Peer Effects in Recovery from Substance Use Disorder

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January 28, 2025

Abstract

We study the impact of peers on recovery from substance use disorders using linked administrative data on Norwegian inpatients. Peers are defined based on overlapping treatment episodes and a common geographic area of residence. Waiting lists provide a source of quasi-experimental variation in peer exposure. We find that peers reduce all-cause and substance-induced mortality, reduce hospital admissions for substance-related disease, and have no discernible impact on admissions for substance-related poisoning. As highlighted by our theoretical framework, these findings are consistent with two mechanisms: peers supporting one another to abstain and, when use does occur, peers reducing harm by using together.

Key words: Peer effects, substance use treatment, mortality, morbidity

JEL codes: I12, D85

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[§]The authors thank Asle Enger and his group at the Department of Substance Abuse and Addiction Treatment, University of Oslo Hospital for providing information and insights into inpatient treatment for SUDs in Norway that was crucial for this research. We thank Davide Dragone for sharing his knowledge of the relevant theoretical literature; Elaine Kelly and other participants of the Essen Health Conference; Miriam Wüst, Torben Heien Nielsen and other seminar participants at the University of Copenhagen Center for Economic Behavior and Inequality; and participants at the Risky Behaviors Workshop, the Australasian and European Workshops on Econometrics and Health Economics, and the University of York Center for Health Economics seminar. We also thank the Applied Micro group at the University of Melbourne for their valuable insights and suggestions.

1 Introduction

Social networks and peer interactions play an important role in substance use behavior. Research has shown that adolescent entry into substance use is influenced by the use of peers (Gaviria & Raphael, 2001), that social networks are key to access (Becker, 1955; Belackova & Vaccaro, 2013; Jacobi & Sovinsky, 2016), and that escalation from experimentation to regular, and potentially disordered use, is more likely when normalized among friends (Becker, 1955; D'Amico & McCarthy, 2006; Lundborg, 2006). Less is known about how peers influence recovery from substance use disorders (SUDs), even for established peer based programs such as Narcotics Anonymous (Eddie *et al.* , 2019). Yet knowledge of whether and under what circumstances peers aid recovery could be pivotal for alleviating treatment gaps. These gaps, which stem from a mismatch between the acute care model used for addiction treatment and the chronic, relapsing nature of SUDs, leave individuals without adequate support outside clinical settings, and lead to low treatment retention, cycles of relapse, and increased mortality (Brorson *et al.* , 2013; Lappan *et al.* , 2020; Stanojlović & Davidson, 2021). In this context, the question of whether peers can be leveraged to support recovery in the community is of significant policy interest.

To address this question, we examine whether peers acquired during treatment can support recovery. We focus on peers acquired during an inpatient episode of SUD treatment. Inpatient peers are a plausible source of support because, in addition to having a common goal to recover, inpatients attend group treatment sessions in which they learn strategies to aid recovery and to reduce harm in the event of relapse (Blanco *et al.* , 2020; Compton *et al.* , 2019). Of course, inpatient peers may themselves relapse and so subvert the recovery of others (Bohnert *et al.* , 2009; Curtis *et al.* , 1995). The net impact of peers from inpatient treatment episodes is thus an empirical question, the answer to which has important implications for the design of post-treatment programs.

To provide structure to our empirical investigation, we extend the theoretical model of addiction by Bernheim & Rangel (2004) to include peers. Peers may influence substance use behavior through three potential mechanisms: (1) they may support the focal individual to abstain from use; (2) they may subvert the focal individual's recovery by inducing use; (3) and when use does occur, they may reduce its harm by using together rather than using alone (to guard against overdose). Each mechanism provides empirical predictions for the impact of peers on the three substance related health outcomes we study: death, hospital admission for substance related poisoning, and hospital admission for substance related disease. Comparing these predictions to our empirical findings allows us to distinguish dominant mechanisms through which peers impact behavior.

Our empirical analysis uses high-quality administrative data linking the Norwegian Patient Registry, Cause of Death Registry and Population Registry. We extract the universe of individuals receiving inpatient treatment for a SUD in 2009 and 2010, who we track until 2013. We define peers as those whose inpatient treatment episodes overlap and who also share the same geographic area of

residence.[1] As with those who have overlapping episodes of incarceration (e.g., Billings & Schnepel (2022)), we expect SUD patients with overlapping episodes of inpatient treatment, and who are resident in the same geographic area, to maintain social ties that can impact their substance use behavior, and hence the health outcomes we observe, in the years following treatment.

Causal interpretation of our findings rests on random exposure to peers during inpatient treatment. As we discuss in detail in the following sections, random exposure through overlapping episodes is plausible given the institutional features that preclude inpatients from controlling when they are able to enter a treatment facility. The date of admission is determined by the interaction of a prioritized waiting list and the discharge dates of those already in treatment. Neither of these can be manipulated by the patient or the referring physician, hence we argue that the admission date is as good as randomly assigned. We isolate this quasi-experimental variation in timing using a model with facility by area of residence fixed effects, and provide empirical evidence that our identification strategy is credible through various balance tests, two placebo tests, and a series of robustness tests designed to address concerns around endogenous exit from treatment and econometric modeling of low probability events such as death.

Our paper contributes three key findings. First, peers reduce mortality in the post-treatment period. We estimate that an additional peer reduces mortality (from all causes) by 10% of its mean in the first year following inpatient treatment. This shows that peers have an immediate impact. Three years following treatment, an additional peer is estimated to reduce mortality by 4%, suggesting that their effect is not only significant, but also long lasting.

Our second key finding relates to how peers reduce mortality. We show that peers reduce substance induced deaths, and more specifically, overdose deaths. This suggests that peers influence substance use behavior. Further, we show that there is no evidence that peers increase hospital admissions for substance related poisoning, so the reduction in substance induced deaths cannot solely be attributed to peers using more safely together. Finally, we show that peers reduce hospital admissions for substance related (liver) disease. This is consistent with peers providing support to abstain from substance use. Overall, the evidence rules out subversion of recovery as the dominant mechanism through which peers influence each other. Rather, peers play a role in supporting one another to abstain from use, and when use does occur, peers reduce harm by using together rather than alone.

Our third key finding relates to the question of which peers matter. We investigate whether peers who were treated for the same type of substance are more or less influential than those treated for a different type. We find that peer type does matter, with individuals who were treated for a drug use disorder benefiting from greater exposure to peers who were also treated for a drug use disorder. These peers reduce mortality from all causes and from overdose, as well as hospital

[1]Given the role of geographically proximate peers in risky behaviors (Adamopoulou *et al.* , 2024; Billings & Schnepel, 2022; Case & Katz, 1991; Christakis & Fowler, 2007; Dustmann & Landersø, 2021; Kling *et al.* , 2005, 2007; Ludwig *et al.* , 2001; Stevenson, 2017), these are individuals with whom relationships formed during inpatient treatment are most likely to persist after exiting inpatient treatment.

admissions for substance related disease. In contrast, peers who were treated for an alcohol use disorder had no discernible impact on those treated for a drug use disorder.

Our work contributes to three distinct literatures. First, we advance the literature on peer effects in substance use. There is extensive research on adolescent entry into substance use (e.g., Ajilore *et al.* (2016); Arduini *et al.* (2019); Case & Katz (1991); Clark & Lohéac (2007); Cutler & Glaeser (2005); Eisenberg *et al.* (2014); Fletcher (2012); Fletcher & Ross (2018); Gaviria & Raphael (2001); Hsieh & Van Kippersluis (2018); Kawaguchi (2004); Li & Guo (2020); Lundborg (2006)) and a growing body of work on adult entry into prescription opioid misuse (Adamopoulou *et al.* , 2024; Seamans *et al.* , 2018). However, there is little work on *exit* from substance use, and to our knowledge, none that uses quasi-experimental variation.[2] Our work also adds to the wider literature on peer effects in risky behaviors such as crime, unsafe sex and truancy (e.g., Card & Giuliano (2013); Díaz *et al.* (2021); Lindquist *et al.* (2024); Patacchini & Zenou (2012)).

Finally, we advance the literature on determinants of mortality and morbidity of people with SUDs. People with SUDs have substantially higher mortality and morbidity than those without, and an estimated two thirds of substance induced deaths in the US are not coded as such in cause of death data (Glei & Preston, 2020). Mortality has been shown to be associated with the type and amount of substance used, and on the route of administration (Degenhardt *et al.* , 2011), macroeconomic conditions (Hollingsworth *et al.* , 2017) and individual characteristics such as sex (Gjersing & Bretteville-Jensen, 2014; Larney *et al.* , 2020). Considering research that uses quasi-experimental variation, Alpert *et al.* (2018), Alpert *et al.* (2022) and Evans *et al.* (2019) show that mortality is reduced by availability of abuse-deterrent opioids, and Swensen (2015) shows that mortality is reduced by an increase in treatment facilities. These papers use state or county level variation in the US. Focusing on the role of peers, Mäckle & Ruenzi (2022) and Cutler & Donahoe (2024) use US county level data to examine cross-county spillovers in opioid use and deaths that could be attributed to peer effects. To our knowledge, ours is the first study using individual level variation to directly examine the role of peers.

2 Theoretical framework

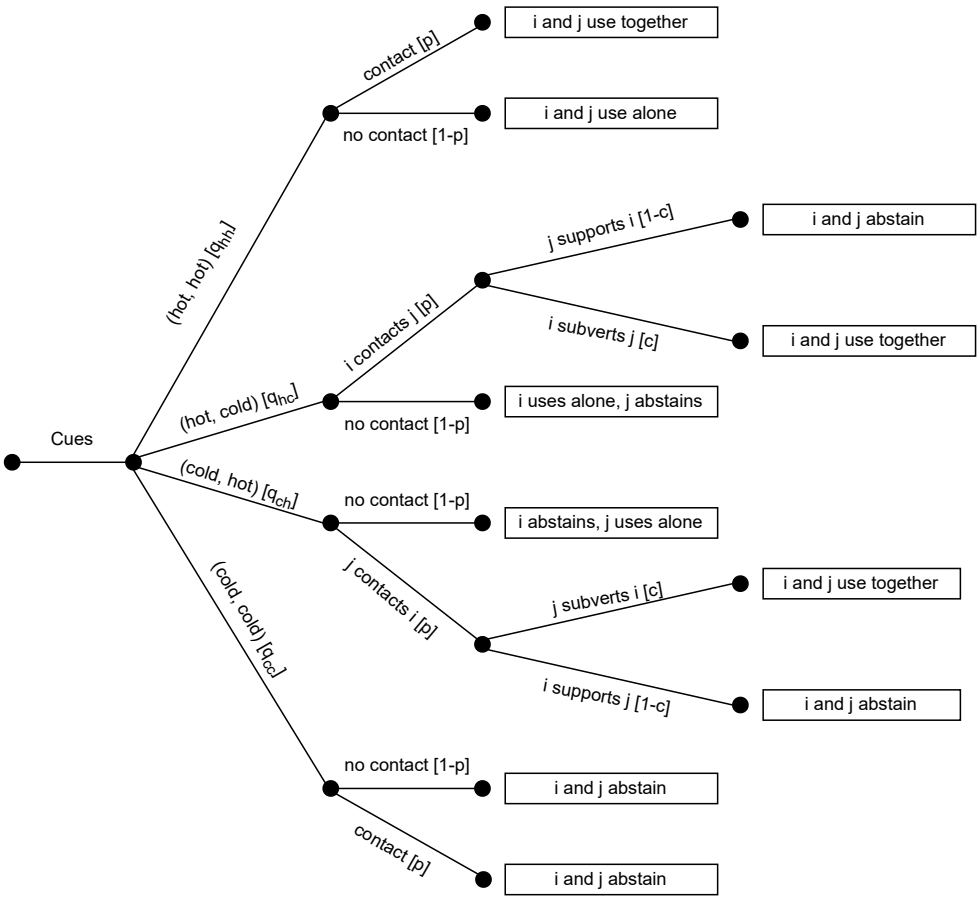
In studying how peers acquired during inpatient treatment for a SUD may influence behavior post-treatment, we take as a starting point the model of addiction and cue triggered decision processes by Bernheim & Rangel (2004). A key feature of this model is that it accounts for the fact that

[2]Previous work has considered associations between the smoking cessation of an individual and their social network (Christakis & Fowler, 2008), their spouse (Palali & Van Ours, 2017) and their household (Jones, 1994). Jason *et al.* (2012) and Bohnert *et al.* (2009) study the association between quitting use among individuals with a SUD and their social network size, and quitting cocaine and heroin use and network composition, respectively. Causal evidence on the effectiveness of mutual support abstinence based programs such as Alcoholics Anonymous and Narcotics Anonymous is limited (Kaskutas, 2009; White *et al.* , 2020) and where it exists, is mixed (Kaskutas, 2009). Whilst one might expect that peers play an important role in such programs, to our knowledge, no study has specifically examined their role.

addictive substances interfere with processes in the brain involved in forecasting near-term rewards. As a consequence, when exposed to strong environmental cues, individuals enter a 'hot' decision making mode in which their cue triggered impulses dominate their cognitive processes, and they consume the substance irrespective of their underlying preferences. Otherwise, individuals are in 'cold' mode and make decisions in line with their preferences. Such decision making states have also been referred to as 'fast' (instinctive and emotional) and 'slow' (deliberative and logical) (e.g., Kahneman (2011)).

To incorporate peers into the model, we consider two representative individuals, i and j, who shared an inpatient treatment episode and who live in the same geographic area, and therefore may plausibly maintain contact in the post-treatment period that we study. In the post-treatment period, environmental cues, which are random, identically distributed (for simplicity) and potentially correlated, are received by both i and j. Sufficiently strong cues result in individuals entering the hot decision making mode. Otherwise, decision making occurs in cold mode. This leads to four

Figure 1: A model of cue triggered substance use among peers



Notes: The four initial states of the world (in parentheses) are the modes of i and j. For example, (hot, cold) means that i is in hot mode and j is in cold mode. The probability of each branch is shown in brackets.

possible states of the world: (cold, cold), (cold, hot), (hot, cold), (hot, hot). i and j's substance use behavior depends on the state of the world and whether there is contact between peers.

Figure 1 shows the set of possible interactions for i and j and the resulting substance use behavior. The first branch captures the state of the world, and subsequent branches capture interactions between i and j. The probabilities of each branch are shown in brackets. Since we study individuals who undertake inpatient treatment for their SUD, we assume that underlying preferences are for recovery. So, if both i and j are in cold mode, both abstain from using whether they are in contact or not. If at least one of them is in hot mode, an individual can influence the other's behavior in one of three ways. First, if the individual is in cold mode and they are contacted by a peer in hot mode, they may *support* the recovery of their peer by helping them manage their desire to use. Peer support such as this forms the basis of well known recovery programs such as Alcoholics Anonymous and Narcotics Anonymous. Second, if the individual is in hot mode and they make contact with a peer who is in cold mode, they may *subvert* the recovery of their peer by causing them to use. This can happen because the desire to use may be contagious across individuals, and captures the canonical 'peer effect' commonly found in the literature on entry into substance use. The third way that peers can influence substance use behavior arises if both i and j are in hot mode. If both are in hot mode and make contact, then they *use together* more safely than they would have had they used alone. Using together is a harm reduction strategy because it reduces the risks associated with using substances, most notably, the risk of death.

A key determinant of the effect of peers in recovery from SUD in the post-treatment period is the probability that i and j are in contact, which we denote by p. We therefore conduct comparative statics in p. We also denote the probability of support given contact by $1-c$, and the probability of subvert given contact by c. To simplify the analysis below, we set $q_{cc}=0$ (because peers play no role in state (cold, cold)), $q_{hh}=q$, and $q_{hc}=q_{ch}=(1-q)/2$.[3]

Our interest lies in the impact of a change in p on i's substance use behavior, and through this, on i's observable health outcomes. Using Y to denote an indicator for i's health outcome, we decompose $\partial E(Y)/\partial p$ as follows,[4]

$$\frac{\partial E(Y)}{\partial p} = \underbrace{\left[\left(\frac{1-q}{2}\right)(1-c)\right]\Delta_{\text{support}}}_{\text{Support recovery mechanism}} + \underbrace{\left[\left(\frac{1-q}{2}\right)c\right]\Delta_{\text{subvert}}}_{\text{Subvert recovery mechanism}} + \underbrace{\left[q+\left(\frac{1-q}{2}\right)c\right]\Delta_{\text{use together}}}_{\text{Use together mechanism}}.$$

Each term on the right hand side above corresponds to one of the three mechanisms through which j changes i's substance use. The effect of each mechanism can be broken down into two parts. The

[3]This symmetry is natural because i and j are representative agents: their cues are identically distributed and their thresholds for entering hot mode are the same.

[4]With a little algebra, this decomposition can be obtained from $E(Y) = P(\text{use alone})E(Y|\text{use alone}) + P(\text{use together})E(Y|\text{use together}) + P(\text{abstain})E(Y|\text{abstain})$, where $P(\text{use alone}) = q(1-p) + \left(\frac{1-q}{2}\right)(1-p)$ is the probability of i using alone, $P(\text{use together}) = qp + (1-q)pc$ is the probability of i using with j and $P(\text{abstain}) = 1 - P(\text{use alone}) - P(\text{use together})$ is the probability that i abstains. Clearly, the case of j's substance use is symmetric to that of i, hence we omit the i subscript from Y.

leading part in brackets is the probability that the mechanism arises. The trailing part denoted by Δ captures the impact of the mechanism on the outcome when it does arise. The support recovery mechanism causes i to abstain when, absent contact with j, i would have used alone, hence $\Delta_{\text{support}} = E(Y|\text{abstain}) - E(Y|\text{use alone})$. The subvert recovery mechanism causes i to use with j rather than abstain, hence $\Delta_{\text{subvert}} = E(Y|\text{use together}) - E(Y|\text{abstain})$. The use together mechanism causes i to use with j rather than alone, hence $\Delta_{\text{use together}} = E(Y|\text{use together}) - E(Y|\text{use alone})$.

To sign the impact of each mechanism (i.e., the Δs) we need to establish relative values for $E(Y|\text{abstain})$, $E(Y|\text{use alone})$ and $E(Y|\text{use together})$ for each health outcome. Reflecting the empirical measures available to us in the administrative data, the health outcomes we consider are death, hospital admission for substance related poisoning, and hospital admission for substance related disease. It is clear that the probability of death and hospital admission should be lowest when abstaining, and so we normalize $E(Y|\text{abstain}) = 0$ and only compare $E(Y|\text{use alone})$ with $E(Y|\text{use together})$. Norwegian public health agencies take a harm minimization approach, explicitly encouraging those with a SUD to use together rather than alone (NIPH, 2019) because using under supervision has a lower risk of death (Caulkins *et al.* , 2019; Levengood *et al.* , 2021; Yoon *et al.* , 2022). Peers can supervise one another to guard against overdose or poisoning, and in the event of overdose or poisoning, they can contact emergency services and place the victim in the recovery position. They can also administer take-home anti-overdose medication such as Naloxone (Chimbar & Moleta, 2018). Denoting the indicator for death by D, these arguments imply that $E(D|\text{use alone}) > E(D|\text{use together})$. When a peer prevents a death whilst using together, it results instead in a (non-fatal) hospital admission for poisoning (denoted H_P). This means that $E(H_P|\text{use together}) = E(H_P|\text{use alone}) + [E(D|\text{use alone}) - E(D|\text{use together})]$.[5] In contrast, hospital admissions for substance related disease (denoted H_D) should depend only on the extent of use rather than whether it takes place alone or with a peer, so $E(H_D|\text{use together}) = E(H_D|\text{use alone})$.[6]

Table 1 summarizes the anticipated sign of the effect that peers have through each mechanism on each health outcome. The support recovery mechanism decreases deaths and both types of hospital admission. The subvert recovery mechanism increases deaths and both types of hospital admission. The use together mechanism decreases deaths, increases hospital admissions for poisoning (since prevented deaths become admissions for poisoning) and has no impact on admissions for disease.

[5] The qualitative conclusions of our analysis are unchanged when only some fraction $\alpha \in (0, 1]$ of deaths become poisoning admissions when using together with a peer. (i.e., $E(H_P|\text{use together}) = E(H_P|\text{use alone}) + \alpha\,[E(D|\text{use alone}) - E(D|\text{use together})]$). This may arise when the poisoning is mild enough to treated by paramedics at the scene or in the emergency room, without leading to subsequent hospital admission.

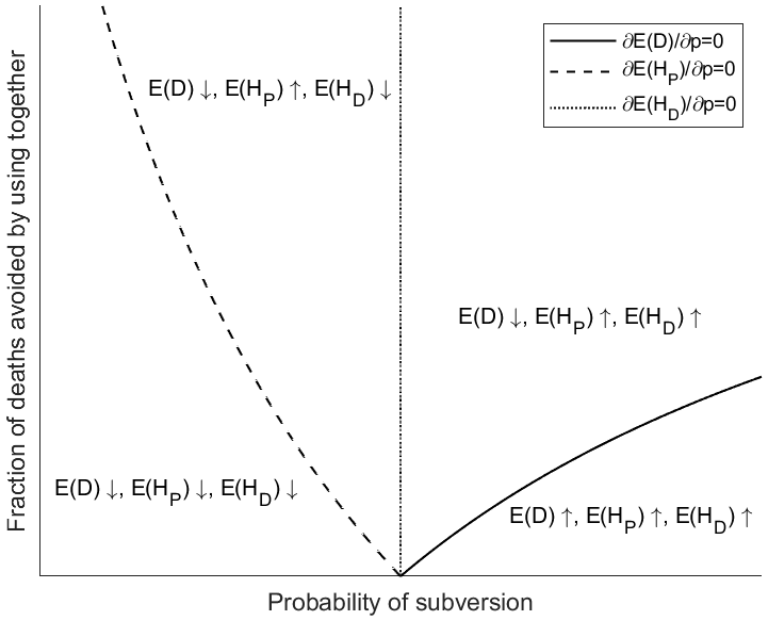
[6] A potential exception to this is among those treated for a drug use disorder involving needle administration. During the period of our data needle exchanges were widely available, accessible, and extensively used (Hordvin, 2012), and harm minimization education formed a core component of inpatient treatment programs. We therefore expect limited potential for needle sharing among peers acquired during inpatient treatment to increase substance related disease.

Table 1: Effect of each mechanism on death and hospital admission

Mechanism	Death (D)	Poisoning admission (H_P)	Disease admission (H_D)
Support recovery	$\downarrow$	$\downarrow$	$\downarrow$
Subvert recovery	$\uparrow$	$\uparrow$	$\uparrow$
Use together	$\downarrow$	$\uparrow$	$\rightarrow$

Our empirical approach captures the probability of peer contact using the (day-weighted) proportion of overlapping inpatients who are from the same geographic location as the focal individual, and estimates the net impact of all three mechanisms (i.e., $\partial E(Y)/\partial p$). When interpreting our findings we can use Table 1 to infer the existence and relative importance of each mechanism. For example, if we were to find empirically that peers increase the probability of death, we could conclude that the subversion mechanism exists. But if we were to find that peers decrease the probability of death, whilst we could not rule out that subversion happens, we could conclude that it is not the dominant mechanism, which must be either supporting recovery or using together. We can then use the empirical results for hospital admissions to establish the extent to which each of these mechanisms is at play.

Figure 2: Net impact of peers on health outcomes



Notes: This figure depicts the net impact of peers on death (D), hospital admission for substance poisoning (H_P) and hospital admission for substance related disease (H_D). The x-axis is the probability of subversion, c. The y-axis is the fraction of deaths avoided by using together, $[E(D|\text{use alone}) - E(D|\text{use together}]/E(D|\text{use alone})$. Here we take $q = 0.3$ and $E(H_P|\text{use alone})/E(D|\text{use alone}) = 5/4$. Varying these parameters changes only the relative size of each region.

We can also make use of Figure 2 when interpreting our empirical findings. Figure 2 depicts how the sign of the net impact of peers ($\partial E(Y)/\partial p$) depends on the primitives c (the probability of subverting recovery) and $[E(D|\text{use alone}) - E(D|\text{use together})]/E(D|\text{use alone})$ (the fraction of deaths prevented by using together rather than alone). The four regions correspond to each feasible combination of signs over our three outcomes.[7] We can use Figure 2 to learn about the primitives of our theory. For example, if we were to find empirically that peers increase the probability of death and both types of hospital admission, we could conclude that we are in the bottom right region, and hence that the probability of subversion is high and the fraction of deaths prevented by using together is low.

2.1 Plausibility of theory: Survey evidence

The link between our theory and empirical analysis hinges on the plausibility that individuals sharing an episode of inpatient treatment maintain contact in the post-treatment period, during which the outcomes of death and hospital admission are measured. To motivate this we draw on evidence from a recent survey of past four week opioid and stimulant users in seven Norwegian cities (Gjersing, 2023), in which we interviewed respondents about social connections formed in inpatient treatment and their role in subsequent recovery. Of the 975 respondents, 72% had previously undertaken inpatient treatment. Among these individuals, 53% reported that they had subsequently been in contact with a peer they met during their last episode of inpatient treatment, suggesting that the formation of long lasting social ties during inpatient treatment is plausible and occurs reasonably frequently. Our theoretical framework also implies that the net impact of peers on substance use is ambiguous.[8] We asked survey respondents whether the peers from their last inpatient treatment episode with whom they stayed in contact affected their substance use in the post-treatment period. Consistent with an ambiguous net impact, 18% reported a decrease in use due to peers and 23% reported an increase in use due to peers.[9]

[7] Since this is fewer than the $2^3 = 8$ empirical outcomes we might observe, our theory can be refuted by the data.

[8] In our model, using the expressions in footnote 4, the impact of peers on any kind of use is $\partial[P(\text{use alone}) + P(\text{use together})]/\partial p = -(1-q)(1-2c)/2$. When $c < 0.5$ peers decrease substance use but when $c > 0.5$ peers increase substance use.

[9] The remaining 59% reported that individuals they met during inpatient treatment and with whom they stayed in contact did not influence their substance use. We also note that one ought not to place any emphasis on the difference between the proportion of respondents reporting that peers increased their consumption and the proportion of respondents reported that peers decreased their consumption. This is because the survey selected past-four-week drug users for interview. In contrast, the administrative sample used in our empirical analysis comprises all individuals who received inpatient treatment for a SUD (including alcohol use disorder), whether or not they continue to use in the post-treatment period.

3 Institutional setting

Norway has a universal public healthcare system funded by general taxation. National policy is determined centrally by the Ministry of Health and Care Services but provision is decentralized to four Regional Health Authorities (Northern Norway, Central Norway, Western Norway and South-Eastern Norway), which oversee 20 Health Trusts.

SUD services are provided directly by hospitals, through psychiatric units at hospitals, by dedicated substance use treatment centers and through privately practicing specialists and non-profit organizations contracted to provide publicly funded treatment. Services include emergency care, detoxification, outpatient treatment and inpatient treatment. We focus on inpatient treatment. Most inpatient treatment occurs at dedicated addiction units but it can also occur within psychiatric units of hospitals. During the period of our data, facilities that provide inpatient treatment ranged in size from 8 to 55 beds, with a median of 19. They often specialize in particular substances (e.g., alcohol), patient sex (e.g., female only) and patient age (e.g., young adults only, typically using a cut off of $\leq$ 26 years old). Facilities also offer different therapies to one another, which may improve patient outcomes, including mortality (Gatti *et al.* , 2020). Patients are typically referred to inpatient treatment by a specialist providing them with another service (e.g., outpatient treatment).[10] The referral may be to a specific treatment facility that the patient is permitted to nominate. Because of differences in the types of patients catered for and therapies offered, patients often do not attend their nearest facility.

Referrals routinely exceed resources. This excess demand is rationed through a prioritized waiting list. During the period of our data, neither the referring physician or the patient observed an estimate of the waiting time at the point of referral. Referrals are sent to a priority-setting unit, which forwards them to a discipline specific assessment panel. Due to the Patients' Rights Act (1999), prioritization for treatment is based solely on clinical need. Assessments do not take into account factors such as sex, ethnicity, socioeconomic status, past harmful behavior, productivity, the available capacity at the facility chosen by the patient, or in the specialist care sector more broadly. Due to these features, a patient cannot choose their date of admission, which is determined instead by the interaction of their referral date and clinical need, the referral dates and clinical needs of others, local resource constraints and the discharge dates of those already in treatment.

Once admitted to a treatment facility and prior to starting treatment, patients undergo detoxification. Detoxification clears substances from the body and manages withdrawal symptoms. It takes around one week for alcohol and cannabis, and two weeks for other illicit drugs. After detoxification, a typical treatment plan comprises therapy (cognitive behavioral therapy and motivational interviews), group therapy, environmental therapy, education (including harm minimization and overdose management) and physical activity. During treatment, patients interact routinely with

[10]Direct referrals from a general practitioner are also possible. While patients cannot be directly referred by the criminal justice system, a small number of individuals may serve a proportion of their incarceration in inpatient treatment.

one another, both informally as residents of the facility and formally as part of their treatment.

A patient may be discharged on completion of their treatment or if they no longer wish to continue or if treatment is ineffective. At discharge, the patient attends a meeting to identify a plan for follow up in the community, to ensure that the patient has a suitable home, and to receive information on community services for continued recovery and how to access future treatment.

4 Data

We access Norwegian administrative data linking inpatient treatment records from the Norwegian Patient Registry (NPR) with the Norwegian Cause of Death Registry and the Norwegian Population Registry. The cohort for which linking was undertaken includes all individuals who were in the specialist healthcare sector with a primary diagnosis of a SUD (ICD-10 codes F10-F16 and F18-F19) between 1 January 2009 and 31 December 2010. These individuals were identified in the NPR, which covers all patients in the specialist healthcare system. For each inpatient episode, the NPR provides information on the diagnosed illness (ICD-10 codes) and type of care (e.g., emergency care or treatment), the start and end dates for the episode, the sector (specialized interdisciplinary or psychiatric), and for treatment episodes in public facilities, the geographic location (Health Trust). The NPR was uniquely linked by Statistics Norway to the Norwegian Cause of Death Registry and to the Norwegian Population Registry, which contains demographic and socioeconomic information, including county of residence.[11] For each patient in our analysis cohort, all episodes in specialist healthcare recorded up to 31 December 2013 were extracted from the NPR, and all records up to 31 December 2013 were extracted from the Cause of Death Registry.

Combining the patient level data with supplementary facility level data on the Health Trust, sector, and the types of patients catered for (sex, age range and substance), we matched patients to the facility at which their treatment was provided. For a minority of patients we do not have sufficient information to uniquely identify the facility, in which case we keep track of the list of facilities that they could have attended. To assess the match quality we can compare the implied occupancy with the number of beds.[12] Since the aggregate demand for care routinely exceeds capacity, low quality matching would to lead to a significant fraction of facilities appearing to operate above capacity. According to our matching, capacity was exceeded on fewer than 5% of facility-days.

[11] County of residence is not recorded at the time of inpatient admission but instead on January 1 2010. However, we do not expect there to be much mobility of patients between counties during our sample period due to the short window considered, the fact that counties are relatively large, and the fact that migration rates are lower among low SES individuals (as measured by education) in Norway (Godøy & Huitfeldt, 2020), such as those in our sample.

[12] For this exercise, if a patient could have attended more than one facility, we distribute them fractionally across each possible facility.

4.1 Sample

The sample is constructed from all inpatient episodes for treatment for a SUD that occurred in one of 39 public facilities and which include at least one day of inpatient treatment during the period 1 January 2009 to 31 December 2010. Since patients do not interact during detoxification, we shift the date of admission forward by the period of detoxification to obtain the start date of treatment. This leads to a sample size of 4,856 treatment episodes. Of these, 41% of episodes are for alcohol use disorders and 59% are for drug use disorders, of which the most common is opioid use (37%), followed by multiple drug use (28%), stimulants other than cocaine (15%) and cannabis use (12%). This sample is used to construct the peer exposure variable (described below).

The analysis sample uses only the first inpatient episode for each individual that starts between 1 January 2009 and 31 December 2010, and ends by 31 December 2010 (N=4,257). These are the patients for which we are able to observe all other patients with an overlapping treatment episode. We exclude a further 369 observations for patients who attend specialized interdisciplinary facilities in Central Norway, as this health region does not report specialist treatment at the Health Trust level (which implies that we cannot match patients to facilities), 16 observations for which there are more than four facilities that they may have attended, and 132 due to missing controls, resulting in a sample size of N=3,838. Of these, we lose another 71 observations from our fixed effects analysis because they are singletons, leading to an estimation sample of N=3,767. We can uniquely identify the facility attended for 57% of these patients. There are two potential facilities for 15% of patients, three potential facilities for 21% of patients, and four possible facilities for 6% of patients.

4.2 Outcomes

We consider outcomes related to death and hospital admission. The death registry data contains the date of death and one or more ICD-10 codes for the underlying cause. The outcomes we consider are indicators for death due to all causes, death due to external causes, and death due to causes other than external causes, which we refer to as internal causes for simplicity. External causes include accidents (e.g., accidental poisonings), intentional self harm (including suicide by intentional poisoning) and homicide. Poisonings (accidental and intentional) include deaths in which an illicit drug overdose is the cause of death. Internal causes of death generally relate to disease.[13]

We also consider substance induced deaths. Substance induced deaths are defined as deaths that are either drug induced or alcohol induced. Drug induced deaths are those caused by a mental-behavioral disorder due to psychoactive substance use (opioids, cannabinoids, cocaine,

[13]Internal causes include those due to infection or parasites, cancer, blood or immune system disorders, endocrine or metabolic system disorders, nervous system disorders, circulatory system disorders, respiratory system disorders, digestive system disorders, skin disorders, musculoskeletal disorders, genitourinary disorders, congenital malformations, and other abnormalities.

Table 2: Descriptive statistics: Outcomes and peer exposure

	Mean			
	within 1 year	within 3 years		
Death (0/1)				
all-causes	0.022	0.067		
internal	0.011	0.033		
external	0.012	0.033		
substance induced	0.007	0.023		
Any hospital admission (0/1)				
poisoning	0.030	0.067		
liver disease	0.038	0.076		
	Mean	Std. dev.	Min	Max
Peers				
days in treatment	28.46	50.66	1	619
num. overlapping inpatients	36.76	49.10	1	700
num. overlapping inpatients from same county	17.70	27.63	0	403
peer exposure	0.55	0.31	0	1
Observations	3,767			

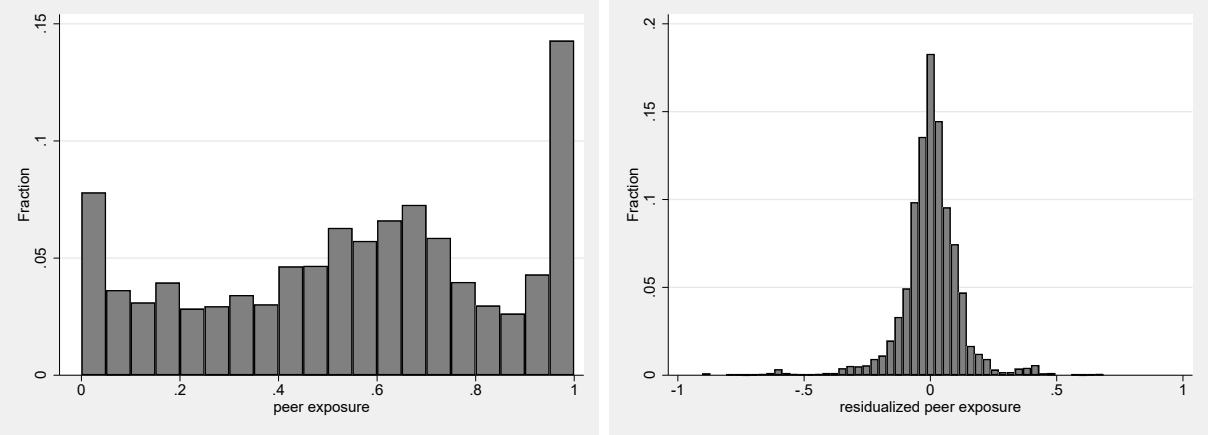
other stimulants, hallucinogens, multiple drug use) or poisoning (accidental, intentional or of undetermined intent) by opium, heroin, other opioids, methadone, other synthetic narcotics, cocaine, other and unspecified narcotics, cannabis, lysergide, other and unspecified psychodysleptics, or psychostimulants.[14] Alcohol induced deaths are deaths from alcohol abuse, degeneration or damage to the nervous system due to alcohol, death due to alcoholic cardiomyopathy, gastritis, liver disease, or pancreatic disorders, and poisoning by alcohol (accidental, intentional, undetermined intent). Table 2 reports descriptive statistics for our death related outcomes. It shows that 2.2% of our analysis sample die within a year of leaving inpatient treatment and 6.7% die within three years. Approximately half of deaths are due to external causes and around a third are substance induced.

We also consider outcomes measuring two kinds of (non-fatal) hospital admission: those due to substance related poisoning, and those due to substance related disease.[15] To measure poisoning admissions, we construct an indicator which takes the value one if we observe at least one hospital admission for which the ICD-10 code indicates either poisoning or acute intoxication induced by drug or alcohol use. To measure disease related admissions we use chronic liver disease, which is

[14]This selection was agreed by the EMCDDA Expert Group on Drug-related deaths. It was called "Selection B" for General Mortality Registries based on ICD-10.

[15]In contrast to mortality, we only observe the calendar year of hospital admission. For a patient leaving treatment in year y we therefore construct indicators for any hospital admission occurring in years $y+1$ to $y+c$, and refer to this as an admission within c years of leaving treatment.

Figure 3: Distribution of peer exposure: Raw and residualized with respect to fixed effects



associated with both drug use and alcohol use (Pateria *et al.* , 2013). We construct an indicator which takes the value one if we observe at least one hospital admission for which the ICD-10 code indicates either toxic liver disease, alcoholic liver disease or chronic hepatitis.

4.3 Measuring peer exposure

We use overlapping episodes of inpatient treatment to define peer exposure. We expect inpatients to form social ties during their inpatient treatment because they live together and interact both informally and formally as part of their treatment. Since the treatment facilities are reasonably small (8 to 55 beds with a median of 19), all patients have the opportunity to form social ties with one another. Following the literature showing the importance of geographically proximate peers in risky behaviors (Adamopoulou *et al.* , 2024; Billings & Schnepel, 2022; Case & Katz, 1991; Christakis & Fowler, 2007; Dustmann & Landersø, 2021; Kling *et al.* , 2005, 2007; Ludwig *et al.* , 2001; Stevenson, 2017), and the importance of geography in forming and maintaining social ties more broadly (e.g., Kim *et al.* (2023)), we define peers as others with whom the focal individual shared their inpatient treatment episode and who are resident in the same geographic area. We expect SUD patients with overlapping episodes of inpatient treatment, and who are residents in the same geographic area, to form social ties that can impact their substance use behavior, and through that, their health outcomes in the post-treatment period. In the appendix we consider definitions of peers based on common age and sex (i.e., homophily) and common age, sex and area of residence. We find that overlapping treatment episodes with geographically proximate individuals drive mortality to a greater extent than those of the same age and sex.

The geographic unit on which our peer measure is based is the county. This reflects the data we have access to, but also ensures a large enough number of individuals receiving inpatient treatment for a SUD residing in each geographic area so as to generate sufficient variation from overlapping

treatment episodes. During the period of our data there were 19 Norwegian counties. A median sized county had around 519 injecting drug users and 13,903 problematic alcohol users.[16] Those with a SUD tend to be concentrated in urban centers (Abuse & Administration, 2022; Gjersing, 2023; Swensen, 2015), of which the median Norwegian county had one, up to a maximum of three.[17] The population of individuals with a SUD in a typical county is thus reasonably small and likely to be concentrated in its urban center. In our estimation sample, the number of inpatients residing in each county ranges from 19 (Nord-Trøndelag) to 815 (Oslo) with a median of 133.

Following the literature on criminal recidivism (e.g., Bayer *et al.* (2009); Damm & Gorinas (2020); Johnsen & Khoury (2024)) we use the proportion of peers rather than the count. The peer count is correlated with the duration of treatment and the occupancy of the treatment facility, hence potentially with severity of illness, and quantity and quality of treatment. We measure peer exposure using the proportion of all inpatients with whom the focal patient's treatment episode overlaps and who reside in the same county, weighted by the number of days overlapped. Day-weighting is appropriate if the opportunities for interaction are approximately equal across all inpatients attending the facility on a given day. If the facility that the patient could have attended is not unique, we use all inpatients who *could* have overlapped to construct the peer variable. This introduces some measurement error, which may attenuate our estimates towards zero.[18]

The bottom panel of Table 2 shows that the mean number of days in treatment is 28.46 (the median is 9). During that time, the mean number of other patients with overlapping episodes is 36.76, of which 17.7 reside in the same county. The mean (day-weighted) exposure to peers from the same county in our sample is 55%. Figure 3 shows that there is ample variation in exposure to peers, even after partialling out the fixed effects that we employ.

4.4 Controls

To absorb residual variation we use the following pre-admission patient characteristics: the type of SUD (drug use or alcohol use), sex, age, education (an indicator for lower secondary only), parents' education (an indicator for both having lower secondary only), an indicator for single marital status, and an indicator for a mental health comorbidity at the point of admission. We also form day-weighted averages of these variables over all others with overlapping treatment

[16] In 2009, the prevalence of injecting drug users among 15-64 year olds was estimated to be about 0.33%. This figure is based on the midpoint of the estimated range of 8800 to 12500 injecting drug users nationally (SIRUS, 2011), and a population of 15-64 year olds of 3,200,276. In 2011, the population size of Norway's 19 counties ranged from 73,417 (Finnmark) to 599,230 (Oslo) with a median of 235,493, where 15-64 year olds account for 66% of the population, implying 519 injecting drug users in a median sized county. The prevalence of alcohol use disorder was estimated to be 7.2% of individuals aged 15 or older (WHO, 2018), which translates to around 13,903 in a median sized county.

[17] This figure is based on Norwegian cities (*byer*) with a population exceeding 30,000 in 2011 (as city status is sometimes conferred on the basis of factors such as historical importance or administrative function).

[18] Measurement error is also a feature of the criminal recidivism literature. For example, Bayer *et al.* (2009) only observe prisoners who are *released* during a given window, and correct for the potential peers that they do not observe using the expected number of days of overlap.

Table 3: Descriptive statistics: Controls

	Variables	Mean	St Dev.	Min	Max
Individual controls					
	drug (0/1)	0.56			
	male (0/1)	0.69			
	minimum ed. (0/1)	0.58			
	both parents min. ed. (0/1)	0.26			
	single (0/1)	0.63			
	mental illness (0/1)	0.19			
	age	41.03	13.38	17	85
	$age^2/1000$	1.86	1.15	0.29	7.23
Contextual controls					
	drug	0.70	0.21	0	1
	male	0.69	0.19	0	1
	minimum ed.	0.58	0.19	0	1
	both parents min. ed.	0.23	0.16	0	1
	single	0.63	0.19	0	1
	mental illness	0.26	0.20	0	1
	young (age$\leq$ 26)	0.19	0.24	0	1
Observations		3,767			

episodes to account for contextual peer effects (Manski, 1993).

Table 3 reports descriptive statistics. Around 56% of patients receive treatment for a drug use disorder, with the remaining 44% treated for an alcohol use disorder. Patients are typically male (69%), have a low level of education (58% have only lower secondary) and are single (63%). They have an average age of 41, 19% have a mental illness, and 26% come from a disadvantaged background, as measured by both parents having only lower secondary education. The contextual controls have similar means but smaller standard deviations owing to their being weighted averages.

5 Empirical strategy

Since we do not observe post-treatment contact among inpatients receiving treatment for a SUD, we model a reduced form relationship between post-treatment outcomes and a proxy for the probability of post-treatment peer contact (the parameter p in our theoretical framework), which we measure using exposure to other inpatients residing in the same geographic area during inpatient

treatment.[19] We use the following linear probability model,

$$Y_{icjt} = \beta PeerExposure_{icjt} + \mathbf{x}'_{icjt}\gamma + \mu_t + \alpha_{jc} + u_{icjt},$$

where Y_{icjt} is an indicator for the health outcome of individual i who resides in county c and who was treated at facility j until exiting on date t within a fixed time period (one or three years) after leaving treatment; $PeerExposure_{icjt}$ measures exposure to geographically proximate peers during inpatient treatment; $\mathbf{x}_{icjt}$ includes controls; μ_t is a fixed effect for month-by-year of exit; α_{jc} is a facility-by-county-of-residence fixed effect; and u_{icjt} is an error term. Our model includes both *contextual peer effects* and *correlated effects* (Manski, 1993). Contextual peer effects arise when the outcome depends on the type of other individuals in the reference group (captured by our contextual controls), whereas correlated effects arise when individuals in the same reference group have similar outcomes due to their exposure to a common environment (captured by the fixed effects).[20]

5.1 Identification

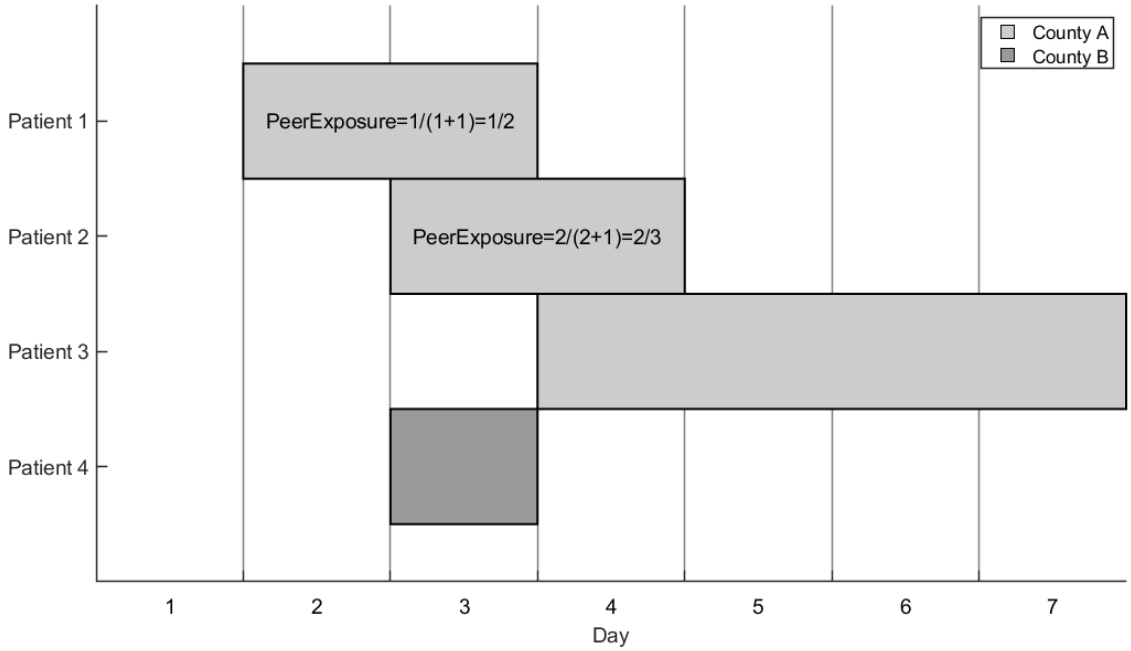
Our identification strategy leverages variation from overlapping inpatient treatment episodes. This type of variation has previously been used in the context of criminal recidivism (Bayer *et al.* , 2009; Billings & Schnepel, 2022; Damm & Gorinas, 2020; Johnsen & Khoury, 2024; Ouss, 2011; Tan & Zapryanova, 2022). Key to our approach is the presence of the facility-by-county fixed effect, α_{jc}. This ensures that the identifying variation comes exclusively from comparison of inpatients attending the same facility and who live in the same county, whose peer exposure varies only due to the timing of their episodes. For this reason our findings are not driven by selection into treatment facilities. Absent county-by-facility fixed effects we might worry, for example, that patients select into treatment facilities located outside their county of residence based on unobservable drivers of their health outcomes, and, so may encounter fewer other inpatients resident in their county, and hence have lower peer exposure.

Figure 4 provides a stylized illustration of the identifying variation that we use. It shows hypothetical inpatient treatment episodes of four patients attending the same facility at around the same time. Patients 1 and 2 both reside in County A but patient 1 enters and exits treatment one day earlier than patient 2. Due to this difference in timing, patients 1 and 2 have different levels of day-weighted peer exposure (i.e., there is within facility-by-county variation). This is because patient 1 exits before patient 3 arrives but patient 2 does not. Due to this, we expect patient 2 to be more likely to interact with others in the post-treatment period. It is this comparison of the

[19] Few studies of peer effects directly observes contact among peers, nor which social ties are actually formed. Instead, as in our case, the focus is on modeling the relationship between outcomes and the social ties that *could* potentially be formed.

[20] Our empirical model does not include *endogenous peer effects*. Endogenous peer effects arise when the outcome of the focal individual depends directly on the outcomes of the other individuals in the reference group.

Figure 4: Within county-by-facility variation in peer exposure



post-treatment health outcomes that facilitates identification of the impact of peers.

We now discuss threats to identification and how we address them. The extent to which an inpatient's episode overlaps with others is driven partly by their own dates of entry and exit, and partly by the entry and exit dates of others. Since we do not think it credible that a given patient may manipulate others' dates, we focus the discussion on the extent to which a patient might select their own entry and exit dates.

The institutional environment implies that the timing of admission to an inpatient facility is determined by the interaction of a prioritized waiting list and the discharge dates of those receiving inpatient treatment prior to the focal patient (i.e., when beds become available). Although we are not able to determine the full time between referral and admission to a treatment facility (because our data does not contain the date of referral), we can measure the wait time from assessment to admission. The mean and median wait times from assessment to admission are 83.35 and 30 days respectively. Around a third of inpatients are admitted within seven days of assessment and around a third are not admitted within 60 days. Given that the expected wait time exceeds 83 days, and that the date of admission is partly driven by others' discharge dates, we consider it unlikely that the inpatients in our sample are able to systematically manipulate their date of entry into inpatient treatment.

A related concern is that individuals with pre-existing social ties might seek to overlap in inpatient treatment. To investigate the extent to which this is feasible, we examine the variation in admission dates among inpatients residing in the same county, attending the same facility, and

assessed in the same week, by residualizing the number of days from assessment to admission with respect to a fixed effect for county-by-facility-by-week of assessment for our estimation sample. The standard deviation of this residual is 47.67 days. This is larger than the mean number of days in treatment, which is 28.46. Since the distributions of wait time and treatment duration are both skewed to the right, we also compare quantile based measures. The interquartile range of the residualized wait time is 28.8 days whereas the median number of days in treatment is 9 days. These statistics imply that individuals seeking to overlap in inpatient treatment would tend to be unsuccessful, which is likely to be because they cannot manipulate the dates that beds become available. For this reason, we do not think it credible that our findings are driven by individuals seeking to overlap in inpatient treatment.

In our view the predominant threat to identification is endogenous exit from treatment. Endogenous exit may be more salient in our context than that of criminal recidivism because, whilst prisoners may indirectly influence their release date through their behavior, inpatients receiving treatment for a SUD may discharge themselves at any time. For example, inpatients could elect to leave treatment once they feel they have acquired a sufficiently supportive peer group, and doing so would cut short their inpatient treatment, which might also determine their post-treatment health outcomes. To address such concerns we conduct three robustness tests. First, we use an alternative measure of peers, where we only use the first day of the focal individual's treatment episode to construct the peer variable. Variation in exposure to peers on the first day of treatment cannot be driven by endogenous exit. Second, we consider treatment duration as an outcome, and show that it does not depend on our primary measure of peer exposure or that based only on the first day. Third, we repeat our analysis but define outcomes to be indicators of death within a fixed time period of *entry* into treatment and use month-by-year of entry fixed effects.

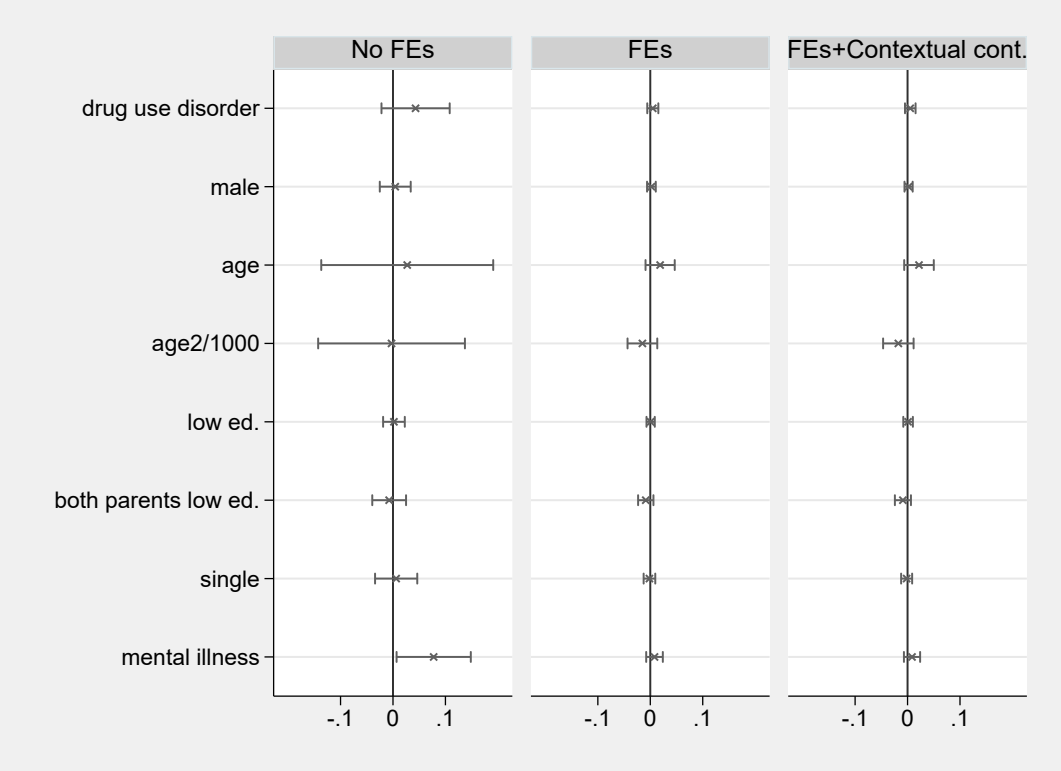
5.2 Balance tests

Figure 5 examines the relationship between peer exposure and observable pre-admission patient characteristics. It reports coefficient estimates and 0.95 confidence intervals from three separate regressions in which peer exposure is the dependent variable. The first includes individual controls, the second adds the full set of fixed effects, and the third also adds contextual controls. In all three specifications, patient characteristics, including those measuring severity of illness (drug use disorder and mental illness) are not individually significantly associated with peer exposure, with the exception of the coefficient on the indicator for mental illness in the specification that does not include fixed effects or contextual controls. Further, when including fixed effects, these individual characteristics are not jointly associated with peer exposure at any conventional significance level. Though we do not report it here, we also find no evidence that peer exposure is associated with any of the contextual controls, including those measuring the severity of illness of other inpatients. We consider two alternative balance tests in the appendix, both of which yield the same conclusion.

6 Results

In this section we begin by considering the impact of peers on death before examining their impacts on hospital admissions. We focus initially on death for two reasons. First, from a social welfare and a public health perspective, death is clearly the most policy relevant health outcome associated with substance use (and its treatment). People with SUDs are at elevated risk of death compared to the general population, and are at greatest risk following a period of abstinence from substance use, such as release from an inpatient treatment facility or from prison (Binswanger *et al.* , 2007; Gossop *et al.* , 2002; Ravndal & Amundsen, 2010). Second, analysis of death allows us to begin to unpick the mechanisms through which peers drive substance use. In Table 1 we showed that we can eliminate subversion of recovery as the dominant mechanism if we can empirically rule out that peers increase the probability of death.

Figure 5: Balance test



Notes: The figure shows coefficient estimates and 95% confidence intervals based on standard errors clustered at the facility level from regression in which the dependent variable is peer exposure. To represent all regressors on the same axis, we rescale the age variables to have standard deviation equal to one. All other regressors are binary.

Table 4: Death

	Death within 1 year of exit			Death within 3 years of exit		
	All	Internal	External	All	Internal	External
peer exposure	-0.061**	-0.011	-0.050**	-0.079***	-0.014	-0.059**
	(0.025)	(0.006)	(0.011)	(0.025)	(0.016)	(0.023)
	[0.07]	[0.36]	[0.07]	[0.02]	[0.36]	[0.06]
Mean dep. var.	0.022	0.011	0.012	0.067	0.033	0.033
+1 peer (%)	-10.25	-3.76	-16.15	-4.41	-1.57	-6.83
Observations	3,767	3,767	3,767	3,767	3,767	3,767
R-squared	0.055	0.052	0.052	0.059	0.074	0.046

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 4.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1. Multiple-testing adjusted p-values are in brackets.

6.1 Mortality analysis

Table 4 reports our findings for the impact of peers on death within one and three years of exiting inpatient treatment. Columns one to three respectively consider all deaths, deaths from internal causes, and deaths from external causes in the first year after exiting inpatient treatment. Columns four to six consider the same outcomes but over a three year horizon, allowing us to evaluate the extent to which deaths are prevented or merely delayed. Standard errors account for clustering at the facility level. Since we consider multiple outcomes, in brackets we provide multiple-testing adjusted p-values for statistical significance using the stepwise method proposed by Romano & Wolf (2005a,b).

Looking at the first column of Table 4, we find that peers reduce mortality risk in the first year after exiting treatment. Without adjusting for multiple testing, the coefficient is statistically significant at the 0.05 level. The multiple-testing adjusted p-value is 0.07. The row denoted '+1 peer (%)' considers the impact of an additional peer.[21] Being treated alongside one additional peer for the duration of the treatment episode reduces all-cause mortality in the year following treatment by around 10% of the mean. Looking at the second and third columns of Table 4 we observe that the peer-induced reduction in all-cause mortality is driven by a reduction in deaths from external causes, with no evidence of any impact of peers on internally caused deaths. Looking at columns four to six of Table 4, we observe only marginally larger point estimates for peer-induced reductions in 3-year mortality compared to 1-year mortality. This suggests that peers have the

[21] When making this calculation we suppose that the additional peer overlapped with the entire duration of the focal individual's episode. Denoting $PeerExposure = m/n$ where m is the total number of days overlapped with home-county peers and n is the total number of days overlapped with all inpatients, the counterfactual peer exposure we consider is $PeerExposure = (m+d)/(n+d)$ where d is the number of days the focal individual received treatment. The difference between peer exposure and its counterfactual varies across individuals, so we report its mean.

greatest impact in the first post-treatment year, and that deaths are prevented rather than delayed, at least within the time horizon available to us.

In the appendix we provide evidence that our baseline mortality estimates are credible through two placebo tests and various robustness tests. The first placebo test shifts the start and end dates of the focal individual's inpatient treatment episode when constructing their measure of peer exposure, assigning them a placebo group of potential peers who received treatment in the same facility at around the same time, but had no (or limited) overlap. We find no impact of 'peers' on mortality (appendix Figures 8 and 9). The second placebo test uses a sample of emergency care episodes. Emergency patients are admitted because they pose a risk to themselves or others. They do not interact with others and do not receive treatment, so should not experience any peer effects. Again, we find no impact of 'peers' on mortality (appendix Table 11). Together, our placebo tests provide empirical evidence that our baseline findings are unlikely to be driven by some artifact of inpatient admission other than peer effects.

Our robustness tests demonstrate that our baseline mortality estimates are unlikely to be driven by endogenous exit. To demonstrate this we show that our findings are robust to the use of an alternative measure of peers based on the first day of the focal individual's treatment episode and to defining outcomes in terms of death within a fixed time period of *entry* into treatment (appendix Table 12), and by showing that treatment duration does not depend on our primary measure of peer exposure or that based only on the first day of the episode (appendix Table 13). The final set of robustness tests show that our findings are not sensitive to the choice of econometric model for the rare outcomes we consider (appendix Table 14). Further details of our placebo and robustness tests are provided in the appendix.

6.2 Do peers influence substance use behavior?

In our theoretical framework, peers alter the substance use behaviors of abstaining from use, using alone, and using together by influencing the relative probabilities of each behavior. Empirically, if peers influence mortality by altering these behaviors, we would expect to observe that exposure to peers has an impact on deaths that are linked to substance use, provided that their probability could reasonably be expected to adjust to changes in substance use over the three year post-treatment time-frame available to us. For example, we might expect the risk of fatal overdose (an external cause) to adjust nearly instantaneously to peer induced changes in substance use, but we would not expect this to be true for mortality due to substance related disease (an internal cause), the risk of which is likely to adjust considerably more slowly, even for diseases which are strongly linked to substance use. The baseline findings in Table 4 show this to be the case, with peers reducing the probability of death from external causes (but not internal causes) starting from the first year post-treatment.

Direct evidence that peers influence substance use behavior can be obtained by examining

Table 5: Substance induced death

	SID within 3 years of exit		
	All	Internal	External
peer exposure	-0.029**	-0.005	-0.024**
	(0.012)	(0.007)	(0.011)
	[0.02]	[0.28]	[0.03]
Mean dep. var.	0.023	0.009	0.014
+1 peer (%)	-4.72	-2.14	-6.42
Observations	3,767	3,767	3,767
R-squared	0.034	0.029	0.049

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 4.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1. Multiple-testing adjusted p-values are in brackets.

deaths intrinsically linked to substance use. Table 5 presents results for the impact of peers on substance induced deaths. We focus on a three year post-treatment time-frame since substance induced deaths are reasonably rare, equating to only 0.7% of sample members in the first year post-treatment but rising to 2.3% by the third. The first column shows that peers reduce the probability of substance induced deaths within three years of exiting treatment. The second and third columns show that the effect is driven by externally caused substance induced deaths (overdoses) as opposed to internally caused substance induced deaths (substance related disease).

6.3 How do peers influence substance use behavior?

Table 1 summarizes the theoretical impacts of peers on health outcomes associated with each of the three potential mechanisms of subvert recovery, support recovery and use together. Comparing these predictions to our empirical finding that peers *reduce* deaths implies that the dominant mechanism cannot be subversion of recovery. However, both the support and use together mechanisms could explain a peer induced reduction in mortality. We can separate these two mechanisms by examining hospital admissions. If peers decrease deaths mainly by supporting one another to abstain, we should find that they reduce hospital admissions for both substance related poisonings and substance related disease. In contrast, if peers decrease deaths mainly through using more safely together, we should find that they increase hospital admissions for poisoning (as deaths are displaced) but have no effect on admissions for substance related disease.

Table 6 reports results for hospital admissions. The first two columns respectively examine admissions for substance related poisoning and liver disease within 1 year of exiting treatment. We find a positive but small in magnitude and statistically insignificant impact of peers on admissions for poisoning and a negative impact of peers on admissions for liver disease, which is statistically significant at the 0.05 level. Columns three and four examine admissions within three years of exit.

Table 6: Substance related hospital admission

	Any admission within 1 year of exit		Any admission within 3 years of exit	
	Poisoning	Liver disease	Poisoning	Liver disease
Peer exposure	0.020	-0.041**	-0.014	-0.071**
	(0.030)	(0.019)	(0.036)	(0.028)
	[0.59]	[0.02]	[0.66]	[0.02]
Mean dep. var.	0.070	0.038	0.157	0.078
+1 peer (%)	1.45	-4.43	-0.42	-3.53
Observations	3,767	3,767	3,767	3,767
R-squared	0.071	0.035	0.084	0.041

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 4.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1. Multiple-testing adjusted p-values are in brackets.

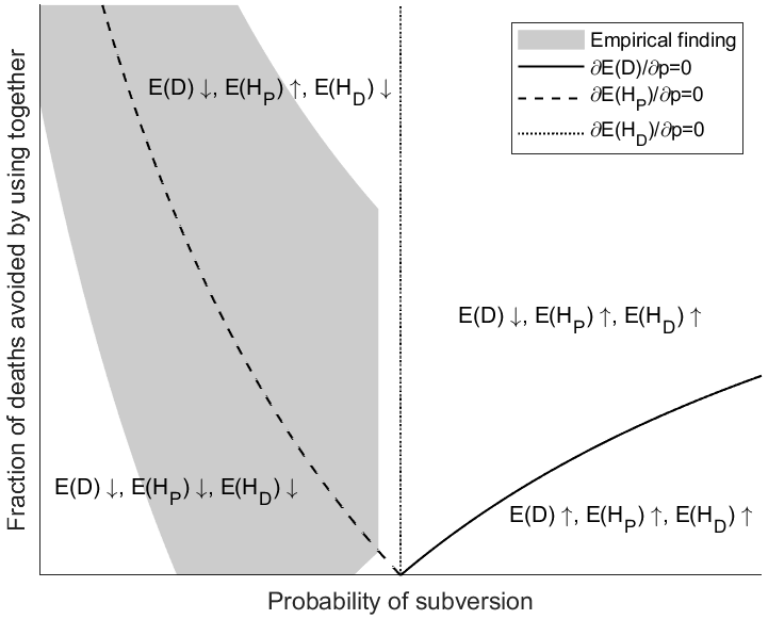
We find a negative but statistically insignificant impact of peers on admissions for substance related poisoning and a negative impact of peers on admissions for liver disease, which is statistically significant at the 0.05 level.

The finding that greater peer exposure reduces hospital admissions for liver disease at one and three years post-treatment provides evidence that peers support recovery from SUDs. And this support to abstain partly accounts for the peer induced reduction in mortality we find. The finding of no effect of peers on admissions for poisoning may reflect the offsetting effects of the support to abstain mechanism and the use together mechanism, since they work in opposing directions.[22]

Figure 6 highlights the region of the theoretical primitives that is compatible with our empirical finding that peers reduce deaths (D) and hospital admissions for substance related disease (H_D). Our findings suggest that peers are more likely to support one another rather than subvert one another (i.e., the probability of subvert is $c < 0.5$ so the probability of support is $1-c > 0.5$). This is because peers can only reduce hospital admissions for disease by reducing total use (since using alone or together with a peer makes no difference for disease), which happens only when $c < 0.5$. However, since we found no empirical evidence of any effect of peers on poisoning admissions, we can learn little about the fraction of deaths avoided by using together with a peer.

[22] An alternate explanation is that overdose is not well measured using hospital admission because a majority of non-fatal overdoses are dealt with through ambulance call outs and emergency department treatment, which is not included in out data. Another explanation is that we do not have sufficient power to detect a small effect. A fourth is survivor bias. If peers prevent deaths (as we find) they may mechanically increase hospital admissions since only those who survive may be admitted to hospital. A fifth is measurement error in the peers variable, since the facility attended cannot always be uniquely determined, as discussed in Section 4.3.

Figure 6: Net impact of peers on health outcomes: Empirical findings



Notes: This figure depicts the net impact of peers on death (D), hospital admission for substance poisoning (H_P) and hospital admission for substance related disease (H_D). The x-axis is the probability of subversion, c. The y-axis is the fraction of deaths avoided by using together, $[E(D|\text{use alone}) - E(D|\text{use together}]/E(D|\text{use alone})$. Here we take $q = 0.3$ and $E(H_P|\text{use alone})/E(D|\text{use alone}) = 5/4$. Varying these parameters changes only the relative size of each region.

6.4 Which peers influence substance use behavior?

A final piece of evidence comes from examining which types of peer drive our findings. Our theoretical framework highlighted three mechanisms through which peers might play a role, each of which we might reasonably expect to be stronger among peers treated for the same substance relative to different substances. Strategies to recover are likely to be substance specific, hence peers with experience of the same substance might more effectively support one another to abstain. Using together with such a peer may be more effective in preventing adverse outcomes since they are likely to be better able to recognize and respond to early signals of distress. However, such peers may also be more likely to subvert recovery, due to access effects, for example. To examine this, we divide our sample into those treated for a drug use disorder and those treated for an alcohol use disorder, and separate the peer variable into exposure to peers with a drug use disorder and an alcohol use disorder.[23]

Table 7 examines deaths and hospital admissions among those treated for a drug use disorder. The first two columns consider all deaths and substance induced deaths within three years of exiting

[23]We additively decompose our baseline peer variable into two parts: the day-weighted proportion of overlapping inpatients residing in the same county and who have a drug use disorder; and the day-weighted proportion of overlapping inpatients residing in the same county and who have an alcohol use disorder.

Table 7: Inpatients treated for drug use disorder

	Death within 3 years of exit				Any admission within 3 years of exit	
	All	Substance induced	All	Substance induced	Liver disease	Liver disease
peer exposure	-0.100***	-0.055***			-0.065	
	(0.034)	(0.020)			(0.040)	
	[0.06]	[0.06]			[0.31]	
drug peer exposure			-0.113***	-0.065***		-0.071*
			(0.039)	(0.021)		(0.036)
			[0.06]	[0.06]		[0.15]
alcohol peer exposure			-0.046	-0.013		-0.041
			(0.066)	(0.038)		(0.079)
			[0.78]	[0.78]		[0.78]
Mean dep. var.	0.053	0.022	0.053	0.022	0.087	0.087
+1 peer (%)	-8.14	-10.68	-14.46	-19.85	-3.23	-5.53
			-8.87	-5.99		-4.82
Observations	2,118	2,118	2,118	2,118	2,118	2,118
R-squared	0.080	0.059	0.081	0.060	0.067	0.067

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 4.4. Standard errors (in parentheses) are clustered by facility. ∗ ∗ ∗: p<0.01. ∗∗: p<0.05. ∗: p<0.1. Multiple-testing adjusted p-values are in brackets.

treatment. Both coefficients are negative and both are statistically significant. An additional peer reduces all cause mortality of patients treated for a drug use disorder by 8% of the mean, and substance induced mortality by 11% of the mean. Columns three and four separate peer exposure by substance type. The estimates suggest that peers with a drug use disorder have the largest impact on our sample of inpatients receiving treatment for a drug use disorder, whereas peers with an alcohol use disorder have a smaller (negative) and statistically insignificant effect. Columns five and six consider hospital admission for liver disease. We find a statistically insignificant negative impact of peers. In column six we find that peers with a drug use disorder have a larger negative impact than peers with an alcohol use disorder. These findings are consistent with peer influence on substance use behavior being stronger among peers receiving treatment for the same substance.

Table 8 provides analogous results but for the sample of patients treated for alcohol use disorder. In contrast to those treated for a drug use disorder, we do not find evidence that peers reduce all cause or substance induced mortality within three years of exiting inpatient treatment. This is likely because the drivers of mortality are different for alcohol use and drug use disorders. As shown in the descriptive statistics in Table 16 in the appendix, individuals with a drug use disorder are more likely to die from external causes (e.g., overdose), especially given that the risk of overdose is elevated following periods of inpatient treatment (Gossop *et al.* , 2002; Havard *et al.* , 2023;

Table 8: Inpatients treated for alcohol use disorder

	Death within 3 years of exit				Any admission within 3 years of exit	
	All	Substance induced	All	Substance induced	Liver disease	Liver disease
peer exposure	-0.066	0.005			-0.103**	
	(0.057)	(0.029)			(0.050)	
	[0.58]	[0.58]			[0.18]	
drug peer exposure			-0.049	-0.005		-0.091*
			(0.046)	(0.030)		(0.045)
			[0.64]	[0.92]		[0.21]
alcohol peer exposure			-0.101	0.023		-0.125
			(0.117)	(0.040)		(0.117)
			[0.69]	[0.73]		[0.64]
Mean dep. var.	0.085	0.025	0.085	0.025	0.066	0.066
+1 peer (%)	-2.35	0.57	-3.19	-1.02	-4.67	-7.69
			-8.19	6.42	-13.03	
Observations	1,631	1,631	1,631	1,631	1,631	1,631
R-squared	0.082	0.052	0.082	0.052	0.059	0.059

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 4.4. Standard errors (in parentheses) are clustered by facility. ∗ ∗ ∗: p<0.01. ∗∗: p<0.05. ∗: p<0.1. Multiple-testing adjusted p-values are in brackets.

Ravndal & Amundsen, 2010). In contrast, those with an alcohol use disorder are more likely to die from internal causes such as disease related to alcohol misuse (e.g., liver disease). Since deaths from overdose respond rapidly to changes in substance use, yet deaths from alcohol related disease respond slowly, it is not surprising that we find stronger evidence that peers impact the mortality of those with a drug use disorder over the reasonably short post-treatment time-frame available to us. Columns five and six examine hospital admission for disease. In column five we find evidence that peers reduce hospital admissions for liver disease. This suggests that peers support recovery but that our post-treatment follow up period is too short to detect any impact on death for individuals recovering from an alcohol use disorder. In column six we find a larger (though imprecisely estimated) impact of peers receiving treatment for an alcohol use disorder than for peers receiving treatment for a drug use disorder. Again, this is consistent with stronger peer influence among those receiving treatment for the same substance.

7 Conclusion

This paper uses linked administrative data from Norway to examine how peers acquired during inpatient treatment influence recovery from substance use disorders. Our empirical analysis is

informed by theory. By extending the model of addiction and cue triggered decision processes by Bernheim & Rangel (2004) to include peer effects, we derive theoretical predictions linking the potential mechanisms through which peers may influence substance use behaviors with the health related outcomes that we study. Our data comprise the universe of inpatients who were treated for a SUD in Norway's specialist health care system in 2009 and 2010, who we track until 2013. We define peer exposure as the (day-weighted) proportion of individuals with overlapping treatment episodes who reside in the same county as the focal individual, and exploit quasi-random variation in the timing of entry into treatment to identify the impact of peers on post-treatment mortality and substance related hospitalizations.

We find that peer exposure reduces mortality in the first year after exiting treatment, and that this effect persists until the third year. We also find that peers decrease hospitalizations for substance related disease, suggesting that peers support one another to abstain from using. Yet we find no impact of peers on hospitalizations for substance related poisoning, which could be due to the offsetting effects of peers supporting abstinence, which is expected to reduce both deaths and poisonings, and peers using more safely together, which is expected to reduce deaths but increase non-fatal poisonings. Overall, the evidence suggests that both the 'supporting abstinence' and the 'safely using together' mechanisms contribute to the peer induced reduction in post-treatment mortality. Finally, we find that peer type matters. Individuals treated for a drug use disorder benefit from exposure to peers who are also treated for a drug use disorder, whereas peers who are treated for an alcohol use disorder have no discernible impact. This may reflect a better knowledge of harm reduction and trigger mitigation strategies by peers who share a drug use disorder.

Substance induced deaths such as overdoses are preventable, yet they remain a leading cause of mortality. For example, in the US context, deaths from opioid overdose have quadrupled in the last twenty years, from 8.2 per 100,000 in 2002 to 32.6 per 100,000 in 2022 (Spencer *et al.* , 2024), yet the long-standing shortage of addiction services workers persists (Nguyen *et al.* , 2021). This implies significant scope for policy intervention. Our findings suggest that treatment gaps following inpatient treatment may be addressed by leveraging the bonds formed among inpatients while in treatment. This could entail, for example, facilitating former patients establishing mutual self-help recovery programs in their local communities, to which patients could be connected with upon discharge. If these types of mutual-help programs accommodate multiple pathways to recovery, they could fill important post-treatment gaps, especially among those for whom abstinence based peer support programs, such as Narcotics Anonymous, are not suitable. Our finding that peer effects are strongest among inpatients being treated for the same substance suggests fostering social ties among such individuals. This could be achieved through mutual-help programs which match based on substance, or increased specialization of treatment facilities by substance.

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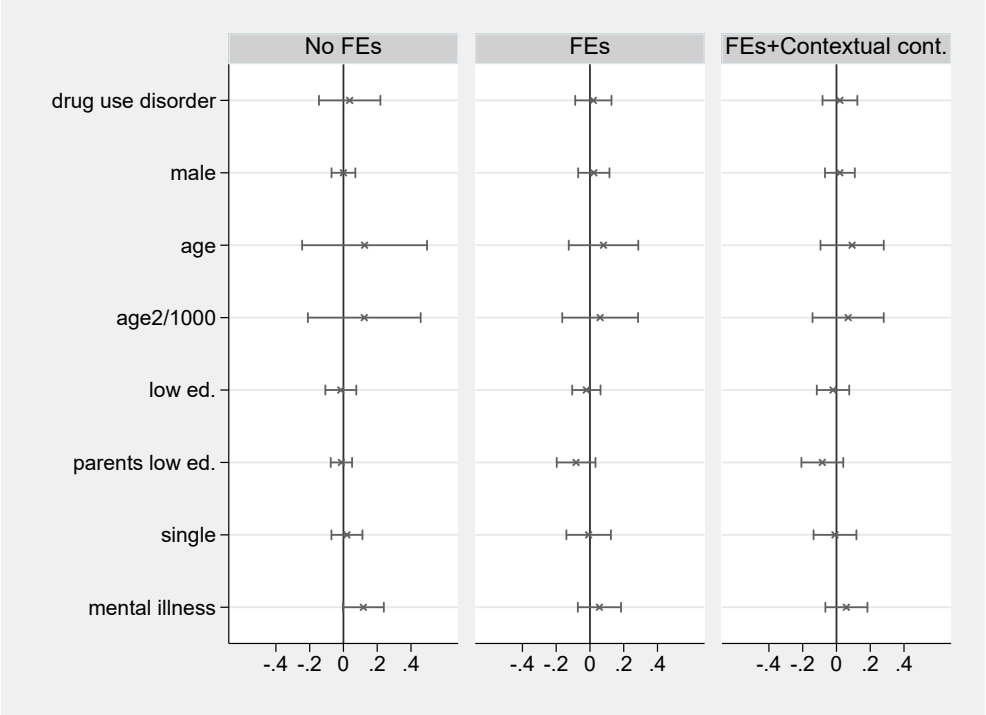
Appendix

Alternative balance tests

In the main text we presented the results of a balance test in which the dependent variable was the variable of interest (the peer measure) and the independent variables comprised patient characteristics, contextual controls, and fixed effects. An alternative sometimes used is to consider regressions where the dependent variable is a patient characteristic and the independent variables comprise the variable of interest (the peer measure), contextual controls and fixed effects. Figure 7 reports the coefficient on the peer measure and its 0.95 confidence interval. In all models it is close to zero and statistically insignificant.

Another type of balance test that is sometimes used is to first predict the outcome of interest (death) using individual level observables, and then to run regressions in which the dependent variable is the fitted values of the outcome of interest and the independent variables are the treatment variable (the peers variable), fixed effects and contextual controls. Table 9 reports the results of this exercise. In models with neither fixed effects nor contextual controls we find an association with the peers variable that is statistically significant at the 0.1 level. However,

Figure 7: Balance test: Controls as dependent variable



Notes: The figure shows coefficient estimates and 95% confidence intervals based on standard errors clustered at the facility level. To represent all coefficients on the same axis, we rescale the age variables to have standard deviation equal to one.

Table 9: Balance test: Fitted values

	Death within 1 year of exit (fitted values)			Death within 3 years of exit (fitted values)		
Peer exposure	0.003*	0.002	0.002	0.007	0.005	0.005
	(0.002)	(0.001)	(0.001)	(0.006)	(0.004)	(0.004)
Mean dep. var.	0.022	0.022	0.022	0.067	0.067	0.067
Observations	3,767	3,767	3,767	3,767	3,767	3,767
FEs	N	Y	Y	N	Y	Y
Contextual controls	N	N	Y	N	N	Y

Notes: Outcomes are fitted values using all controls listed in Section 4.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1.

with fixed effects and contextual controls the magnitude of the coefficient on the peers variable is reduced, and it is not statistically significant.

Placebo tests

To provide evidence that our baseline findings are driven by genuine peer effects, rather than some other artifact of inpatient admission, we conduct two placebo tests. The first placebo test shifts the treatment episode of the focal individual to a different point in time when constructing all

Figure 8: Placebo test: Shifted treatment episode (death within 1 year)

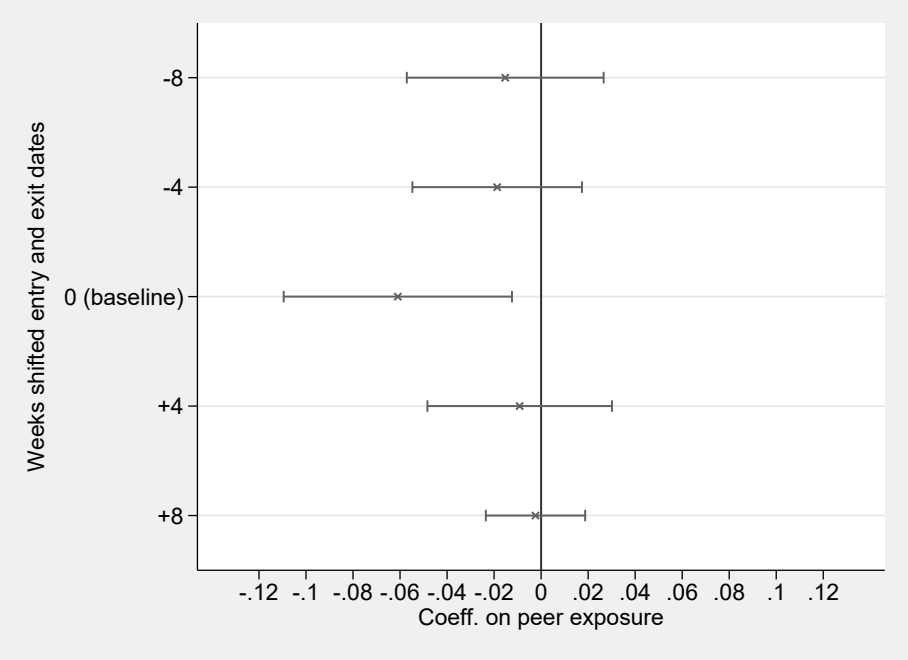
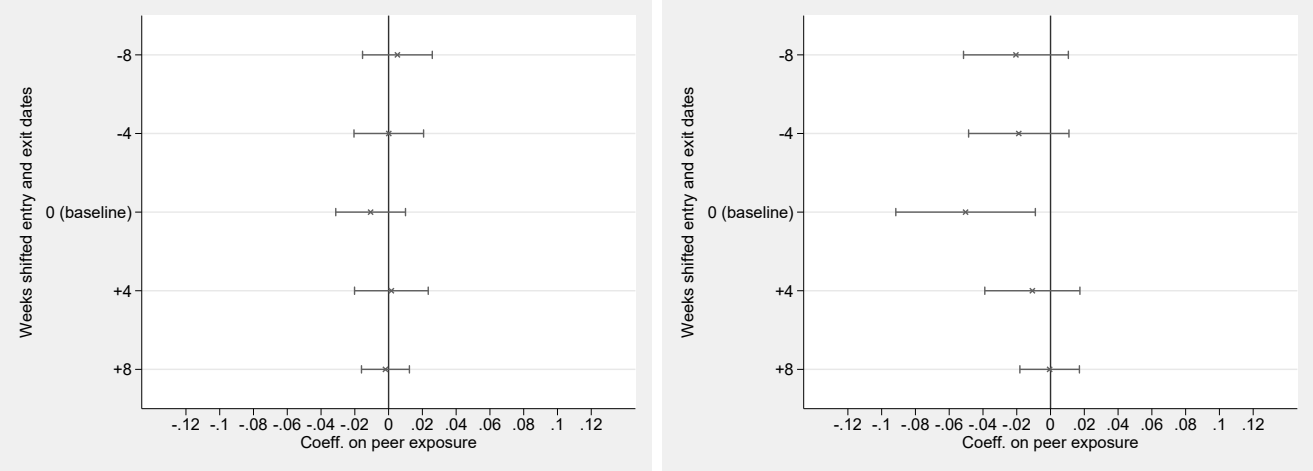


Figure 9: Placebo test: Internal (left) and external (right) cause deaths within 1 year



variables, including our measure of peer exposure. This assigns each inpatient a placebo group of other inpatients who received treatment at the same facility at around the same time, but whose treatment episodes had no (or limited) overlap. We shift episodes by four weeks and eight weeks. Shifting by four weeks implies that the shifted episode would have no overlap with the actual episode for an individual with median treatment duration (9 days) and little overlap for an inpatient with mean treatment duration (28 days). Shifting by eight weeks implies that the gap between the actual and shifted episode is approximately double the mean treatment duration. This means that the group of overlapping inpatients will tend to be entirely different in the shifted episode relative to the unshifted episode.

Figure 8 reports the coefficient and 0.95 confidence intervals for the impact of peer exposure on mortality in the first year after leaving treatment. The coefficient is small in magnitude and not statistically distinguishable from zero whenever the episode is shifted. This contrasts with our baseline findings of a statistically significant negative coefficient. Results for deaths from internal and external causes are reported in Figure 9. Again, we find no impact of placebo exposure to peers.

The second placebo tests replicates our baseline findings using a sample of patients admitted into inpatient treatment facilities as emergency patients. Emergency care is used when people with a SUD are a danger to themselves or others, and involves detoxification but not treatment to address disordered substance use. As patients admitted to inpatient facilities for emergency care do not interact with other patients, we should find no impact of overlapping patient episodes on the focal patient's mortality risk.

The sample of emergency patients is constructed as follows. We have 3,787 emergency care inpatients, of whom 42% are admitted for an alcohol use disorder and 58% for a drug use disorder. Among the latter, 55% were admitted with a polysubstance diagnosis, 14% for stimulants other

Table 10: Descriptive statistics for emergency patients: Outcomes and peer exposure

	Mean			
	within 1 year	within 3 years		
Death				
all-causes	0.020	0.060		
internal	0.007	0.030		
external	0.012	0.027		
substance induced	0.007	0.020		
	Mean	Std. dev.	Min	Max
Peers				
days admitted	9.65	20.88	1	349
num. overlapping inpatients	15.38	20.91	1	265
num. overlapping inpatients from same county	10.52	15.26	0	219
peer exposure	0.71	0.31	0	1
Observations	3,462			

than cocaine, and 11% with an opioid related diagnosis. Around 10% of emergency patients have a cannabis use disorder. From this sample of emergency patients, we define the analysis sample as the first inpatient episode that starts in 2009 or 2010 and ends by 31 December 2010 (N=3,658), and exclude 11 patients for whom the potential facilities attended are greater than four, 6 individuals from Central Norway, and 6 more due to missing observations on the control variables, resulting is a sample size of N=3,553. A further 91 observations are lost due to their being singletons in our fixed effects analysis, resulting in an analysis sample of N=3,462. Table 10 summarizes the sample.

Table 11 repeats our baseline analysis of treatment patients using the sample of emergency patients. The point estimates of interest are reasonably small in magnitude and do not approach statistical significance at conventional levels for any cause of death or time horizon. Our main results for treatment patients within 1 year of exit found a negative coefficient that was statistically significant at the 0.05 level and implies that exposure to an additional peer reduced mortality in the first year after exit by 10% relative to the mean. For the sample of emergency patients the coefficient is close to zero, statistically insignificant (despite the similar sample size), and implies that the effect of an additional peer (+3%) is considerably smaller than our baseline findings. Over three years, the coefficients for the placebo sample are even smaller in magnitude. For all cause mortality the effect size of an additional peer is (+0.4%), which is an order of magnitude smaller than for our main sample (-4.4%).

Table 11: Placebo test: Sample of emergency care patients

	Death within 1 year of exit			Death within 3 years of exit		
	All	Internal	External	All	Internal	External
peer exposure	0.019	-0.002	0.011	0.007	-0.004	0.004
	(0.022)	(0.014)	(0.013)	(0.027)	(0.018)	(0.016)
Mean dep. var.	0.020	0.007	0.012	0.060	0.027	0.030
+1 peer (%)	3.41	-0.76	3.50	0.43	-0.58	0.43
Observations	3,462	3,462	3,462	3,462	3,462	3,462
R-squared	0.056	0.049	0.048	0.058	0.083	0.040

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 4.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1. Multiple-testing adjusted p-values are in brackets.

Robustness tests

Here we perform various robustness tests designed to address the threats to identification identified in Section 5.1. First, we may be concerned about endogenous exit from treatment. The first panel

Table 12: Robustness: First day peers and model based on entry date

	First day peers only					
	Death within 1 year of exit			Death within 3 years of exit		
	All	Internal	External	All	Internal	External
peer exposure	-0.058**	-0.007	-0.051*	-0.069**	-0.010	-0.055*
(first day only)	(0.026)	(0.006)	(0.009)	(0.028)	(0.015)	(0.028)
Observations	3,767	3,767	3,767	3,767	3,767	3,767
	Model based on entry date					
	Death within 1 year of entry			Death within 3 years of entry		
	All	Internal	External	All	Internal	External
peer exposure	-0.050***	-0.009	-0.041**	-0.078***	-0.010	-0.061**
	(0.022)	(0.010)	(0.019)	(0.028)	(0.016)	(0.024)
Observations	3,767	3,767	3,767	3,767	3,767	3,767
	First day + entry date					
	Death within 1 year of entry			Death within 3 years of entry		
	All	Internal	External	All	Internal	External
peer exposure	-0.048**	-0.004	-0.044*	-0.066**	-0.007	-0.055*
(first day only)	(0.024)	(0.005)	(0.024)	(0.029)	(0.015)	(0.028)
Observations	3,767	3,767	3,767	3,767	3,767	3,767

Notes: All models include county-by-facility and month-by-year of exit (top panel) or entry (bottom two panels) fixed effects, and all controls listed in Section 4.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1.

Table 13: Robustness: Duration of treatment

	duration	duration	log duration	log duration
peer exposure (first day only)	-0.542 (8.473)		0.246 (0.196)	
peer exposure (all days)		-0.402 (4.638)		0.221 (0.185)
Mean dep. var.	28.46	28.46	2.279	2.279
+1 peer (%)	-0.07	-0.05	0.41	0.36
Observations	3,767	3,767	3,767	3,767

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 4.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1. Multiple-testing adjusted p-values are in brackets.

of Table 12 addresses this by repeating our baseline analysis, but using only other inpatients encountered on the first day of the treatment episode to construct the peer variable, which cannot be driven by endogenous exit. The estimates are similar to our baseline results, which may reflect those individuals encountered on the first day playing a more central role than those encountered later during the treatment episode. The second panel of Table 12 reports results measuring death from the (as good as randomly assigned) date of entry into treatment and using fixed effects for month-by-year of entry, rather than exit. We find very similar results to our baseline analysis. The third panel of Table 12 combines the models from the first two panels: we measure peers using only the first day of inpatient treatment and consider the model built around date of entry rather than date of exit. Again, the results remain nearly identical to our baseline results.

To further assess the extent to which endogenous exit may drive our findings, Table 13 considers the duration of treatment as the dependent variable, and estimates models in which the variable of interest is either peer exposure on the first day of the episode, or peer exposure throughout the episode. Since the treatment duration is skewed to the right, we also estimate models where the dependent variable is log duration. If inpatients systematically extend or curtail their treatment based on the social ties that have or have not formed, we would expect the coefficients in Table 13 to be different from zero. We find no evidence of any systematic relationship between peer exposure and duration. All of the coefficients are statistically insignificant and all of the implied effect sizes of an additional peer are close to zero.

Another concern relates to econometric modeling of low probability events. This applies mainly to mortality by cause within one year of exiting treatment, since only 2.2% of sample members die over this time-frame. Though the linear probability model is the workhorse empirical model in a setting where identification rests on fixed effects, and has been shown to perform well when modeling rare events (Timoneda, 2021), alternatives that may be more suitable are the complementary

Table 14: Robustness: Alternative models for rare events

	Death within 1 year of exit					
	Complementary log-log			Penalized Logit		
	All	Internal	External	All	Internal	External
peer exposure	-0.099***	-0.046*	-0.113***	-0.095***	-0.037	-0.085***
	(0.024)	(0.026)	(0.028)	(0.027)	(0.027)	(0.026)
Observations	3,767	3,767	3,767	3,767	3,767	3,767

Notes: Average marginal effects are reported. All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 4.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1.

log-log model and the penalized Logit model proposed by Firth (1993). Table 14 reports the results for these models. For ease of comparison with the linear probability model, we report average marginal effects. The signs of the average marginal effects are the same as those for the linear probability model in the main text. If anything, the average marginal effects for these alternative models are larger than our baseline linear probability estimates.

Geography or homophily?

In our baseline analysis we define peers based on geographic proximity. It is possible, however, that geographic proximity is not what drives the formation of long-run ties per se, but rather the

Table 15: Geography or homophily?

	Death within 1 year of exit			Death within 3 years of exit		
	All	Internal	External	All	Internal	External
peer exposure	-0.012	-0.011	-0.001	0.014	0.015	-0.001
(sex and age only)	(0.024)	(0.011)	(0.019)	(0.038)	(0.016)	(0.033)
Mean dep. var.	0.022	0.011	0.012	0.067	0.033	0.033
+1 peer (%)	-3.66	-7.15	-0.48	1.40	3.11	-0.29
Observations	3,767	3,767	3,767	3,767	3,767	3,767
peer exposure	-0.062***	-0.023	-0.038**	-0.061	-0.006	-0.054
(sex, age and county)	(0.021)	(0.016)	(0.018)	(0.039)	(0.025)	(0.035)
Mean dep. var.	0.022	0.011	0.012	0.067	0.033	0.033
+1 peer (%)	-23.00	-18.45	-27.14	-7.66	-1.57	-13.97
Observations	3,767	3,767	3,767	3,767	3,767	3,767

Notes: All models include month-by-year of exit fixed effects and all controls listed in Section 4.4. When defining peers based on sex and age (top panel), all models include sex-by-age-by-facility fixed effects. When defining peers based on sex, age and county (bottom panel), all models include sex-by-age-by-county-by-facility fixed effects. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1.

tendency of similar individuals to form ties with one another (Jackson *et al.* , 2017), including in the context of risky behaviors (Johnsen & Khoury, 2024; Mastrobuoni & Rialland, 2020), a phenomenon known as homophily. In this case, geography may be one dimension along which homophily operates, but others such as sex or age may also matter.

We explore this issue by considering two additional definitions of peers: one in which peers are overlapping inpatients who share the same sex and age (defined by an indicator for age$\leq$ 26), and another in which peers share the same sex, age and county of residence. In each model, to ensure that identification comes from timing of entry/exit alone, the fixed effects are for the dimensions along which we define peers. When we define peers using sex and age only, the fixed effects are for sex-by-age-by-facility. When we also require that they reside in the same county, the fixed effects are for sex-by-age-by-county-by-facility. Table 15 reports the results. The first panel defines peers based on common age and sex only. The second defines peers based on common age, sex and county of residence. When peers are defined based on age and sex only, we do not find statistically significant evidence the peers impact mortality. When peers are defined based on sex, age and county of residence, within one year of exiting inpatient treatment we find a statistically significant impact of peers on all cause and external cause mortality, but we find no statistically significant effects at a three year horizon (though the coefficient of interest remains negative for all cause and external cause mortality).

Descriptive statistics by SUD type

Table 16 provides descriptive statistics for those treated for drug and alcohol use disorders. For those with a drug use disorder, around three quarters of deaths are due to external causes (e.g., overdose), whereas the reverse holds for those with an alcohol use disorder (e.g., liver disease). A greater fraction of deaths are substance induced among those treated for a drug use disorder relative to an alcohol use disorder, especially over a three year time-frame since exit (42% vs 29%). Those with a drug use disorder have longer treatment episodes (mean of 35.89 days vs 18.85 days) but have similar peer exposure (0.56 vs 0.54), exposure to peers with a drug use disorder (0.39 vs 0.36) and exposure to peers with an alcohol use disorder (0.17 vs 0.18).

Table 16: Descriptive statistics by SUD type: Outcomes and peer exposure

	Drug use disorder		Alcohol use disorder	
	Mean within 1 year	Mean within 3 years	Mean within 1 year	Mean within 3 years
Death				
all-causes	0.021	0.053	0.024	0.085
internal	0.005	0.014	0.018	0.057
external	0.016	0.038	0.006	0.025
substance induced	0.008	0.022	0.008	0.025
Any hospital admission				
poisoning	0.029	0.065	0.032	0.069
liver disease	0.040	0.086	0.035	0.063
	Mean	SD	Mean	SD
Peers				
days in treatment	35.89	61.25	18.85	29.53
num. overlapping inpatients	37.61	58.92	35.67	32.20
num. overlapping inpatients from same county	18.26	32.94	16.98	18.62
peer exposure	0.56	0.32	0.54	0.30
drug peer exposure	0.39	0.28	0.36	0.25
alcohol peer exposure	0.17	0.18	0.18	0.19
Observations	2,125		1,642	