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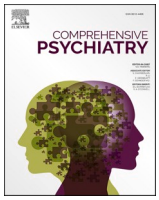

Exercise moderates longitudinal group psychopathology networks in individuals with eating disorders

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ABSTRACT

Individuals with eating disorders (EDs) often engage in exercise no matter potential negative long-term outcomes (e.g., weight loss, injury). Yet exercising may temporarily attenuate ED symptoms, but whether exercise also affects network structure and pairwise associations of ED symptoms remained unclear. We used a novel approach called Moderated Multilevel Graphical Vector Autoregression to estimate changes in psychopathology networks from before to after exercising in ecological momentary assessment data from 102 individuals with EDs across multiple days (M = 22.14, SD = 5.40; range: 6–22 days) at 4 times daily. Between-person and within-person temporal networks were computed, obtaining stable centrality coefficients for temporal networks only. In those, autoregressive effects of several symptoms, including binge-eating, overeating, or weighing oneself, were attenuated when participants previously exercised. Exercise mostly downregulated temporal effects of ED symptoms on other symptoms, including effects of binge eating and other compensatory behaviors on feeling guilty after the most recent meal, vomiting on weighing oneself, and overeating on fear of weight gain. Our study highlights the complex dynamic effects of exercise on ED symptoms in daily life and calls for novel studies investigating mechanisms of exercise to inform treatments targeting detrimental long-term effects of exercise in EDs.

1. Introduction

Eating disorders (EDs) are severe mental disorders resulting in high mortality, morbidity, and healthcare costs [18,52]. EDs primarily affect young people and up to 17.9 % of women and 2.4 % of men have experienced an ED by early adulthood [44], with a further increase during the COVID-19 pandemic [50]. In addition to ED symptoms like binge eating, restrictive eating or purging behaviors, many individuals with EDs frequently engage in exercise, which is often described as maladaptive. Maladaptive exercise is defined as exercise that is conducted in a driven or compulsive manner, conducted to control weight and shape, to compensate for calorie intake and/or excessive in frequency or duration, although no consensus on a definition has been reached yet [27]. Depending on the type of ED, between 16.7 % and 83.5 % of individuals with EDs are affected by maladaptive exercise, with highest rates for individuals with anorexia nervosa (AN) and bulimia nervosa (BN; [7,16,47]). Individuals with EDs who engage in maladaptive exercise describe exercising mostly due to feeling compelled or driven to exercise, to follow rigid regimes, or to control weight or shape [26] despite potentially severe consequences, such as pain, risk of injury, or impaired daily functioning [35]. Although individuals with EDs also engage in adaptive exercise, new research shows that specific exercise episodes are generally experienced as maladaptive by individuals with EDs [26].

Theoretical models of maladaptive exercise in EDs highlight the short-term positive consequences of exercising for these individuals that most likely reinforce the behavior over time [22,34]. In fact, individuals indicate that maladaptive exercise serves both affective and social positive and negative reinforcement functions across populations with varying degrees of ED severity [24]. Evidence from an Ecological Momentary Assessment (EMA) study corroborates this further [11]. These effects were also found when investigating exercise and physical activity more broadly, instead of specifically maladaptive exercise: Prior increases in physical activity led to subsequent higher levels of positive

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and lower levels of negative affect in adults and adolescents with anorexia nervosa, but only to more positive affect in healthy adolescents [23]. Reichert et al. [38] showed that improved mood and lower momentary levels of ED cognitive symptoms, such as body dissatisfaction and drive for thinness occurred after exercising in adults with EDs, but only mood was changed in healthy adults. However, another study investigating trajectories of body dissatisfaction pre- and post-exercise in individuals with binge-spectrum EDs only found an increase in body dissatisfaction prior to but no decrease after exercising [46]. In addition, exercising, regardless of being adaptive or maladaptive, may induce several more general non-ED-specific physiological and psychological changes that could possibly influence ED symptoms temporally, such as changes in cortisol secretion [21], emotion regulation capacities [1] or the endocannabinoid system ([8]; for an overview see [22]). Yet, to our knowledge, there is no research on whether exercise influences pairwise associations between ED symptoms, such as modifying the influence of binge eating on subsequent vomiting or vice versa, which is a common temporal sequence of symptoms in bulimia nervosa [28].

A potentially useful framework for understanding dynamic changes in pairwise associations between symptoms is the network theory of mental disorders [2]. Network theory posits that symptoms and their dynamic relations constitute and maintain mental disorders, rather than being caused by common latent factors. More specifically, symptoms are activated by external or internal events (e.g., social interactions, physiological states like hunger) which subsequently activate or inhibit further symptoms. Mental disorders can thus be considered more or less stable states of symptom networks (see e.g. [6], as an example for understanding major depression as a complex dynamical system). In addition, treatments, activities, or events that are not normally part of the symptoms network, such as whether an individual was exercising, can be conceptualized as moderators of specific associations between symptoms in the network. Conjoint with network theory, network analysis was extended as a statistical approach to model symptom networks. Several studies already have used network analysis to further our understanding of EDs. Many cross-sectional studies highlighted that cognitive-affective ED symptoms such as overevaluation of weight and shape, body dissatisfaction and fear of weight gain are most central (i.e., most interrelated with and influential to other symptoms in the network) for ED psychopathology [9,30,41,45], and other studies showed that central symptoms predict clinical and treatment outcomes in individuals with EDs [10,29]. However, much less research has used network analysis together with intensive longitudinal data (i.e. EMA) to investigate short-term temporal associations between symptoms. With EMA, behaviors, cognitions, and mood are assessed directly in the daily life of participants across several measurement time points (i.e., several times a day for multiple days). Therefore, EMA increases ecological validity, reduces retrospective bias, and allows to capture and model temporal associations that allow for better approximations of causality compared to cross-sectional studies [43]. Levinson et al. [33], using EMA data, found similar results as cross-sectional studies regarding the centrality of cognitive-affective symptoms, such as fear of weight gain, desire for thinness or feeling guilty in within-person networks modelling temporal associations. Remarkably, these symptoms also predicted ED outcome six months later. They also found that individual network models were highly heterogeneous and can be potentially used to identify personalized treatment targets for ED treatment [32], further highlighting the potential of network analysis in EDs. These authors also found that when averaged, maladaptive exercising had the highest strength centrality (i.e., high interconnectedness to other symptoms on average), which indicates that maladaptive exercise might be an influential symptom in ED symptom networks [33]. However, including exercise as a symptom in these network models allowed only for investigating direct associations between exercise and other symptoms, but not for modelling the potential influence of exercise on the associations between two other symptoms. Investigating whether exercise in general moderates pairwise associations between other symptoms, for example whether exercising interrupts the frequent cycle of binge eating followed by vomiting or the association of the desire to be thinner with thinking about dieting, is important for better understanding why individuals with EDs maintain exercise regimes that are maladaptive due to being severely underweight or currently injured. Then, better treatment options can be developed that not merely prohibit exercising but rather provide patients with alternatives to exercise that effectively alleviate other symptoms and their associations.

We hypothesize that exercise changes the network structure of ED pathology given that exercising, regardless of being adaptive or maladaptive, results in several acute physiological and psychological changes, both in general and specific to EDs. To model these possible changes on associations among key ED cognitions, affect, and behaviors, we analyzed intensive longitudinal data from Levinson et al. [33] with a novel network analysis method allowing to estimate moderated pairwise associations. To our knowledge, this is the first time that moderated network analysis is used to investigate short-term effects of a specific behavior on symptom associations in a psychopathology network. We identified top central symptoms and strongest pairwise associations when previously exercising or not exercising, as well as strongest moderation effects in both average and temporal network models. We compared network density of moderated networks when exercising or not exercising. We also inspect stability of centrality measures, pairwise associations and moderation effects in all network models.

2. Methods

2.1. Participants and procedure

We analyzed data from Levinson et al. [33] for this study. Participants were $N = 102$ individuals diagnosed with an ED and aged 16–61 years ($M = 29.55$, $SD = 9.26$ years). The sample was predominantly female ($n = 96$ female, $n = 4$ male, $n = 1$ nonbinary and $n = 1$ missing gender) and mostly non-Hispanic white ($n = 86$; Asian or Asian-American: $n = 5$, Hispanic: $n = 4$, multi−/bi-racial: $n = 4$, non-Hispanic Black: $n = 3$). ED diagnoses were $n = 29$ anorexia nervosa, $n = 13$ bulimia nervosa, $n = 4$ binge-ED, $n = 38$ otherwise-specified feeding and EDs and $n = 18$ EDs in remission. At time of participation, $n = 60$ participants were receiving any type of treatment ($n = 3$ partial inpatient, $n = 3$ intensive outpatient and $n = 55$ outpatient treatment). Data collection for the study was approved by the University of Louisville Institutional Review Board (no. 16.1077). All participants gave informed consent prior to participation and the study was conducted in line with the Declaration of Helsinki.

Participants were recruited between April 2017 and September 2019 across the US for a study on daily habits of people with EDs. Participants completed two semi-structured interviews (see measures) via teleconference to assess inclusion/exclusion criteria. They were included if they reported a current or past diagnosis of an ED that was still only in partial remission. Exclusion criteria were current suicidality, psychosis, or mania. Diagnoses were double-checked by four independent raters prior to inclusion. After inclusion, participants completed baseline self-report measures using an online questionnaire platform (REDCap; [20]) and then completed EMA measures four times a day for 25 days (three to five minutes at each assessment time point). Assessment days were not fully consecutive, with two assessment bursts over 52 days.

2.2. Measures

2.2.1. Structured Clinical Interview for DSM-5 Eating Disorder Module (SCID-5-RV)

The SCID-5 is a semi-structured interview for assessing DSM-5 diagnoses [17]. The ED module was used to determine ED diagnoses.

2.2.2. Mini-International Neuropsychiatric Interview 5.0 (MINI 50.0)

The MINI 5.0 is a semi-structured interview to make DSM-5

diagnoses [42]. It was used to assess active suicidal intent, mania, and psychosis; for exclusion of participants.

2.2.3. Ecological momentary assessment measures

We used the Daily Habits Questionnaire [31] to assess symptoms relevant to people with EDs using an EMA assessment schedule with four-hour semi-fixed intervals (four beeps per day) over 25 days. All questions were answered on a scale from 1 – not at all to 6 – extremely, except for the items 'anxiety' and 'guilt', which were measured from 1 – no anxiety/guilt to 6 – extreme anxiety/guilt. In line with Levinson et al. [33], 12 core cognitive, behavioral and affective ED symptoms were selected a-priori as in the original study from the Daily Habits Questionnaire for our analyses (see Table 1). Exercise was assessed by asking participants directly whether they engaged in any type of exercise (yes/no) since the last beep (including adaptive and maladaptive exercise; aerobic, strength exercise, etc.). Given that most exercise episodes can be considered maladaptive [26], effects on ED symptoms extend to physical activity and general exercise, and that assessing engagement in a behavior is less demanding than qualifying this behavior as either adaptive or maladaptive during EMA, we assessed moderation effects using this item.

2.3. Statistical analyses

2.3.1. (Non-)stationarity of the time series data

Multi-level graphical vector autoregressive models (mlGVAR) assume stationarity of the underlying data, an assumption that might be violated in time-series data of a sample with nearly 60 % of the participants receiving treatment. However, the effects of non-stationarity especially on cross-lagged associations in network models appear to be difficult to detect and approaches that directly model non-stationarity in network dynamics remain challenging [5]. To account for and assess potential bias introduced by non-stationarity of time-series data, we (1) conducted Kwiatkowski-Phillips-Schmidt-Shin (KPSS; [25]) tests to assess the stationarity of univariate time series (i.e., for each symptom by each individual separately) using the *aTSA* package v3.1.2.1 [36], (2) detrended all univariate time series that are trend-level stationary (i.e., follow a deterministic trend) by replacing the data with the residuals of a simple linear model that regresses the symptom variable on time as recommended by Epskamp et al. [14] and (3) assessed stationarity again after detrending.

Table 1
Abbreviations of symptom nodes.

Node name	Corresponding item	Scale
Exerc	Did you exercise since the last check in	*Yes – no*[a]
Binge	Engaged in binge eating since the last check in	1 = *not at all* to 6 = *extremely*
Bodycheck	Engaged in body checking since the last check in	1 = *not at all* to 6 = *extremely*
Compens	Engaged in other compensatory behaviors since the last check in	1 = *not at all* to 6 = *extremely*
Deserve	I had the thought I don't deserve to eat during the most recent meal	1 = *not at all* to 6 = *extremely*
Diet	I am thinking about dieting	1 = *not at all* to 6 = *extremely*
Feargain	I am terrified of gaining weight	1 = *not at all* to 6 = *extremely*
Guilty	I felt guilty during the most recent meal	1 = *not at all* to 6 = *extremely*
Overeat	I feel like I have overeaten since the last check in	1 = *not at all* to 6 = *extremely*
Restrict	Engaged in restriction since the last check in	1 = *not at all* to 6 = *extremely*
Thinner	I am preoccupied with the desire to be thinner	1 = *not at all* to 6 = *extremely*
Vomit	Engaged in vomiting since the last check in	1 = *not at all* to 6 = *extremely*
Weigh	I have weighed myself since the last check in	1 = *not at all* to 6 = *extremely*

Note: [a]For continuous moderation analyses, exercise was also answered on a scale from 1 = *not at all* to 6 = *extremely*.

2.3.2. Network estimation

We constructed between-person, contemporaneous and temporal moderated network models at the group level with *R*, v4.0.3 [37] and *RStudio*, v1.3.1093 [40], using the *modnets* package v0.9.0 [49]. We fitted mlGVAR models to our data that incorporated exercise as an exogenous moderator to the network. Exogenous moderators will not be modelled as nodes in a network and instead are included in the model as interaction terms. Moderated mlGVAR uses a pseudo-mixed effects estimation technique, in which fixed effects are fully estimated and random effects are approximated (for details, see [15]). To account for correlated errors when including interaction terms, a seemingly unrelated regression model was used instead of graphical least absolute shrinkage and selection operator (GLASSO) regularization that is typically used in mlGVAR models (see Chapter 5 of Swanson [48] for technical details on this approach). Missing data were handled by listwise deletion, as multiple imputation techniques are currently not available for estimating moderated mlGVAR models.

2.3.3. Network models

Two group-level networks were created. Within these models, ED symptoms are represented as *nodes*. Associations between nodes are called *edges*, which reflect linear associations between two nodes, conditioned on all other nodes. Except for temporal networks, these associations are undirected. Missing edges indicate independence of two nodes after conditioning on all other nodes with $p < .05$, as recommended by Epskamp and Fried [12]. Statistical significance was calculated separately for each conditional level (i.e., whether participants previously exercised or not) and for interaction terms. Significant interactions reflect that exercise moderates the association between two nodes.

2.3.3.1. Between-person network. First, we created the between-person network that represent average relations of symptoms with other symptoms and their average moderation by exercise across all participants. Between-person networks are similar to cross-sectional networks, although they incorporate longitudinal information to create stable means across time for each individual. Significant interactions in between-person networks indicate that on average the association between two symptoms is moderated by exercise. We plotted between-person networks including direct effects of the moderator as well as at different levels of the moderator to highlight changes in edges due to the moderator (conditional network models).

2.3.3.2. Contemporaneous network. Contemporaneous networks cannot be moderated and are therefore only presented in the supplements (Supplement S1).

2.3.3.3. Temporal network. In the temporal network, nodes predict other nodes from one time point (*t-1*) to the next (*t0*), that is four hours in our data. Moderation indicates that the moderator (i.e., exercise) significantly changes the temporal association of two nodes. Moderator variables are lagged by four hours. Thus, a significant interaction effect indicates that exercising in between *t-2* and *t-1* (since the exercise item asks whether someone exercised *since the last check-in*) moderates the temporal association of symptom *a* at *t-1* (or, more precisely, in between *t-2* and *t-1* for items asking *since the last check-in*) on symptom *b* at *t0* (for technical details see [48], pp. 90–92). Moderated temporal networks are also plotted including direct effects of the moderator as well as at different levels of exercise to highlight changes in edges due to the moderator.

2.4. Network inference, density and stability

To assess overall network structure, we computed strength and expected influence as centrality indices for all networks. We did not compute betweenness and closeness, as these centrality measures are not appropriate for psychopathology networks [4]. In contemporaneous and between-person models, node strength is defined as the absolute sum of all edges of a given node to all other nodes, indicating which symptoms are most interconnected. Expected influence is defined as the summed weight of a node's edges shared with remaining nodes. The main difference between strength and expected influence is that expected influence accounts for negative edge weights. In psychopathology networks, symptoms that have many negative edges (i.e., attenuate rather than activate other symptoms) may be less problematic than symptoms with many positive edges [39]. Thus, high positive expected influence indicates that a given symptom has a strong activating influence in a network, whereas high negative expected influence indicates an attenuating influence. In temporal networks, centrality measures must consider directionality of edges. Thus, in-strength/in-expected influence as well as out-strength/out-expected influence can be computed, whereby in-strength/in-expected influence are measures of which symptoms are influenced by other symptoms, whereas high out-strength/out-expected influence indicate symptoms that are highly influential to other symptoms in the network (again, expected influence accounts for negative edge weights). Centrality measures were also computed for interaction terms, although the interpretation of these measures is still up to debate and outside the scope of this manuscript ([48], pp. 53–54).

Network density was computed for between-person and temporal networks as the absolute value of all pairwise and autoregressive edge weights divided by the number of all possible pairwise and autoregressive edges (i.e., network density reflects the proportion of possible absolute edge weights). This approach was first described by Bringmann et al. [3]. To compare network density of between-person and temporal networks when individuals were exercising versus not exercising previously, we evaluated the ratio of the difference in network density of the two conditional networks to its estimated variance. We therefore computed a z-score defined as $Z = \frac{\delta_0 - \delta_1}{sd(\delta_0 - \delta_1)}$, where δ_0 and δ_1 reflect the network density at either previously or not previously exercising, divided by the standard deviation of the differences $sd(\delta_0 - \delta_1)$. The standard deviation is based on δ_0 and δ_1 as estimated de novo for each of $N = 1000$ bootstrapped samples (for details see below). *P*-values for the *z*-statistic were obtained by referencing z against the normal distribution.

The stability of centrality measures and the accuracy of edge-weights were estimated by drawing bootstrapped confidence intervals using the nonparametric bootstrapping function *bootNet* of the modnets package v0.9.0 [49], which is an extension of the *bootnet* package [13] to moderated mlGVAR network models. Stability is assessed with the correlation stability coefficient (CS), which reflects the number of cases that can be dropped from a network model to maintain the correlation between the original measures and those of the bootstrapped subsets at $\rho = 0.70$ or higher with 95 % probability [13]. In line with Epskamp et al. [13], correlation stability should not be lower than $CS = 0.25$ for interpretation.

2.5. Availability of data and code

Analyses were not preregistered. Anonymized data and code to reproduce the network analyses are available at https://osf.io/btzvj/.

3. Results

One hundred and two participants provided in total 6959 assessments (68.2 % of 10,200 planned assessments) that were included in our analysis (Mean assessments per participant: $M = 71.75$, $SD = 24.36$; range: 20–123 assessments).[1] On average, participants reported that they exercised at 9.65 days ($SD = 8.26$ days; range: 0–30 days) resulting in $M = 13.11$ ($SD = 14.64$; range: 0–85) exercise assessments per participant. KPSS tests revealed that 63 % of symptoms were stationary across participants (range 38 % – 79 % per symptom) which increased to 86 % (range 51 % – 96 % per symptom) after detrending univariate symptom time series that followed a deterministic trend. We consider our data to be sufficiently stationary for conducting mlGVAR models after detrending. We then investigated the effects of exercise as a moderator on eating psychopathology networks.

3.1. Between-person moderated networks

Network graphs for between-person networks are displayed in Fig. 1. Correlation stability of edge weights accuracy and expected influence (both $CS = 0.21$) of pairwise edges was marginally lower than the recommendation of CS $\geq$ 0.25 by Epskamp et al. [13], but extremely low for strength of pairwise edges and both edge weights and centrality measures of interactions (all $CS \leq 0.05$). This is likely due to the dense structure of the between network with generally low edge weights. Thus, only expected influence will be carefully interpreted. Fig. 2 displays centrality measures for symptoms in networks with and without exercising (detailed centrality indices are shown in Supplement S2). *Overeat* (expected influence [*EI*] = 2.19) and *bodycheck* (*EI* = 1.01) were the most positively interconnected symptoms when not previously exercising (see Fig. 1A), and *diet* (*EI* = 1.65) and *guilty* (*EI* = 1.04) when previously exercising. The interconnectedness of the overall network decreased with exercising, but for example *overeat* (*EI* = −1.79) switched to being strongly *negatively* interconnected (see Fig. 1B).

In general, the network appears slightly denser for the "exercise not present" conditional network when looking at statistically significant edges only. However, network density based on absolute edge weights was larger when previously exercising (density = 0.685) compared to not exercising (density = 0.178), although the difference was not statistically significant ($z = -0.191$, $p = .849$). When looking at specific edges (see Table 2 for pairwise edge and interaction weights), no significant direct effects of exercise emerged. Strongest positively associated was *feargain – thinner* ($\beta = 0.73$) and strongest negatively associated was *guilty – binge* ($\beta = -0.42$) in the "exercise not present" conditional network; whereas *restrict – vomit* ($\beta = 2.15$) was strongest positively associated and *binge – overeat* ($\beta = -2.11$) was strongest negatively associated in the "exercise present" network. Only four edges persist at both levels of exercise: 1) *binge – overeat*, 2) *guilty – bodycheck*, 3) *diet – thinner*, and 4) *restrict – weigh* (see Fig. 1A and B). Of those, *diet – thinner* was decreased in strength but remained positively associated, whereas *binge – overeat*, *guilty – bodycheck* and *restrict – weigh* switch signs from positive to negative, indicating a significant negative moderation by exercise. Several other edges were statistically significantly moderated such that the resulting pairwise edge only reached statistical significance when participants exercised (*compens – weigh*, *compens – restrict*, *compens – vomit*, *restrict – vomit*, *deserve – bodycheck*, *overeat – feargain*, *guilty – feargain*, and *diet – binge*) or did not exercise previously (*guilty – binge*, *restrict – bodycheck*, and *weigh – binge*). However, these moderation effects have to be interpreted carefully as interaction terms were unstable. Associations between binge eating and other compensating behaviors, vomiting, and body checking as well as between thinking not deserving to eat during the most recent meal and feeling guilty during

[1] Due to technical issues (e.g., smartphone loss or change of assessment id), several participants reported on more days or over a longer assessment period than preplanned: 13 participants provided assessments on 25 days or less within longer range than 52 days; and 10 participants reported on more than 25 days, with six of those completing more than 100 total assessments. We used all available data from all participants.

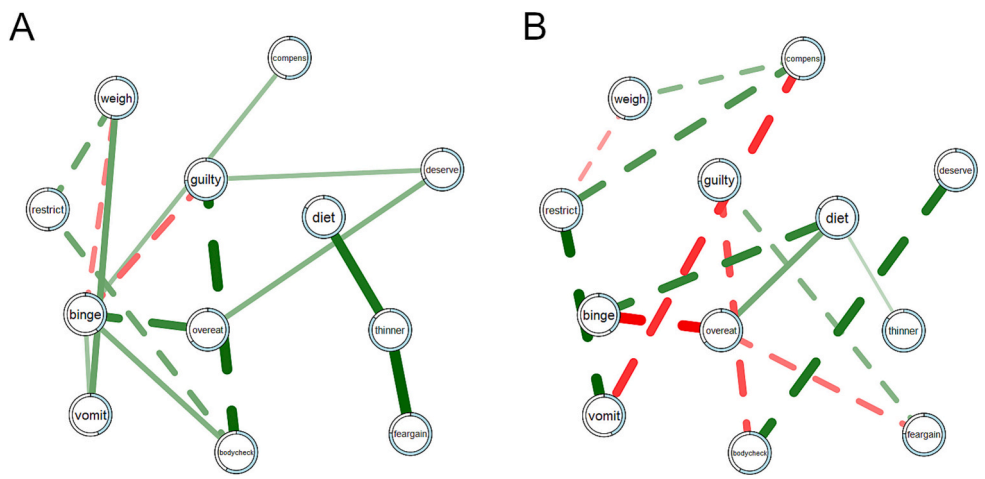

Fig. 1. Network graphs for between-person networks as conditional network graphs A) when not previously exercising and B) when previously exercising. All edges (lines) are significant at $p < .05$. Red lines indicate negative and green lines positive pairwise associations between symptoms. Significant interaction (moderation) effects are depicted as dashed lines. Thickness of lines represents strength of the pairwise association. Pie graphs around nodes (symptoms) reflect R^2 as a measure of prediction error. For descriptions of the symptom abbreviations see Table 1. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

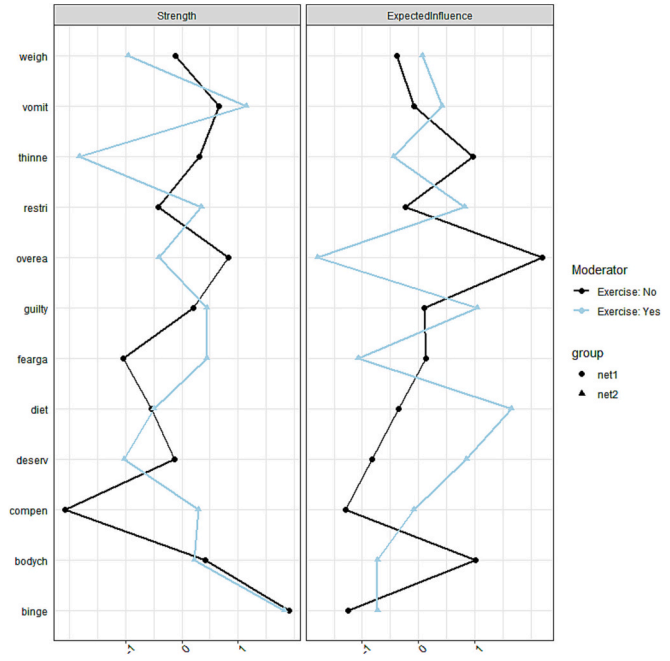

Fig. 2. Centrality plot for the between-person networks. Black line indicates centrality measures of the nodes (symptoms) in the absence of exercise, whereas the blue line indicates centrality measures incorporating interaction effects of exercise. Strength is defined as the absolute sum of all edges (associations) of a given node to all other nodes, whereas expected influence accounts for whether an edge weight is positive or negative and thus is the summed weight of a node's edges shared with remaining nodes. Of note, correlation stability is below the recommended threshold (CS < 0.25) for centrality measures. Therefore, they should be interpreted with caution. For descriptions of the symptom abbreviations see Table 1. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

the most recent meal, thinking not deserving to eat during the most recent meal and overeating, and desire to be thinner and fear of weight gain were all positive when participants did not exercise but did not reach statistical significance when participants were exercising. Only the association between thinking about dieting and overeating was positively associated exclusively when participants were previously exercising.

3.2. Temporal moderated networks

Correlation stability was excellent for edge weights ($CS = 0.75$), good for in-strength (in-S; $CS = 0.52$), out-strength (out-S; $CS = 0.44$), in-expected influence (in-EI; $CS = 0.52$) and out-expected influence (out-EI; $CS = 0.52$) of pairwise edges, and acceptable for edge weights, in- and out-expected influence ($CS \geq 0.28$) but not in- and out-strength ($CS = 0.21$) of interactions. Fig. 3 displays centrality measures of pairwise edges for the temporal networks at both levels of exercise. *Overeat* showed the highest in-S (*in-S* = 1.87), followed by *guilty* (*in-S* = 1.63) which also showed the highest in-EI (*in-EI* = 1.55) when participants were not exercising previously. After exercising, however, only *overeat* maintained high in-S (*in-S* = 1.39), whereas *deserve* and *diet* showed the highest in-EI ($1.14 \leq$ *in-EI* ≤ 1.77), indicating that they now were the symptoms that were mostly influenced by other symptoms. When participants did not exercise previously, *vomit* (*out-S* = 1.89) and *thinner* (*out-S* = 1.29) were the most influential symptoms regarding out-S, but only *thinner* remained influential with positive edges (*out-EI* = 1.50), whereas *vomit* showed mostly negative edges (*out-EI* = −2.23). When previously exercising, *vomit* (*out-S* = 1.17), *guilty* (*out-S* = 1.51) and *deserve* (*out-S* = 1.48) were central nodes regarding out-S, but only *guilty* remained highly influential to other symptoms when controlling for negative edge weights (*out-EI* = 1.87). Detailed centrality indices are shown in Supplement S3, including centrality measures for interactions.

When inspecting statistically significant edges only, the network appeared much denser when participants did not exercise. However, the difference was not statistically significant when comparing absolute edge values (not previously exercising: density = 0.0474; previously exercising: density = 0.0465; $z = 0.108$, $p = .914$). Significant direct effects of exercise were observed on *compens*, *feargain* and *weigh* ($-0.07 \leq \beta \leq -0.06$) (Fig. 4; see Table 3 for all edge weights). At the "no previous exercise" level, all auto-regressive edges were statistically significant and had the highest positive edge weights ($0.13 \leq \beta \leq 0.34$) of all, indicating that from *t-1* to *t0* symptoms tend to increase in severity when participants did *not* exercise previously (see Fig. 5A). The three strongest positive edges were *binge* → *guilty*, *compens* → *guilty*, and *thinner* → *diet* (all $0.11 \leq \beta \leq 0.13$). The strongest negative edges were *vomit* → *binge* ($\beta = -0.18$), *vomit* → *overeat* ($\beta = -0.13$) and *vomit* → *bodycheck* ($\beta = -0.10$).

Table 2
Edge weights for pairwise edges and interactions in the between-person network with exercise as a dichotomous moderator.

A: pairwise edges when exercise is not present

	Binge	Bodycheck	Compens	Deserve	Diet	Feargain	Guilty	Overeat	Restrict	Thinner	Vomit	Weigh
Binge	–											
Bodycheck	**0.35**	–										
Compens	**0.28**	0.08	–									
Deserve	0.01	−0.27	−0.14	–								
Diet	−0.16	0.02	−0.13	−0.21	–							
Feargain	0.20	0.04	−0.04	−0.13	−0.08	–						
Guilty	**−0.42**	**0.70**	0.03	**0.29**	0.12	0.00	–					
Overeat	**0.56**	−0.09	0.07	**0.34**	0.25	0.18	0.19	–				
Restrict	−0.15	**0.40**	0.08	0.18	0.02	0.06	−0.18	0.23	–			
Thinner	−0.14	−0.01	0.05	0.28	**0.65**	**0.73**	−0.04	−0.10	−0.08	–		
Vomit	**0.27**	−0.20	0.29	0.08	0.22	0.11	0.24	−0.26	−0.17	−0.15	–	
Weigh	**−0.35**	0.16	−0.12	0.16	0.07	−0.16	−0.02	0.20	**0.40**	−0.03	**0.42**	–

B: pairwise edges when exercise is present

	Binge	Bodycheck	Compens	Deserve	Diet	Feargain	Guilty	Overeat	Restrict	Thinner	Vomit	Weigh
Binge	–											
Bodycheck	−1.05	–										
Compens	−0.80	0.79	–									
Deserve	0.67	**1.84**	−0.39	–								
Diet	**1.67**	0.69	0.25	0.08	–							
Feargain	−1.16	−0.21	−0.64	0.59	0.75	–						
Guilty	1.71	**−1.45**	1.02	0.78	−1.07	**1.01**	–					
Overeat	**−2.11**	−0.66	−0.44	−0.05	**1.13**	**−1.15**	0.80	–				
Restrict	1.19	−0.91	**1.42**	0.07	0.28	0.67	−0.36	−0.38	–			
Thinner	−0.20	0.06	0.45	−0.70	**0.53**	−0.09	0.24	0.11	−0.72	–		
Vomit	−0.92	0.83	**−1.70**	0.28	0.24	−1.81	0.55	0.50	**2.15**	0.87	–	
Weigh	0.72	−0.23	**0.96**	−0.52	−0.43	1.12	−0.21	0.00	**−0.81**	−0.27	0.87	–

C: interactions

	Exercise × binge	Exercise × bodycheck	Exercise × compens	Exercise × deserve	Exercise × diet	Exercise × feargain	Exercise × guilty	Exercise × overeat	Exercise × restrict	Exercise × thinner	Exercise × vomit	Exercise × weigh
Binge	–											
Bodycheck	−1.40	–										
Compens	−1.08	0.71	–									
Deserve	0.66	**2.11**	−0.25	–								
Diet	**1.84**	0.68	0.38	0.29	–							
Feargain	−1.37	−0.24	−0.60	0.72	0.83	–						
Guilty	**2.12**	**−2.15**	0.99	0.49	−1.18	**1.01**	–					
Overeat	**−2.67**	−0.57	−0.51	−0.39	0.89	**−1.32**	0.61	–				
Restrict	1.34	**−1.31**	**1.35**	−0.11	0.26	0.61	−0.18	−0.62	–			
Thinner	−0.06	0.06	0.40	−0.98	−0.12	−0.82	0.28	0.21	−0.63	–		
Vomit	−1.19	1.03	**−1.99**	0.20	0.02	−1.91	0.31	0.77	**2.32**	1.01	–	
Weigh	**1.07**	−0.38	**1.08**	−0.68	−0.50	1.28	−0.19	−0.20	**−1.21**	−0.24	0.45	–

Note: Edge weights and interaction weights with $p < .05$ are bold. Edge and interaction weights are averaged and thus reflect the average of the two corresponding edges symptom a → symptom b and symptom b → a (or exercise × symptom a → symptom b and exercise × symptom b → symptom a, respectively). Interaction weights (i.e., exercise × symptom) are thus the difference between pairwise edge at "exercise not present" – pairwise edge at "exercise present". For descriptions of the symptom abbreviations of symptoms see Table 1.

When participants were exercising previously (Fig. 5B), autoregressive edges remained mostly stable for *bodycheck*, *diet*, *guilty*, *restrict*, *thinner* and *vomit* ($0.13 \leq \beta \leq 0.34$; all estimates at the not exercising level) with non-significant interaction terms. Only few other edges remained stable, unmoderated, and statistically significant at both levels of exercise: *guilty* → *overeat* ($\beta = 0.10$), *restrict* → *weigh* ($\beta = -0.04$), and *bodycheck* → *weigh* ($\beta = 0.03$; all estimates at the not previously exercising level). Looking at the interaction terms, most of the statistically significant interactions were negative (see Table 4), indicating that exercise had a mostly attenuating effect on pairwise associations of ED symptoms. Autoregressive edges of *binge*, *compens*, *deserve*, *overeat* and *weigh* were significantly attenuated by exercise (interaction terms: $-0.11 \leq \beta \leq -0.29$) and even did not reach statistical significance for *binge*, *overeat* and *weigh* (all $\beta \leq 0.05$, $p \geq .07$) when previously exercising. Exercise also significantly moderated the autoregressive edge of *feargain* which increased after exercise ($\beta = 0.34$; interaction term: $\beta = 0.12$). Exercise also statistically significantly attenuated edges between *binge* → *guilty*, *compens* → *deserve*, *compens* → *guilty*, *compens* → *weigh*, *deserve* → *feargain*, *deserve* → *overeat*, *deserve* → *thinner*, *overeat* → *guilty*, *thinner* → *compens*, *thinner* → *diet*, and *vomit* → *weigh* (interaction term: $-0.20 \leq \beta \leq -0.07$). Of those, only the edge *compens* → *guilty* remained at both levels of exercising, whereas all other edges did not reach statistical significance at either with or without previous exercise. Four statistically significant interactions were positive, indicating that the corresponding edges were increased by exercise (interaction term: $0.07 \leq \beta \leq 0.18$). However, only three of the corresponding edges appeared either when or when not previously exercising (*guilty* → *feargain*, *vomit* → *binge and vomit* → *compens*), whereas the edge *restrict* → *deserve* did

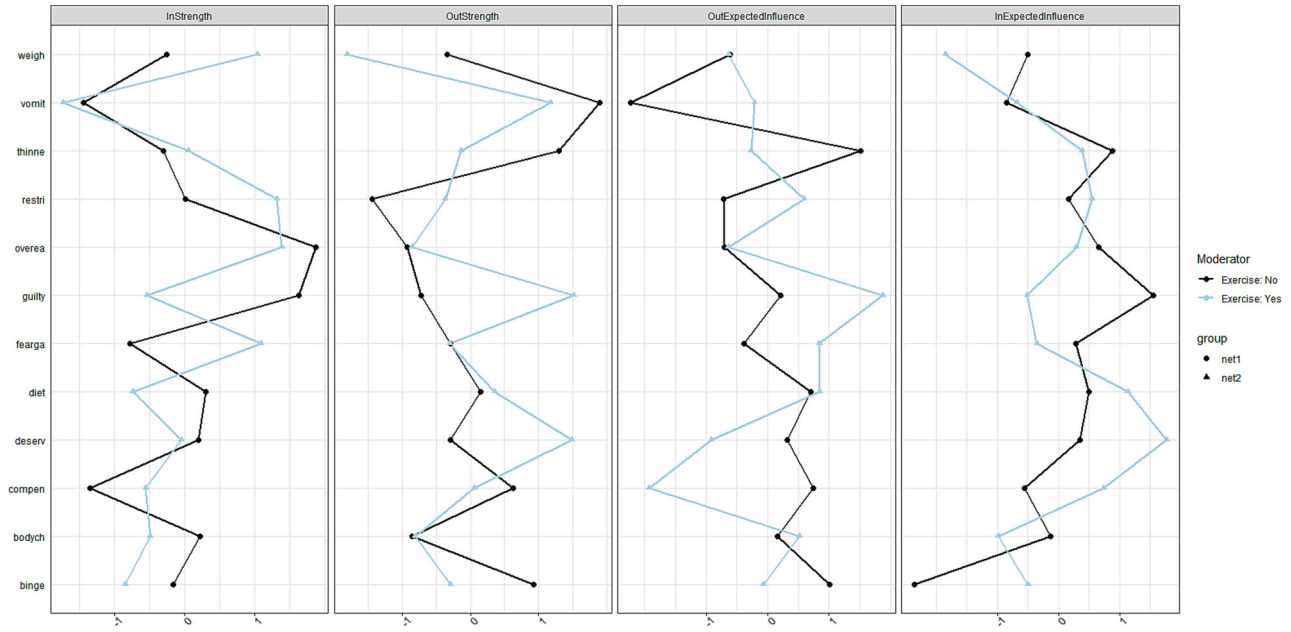

Fig. 3. Centrality plot for the temporal networks. Black line indicates centrality measures of the nodes (symptoms) when not previously exercising, whereas the blue line indicates centrality measures when participants previously exercised. In-strength is the absolute weight of all edges (associations between symptoms) connecting to this node from all other nodes, whereas out-strength reflects the absolute weight of all edges connecting this node to all other nodes. Similarly, in-expected influence and out-expected influence are the summed weights (i.e., accounting for positive/negative edges) of all edges connecting to this node from all other nodes or from this node to all other nodes, respectively. For descriptions of the symptom abbreviations see Table 1. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

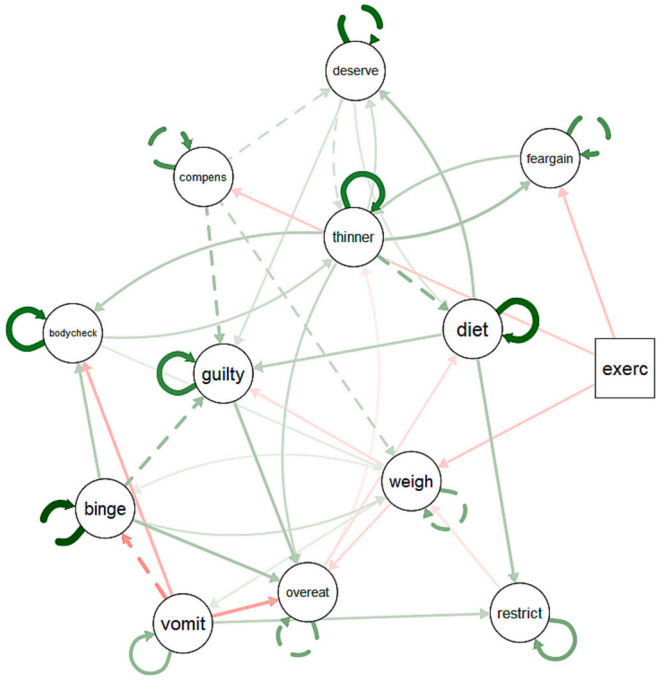

Fig. 4. Network graph for the temporal network including direct effects of exercise.
All edges (lines) are significant at $p < .05$. Red lines indicate negative and green lines positive pairwise associations between symptoms. Edges are directed and depict associations from symptom *a* at *t-1* to symptom *b* at *t0*. Significant interaction (moderation) effects are depicted as dashed lines. Thickness of lines represents strength of the pairwise association. Pie graphs around nodes (symptoms) reflect R^2 as a measure of prediction error. For descriptions of the symptom abbreviations see Table 1. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

not reach statistical significance at all (*ps* $\geq$ 0.096).[2]

4. Discussion

We used moderated network analyses to investigate whether associations between ED symptoms differ when individuals previously exercised compared to when they did not, in individuals with EDs that provided multiple daily assessments over several weeks. We analyzed both average between-person and temporal within-person associations between symptoms. Several differences between between-person and within-person networks were observed.

4.1. Between-person moderated networks

Exercising had no direct average effects in the between-person network. Surprisingly, the network was less dense when not exercising compared to exercising, although this difference was not statistically significant. Conditional between-person networks varied substantially when participants previously exercised compared to when they did not. Previously exercising mostly attenuated average associations between other ED symptoms. However, these findings must be interpreted carefully as both interaction terms and pairwise edge weights did not show sufficient correlation stability, indicating substantial variation in individual networks. Only the association between desire to be thinner and thinking about dieting remained stable across both conditional networks.

[2] In Levinson et al. [40], exercise was additionally assessed as "Engaged in excessive exercise since the last check in" ranging from 1 – not at all to 6 – extremely. However, the wording of the item made it less clear whether *intent* to exercise or *actually* exercising was the main focus, and participants were not given a definition of what "excessive" means in this context. This resulted in only small overlap with the dichotomous exercise item and less stable centrality measures when using this item as a moderator. For the sake of completeness, results are reported in Supplement 4.

Table 3
Edge weights for pairwise edges in the temporal network with exercise as a dichotomous moderator.

A: Pairwise edges when exercise is not present

	Intercept	Direct effect of exercise	Binge	Bodycheck	Compens	Deserve	Diet	Feargain	Guilty	Overeat	Restrict	Thinner	Vomit	Weigh
Binge	0.01	−0.04	**0.34**	0.01	−0.02	0.02	−0.02	−0.02	0.01	0.02	0.00	0.01	**−0.15**	**0.03**
Bodycheck	0.00	−0.06	**0.09**	**0.28**	−0.01	0.00	0.02	−0.01	0.00	−0.01	0.00	**0.09**	**−0.10**	0.04
Compens	**0.02**	**−0.06**	0.01	0.01	**0.22**	0.03	0.00	0.01	0.00	0.00	0.00	0.03	−0.04	0.02
Deserve	0.02	0.02	0.01	−0.01	**0.06**	**0.29**	**0.09**	−0.04	0.02	0.02	−0.02	**0.05**	−0.03	−0.02
Diet	0.02	−0.01	0.02	0.03	0.03	**0.04**	**0.31**	−0.03	0.02	**−0.04**	0.00	**0.13**	0.00	−0.03
Feargain	**0.03**	**−0.07**	0.01	0.02	0.03	0.01	0.00	**0.22**	0.02	−0.01	0.00	**0.10**	−0.03	0.00
Guilty	**0.05**	−0.07	**0.12**	0.00	**0.11**	**0.05**	**0.08**	−0.04	**0.24**	0.01	0.01	0.04	−0.06	**−0.05**
Overeat	**0.08**	−0.06	**0.10**	−0.01	0.06	0.03	0.01	0.03	**0.10**	**0.17**	−0.03	**0.07**	**−0.13**	**−0.05**
Restrict	−0.01	−0.05	−0.04	0.01	−0.01	0.02	**0.10**	0.03	−0.02	−0.03	**0.17**	0.00	**0.08**	0.00
Thinner	**0.03**	−0.03	−0.01	**0.06**	0.02	**0.05**	0.02	**0.08**	0.01	**−0.03**	0.00	**0.26**	0.00	−0.01
Vomit	0.01	−0.04	0.02	0.01	−0.03	−0.01	0.01	0.00	0.01	0.00	0.00	−0.01	**0.13**	**0.03**
Weigh	**−0.04**	**−0.07**	**0.05**	**0.03**	**0.07**	−0.03	−0.01	0.00	0.00	0.02	**−0.04**	0.01	−0.03	**0.16**

B: pairwise edges when exercise is present

	Intercept	Direct effect Of exercise	Binge	Bodycheck	Compens	Deserve	Diet	Feargain	Guilty	Overeat	Restrict	Thinner	Vomit	Weigh
Binge	0.01	−0.04	0.05	−0.03	−0.02	−0.04	−0.01	0.01	0.05	−0.02	0.03	−0.03	0.03	0.02
Bodycheck	0.00	−0.06	0.04	**0.29**	−0.06	−0.03	−0.08	0.02	0.00	0.02	0.04	−0.01	−0.01	−0.01
Compens	**0.02**	**−0.06**	0.02	−0.03	**0.11**	0.01	−0.01	0.04	0.00	0.00	0.00	−0.05	**0.14**	0.01
Deserve	0.02	0.02	0.05	0.00	−0.03	**0.14**	**0.09**	−0.02	**0.10**	0.02	0.04	0.00	0.01	0.00
Diet	0.02	−0.01	0.04	−0.01	−0.03	0.03	**0.24**	0.07	0.03	−0.01	0.03	0.00	0.03	−0.01
Feargain	**0.03**	**−0.07**	0.04	0.00	−0.04	**−0.07**	−0.01	**0.34**	**0.11**	**−0.08**	0.03	0.06	0.02	−0.05
Guilty	**0.05**	−0.07	−0.07	0.01	**−0.08**	0.01	0.02	0.02	**0.25**	0.00	0.06	0.03	0.00	−0.01
Overeat	**0.08**	−0.06	0.00	0.04	0.00	**−0.12**	0.03	0.03	**0.12**	0.04	0.02	0.06	−0.07	−0.03
Restrict	−0.01	−0.05	−0.02	0.04	−0.04	**0.13**	0.02	0.05	−0.07	0.06	**0.20**	−0.08	0.02	−0.01
Thinner	**0.03**	−0.03	−0.03	0.03	0.02	−0.06	**0.10**	−0.01	**0.08**	−0.03	−0.01	**0.26**	0.01	−0.01
Vomit	0.01	−0.04	−0.04	0.01	0.00	−0.02	0.02	0.03	0.00	−0.01	−0.02	−0.03	**0.21**	0.01
Weigh	**−0.04**	**−0.07**	0.01	**0.08**	**−0.05**	0.02	0.03	−0.04	0.00	−0.02	**−0.06**	0.02	**−0.17**	0.05

Note: Edge weights with $p < .05$ are bold. Edges are directed; therefore, columns represent predictors and rows outcomes. For descriptions of the symptom abbreviations of symptoms see Table 1.

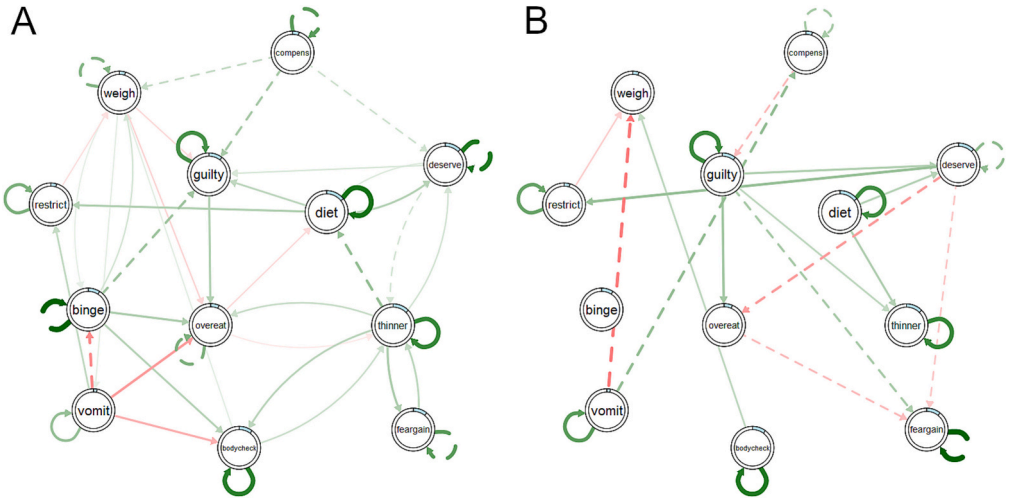

Fig. 5. Conditional network graphs when A) previously not exercising and B) when previously exercising. All edges (lines) are significant at $p < .05$. Red lines indicate negative and green lines positive pairwise associations between symptoms. Edges are directed and depict associations from symptom *a* at *t-1* to symptom *b* at *t0*. Significant interaction (moderation) effects are depicted as dashed lines. Thickness of lines represents strength of the pairwise association. Pie graphs around nodes (symptoms) reflect R^2 as a measure of prediction error. For descriptions of the symptom abbreviations see Table 1. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

4.2. Temporal moderated networks

The temporal within-person network was denser and more interconnected when participants were not exercising previously, although the difference to when exercising was not statistically significant. This indicates that exercise might reduce short-term symptom spread across the network, but the effect was small and most likely not clinically relevant. Previously exercising directly decreased levels of other compensatory behaviors, weighing oneself and fear of weight gain at the subsequent time point. Most likely, participants perceived exercising as a weight control behavior such that fear of weight gain decreased and other weight control behaviors seemed unnecessary. Surprisingly, experiencing fear of weight gain prior to exercising was more likely leading to higher levels of fear of weight gain following exercise, whereas many other symptoms' increases over time were attenuated when previously exercising, as autoregressive effects decreased.

Table 4
Edge weights for interactions in the temporal network with exercise as a dichotomous moderator.

	Exercise × binge	Exercise × bodycheck	Exercise × compens	Exercise × deserve	Exercise × diet	Exercise × feargain	Exercise × guilty	Exercise × overeat	Exercise × restrict	Exercise × thinner	Exercise × vomit	Exercise × weigh
Binge	**−0.29**	−0.03	−0.01	−0.07	0.00	0.03	0.04	−0.03	0.03	−0.04	**0.18**	−0.01
Bodycheck	−0.05	0.01	−0.05	−0.03	−0.10	0.03	0.00	0.04	0.04	−0.09	0.09	−0.05
Compens	0.01	−0.05	**−0.11**	−0.01	−0.01	0.03	0.00	0.00	0.00	**−0.07**	**0.17**	−0.01
Deserve	0.03	0.00	**−0.10**	**−0.15**	0.00	0.02	0.07	0.00	**0.07**	−0.05	0.04	0.01
Diet	0.02	−0.03	−0.06	−0.01	−0.06	0.09	0.01	0.03	0.03	**−0.13**	0.03	0.02
Feargain	0.03	−0.01	−0.07	**−0.08**	−0.01	**0.12**	**0.09**	**−0.07**	0.02	−0.04	0.05	−0.05
Guilty	**−0.20**	0.01	**−0.19**	−0.04	−0.06	0.06	0.01	−0.01	0.05	−0.01	0.06	0.04
Overeat	−0.10	0.05	−0.06	**−0.15**	0.03	0.01	0.02	**−0.13**	0.04	−0.01	0.05	0.02
Restrict	0.02	0.02	−0.03	0.11	−0.08	0.02	−0.05	0.09	0.03	−0.08	−0.05	0.00
Thinner	−0.02	−0.03	0.00	**−0.11**	0.07	−0.09	0.07	−0.01	−0.01	0.00	0.01	0.01
Vomit	−0.06	0.00	0.02	−0.01	0.01	0.03	−0.01	−0.01	−0.02	−0.02	0.08	−0.02
Weigh	−0.04	0.04	**−0.12**	0.05	0.04	−0.04	0.00	−0.04	−0.02	0.01	**−0.14**	**−0.11**

Note: Interaction weights that reach significance with $p < .05$ are presented in bold. Interactions are directed; therefore, columns represent predictors (i.e., exercise × symptom) and rows outcomes. Interactions are the difference between pairwise edge at "exercise not present" – pairwise edge at "exercise present". For descriptions of the symptom abbreviations of symptoms see Table 1.

Generally, these autoregressive effects were strong, indicating that when not previously exercising, symptoms tend to worsen over four-hour intervals during daytime. Exercise may serve as a short-term circuit-breaker that interrupts symptom deterioration for short periods, which is in line with other research indicating that maladaptive exercise may be maintained via negative reinforcement [24]. Exercise more strongly downregulates positive temporal associations involving behavioral symptoms, indicating that exercise might replace other behaviors appearing as less adequate in a given moment. For example, it might be more socially acceptable for individuals with EDs to exercise after overeating at a work lunch than vomiting at the workplace. This idea is in line with a study finding that symptom-shifting frequently occurs in individuals with EDs [19]. For example, other compensatory behaviors were increasing thinking not deserving to eat, weighing oneself and feeling guilty when not exercising. These associations were either completely attenuated or negative when participants were exercising previously, as well as the effect of binge-eating on feeling guilty. In addition, negative effects of vomiting on weighing oneself, and thinking not deserving to eat as well as overeating on fear of weight gain emerged when participants were previously exercising. Altogether, the disappearance of many positive effects that were increasing other symptoms over time and the emergence of other negative associations between symptoms after previous exercise underline the attenuating effect of exercise on ED symptoms and their inter-relations within few hours. Our finding that binge eating did not show significant associations with any symptom after exercising may indicate that at least for some individuals or situations exercise may buffer detrimental effects of later binge eating. Individuals with EDs may interpret having exercised as a carte blanche to binge eating without feeling guilty at the next meal or body checking afterwards, as excess calorie intake during the binge eating episode has already been "pre-burned".

4.3. Implications for research and clinical practice

Our study has several implications for research and clinical practice. We examined short-term effects of exercise on associations between ED symptoms and found important changes in symptom associations at different exercise levels, though overall network density did not change significantly. Since we only investigated short-term effects over four hours, more global effects (e.g., changes in network density) might be less stable over longer periods than few minutes to an hour and stronger for changes of associations between general psychological symptoms (e.g., affect), which we did not study. Future research should explore changes in network density from before to after exercising with shorter intervals (e.g., hourly) and investigate how ED symptom networks change after other ED behaviors like vomiting or binge-eating. The moderated networks framework used in our study could also be applied to assess short-term treatment effects on symptom networks, especially of just-in-time adaptive interventions, or effects of current weight on day-level symptom networks. Additionally, investigating whether exercise moderates symptom networks on an individual level may be promising and could provide insights into idiographic maintenance mechanisms of driven exercise [33], especially when using objective measures of exercise intensity such as heart rate or actiometric data. Finally, experimental studies should test potential moderating effects of exercise on symptom associations, such as whether effects of exercise on the link between overeating and fear of weight gain are dose-dependent [22]. Clinicians should emphasize both the long-term negative but also the short-term positive effects of exercise on ED symptoms, fostering better understanding and adherence to exercise limitations during treatment.

4.4. Strengths and limitations

Several strengths of our study have to be noted. To our knowledge, it is the first study that models exercise effects on several ED symptoms and their temporal associations simultaneously. Using a large intensive longitudinal dataset enabled us to obtain mostly stable estimates, especially for the temporal network. In addition, it is, to our knowledge, the first study to statistically compare network density of two networks using a z-statistic test. A limitation is that we did not disentangle adaptive and maladaptive exercise in our study. Future studies should therefore directly ask participants to indicate whether they were currently engaging in adaptive or maladaptive exercise, as previous EMA research indicates that participants were able to reliably differentiate these, albeit most episodes were described as maladaptive [26]. Our participants predominantly identified as female and white, limiting generalizability to male individuals or more diverse samples. Unfortunately, centrality measures for between-person networks were unstable for most measures, potentially due to including many symptoms in our network models. Heterogeneity in endorsement of symptoms was high, which prevented us from estimating idiographic networks, as current statistical procedures require ratings of at least three different levels of each symptom of each individual to compute idiographic networks. As our study is a secondary analysis of data from Levinson et al. [33], the intervals between repeated measurements might not be optimal for capturing exercise effects on ED symptoms, and future studies might consider shorter intervals. Different to Levinson et al. [33], we did not use multiple imputation for dealing with missing data as there are currently no validated procedures available for moderated network analysis regarding the pooling of parameter estimates resulting from separate analyses on multiple imputed datasets [51]. This might result in

slightly biased parameter estimates, especially when missingness did not occur completely at random (e.g., beeps during exercise were missed more often), or in the lack of detecting all significant edges due to lower power for small associations. However, given that more than two thirds of all planned assessments were recorded and participants responded with a minimum of 20 assessments, our dataset is still sufficiently large for network analysis. Finally, the statistical method that we used to estimate moderated network models is fairly new and therefore comes with inherent limitations, such as that currently no work has been conducted on how to best interpret centrality measures for interactions.

5. Conclusion

We found that exercise attenuates the increase from one time point to the next for several symptoms, including binge eating, compensatory behaviors, deserving to eat, and overeating. Several temporal associations between symptoms were also attenuated when previously exercising, including binge eating on feeling guilty after the most recent meal, and vomiting and other compensatory behaviors on weighing oneself. Our findings highlight that exercise might serve as a short-term remedy for symptoms in individuals with EDs, and that this attenuating effect is not limited to direct effects but also extends to more complex relations between symptoms. Given that exercise mostly affects behavioral ED symptoms and their associations, it may function as a replacement for other more severe ED behaviors. Our findings further underline the need for both experimental and EMA studies that investigate physiological mechanisms of exercise in EDs to then inform more precise treatments targeting the detrimental long-term effects of maladaptive exercise.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.comppsych.2024.152543.

Author contributions

Conceptualization: DRK; Methodology: DRK, TS; Formal Analysis: DRK; Investigation: CAL, CRN; Resources: CAL, CRN; Data Curation: CAL, CRN, DRK; Writing – Original Draft: DRK; Writing – Review & Editing: CAL, CRN, DRK, TS; Project Administration: CAL, DRK.

CRediT authorship contribution statement

David R. Kolar: Writing – review & editing, Writing – original draft, Project administration, Methodology, Formal analysis, Data curation, Conceptualization. **Christina Ralph-Nearman:** Writing – review & editing, Resources, Investigation, Data curation. **Trevor Swanson:** Writing – review & editing, Methodology. **Cheri A. Levinson:** Writing – review & editing, Resources, Project administration, Investigation, Data curation.

Declaration of competing interest

Authors have no conflicts of interest to declare.

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