associated with eating disorder psychopathology such as binge eating [69–71]. Thus, in theory, mindfulness-based interventions might affect the dynamic organization of the DAN as well as reduce binge eating frequency in patients with BN. Indeed, though cognitive behavioral and interpersonal therapy have received the largest empirical support in adults with BN, there are studies which suggest that mindfulness-based cognitive therapy as well as dialectic behavioral therapy can be effective in treating BN [72]. However, it is unclear whether changes in the static and dynamic embeddedness of the DAN and other brain subnetworks might mediate this effect, and future studies should explore whether this is the case. Additionally, treatment with antidepressants can affect the dynamic reconfiguration of brain subnetworks [73, 74]. As such, this opens up the possibility to tailor future treatments based on the observed pattern of brain network dynamics, thereby facilitating personalized treatment, but this would require

more insight into the unique effects of specific treatments on brain network dynamics.

The conclusions of this study should be made in light of several limitations. First, increasing the sample size and using a longer scan time further could beneficially impact the power of the analyses. Second, the sample consists of exclusively female participants that are mostly young and of Caucasian descent, which could limit the generalizability of the results to all patients with AUD or BN [75, 76]. Third, the current study explored dynamic functional connectivity using a sliding-window technique, which can suffer from an excess in variability when the window is too short [56]. This is why a validation analysis was included, which showed nonspurious dynamics with the current parameters (e.g. window size and shape), indicating that the observed dynamics do not simply represent static network organization or noise. Fourth, careful exclusion of brain regions showing distorted signal was performed to further improve data quality, but this meant that dynamic functional connectivity of the limbic network could not be investigated, which is unfortunate as the limbic system plays an important role in stress reactivity [77]. Fifth, the current study has a cross-sectional design, though the duration of AUD and BN could impact the dynamic functional connectivity of brain subnetworks, which should be assessed with a longitudinal design.

Conclusion

This study showed that people with BN had more widely dispersed dynamic network reconfigurations compared to HCs, particularly for regions of the DAN. In contrast, people with AUD showed no changes in reconfiguration dynamics. This suggests that patients with BN, in particular, have a less focused dynamic integration of information from the DAN to the rest of the brain. This pattern might be important for the attentional bias to food and high impulsivity that are seen in patients with BN. In addition, the current findings suggest a possible link between disturbances in DAN network dynamics and stress, which might leave individuals with BN more vulnerable to stressors. Future studies could further investigate the negative effects of alcohol misuse and binge eating on network dynamics in a large-scale longitudinal cohort, as well as explore treatments targeting network dynamics in patients with AUD or BN.

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Author contributions Nicolas Leenaerts had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Concept and design: Leenaerts, Broeders. Acquisition, analysis, or interpretation of data: Leenaerts, Broeders. Drafting of the manuscript: Leenaerts, Broeders Critical revision of the manuscript for important intellectual content: Leenaerts, Broeders, Sunaert, Ceccarini, Vrieze, Schoonheim, Vinkers, Obtained funding: Vrieze, Ceccarini Supervision: Vrieze, Schoonheim.

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Data availability The data and scripts that support the findings of this study are available upon request.

Declarations

Conflict of interest A C1 grant (grant number ECA-D4671-C14/18/096) of the Special Research Fund KU Leuven to Vrieze and Ceccarini served as a PhD Scholarship for Leenaerts. Ceccarini was supported by a postdoc grant from FWO (grant number 12R1619N). No other grant of any kind was received. No other disclosures were reported.

Ethical approval The authors assert that all 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 2008.

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