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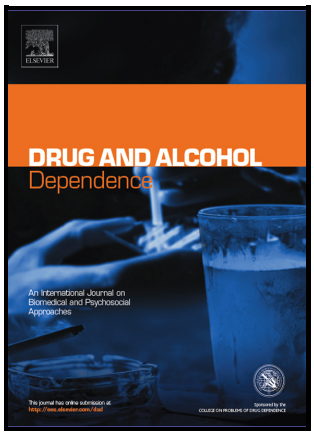

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A network analysis of alcohol-related harms: An exploratory study in United Kingdom adolescents

Short Title: Network analysis of alcohol-related harms in adolescents

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ABSTRACT

Background. Adolescent drinkers experience various harms as a result of alcohol consumption. This study applied a network analysis approach to the study of individual self-reported alcohol-related harms (ARHs) across four waves of data.

Methods. Data were from a large clustered randomised control trial (*N* = 12,738) involving 105 schools. Data were collected at 4 time points over 4 academic years (mean age 12.5 [Time 0], 13.5 [T1], 14.5 [T2], and 15.3 years [Time 3]). Data were gathered on the experience of 16 separate ARHs experienced during the previous six months, and these were dichotomised (yes/no). We estimated cross-lagged panel networks for the 16 ARHs, capturing both the auto-regressive relationships (a harm predicting itself at follow up) and the cross-lagged relationships (a harm predicting another harm at follow-up) across the study (T0 ➔ T1; T1 ➔ T2; T2 ➔T3).

Results. Exposure to all ARHs increased with age. However, the most serious ARHs (e.g., getting in trouble with the police because of your drinking) remained relatively rare, even at age 15. Actively planning to get drunk, coupled with an inability to control levels of intoxication (drinking more than planned) appeared central to each network, facilitating the emergence of all other ARHs. While the prevalence of ARHs increased with age, network complexity declined, and networks becoming more stable.

Conclusions. Interventions aimed at improving the capacity to self-regulate alcohol consumption, and actively challenging the planning of drunken episodes, may be pivotal in reducing the emergence of both acute and chronic ARHs in adolescence.

Keywords: Network analysis; Alcohol; Alcohol-related harms; Adolescence.

Introduction

The deleterious short- and long-term effects of early-onset adolescent drinking are well established (Clark, 2004; Kim et al., 2017; McCambridge et al., 2011). In 2019, alcohol use was said to be the leading risk factor for the attributable burden of disease among people aged 25 to 49, the second-leading risk factor among those aged 10 to 24, and the ninth-leading risk factor among all ages (Global Burden of Disease [GBD], 2020). Further, in 2019, alcohol use accounted for 2.07 million male deaths, and 374,000 female deaths, globally (GBD, 2020). Among adolescents, alcohol-related harms (ARHs) generally show a dose response relationship with consumption (Miller et al., 2007), with a greater likelihood of ARH associated with heavy episodic drinking (HED), and more frequent drinking in adolescence (Esser et al., 2012; Read et al., 2008). Acute ARHs include alcohol poisoning, alcohol-related physical injuries, involvement in car crashes, physical and sexual assault, and problems at school or work, and often arise from the effects of acute intoxication (Hingson & White, 2014; Percy et al., 2011). Chronic ARHs can include depression (Brière et al., 2014; Edwards et al., 2014), increased engagement in self-harm (Herbert et al., 2015), and risky sexual behaviour (Wagenaar et al., 2018).

The biopsychosocial model of health (e.g., Engel, 1977) posits that biological, psychological, and sociological factors interact and overlap to impact each individual's well-being and risk for illness. A range of physical and psychosocial factors that often interact with alcohol consumption make adolescents much more vulnerable than adults to the adverse effects of alcohol (e.g., Newbury-Birch et al., 2009). These interactions can involve; (a) neurological factors resulting from changes in the developing adolescent brain after alcohol exposure (e.g., Jacobus &

Tapert, 2013; Spear, 2018; Squeglia & Gray, 2016), (b) impaired cognition, decision making, and impulse control due to age, and the psychoactive effects of alcohol, increasing the likelihood of accidents and trauma (e.g., Percy et al., 2011; Rodham et al., 2005), (c) social factors that arise from HED that leads to both excessive intoxication and increased adolescent risk-taking (Erskine-Shaw et al., 2017; MacArthur et al., 2012), and (d) physiological factors resulting from a typically lower body mass and less efficient metabolism of alcohol (Van Zanten et al., 2013; Wall et al., 2016).

Drinking patterns established in adolescence tend to persist well into adulthood (Blinded for review), and both frequent drinking, and HED in adolescence are associated with adverse outcomes in adulthood including problem drinking, substance use, and antisocial behaviour, even after controlling for shared risk factors (Najman et al., 2019; Silins et al., 2018). In a latent class analysis of alcohol use trajectories from mid adolescence to early adulthood, Yuen and colleagues (2020) reported that those in the early-onset heavy drinking group had over seven times the odds of reporting an alcohol use disorder than those in lower use trajectories.

While measures of frequency and quantity of consumption in adolescents appear robust, there is little evidence that measures of the adverse consequence of their consumption are valid (Toner et al., 2019). Often studies examining ARHs in adolescence simply count self-reports of different harms experienced (e.g., Mattick et al., 2018; McKay et al., 2018). Such an approach has its limitations in that each harm is given equal weighting in its harmfulness and little or no account is taken of potential interdependencies amongst harms. As a result, little is known about the emergent nature of ARHs in young people, the inter-relatedness of different ARHs and how this relates to developmental outcomes.

A network approach to ARHs

Additional to the existence of social networks among or between individuals, researchers have begun to examine the inter-relatedness of individual symptoms in the prediction and development of illness. This network approach (Borsboom et al., 2013; Cramer et al., 2010) conceptualises symptoms to be mutually interacting entities, which form a complex network where it is possible that some sub-sets of symptoms can be mutually reinforcing (Borsboom et al., 2017; Bringmann et al., 2018; 2021; Funkhouser et al., 2021).

This network approach contrasts with more traditional 'common cause' approaches where symptoms are interpreted as a product of an underlying latent disorder (Bringmann et al., 2018). Within a common cause model, the latent condition gives rise to the existence of symptoms and the summed score of symptoms is typically used to identify and quantify the severity of the underlying condition. In contrast, under a network conceptualisation, a disorder is not a function of the total number of symptoms, rather the disorder exists, or may exist, as a function of the relatedness of networks of symptoms (Borsboom et al., 2013). In this sense, symptoms are understood to be active causal ingredients in a disorder, rather than passive independent receptors of the causal influence of a medical condition. Further, within the network approach, greater attention is paid to the relatedness of symptoms than to the severity, or mean score, of a given set of symptoms (Bringmann et al., 2021).

Network analyses of psychopathology symptoms have predominantly involved adult participants (e.g., Conlin et al., 2022; Haag et al., 2017; Huth et al., 2022; van

Borkulo et al., 2015; van Rooijen et al., 2017). However, analyses involving adolescent participants are beginning to emerge, and these analyses have shed light both on the nature of symptom networks (for example, which symptom is most important or central in a given network), as well as the relationship between the overall network and level of disorder (Funkhouser et al., 2021; Martel et al., 2021; Russell et al., 2017). Indeed, unique longitudinal relationships have been observed in adolescent samples, between psychopathology symptoms, both within, and across symptom domains (Funkhouser et al., 2021). Longitudinal analysis of adult alcohol use disorder symptoms have suggested significant inconsistencies between cross-sectional and longitudinal network models of alcohol use disorder symptoms (Conlin et al., 2022).

In a recent clustered RCT (Blinded for review), the authors reported a significant effect for one primary outcome measuring HED, but not for the second primary outcome measuring ARH. In the data analysis plan (Blinded for review), ARH were conceptualised as individual entities, and a total harm score was computed by simply counting the number of individual harms experienced in the past six months. It seems counterintuitive that an intervention would have a significant impact on drinking behaviours, but not ARHs. Subsequently secondary analyses of these data (Blinded for review), employing a growth mixture modelling analytical approach, again yielded a similar non-significant intervention effect on ARHs. The present study sought to better understand the inter-relatedness of these harms using network analysis, and potentially gain insight into young drinkers' experience of ARHs.

Materials and methods

Study design and participants

Participants (*N* = 12,738; Male = 6389 [50.2%]) were from a total of 105 schools in Country A (*k* = 70; *N* = 7742 [60.8%]), and Country B (*k* = 35; *N* = 4996 [39.2%]). Prior to randomization, all schools were stratified on Free School Meal (FSM) entitlement at the school level (low [33.7%], moderate [39.3%], high [27.0%]), which was taken as a proxy for socio-economic status (Hobbs & Vignoles, 2007). Schools in [country A] were also stratified by school type (male only/female only/coeducational), where the majority were co-educational, whereas, of the 35 schools in country B, only one school was a single-sex school.

Participants were eligible students in the randomized schools, who consented to participate. Opt-in consent was obtained from school head teachers/principals before randomization. Opt-out consent from participants and their parents/guardians was obtained after randomization. Data were self-reported under examination-like conditions on school premises at baseline (T0) and at three follow-ups: +12 months (T1), +24 months (T2), and +33 (T3) months. At T0 the mean age of the participants was 12.5. By T3, the mean age of participants was 15.3.

Measures

The ARHs were measured using a 16-item scale (internal consistency 0.9; McBride et al., 2004). Participants were asked to indicate on a Likert scale how many times in the past six months they had experienced the individual harm. The ARHs included having a hangover after drinking or getting into a physical fight when drinking. A full list of harms is included in Table 1. For this investigation all harms were categorised as yes (having experienced the harm in the last 6-months), or no

(not having experienced the harm in the last six months). All 16 items used in the *blinded* trial were used in each analysis. For ease of legibility of the items within the network, these items were thematically grouped as follows; (a) personal difficulties, (b) externalising behaviours, (c) relationship consequences, and (d) serious consequences. Pairwise deletion was used for each analysis.

Data Analysis

For each time point, we used a cross-lagged panel network to examine the relationship between the ARHs. These relationships were based on the coefficients obtained in node-wise logistic regression, which was used to estimate the auto-regressive relationships (a harm predicting itself at follow up) and the cross-lagged relationships (a harm predicting another harm at follow-up) across all waves of data (T0 → T1; T1 → T2; T2 → T3). Thus, each estimate represents the effect of one harm on another after controlling for all other harms. In the initial T0 → T1 model, the covariates used in randomisation (location, FSM, and gender of the school) were also included. However, this demonstrated that the covariates had little to no effect on the network structure and were dropped from all subsequent analyses.

Similar to Funkerhouse and colleagues (2021), we converted the log-odds to odds ratios (ORs) to increase interpretability. ORs surpassing the 95% confidence interval were included in the network. The networks were plotted using qgraph package in R (Epskamp et al., 2012) with two different visualisations for node placement; (a) Fruchterman and Reingold's (1991) algorithm, which places more strongly connected nodes closer together, and (b) a circular layout so that the evolution of the ARHs can be assessed across development. All networks were directional and included auto-regressive effects. We estimated several centrality

metrics including closeness, betweenness, out-expected influence, and in-expected influence. We examined the differences between the networks over time using several network difference tests (Epskamp et al., 2018) including edge weight difference tests and centrality difference tests.

Some harms will have a longitudinal inverse effect on other harms. To accommodate these into the network and to balance the effect size, we calculated the negative reciprocal of these effect sizes. For example, an OR of 0.5 became -2. Significant inverse associations were presented in red edges with significant positive associations presented in green edges.

Results

The most frequently reported harms at all time points were *planning to get drunk* and *consuming more than planned* (Table 1). As expected, the more serious the harm, the less frequently it was reported. Over the course of the study the frequency of all ARHs increased, with most harms doubling in prevalence. By age 15, around a quarter of young people reported drinking more than they had planned to. The most serious harms were relatively rare, even at age 15. For example, around 5% reported *getting into a fight* when drinking, *getting in trouble with the police* or *being sexually harassed when drinking*. Less than 2% reported requiring medical assistance after consuming alcohol.

T0 → T1 network (age 12.5 to 13.5)

Figure 1 visualises the network structure for the effects of T0 harms on T1 harms. All lines represent a significant effect. Dark green arrows indicate positive

effects and red arrows indicates inverse effects. Arrow width indicates the strength of the effect, for example, Node3 → Node13 (OR = 1.8); Node1 → Node11 (OR = 4.6); and Node1 → Node9 (OR= 6.7).

Personal difficulties (light green nodes) had the greatest influence on subsequent harms. This is reflected in the high centrality metrics in closeness, betweenness, and out-expected influence (see Figure 2). The strongest predictors of subsequent personal difficulties at T1 came from other personal difficulties or autoregressive effects, for example planning to get drunk (node1) at T0 increase the risk of *planning to get drunk* (node 1) at T1.

Relationship consequences (teal nodes) were more often a predicted consequence of other harms rather than a cause of other harms (with the exception of *trouble with boyfriend/girlfriend* - node12). This is reflected in the higher in-expected influence rather than out-expected influence. The strongest predictor of relationship consequences was *planning to get drunk* (node1) which significantly influenced all relationship consequences at T1.

The three externalising behaviours (red nodes) were affected through *verbally abusing someone* (node6), which had an effect on later *physical fighting* (node7) and *damaging property* (node8) as well as an autoregressive effect. *Drinking more than planned* (node1) also influenced all later externalising behaviours. There were some

weak inverse associations between the externalising problems and subsequent harms, for example having previously *damaged property* (node8) was associated with a reduced risk of *getting in trouble with the police* (Node15) and visa-versa, possibly due to the more immediate temporal (cross-sectional) consequences of these actions.

Serious consequences (purple nodes) also tended to be consequences of other harms rather than causes of other harms. This is reflected in the high in-strength value but with low scores on other centrality metrics. Personal difficulties had the strongest influence on serious consequences, particularly *planning to get drunk* (node1). There were some within group effects with both previous *sexual harassment* (node9) and problems with *school performance* (node10) leading to an increased risk of having to *attend the doctor or hospital* (node16) for alcohol problems the following year.

T1 → T2 network (age 13.5 to 14.5)

Figure 3 presents the network structure for the effects of T1 harms on T2 harms. Personal difficulties (light green) continued to exert the greatest influence on subsequent harms, particularly *planning to get drunk* (node1) and *drink more than planned* (node2). This is reflected in the high centrality metrics in out-expected influence (see figure 4). *Being sick* after alcohol (node3) also displayed high betweenness, indicating its importance on the pathway between nodes. These three personal difficulties had a strong predictor effect on subsequent personal difficulties at T2 as well other types of harms. All personal difficulties had an autoregressive effect (e.g., planning to get drunk (node1) at T1 increased the risk of planning to get drunk at T2).

Relationship consequences (teal) had a range of effects both in-expected influence and out-expected influence, with positive as well as negative paths. *Trouble with friends* (node11) and *trouble with boyfriends/girlfriends* (node12) were mostly consequences of a range of other harms. *Trouble with parents* (node13) was both a cause and a consequence of other harms, for example it had autoregressive effects, increasing the risk of subsequent incidence of reporting *hangovers* (node4) and *being involved in physical fights* (node7) at T2, but was also a consequence of personal difficulties and *being unable to remember things* (node5). Surprisingly, *trouble at school* (node14) had several strong negative effects such as being associated with a reduced risk of subsequently *verbally abusing someone* (node6), *drinking more than planned* (node2), and *getting in trouble with a boyfriend/girlfriend* (node12).

The three externalising behaviours (red) displayed autoregressive effects and were positive influenced by a range of other themes as well as displaying some within theme influence. For example, both *verbally abusing someone* (node6) and *damaging property* (node8) were associated with an increased incidence of *getting into physical fights* (node7) with others at T2.

Serious Consequences (purple), as expected, were most often consequences of other harms rather than a cause of other harms. This is reflected in the high in-expected influence of both *trouble with the police* and *having to attend a doctor/hospital*. *Trouble with the police* (node15) was also had high closeness indicating its importance as a link variable with other harms particularly *school*

performance affected (node10) and *trouble at school* (node14). *Being unable to remember things* (node5) was also a frequent consequence of other problems, particularly personal difficulties.

T2 → T3 network (age 14.5 to 15.3)

The network structure for the effects of T2 harms on T3 harms is presented in figure 5. Again, *planning to get drunk* (node1) had the highest, closeness, betweenness, and out-expected influence of any node in the network, indicating its pivotal role in subsequent ARHs. Notably it was associated with all serious consequences (purple nodes), and externalising behaviours (red nodes). Being *sick after drinking* (node3) and having a *hangover* (node4) had a high in-expected influence from other variables within the network, mostly from other personal difficulties (light green nodes). All personal difficulties also had an autoregressive effect.

Relationship consequences (teal) had high in-expected influence and were mostly a consequence of personal difficulties and autoregressive effects. *Trouble with friends* (node11) had the largest out-expected influence and mostly effected personal difficulties and *initiating verbal abuse* (node6).

There was an increased incidence of *physical fighting* (node7) and *damaging property* (node8) in those who previously reported other harms such as *planning to get drunk* and those who got in *trouble with parents* at T2 (node13). Serious

Consequences (purple) were most often consequences of other harms rather than a cause of other harms. This is reflected in the high in-expected influence of *trouble with the police* as a consequence of *physical fights*, *planning to get drunk*, and *not remembering things*. *Trouble with the police* (node15) was also associated with subsequent *sexual harassment* (node9). Again, there were also significant autoregressive effects.

Discussion

This network analysis reveals the centrality, interconnectedness, and evolution of ARHs in early adolescence (from age 12 to 15) in a large and diverse sample of European children, thus providing a unique insight into the emergence of ARHs in young drinkers.

The analyses revealed the central importance of drunkenness, and in particular adolescents' active plans to seek intoxication, in the emergence of all other ARHs. This is closely aligned with drinking episodes where young people consume more than they had initially planned to. Thus, perhaps unsurprisingly, planning to get drunk and an inability to manage levels of intoxication are at the core of all other ARHs. These behaviours are also highly self-supporting, with a large autoregressive effect year on year.

Previous research has highlighted the 'intoxication tightrope' walked by young drinkers as they try to get drunk enough to enjoy the (positive) mood-altering and social lubricant effects of intoxication without being caught drinking by their parents

or other adults and/or suffering the negative consequences of drinking too much (Percy et al., 2011). While personal difficulties (getting drunk, getting too drunk, having a hangover, or being sick after drinking) are the most common consequences of drinking reported by the young people within the sample, such outcomes are generally considered undesirable drinking outcomes amongst young people (Ander et al., 2017) and a clear sign of an immature drinker (Hennell et al., 2021; Percy et al., 2011). Interestingly, by age 15 drinking more than planned was less central to the network of ARHs, being replaced by other negative consequences, such as being unable to remember things, and having a hangover, both of which had been towards the periphery of early networks. This could be a by-product of an increased volume of alcohol consumed.

Compared with personal difficulties resulting from intoxication, externalising behaviours (abusing someone, fighting, or damaging property) and relationships consequences (getting in trouble with friends, boy/girlfriend, parents, or school) were relatively rare across all waves of the study. However, they do increase in prevalence with age. Fortunately, few children experience the more serious ARHs such as requiring medical assistance, getting in trouble with the police, or being sexual harassed.

There were notable differences in the overall network structures at the different stages of development. The initial longitudinal network (T0→T1) displayed a heightened complexity relative to later network structures. This interrelatedness between harms may in fact be due the lack of experience with alcohol amongst these young drinkers leading to numerous proximal adverse consequences. Therefore, even though few might indulge in excessive consumption at this age, this engagement may result in a cascade of different harms due to an inability to manage

intoxication. It has been argued that young people learn to self-regulate consumption through a process of trial and error (Percy, 2008). In essence, it is argued that young drinkers learn from their mistakes and the mistakes of their drinking friends. This has not only been observed in relation to alcohol (Percy et al., 2011), but also in relation to the consumption of other substances (Jakub et al., 2022).

Results demonstrated that prevalence of both HED and ARHs increased with age. This is accompanied by a reduction in network complexity and an increase in network stability. This may reflect both the expanding numbers of adolescents engaging in alcohol use (i.e., consumption behaviour becomes more established and more stable within drinking groups) and a stabilising of the associations between ARHs themselves. For example, we observed a gradual increase in the autoregressive effects across all harms with increasing age (with requiring medical assistance the one exception).

Surprisingly, few negative paths were observed. Overall, experiencing the negative effects of drinking does not appear to significantly reduce young peoples' willingness to engage in alcohol consumption and the associated consequences of alcohol intoxication. There are some examples where encountering a significant harm was associated with a reduction in the occurrence of other harms over the following year. For example, between T0 and T1 getting in trouble with the police and criminal damage had a negative feedback loop, where experiencing either at T0 reduced the likelihood of experiencing the other at T1. Between T1 and T2, getting into trouble at school, while relatively rare, appeared to substantively reduce future occurrences of three other harms (drinking more than you planned, verbally abusing someone, and getting into trouble with your girl/boyfriend). While this suggests that, in certain circumstances, serious consequences may result in reduction of ARHs,

these effects appear relatively short lived as they were not observed at the subsequent network (T2-T3). Moreover, negative paths were relatively rare compared with autoregressive effects or positive paths between harms. But as the young people grow older we do see a reduction in the number of significant paths between harms and a reduction in the effective size of those paths which may reflect maturing, yet still relatively poor, regulation of alcohol consumption and its associated harms.

The study confirms that ARHs in adolescents are not all equal in their interaction with other harms, either in cross-sectional or longitudinal networks. From a measurement perspective a simple summative score fails to capture the complexity of the relationships that exist between emergent harms. Rather than treating individual harms as separate entities formed by individual drinking patterns (and thus able to be simply counted), there is a need to recognise that certain harms (e.g. drinking more than planned to, being sick while drinking) are largely facilitators of other (usually more serious) harms, while these harms appear to operate more as end points (e.g. trouble with the police), having little impact on other subsequent harms.

Given that, somewhat unsurprisingly, serious ARHs have their origins in episodes of uncontrolled intoxication, interventions aimed at providing young drinkers with the necessary skills and strategies to improve their regulation of their own consumption may be a way of disrupting network connections that accelerate the emergence of more serious ARHs in young people. Minimising occasions when young drinkers overshoot their desired level of intoxication may consequently reduce their exposure to the more serious negative consequences of alcohol consumption. It has been argued that without such intervention, we simply leave young people to

acquire these skills through trial and error experimentation, usually in the company of other naive young drinkers, as they seek to become competent consumers (Percy et al., 2011), a process which this analysis would suggest is likely to increase the transition to more serious ARHs.

Strengths ad limitations

While the data analysed herein are longitudinal, and arise from a well-constructed and supervised cRCT, and the research is one of the first network analysis of ARHs in adolescents, the study is not without limitations. Firstly, this study is exploratory in nature, utilising data not specifically collected to facilitate the network analysis of adolescent ARHs. Therefore, the results should be interpreted with caution. Secondly, while the measure of ARHs employed within the study is well regarded, it may not capture the full range of harms experienced by young drinkers. It was designed to assess variations in ARHs (via a summed score) for use as a primary outcome within controlled prevention trials rather than longitudinal network analysis. Therefore, it is possible that the measurement of individual harms could be improved. Thirdly, this longitudinal study of harms relies on retrospective data (over the previous six-months) collected on an approximate annual basis. As such it may fail to capture the more subtle temporal shifts in young people's encounters with ARHs that may arise over shorter time periods. Future research may consider examining changes in the network structure of ARHs via weekly or monthly observations. And finally, many of the network harms reported by young people are highly context dependant. For example, the chances of young drinkers encountering local police officers may be as much driven by police policy and operations in relation to underage drinking, than by the drinking behaviour of the young people themselves. This study did not account for these external contextual factors that may

shape the evolution of ARH networks. Additionally, it is worth noting that this study was undertaken in the UK, a high-income country. The generalizability of findings to data from low- to-middle, or developing countries remains unclear.

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Figure 1: Longitudinal network of alcohol related harms, age 12.5 (T0) to age 13.5 (T1).

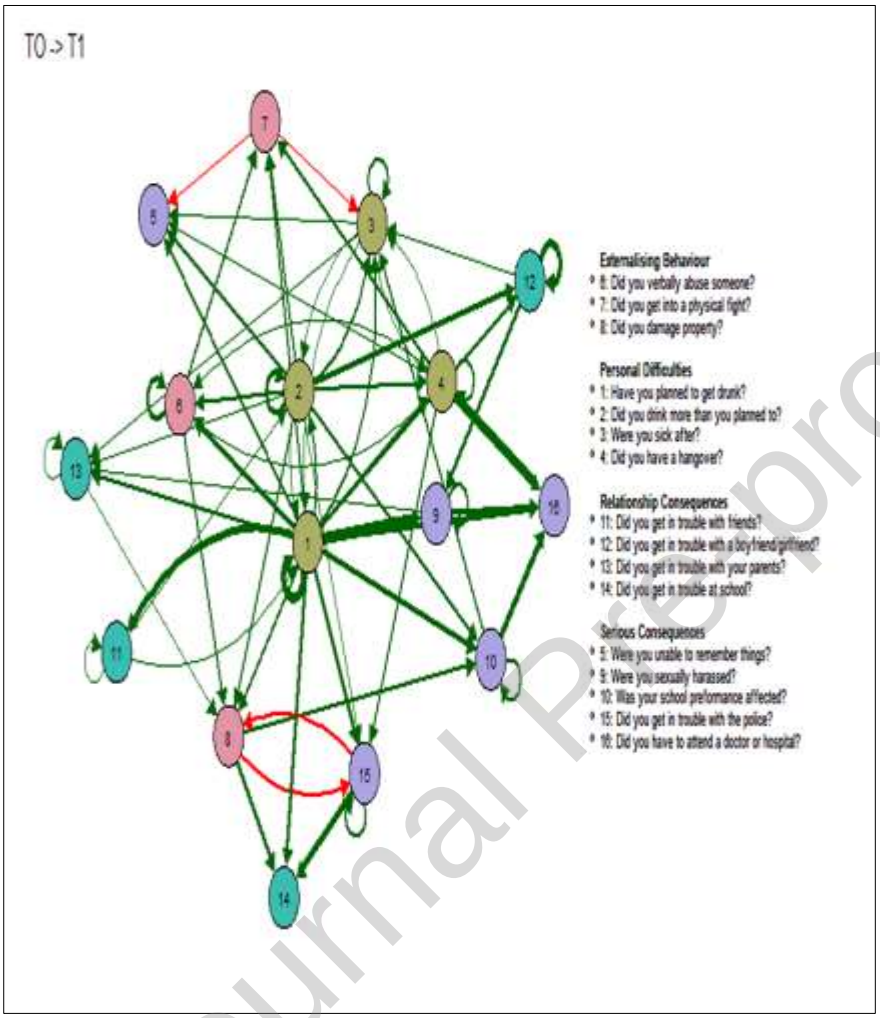

Figure 2: Centrality metrics for T0→T1 network

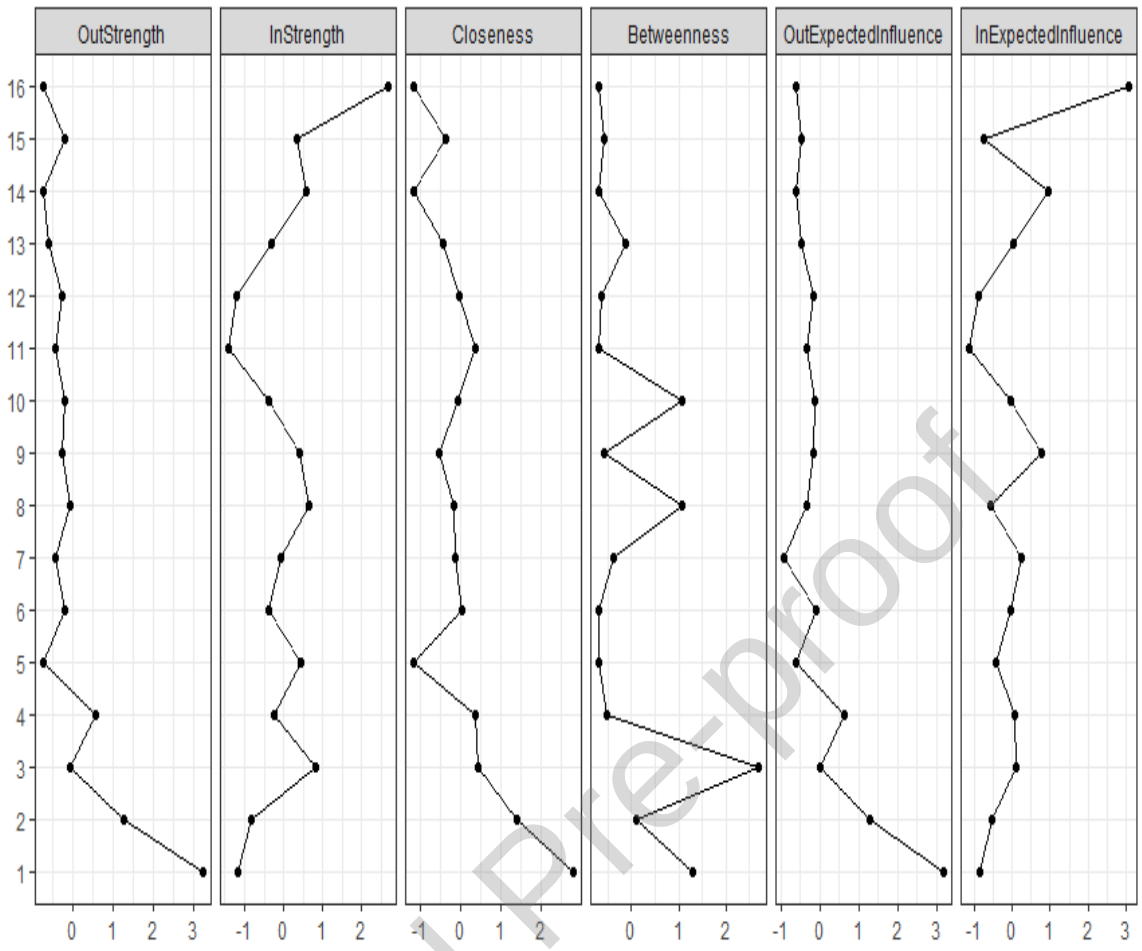

Figure 3: Longitudinal network of alcohol related harms, age 13.5 (T1) to age 14.5 (T2).

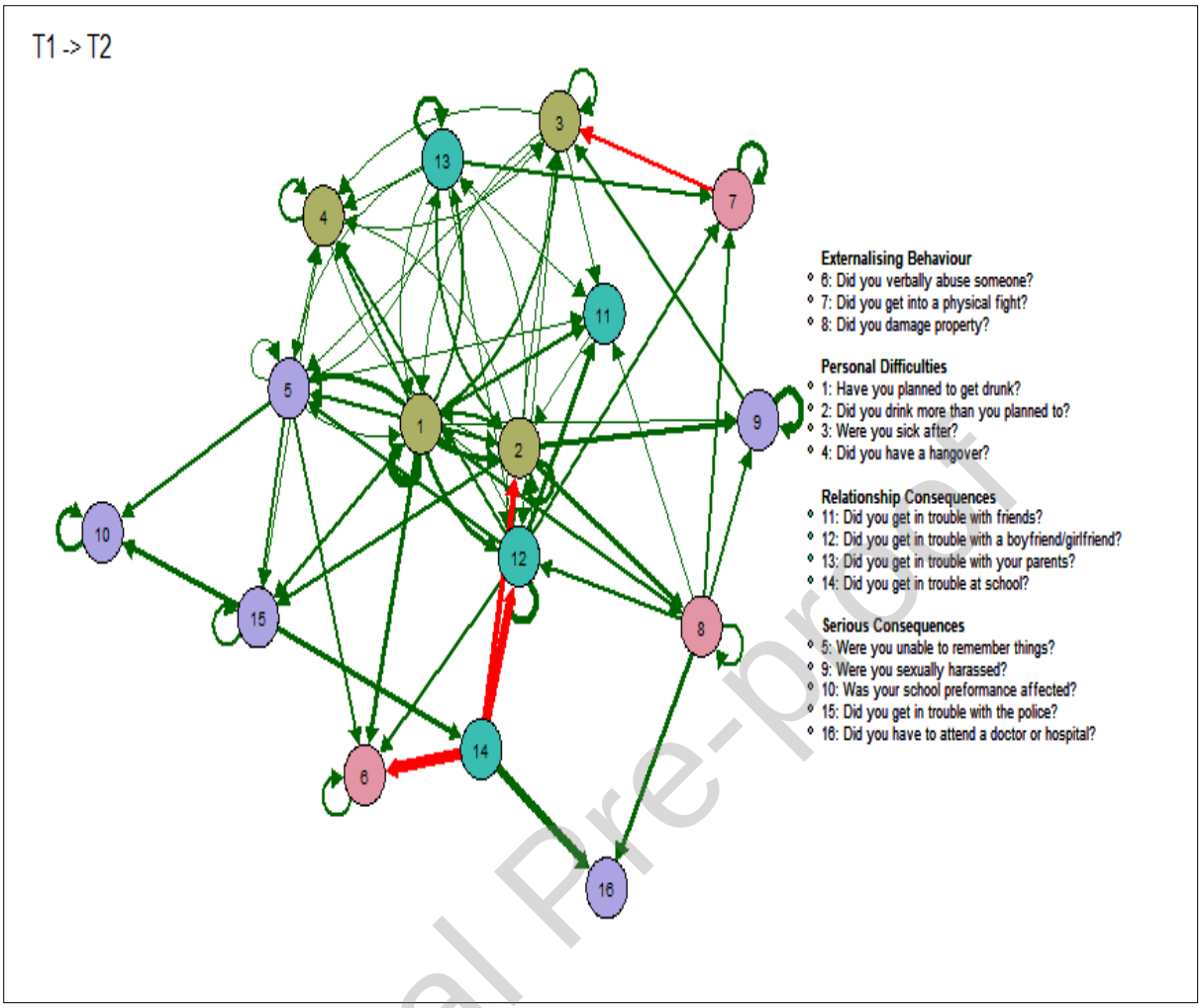

Figure 4: Centrality metrics for T1→T2 network

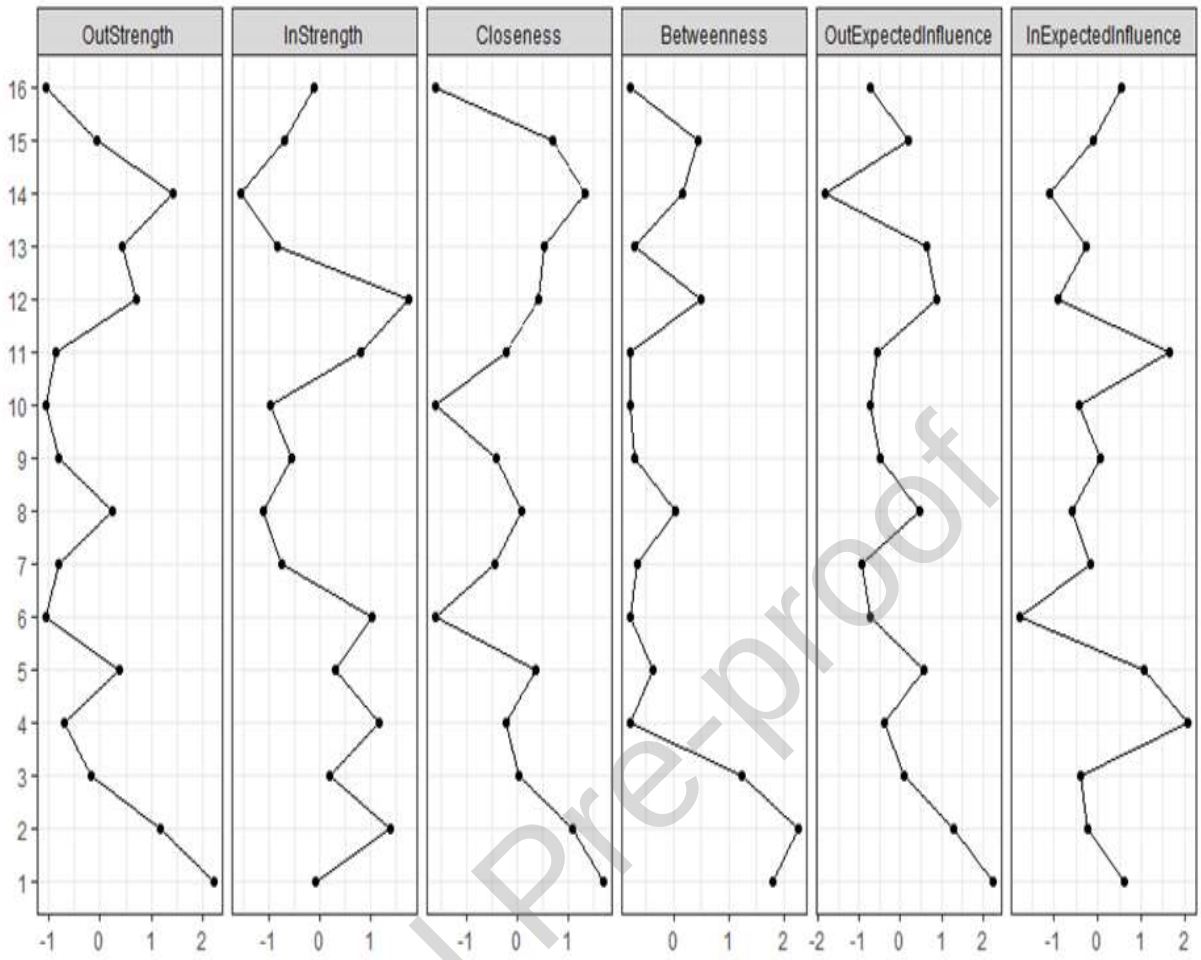

Figure 5: Longitudinal network of alcohol related harms, age 14.5 (T2) to age 15.3 (T3).

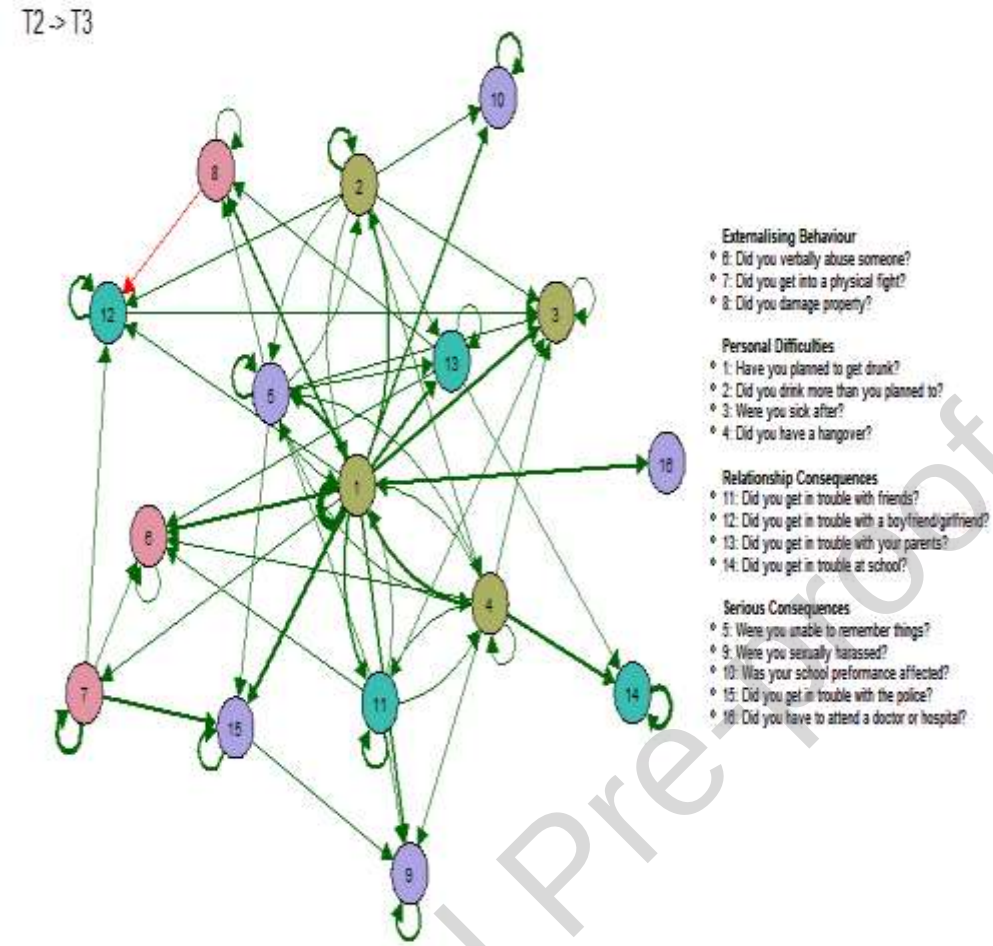

Figure 6: Centrality metrics for T2→T3 network

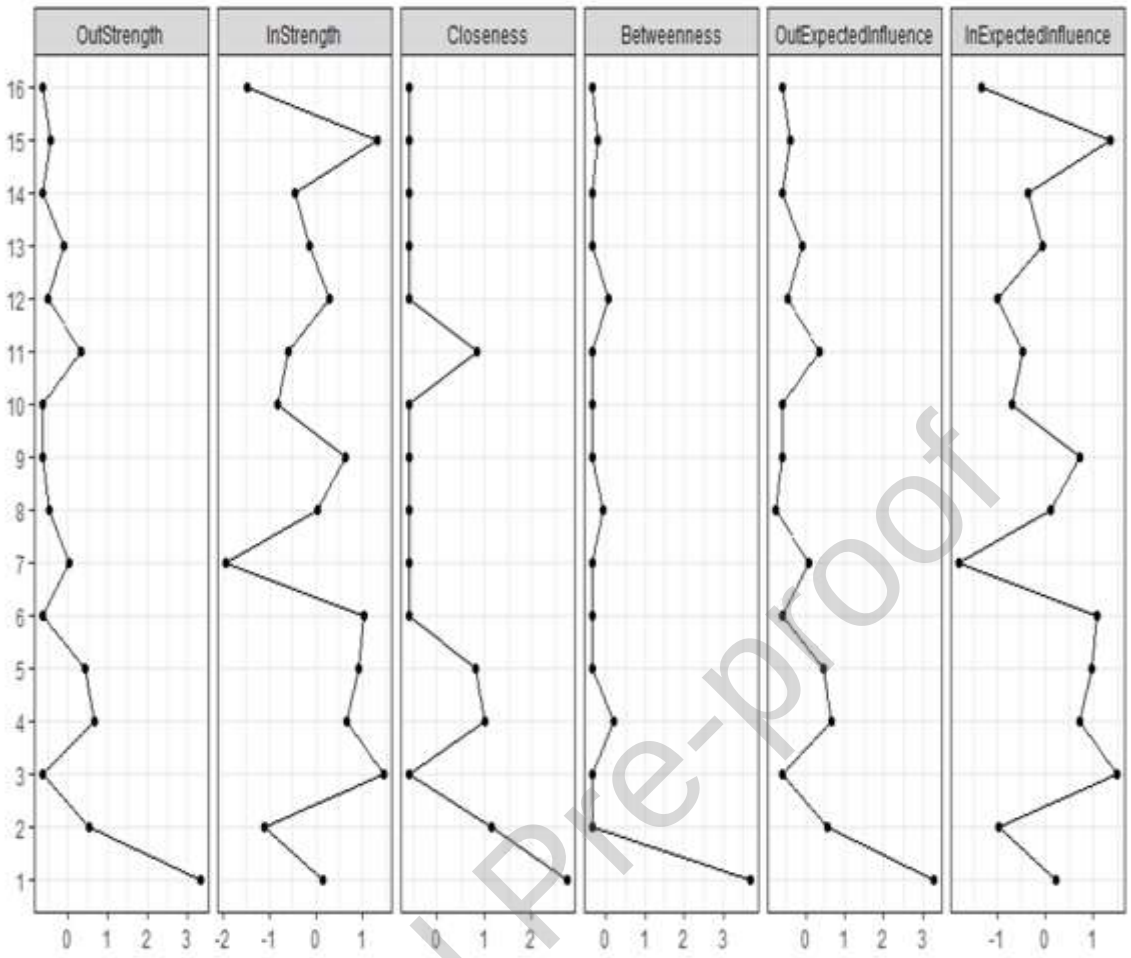

Table 1. Prevalence of experience alcohol related harm over last 6 months

In the past 6 months…	Age 12.5 *N* = 11,316 (T0) %	Age 13.5 *N* = 11,140 (T1) %	Age 14.5 *N* = 10,534 (T2) %	Age 15.3 *N* = 10,405 (T3) %
Personal difficulties				

Have you planned to get drunk?	9.8	12.5	18.8	28.7
Did you drink more than planned to?	10.4	11.5	16.6	27.3
Were you sick after you had been drinking?	8.8	8.4	11.8	16.0
Did you have a hangover after you had been drinking?	8.6	8.6	12.3	19.0
Externalising Behaviours				
Did you verbally abuse someone when you had been drinking?	4.3	4.8	6.2	8.8
Did you get into a physical fight when you had been drinking?	3.3	2.8	3.7	5.6
Did you damage property when you had been drinking?	3.5	3.6	4.7	5.9
Relationship Consequences				
Did you get in trouble with friends because of your drinking?	4.3	4.6	6.9	8.4
Did you get in trouble with a boyfriend/ girlfriend because of your drinking?	3.4	3.8	4.9	7.9
Did you get in trouble with your parents because of your drinking?	5.4	5.4	8.0	11.4
Did you get in trouble at school because of your drinking?	0.9	0.7	1.3	1.7
Serious Consequences				

Did you get in trouble with the police because of drinking?	2.6	2.9	5.0	5.7
Did you have to attend a doctor or hospital because of your own drinking?	0.8	0.9	1.1	1.6
Was your school performance affected (e.g. day off) because you had been drinking?	1.5	2.0	2.5	3.3
Were you sexually harassed when you had been drinking?	1.6	2.1	3.1	5.8
Were you unable to remember things that had happened when you had been drinking?	6.7	8.1	11.7	18.4

Declaration of Competing Interest

The authors have no conflicts of interest to declare.

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Highlights

- Alcohol use among adolescents is associated with a range of harmful outcomes.
- Longitudinal data from a RCT were used to explore harms networks.
- The number of self-reported harms increased with age.
- Planning to get drunk, and drinking more than planned were central to harm networks.
- Network simplicity and stability increased with time.