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Modeling alcohol use disorder as a set of interconnected symptoms – Assessing differences between clinical and population samples and across external factors

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ABSTRACT

Alcohol use disorder is argued to be a highly complex disorder influenced by a multitude of factors on different levels. Common research approaches fail to capture this breadth of interconnecting symptoms. To address this gap in theoretical assumptions and methodological approaches, we used a network analysis to assess the interplay of alcohol use disorder symptoms. We applied the exploratory analysis to two US-datasets, a population sample with 23,591 individuals and a clinical sample with 483 individuals seeking treatment for alcohol use disorder. Using a Bayesian framework, we first investigated differences between the clinical and population sample looking at the symptom interactions and underlying structure space. In the population sample the time spent drinking alcohol was most strongly connected, whereas in the clinical sample loss of control showed most connections. Furthermore, the clinical sample demonstrated less connections, however, estimates were too unstable to conclude the sparsity of the network. Second, for the population sample we assessed whether the network was measurement invariant across external factors like age, gender, ethnicity and income. The network differed across all factors, especially for age subgroups, indicating that subgroup specific networks should be considered when deriving implications for theory building or intervention planning. Our findings corroborate known theories of alcohol use disorder stating loss of control as a central symptom in alcohol dependent individuals.

1. Introduction

Alcohol use disorder (AUD) belongs to the most common but at the same time most untreated mental health disorders (Rehm et al., 2015). Affected individuals struggle to control their alcohol consumption despite detrimental effects on their physical and/or mental health and that of their close social circle (Connor et al., 2016; Rehm et al., 2009). Difficulty in studying and treating addictive behaviors lies in its complex nature; like other mental health disorders, AUD is influenced by a multitude of factors on different social, psychological and biological levels. Common approaches of assessing clinical disorders are unable to capture this multitude of interconnected, highly dependent symptoms (Fried & Nesse, 2015).

Therefore, research in the last decade suggests to model psychological disorders as a set of interacting entities (Kendler, 2016). This direction was introduced in the field of clinical psychology as the network theory of mental disorders (Borsboom, 2017; Cramer et al., 2016; Robinaugh et al., 2020) and has since gotten increased attention in research (e.g., Burger et al., 2020; Elliott et al., 2020; Fried et al., 2015; Heeren et al., 2018; Hoorelbeke et al., 2016; Isvoranu et al., 2016). The network theory models mental disorders as entities (e.g., symptoms) that interact and causally influence each other (Robinaugh et al., 2020). For example, for depression, rumination could lead to difficulty sleeping which in turn leads to difficulties concentrating. In comparison, previous research lines view mental disorders as symptoms loading on a common underlying factor without considering the interaction between those symptoms. Despite the ample research in this area, there is little evidence to support the theory of a common underlying

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factor while the significance of interaction between symptoms has become more apparent (van Bork et al., 2017).

1.1. The network perspective to alcohol use disorder

Researchers have also applied the network theory to substance use disorder, leading to several interesting findings. First, symptom interactions of substance use disorder depend on the drug used, indicating that underlying mechanisms of the same disorder differ heavily between drugs (Baggio et al., 2018). For all drugs, the symptoms were densely connected, however, the strength of connection depended on the drug used (Rhemtulla et al., 2016). Second, the most central node was commonly 'used more/longer than planned' across all drugs, which is an indication of loss of control, fundamental in theories on addiction (e.g., Goldstein & Volkow, 2011). Third, networks based on longitudinal data suggest that fundamental symptoms like compulsion or withdrawal occur primary, while symptoms related to giving up important activities may indicate advanced AUD (Conlin et al., 2021). Fourth, AUD has been assessed together with internalizing disorders, like depression. In a population sample, few interactions were found between substance use and depression symptoms (Wasil et al., 2019), indicating that both disorders are independent. Anker et al. (2017) further suggested that both disorders are dependent through the connection of the symptom drinking-to-cope. Thus far, to the best of our knowledge all network analyses on AUD have been conducted on healthy population level data, limiting ecological validity for AUD patients in treatment, who mostly have severe AUD.

1.2. The current study

In this paper we aim to provide a better understanding of AUD as a complex disorder, focusing on two aspects. In the first part of the paper, we explore differences between clinical individuals and the general population. This is as far as we know, the first study assessing a clinical sample on AUD in a network framework. Here, we aim to shed light on two questions. First, we analyse whether according to postulations of the network theory the clinical subgroup shows a denser network compared to the general population (Borsboom, 2017). To answer this question, we adapt a Bayesian framework as it quantifies the evidence of edge absence and therefore network sparsity (Marsman et al., 2020; Williams et al., 2021). Second, we assess which symptoms are central in each sample's network. While for the clinical sample a central symptom could be considered a treatment target, in the population sample, it could be a target of preventive action.

In the second part of the paper, we assess measurement invariance of the symptom interactions of the population sample across several factors. Investigating and modeling measurement invariance leads to stable parameter estimates that are key for theory development and treatment design (Breslau et al., 2008; Kapur et al., 2012; van de Schoot et al., 2015). We focus on individual factors that are related to mental health disorders, specifically: gender (e.g., McHugh et al., 2018), age (e.g., Lemstra et al., 2008; Phillips et al., 2017), ethnicity (e.g., Brewer et al., 1998; Grucza et al., 2018; Lansky et al., 2014), and income (e.g., Phillips et al., 2017; Richardson et al., 2013).

Given the limited knowledge regarding our analyses, we refrain from generating hypotheses and solely perform exploratory analyses. Exploring the symptom-level interconnections is essential to further spark ideas for confirmatory research.

2. Method

2.1. Participants

We analysed datasets from two different sources to investigate both a population and a clinical sample.

2.1.1. Population sample

The population-based survey compromises the National Survey of Drug Use and Health (NSDUH) data, a US-nationwide survey that collects information on prevalence and correlates of drug use. Participants were surveyed by trained staff using supportive computer assisted interviewing (i.e., for details see United States Department of Health and Human Services, 2016). We utilized the dataset from 2006 to keep external influences comparable to the clinical sample. Networks estimated on the following years do not differ from the chosen year and are included in the online appendix in Fig. S1. We excluded participants who never had a drink ($N = 15{,}114$) or drinks on less than six occasions in the recent year ($N = 12{,}288$), as they did not answer the questions included in our analysis. The final sample size contains 23,591 participants. Of the sample roughly 50 % reported no AUD symptoms at all, whereas 27% indicated at least two symptoms to be present, indicative of a mild AUD and 6.5 % reported four or more symptoms, indicative of a moderate AUD. The sample consists of 48 % female participants ($N = 12{,}105$) and includes individuals of mixed ethnic background (African American: $N = 2{,}443$, 10.36 %; Hispanic: $N = 2{,}985$, 12.65 %; White: $N = 16{,}582$, 70.29 %; Other: $N = 1{,}581$, 6.70 %).

2.1.2. Clinical sample

We utilized data from the Addiction Health Evaluation and Disease (AHEAD) management study conducted in Boston between 2006 and 2010. The study assessed adults above the age of 18 years with a current diagnosis of drug or alcohol dependence. The AHEAD study aimed to evaluate the chronic disease management program, aiming to facilitate collaboration between various professionals involved in treatment (i.e., for details see Saitz & Samet, 2017). In total 563 participants were included in the baseline study and randomly assigned to treatment groups, followed up after 3, 6, and 12 months. We here only use the baseline data and include participants who were treated for AUD solely, as Rhemtulla et al. (2016) showed that networks differ greatly between various drugs used, thus, should be analysed separately. The final sample size consists of 483 participants of which 75 % were male ($N = 358$) and from different ethnic backgrounds (American Indian or Alaska Native: $N = 14$, 2.30%; Asian: $N = 2$, 0.41%; Black or African American: $N = 171$, 35.40%; Native Hawaiian or other Pacific Islander: $N = 1$, 0.02%; White: $N = 220$, 45.55%; Other: $N = 75$, 15.53%).

2.2. Outcome measures

AUD during the past 12 months was assessed through 21 questions in the population sample. The questionnaire was developed by the NSDUH for use in their yearly survey. In the clinical sample, AUD in the past 12 months was assessed through the CIDI-SF for alcohol dependence, a short form questionnaire consisting of eight questions including an initial screening question (Kessler et al., 1998). Both questionnaires ask participants to affirm or negate questions on symptom presence (i.e., binary response, 'yes' and 'no'). Including the same symptoms across the population and clinical sample is a prerequisite to contrast both subgroups. Therefore, we compared questions in both samples to criteria specified in the DSM (American Psychiatric Association, 2013) and decided on variables that were most similar and capture important features of AUD. Our final network consisted of six variables including *time spent*, *tolerance*, *loss of control*, *emotional problems*, *work problems*, and *dangerous activities*. A table with the exact phrasing of the symptom question for each subgroup can be found in Table 1 in the supplementary material.

External factors relevant for our second subgoal, were all assessed through single item questions. Age and income were both administered as categorical variables.

2.3. Statistical analysis

All analyses were conducted in R (R Core Team, 2020) and respective

code can be found in the OSF project repository.[1]

2.3.1. Comparison population and clinical sample

To address our first question, we estimated an Ising model (i.e., a binary network model) separately for each sample and compared strength of connection as well as the underlying topological structures. To estimate each model, we used a recently developed Bayesian approach implementing a shrinkage prior to reduce weak links in the network to zero (Marsman et al., 2020). A major advantage of this Bayesian approach over the classical lasso approach is the ability to express the uncertainty of the estimated structure and of edge in- or exclusion.

We followed the procedure detailed in Marsman et al. (2020), using its respective R-package *rbinnet* based on a Gibbs-variable selection approach (George & McCulloch, 1993). A continuous spike and slab mixture of a zero-centered normal distribution was stipulated on the interactions in the network. The spike and slab components' variances are set by matching their intersection to an approximate credible interval about zero. The width of this interval can be adapted through a precision parameter. The results for a precision of 0.975 are reported here, and for 0.997 in the supplementary material. Furthermore, a standard normal prior for the thresholds and a uniform prior on the structures were used.

Additionally, the strength centrality of each node in both populations was calculated, where strength centrality is the summed absolute strength of a node's links with all other nodes in the network.

2.3.2. Measurement invariance of external factors

There are two common ways to assess whether parameters differ between subgroups. First, variables can be included in the network and the resulting edges with symptoms interpreted. Second, the data is split according to the variable of interest (e.g., for gender into "Female" and "Male or Other") and resulting networks assessed for significant differences in parameter estimates. We were interested, whether interactions differ between subgroups, not how external factors interact within the network. Therefore, we chose the second approach and ran a structural change test - a score-based test for measurement invariance (e.g., Hjort & Koning, 2002; Merkle & Zeileis, 2013). This method assesses whether parameters differ between subgroups by testing for structural differences of the scores (i.e., partial derivative of the log-likelihood with respect to a parameter). It was recently developed for Gaussian graphical models (Jones et al., 2020), but here we report the results for a novel application to Ising models using a Monte-Carlo permutation test approach (Huth et al., 2020). We assessed structural changes instantiated by gender, age, ethnicity, and income. Since the estimates of small subgroups are unreliable, we aggregated subgroups with fewer than 600 observations into a single group called 'Other'. This only occurred for ethnicity. Because of too few observations, we did not assess the subgroup differences for the clinical sample. To account for the various tests we ran, we controlled the family wise error rate through Bonferroni correction. We adjusted the critical value α (i.e., $\alpha = 0.05$) by the number of tests run, resulting in a significance threshold of $\alpha = 0.0125$. Lastly, we calculated the parameter estimates and their respective 95 % confidence interval for each subgroup.

3. Results

3.1. Comparison population and clinical sample

Graphical results of the network analysis and strength centrality are shown in Fig. 1. In the population sample, prevalence rates range from 5% to 25%, with *work problems* being endorsed the least and *loss of control* the most. The median probability model (i.e., see Fig. 1a) is densely connected; all 15 possible interactions between symptoms are present. Strongest associations were found between *time spent* on the one hand and *tolerance* and *dangerous activities* on the other, and between *work problems* on the one hand and *emotional problems* and *dangerous activities* on the other. The weakest association were found between *loss of control* and all other nodes. Observe that all established connections are positive. The data provide full support for all of the network's edges. In the population network, *time spent* had the highest strength centrality and *loss of control* the lowest.

For the clinical sample, prevalence rates of symptoms range from 64% to 90%; *setting limits* being endorsed the most, *dangerous activities* the least. The median probability is depicted in Fig. 1b. *Loss of control* is most strongly connected within the network, with the strongest interaction to *dangerous activities* and *tolerance*. Notably, the network is sparser including 9 out of 15 possible links. However, as seen in Fig. 1d, we only observe sufficient evidence (i.e., exclusion Bayes Factor > 10) for excluding the interaction between *dangerous activities* to *time spent*. Even though the median probability model excludes a variety of further links, there is insufficient evidence to conclude their exclusion (i.e., grey edges; exclusion Bayes Factor < 10). In total we can only conclude on the presence or absence of seven edges and are therefore very uncertain about the true sparsity of the network structure. With a prior precision of 0.997, we find an even sparser network for the clinical sample and overall less evidence for inclusion and exclusion of edges (see Fig. S3 in the supplementary material).

In comparison, the clinical network appears to be sparser than the population network, even though for most of the network's links there was little evidence to draw conclusions about the absence. Nonetheless, the two samples show different underlying topologies. Strong links found in the population network (e.g., *time spent* to *dangerous activities*) were not supported in the clinical population. Vice versa, the link between *loss of control* and *dangerous activities* was very pronounced in the clinical network, however, very weak in the population data.[2] A striking difference was found for the strength centrality of *loss of control*: being most central in the clinical sample, it was least central in the population sample (see Fig. 1e). Here, the *time spent* was most strongly connected.

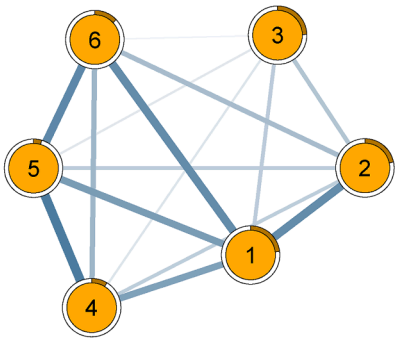
Naturally, the question arises how the network of severe individuals from the population sample compares against the clinically diagnosed sample. To identify severe individuals, researchers commonly need to use a cut-off of the symptom sum-score (e.g., individuals with four or more active symptoms). However, this sum-score filtering might introduce biased edge estimates - the network would suppress or induce negative edges (de Ron et al., 2021; Haslbeck et al., 2020). In an additional analysis we estimated a network based on individuals that would be diagnosed with alcohol use disorder using a sum-score cut-off. Indeed, the network consists of implausible, negative edges (see Figure S4 in the supplementary material). Therefore, while this comparison would be of interest, it is unwarranted through biased estimates.

3.2. Measurement invariance of external factors

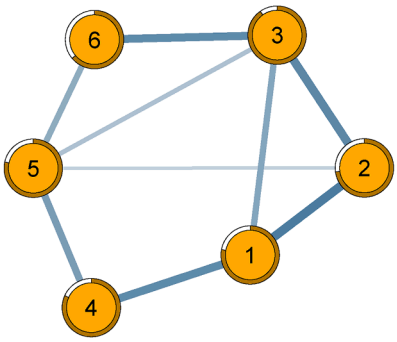
The structural change test of the population sample was significant for all external factors, for all $p < .001$. Thus, the AUD network is measurement non-invariant with regards to all external factors. The variation in edge estimates for each external factor is illustrated in Fig. 2. Most variation between edge weights can be found for the external factor age; for gender the edge weights differ only slightly. For age subgroups the edges from *work problems* on the one hand to *time spent*, *emotional problems*, and *dangerous activities* on the other hand show most variation. The variation on the edge *work problems* to *emotional problems* is similarly found for ethnic subgroups. Furthermore, the variation for

[1] The analysis code can be found in the OSF project repository: https://osf.io/gm6eh/.

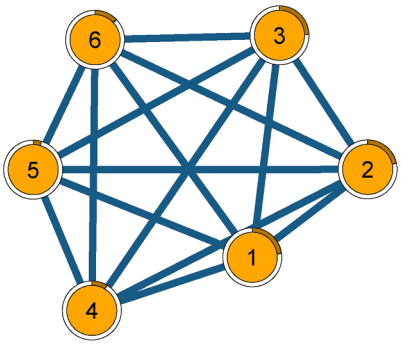
[2] Highest density intervals for all interaction parameters can be found in Fig. S2 in the supplementary material.



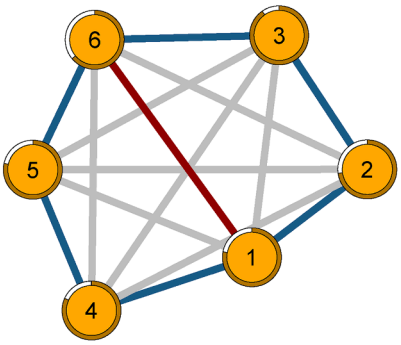
(a) *Median Probability Structure - Population*



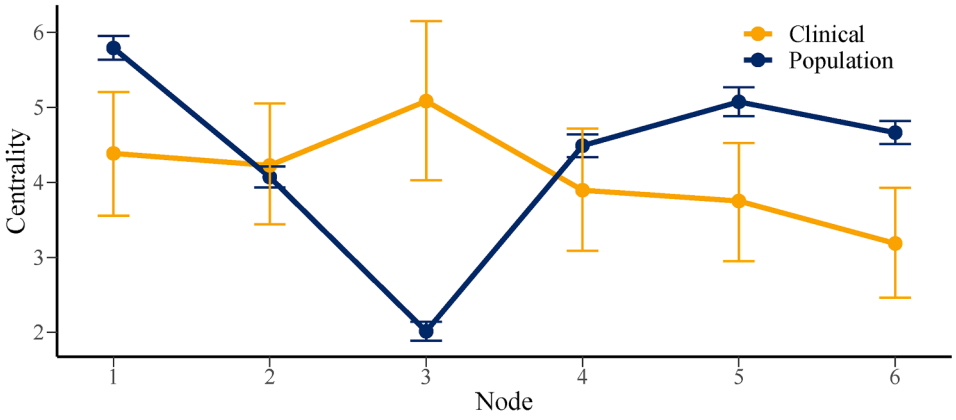
(b) *Median Probability Structure - Clinical*



(c) *Inclusion Bayes Factor - Population*



(d) *Inclusion Bayes Factor - Clinical*



(e) *Strength Centrality*

Fig. 1. Comparison of the population and clinical sample. 1 – Time spent, 2 – Tolerance, 3 – Loss of Control, 4 – Emotional Problems, 5 – Work Problems, 6 – Dangerous Activities; Panel a) and b) show the median probability network, eliminating edges with an inclusion probability < 0.5. Here, the thicker the edge, the stronger is the interaction between two nodes. Pie graphs around each node indicate its endorsement rate. Panel c) and d) show the inclusion Bayes Factor for each edge indicating the evidential strength for inclusion /exclusion. Here, red edges indicate evidence for exclusion, blue edges evidence for inclusion, and grey edges absence of evidence with a Bayes Factor < 10. Panel e) depicts the strength centrality for both the clinical and population subgroup. The higher the centrality score of a node, the stronger it is connected within the network. Networks were illustrated using qgraph (Epskamp et al., 2012). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

the edge *work problems* to *dangerous activities* can be found for income subgroups. To explore the large variation of estimates across the external factor age, we depict the interaction estimates per subgroup in Fig. 3 and for all other external factors in the supplementary material (i.e., Figs. S5–7). The group of participants being 50 years or older shows the most diverging behavior with three edges estimated to be absent. Particularly, the strong interaction between *time spent* and *work problems* found in all other subgroups was estimated to be absent. No post-hoc tests were performed, therefore no conclusions about significance of individual edge differences can be derived.

In sum, parameter estimates differed for various external factors. Therefore, apart from the symptoms of the disorder itself, external factors influence the occurrence and strength of symptom interaction.

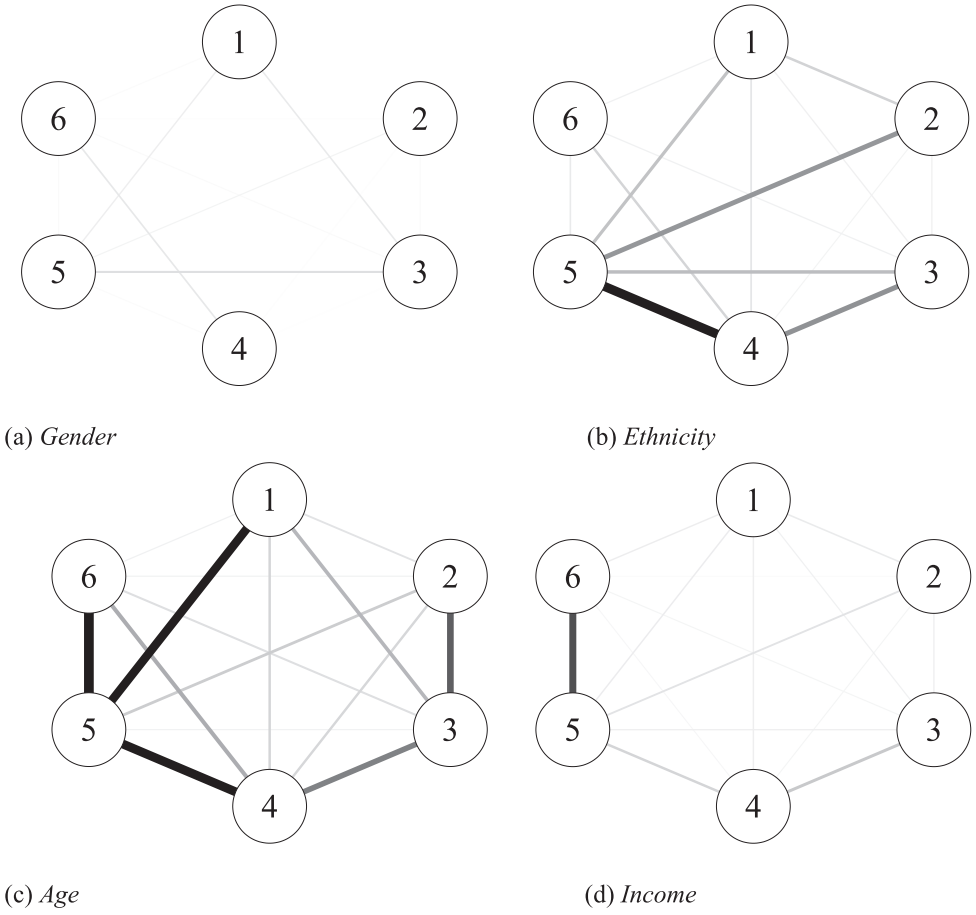


Fig. 2. Edge differences for subgroups in the population sample. 1 – Time spent, 2 – Tolerance, 3 – Loss of Control, 4 – Emotional Problems, 5 – Work Problems, 6 – Dangerous Activities. Networks showing variation in edge estimates for each external factors based on averaged squared differences between edge weight matrices for each subgroup. The thicker an edge, the higher the variation in edge estimates; maximum edge thickness is set equal in all networks to the highest overall edge variation.

4. Discussion

In this paper, we assessed differences between the symptom network of a clinical and population sample and further dependencies on factors like age, gender, ethnicity, and income. Results show that loss of control is a central node in the clinical network, whereas the time spent was most central in the population network. The network topology of the clinical sample is less densely connected than the population counterpart. Furthermore, in the population sample the network interactions are measurement non-invariant and depend on external factors, especially on age subgroups.

Our findings provide several implications for AUD research. In the population sample, the *time spent* drinking/obtaining alcohol is most strongly connected with all other symptoms and therefore central to the disorder, consistent with previous studies (Conlin et al., 2021; Rhemtulla et al., 2016). Central symptoms have been shown to predict future diagnoses (Boschloo et al., 2016), suggesting they could be an opportune target in preventive programs. In the clinical sample, *loss of control* is most strongly connected, corroborating known theories on addiction that define loss of control as a cardinal feature of AUD (Goldstein & Volkow, 2011). Furthermore, we found large heterogeneity in symptom associations due to external factors like age, gender, ethnicity, and income. This finding supports previous research on the disorder-level suggesting subgroup differences across various external factors (e.g., Grucza et al., 2018; Lemstra et al., 2008; Phillips et al., 2017). Specifically, the differences in the age range 50 years and older has been noted before (Veerbeek et al., 2019). Future research needs to replicate the measurement non-invariance of the population sample for a larger clinical population. In our analysis, the clinical subgroup was too small and parameter estimates too unstable to further split the data into subgroups. If measurement non-invariance is confirmed, our results suggest that incorporating those parameter differences into prevention or intervention is of key importance if one aims to be effective. For example, an intervention aiming at reducing the relation between *loss of control* and *emotional problems* could be effective. However, if this relation is not present for individuals above the age of 50, the intervention would miss its intended effect.

Our study furthermore provides implications for the broader field of symptom network modelling. First, the clinical sample suggested a sparser network topology. This finding would be contrary to initial postulations of the network theories of psychopathology (Borsboom, 2017; Cramer et al., 2016; Kendler, 2016), which suggest a denser connection of symptoms for clinically severe individuals. Commonly researchers interpret an absent edge as evidence for conditional independence of symptoms. However, this conclusion is an erroneous misconception from null-hypothesis significance testing (Williams et al., 2021). This predicament is perfectly illustrated in our study: the clinical topology seemed sparser, however, there is insufficient evidence to conclude the edge in- or exclusion. Therefore, to assess the true network sparsity, future studies based on a larger clinical sample using a Bayesian framework should be conducted. Second, a professional diagnosis much better captures the clinically significant impairment of addicted individuals as opposed to self-reported questionnaires. The network of the clinically diagnosed individuals was likely not influenced by Berkson's bias (de Ron et al., 2021), contrary to the network of severe population individuals filtered using a sum-score cut-off. This finding is in line with Haslbeck and colleagues (2020) showing that biased estimates are introduced when using a sum-score cut-off to approximate a latent variable (e.g., "healthy" and "unhealthy"). Lastly, the dependence of estimates on external factors might be of relevance for the replicability

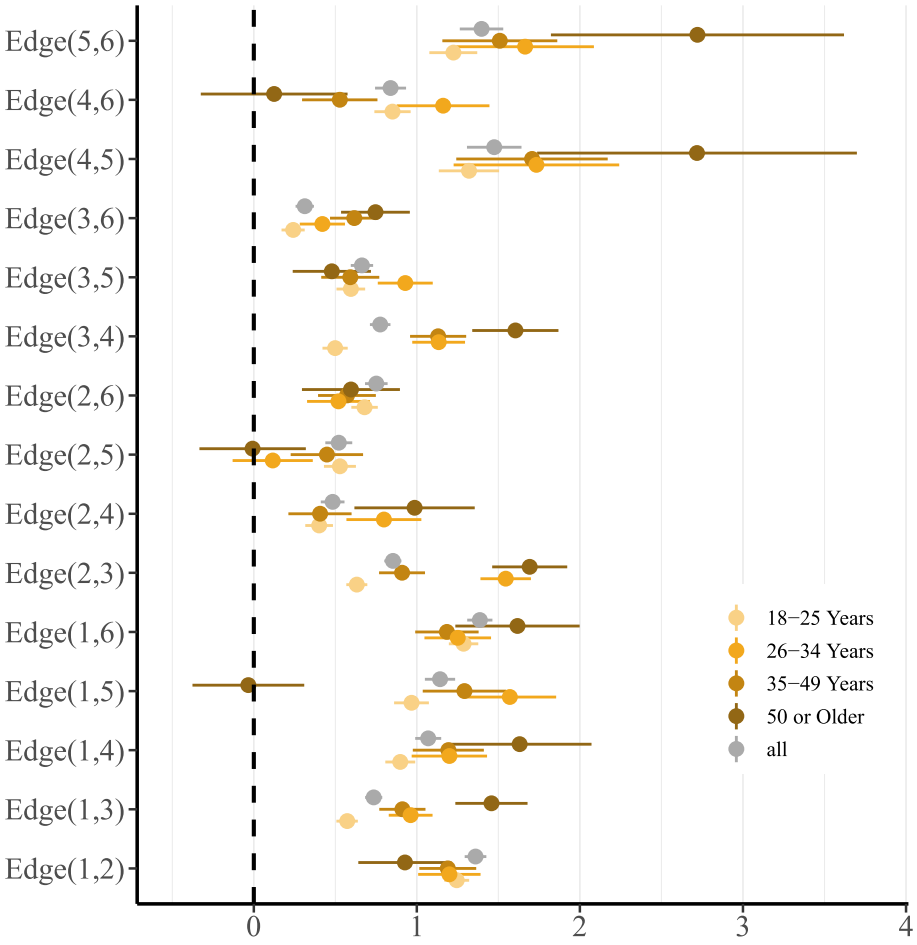


Fig. 3. Edge estimate for age subgroups in the population sample. 1 – Time spent, 2 – Tolerance, 3 – Loss of Control, 4 – Emotional Problems, 5 – Work Problems, 6 – Dangerous Activities. Interaction estimate with respective 95 % confidence interval (CI) for each subgroup across the external factor age: 18–25 Years: N = 12,105, 26–34 Years: N = 3672, 35–49 Years: N = 4885, 50 or Older: N = 2929, and all: N = 23,591. The larger the estimate (depicted as circles), the stronger the interaction between two nodes, the wider the CI (depicted as lines), the less stable the parameter estimate.

debate of network analysis (Forbes et al., 2019; Forbes et al., 2020). Critics argue that networks replicate poorly and question the conclusions drawn from them. As seen, parameters can indeed differ across factors like age, gender, and ethnicity. Since samples across different studies are usually made up of varying demographics, this could well contribute to non-replicability between studies.

There are some limitations to our study. First, cross-sectional data like the ones we used, don't allow for conclusions about the direction of effect. While we learned about the structural differences of population and clinical samples, we could not investigate the mechanisms leading from the recreational to the clinical use of alcohol and which mechanisms further support the stable state of abusing alcohol. To draw conclusions of this type of nature, a longitudinal dataset would be necessary. Second, to compare the clinical and population network, we chose to include symptoms collected in both studies. However, hereby we might have excluded some symptoms relevant for AUD like withdrawal or continuous use despite emotional or work problems. Those could be of relevance in modelling and intervening on AUD and should therefore be incorporated in future analyses. Third, despite aiming to assess the same symptoms, scales in both studies used different question phrasings. Therefore, assessed symptoms might differ between both subgroups, altering their network structure. Future research should aim to replicate our findings by using the same questionnaire across both groups. Fourth, even though individuals reported the occurrence of symptoms in the same time-frame – past 12 months activated symptoms could have occurred months apart and therefore be independent. However, in practice, questionnaires focus on a certain time frame (e.g., past 12 months), as symptoms usually target behavior that is experienced over a certain time period, and not likely time-limited. Nonetheless, future dynamic network modelling should assess the timely co-occurrence with more frequent symptom assessment. Lastly, our analyses were all exploratory. Taking our findings together with previous research and forming a hypothesis-driven analysis are important to confirm our conclusions.

This study compared the symptom connections of both recreational and dependent alcohol users, showing that the time spent drinking alcohol is most strongly connected in recreational alcohol drinkers and loss of control in alcohol dependent individuals. Furthermore, we could confirm that for recreational drinkers the symptom interactions are influenced by external factors. The novel insight these network analyses provided are a starting point for exploring mechanisms underlying recreational alcohol use and alcohol use disorder.

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CRediT authorship contribution statement

K.B.S. Huth: Conceptualization, Methodology, Data curation, Visualization, Software, Formal analysis, Writing – original draft, Writing – review & editing. **J. Luigjes:** Conceptualization, Writing – review & editing. **M. Marsman:** Conceptualization, Methodology, Software, Formal analysis, Writing – review & editing. **A.E. Goudriaan:** Conceptualization, Writing – review & editing. **R.J. van Holst:**

Conceptualization, Supervision, Writing – review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.addbeh.2021.107128.

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