HEDG

Health, Econometrics and Data Group

The University of York

WP 24/18

Do Peers Support or Subvert Recovery from Substance Use Disorders

Christiern Rose; Jenny Williams and Anne Line Bretteville-Jensen

August 2024

http://www.york.ac.uk/economics/postgrad/herc/hedg/wps/

Do Peers Support or Subvert Recovery from Substance Use Disorders

Christiern Rose * Jenny Williams † Anne Line Bretteville-Jensen ‡

April 16, 2024

Abstract

We study peer effects in recovery from substance use disorders. We focus on peers who share an inpatient treatment episode and who reside in the same county, reflecting the salience of geographic proximity for peer influence in risky behaviors, and examine peer effects on post-treatment mortality. We access linked administrative data on death for the universe of individuals who are admitted to inpatient treatment for a substance use disorder in Norway in 2009-2010. The impact of peers is identified using variation in the timing of admissions into treatment, which institutional factors ensure is conditionally exogenous. Patients exposed to a greater share of peers from their home-county have a lower mortality risk. A standard deviation increase in the share of home-county peers reduces mortality by 36% relative to the mean, with one additional peer leading to a 5% reduction. The peer-induced reduction in mortality is concentrated amongst individuals admitted for treatment for a drug use disorder (as opposed to an alcohol use disorder). This is driven by peers who themselves receive treatment for a drug use disorder, and is consistent with peer effects working through two potential channels; reduced illicit drug use and safer illicit drug use. Examining hospital episodes for intoxication and (non-fatal) overdose indicates a limited role for safer drug use, suggesting that peers primarily reduce mortality by reducing drug use. We conclude that peers from inpatient treatment episodes can be instrumental in supporting recovery outside of clinical settings.

Keywords: Peer effects, substance use treatment, mortality

JEL-codes: I12, D85

*Department of Economics, University of Queensland; email: christiern.rose@uq.edu.au

†Department of Economics, University of Melbourne and IZA; email: jenny.williams@unimelb.edu.au

‡Norwegian Institute of Public Health; email: AnneLine.Bretteville-Jensen@fhi.no

1 Introduction

Substance use disorders (SUDs) are a major source of morbidity and mortality, and are associated with social marginalization and economic disadvantage, as measured by low income, unemployment, and engagement in crime (Aldridge *et al.* , 2018; Room, 2005). While evidence based treatments can be effective, relapse is common, even for those able to access high quality treatment. This is because much of the effort to sustain long term remission occurs outside of the healthcare system, and there are substantial gaps in knowledge around factors that support or subvert recovery. We investigate one such potential factor: the influence of peers.[1]

We study peer influence in recovery from SUDs among individuals who share an inpatient SUD treatment episode. In addition to the common goal of addressing their problematic substance use, individuals with overlapping episodes of treatment at an inpatient facility also share group treatment sessions in which they learn strategies to support recovery and to mitigate risks in the event of relapse. The combination of common goals and shared skills and knowledge to navigate recovery make peers gained during an inpatient episode of treatment a plausible source of support in recovery after the treatment episode ends. However, peers acquired during inpatient SUD treatment may also pose a risk to recovery. For example, peers may provide access to substances (Bohnert *et al.* , 2009; Curtis *et al.* , 1995) or there might be complementarities in using substances jointly with peers.[2] Whether the net effect of peers from inpatient treatment is to support or subvert recovery from SUDs is an empirical question, the answer to which has important implications for the design of post-treatment programs.

In studying the influence of peers acquired during inpatient treatment, our focus is on the period directly following the inpatient episode, and the main outcome we study is mortality. Mortality is a salient measure because people with SUDs are at elevated risks 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). Informed by the literature emphasizing the role of geographically proximate peers in risky behaviours (Adamopoulou *et al.* , 2024; Billings & Schnepel, 2022; Case & Katz, 1991; Christakis & Fowler, 2007; Kling *et al.* , 2007), and the

[1] The idea that peers with lived experience of SUDs and recovery can be instrumental in supporting the recovery of others is not new. It is the basis of well known and long established abstinence-based peer support programs such as Alcoholics Anonymous and Narcotics Anonymous, for example. In the US, more structured forms of support, such as Peer Recovery Support Services (PRSS), are being incorporated into healthcare settings as a means of bridging gaps in support outside of clinical settings. This is occurring despite a dearth of evidence as to whether, when, and for whom these services are effective at supporting recovery from a SUD (Eddie *et al.* , 2019).

[2] Access has been found to be an important driver of SUDs. For example, Conover & Scrimgeour (2013) show that a reduction in the legal age to purchase alcohol increased emergency room admissions among newly eligible buyers.

importance of geography in forming and maintaining social ties more broadly (e.g., Kim *et al.* (2023)), we consider peers to be individuals with whom the focal individual shared their inpatient treatment episode and who are resident in the same geographic area. These are individuals with whom relationships formed during inpatient treatment are likely to persist after exiting inpatient treatment, and hence may influence recovery outside of the clinical setting in the post-treatment period.

Our empirical analysis draws on high quality linked administrative data from Norway's National Patient Registry (NPR) and Cause of Death Registry. We observe the universe of individuals who receive inpatient treatment for a SUD in 2009 and 2010, who we are able to track until 2013. From the NPR data we are able to construct our measure of peer exposure, defined by the proportion of other inpatients with overlapping spells at the same treatment location who reside in the same county as the focal patient. Using the linked death registry data for the period 2009-2013 we construct indicators of death from all-causes and specific causes, including substance related causes, out to a three year horizon post-treatment. To explore mechanisms we also consider intermediate outcomes such as hospital episodes for intoxication and overdose. As with people who have overlapping episodes of incarceration (Billings & Schnepel, 2022), we expect SUD patients with overlapping episodes of inpatient treatment, and who are resident in the same geographic area, to form social ties that can impact their substance use behaviour, and through that, outcomes related to mortality and hospital admissions in the post-treatment period.

In studying the impact of peers, causal interpretation rests on random exposure to peers. As we discuss in detail in the following sections, random exposure is plausible given the institutional features that preclude patients from controlling when they are able to enter a treatment facility. The date of entry is determined by congestion and by a waiting list, which cannot be manipulated by the patient or their physician. Because of this, conditional on facility and county of residence, we argue that exposure to home county peers through overlapping treatment episodes is plausibly exogenous. We provide empirical evidence in favour of random peer exposure through various balance tests, which show that it is not associated with a patient's pre-admission observables.

We find that peers reduce mortality among inpatients with an SUD at both the one and three year horizons after exiting inpatient treatment. The reduction in mortality is concentrated in the first year, suggesting an immediate impact of peers on recovery. A standard deviation increase in the share of home-county peers reduces mortality by 36% relative to the mean. One additional home-county peer leads to a 5% reduction. We show that this peer-induced reduction in mortality is concentrated amongst individuals admitted for treatment for a drug use disorder, as opposed to an alcohol use disorder.

We then investigate the underlying mechanisms. We find a large impact of peers on illicit drug induced deaths. This suggests that peers reduce mortality through influencing patterns of illicit drug use. Next we show that the reduction in drug induced deaths is driven by peers who themselves receive treatment for a drug use disorder. This suggests that peers influence patterns of drug use through supporting a reduction in illicit drug use (for example, drawing on effective abstention strategies for illicit drug use learned during treatment) or through supporting safer use (e.g., strategies learned during treatment such as not using alone to guard against overdose). Finally we show that peers do not increase hospitalizations for intoxication or overdose. This suggests that peers reduce mortality primarily by reducing drug use rather than by facilitating safer drug use.

Our findings are supported by two placebo tests. First, we shift the entry and exit dates of each inpatient receiving treatment by a fixed number of weeks ($\pm$ 4 or 8) when constructing the peer exposure variable. This assigns each inpatient a placebo group of other inpatients (potential peers) who received treatment at the same facility at around the same time, but whose treatment episodes had no (or limited) overlap. Consequently, exposure to 'peers' ought to have no (or limited) impact on post-treatment outcomes. Second, we repeat our analysis of treatment patients using instead a sample of patients receiving emergency care. Emergency patients are admitted to undergo detoxification or because they pose a danger to themselves or others. They do not receive treatment and do not interact with other patients, hence should not acquire peers. In both placebo tests we find no impact of 'peers', suggesting that our main findings reflect genuine peer effects as opposed so some other artifact of admission to an inpatient facility.

We also conduct various robustness tests. Though the date of entry into treatment is as good as randomly assigned we might be concerned about endogenous exit. Endogenous exit may arise, for example, if individuals choose to leave or remain in treatment based on the social ties they form, in which case any reduction in mortality associated with peers might be driven by the treatment received, rather than by peers. We rule this out by showing that the impact of peers encountered on the first day of inpatient treatment is nearly identical to that of peers enountered across the whole treatment episode. Exposure to peers on the first day of treatment cannot be driven by endogenous exit from treatment. We also show that our findings are robust to measuring the incidence of death from the (randomly assigned) date of entry, as opposed to the date of exit. Whilst our main specification uses fixed effects for the county of residence and treatment facility, we also show that our findings are unlikely to be driven by unobserved heterogeneity which varies at the county-by-facility level (e.g., the existence of post-treatment support programs) by additionally controlling for the county-by-facility average of the peer variable. Finally we apply the complementary log-log and penalized Logit models that are arguably better suited to modeling low probability outcomes than

the linear probability model we employ. We find slightly larger average marginal effects relative to the linear probability model.

Our work contributes to two distinct literatures. First, we contribute to the literature on peer effects in risky behaviours. There is much work looking at adolescent entry into alcohol, tobacco and cannabis use (e.g., Ajilore *et al.* (2016); Arduini *et al.* (2019); Case & Katz (1991); Eisenberg *et al.* (2014); Fletcher & Ross (2018); Gaviria & Raphael (2001); Hsieh & Van Kippersluis (2018); Kawaguchi (2004); Kremer & Levy (2008); Li & Guo (2020); Lundborg (2006)), sex and truancy (e.g., Card & Giuliano (2013)), and crime (e.g., Díaz *et al.* (2021); Patacchini & Zenou (2012)). Peer effects in adult entry into prescription opioid abuse are examined by Adamopoulou *et al.* (2024). However, there is little existing work on *exit* from risky behaviours, and to the best of our knowledge, none that seeks to identify causal effects using quasi-experimental variation.[3] Causal evidence on the effectiveness of mutual support 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 the role of peers.

Second we contribute to the literature on determinants of mortality of people with SUDs. People with SUDs have higher mortality risk than those without, and an estimated two thirds of SUD related deaths in the US are not coded as such in cause of death data (Glei & Preston, 2020). Mortality has been shown to depend on availability of treatment (Swensen, 2015), availability of abuse-deterrent opioids (Alpert *et al.* , 2018, 2022; Evans *et al.* , 2019), macroeconomic conditions (Hollingsworth *et al.* , 2017) and individual characteristics such as gender (Gjersing & Bretteville-Jensen, 2014; Larney *et al.* , 2020). A closely related paper is Swensen (2015), who exploits county level data in treatment availability in the US and the opening and closing of treatment facilities, finding that, at the county level, a 10% increase in facilities lowers drug-related mortality by 2%. Our individual level data allows us to track patients both during and after their inpatient spells, and enables us us to study the role of peers in SUD mortality.

2 Institutional setting

Norway has a universal public healthcare system funded by general taxation. All Norwegians can access healthcare at close to zero direct cost. National policy is determined centrally by the Ministry of Health and Care Services but provision and administration is decentralized to

[3]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.

four Regional Health Authorities (Northern Norway, Central Norway, Western Norway and South-Eastern Norway), which oversee 20 Health Trusts delivering healthcare.

The specialist healthcare sector provides treatment for SUDs, which is known as 'interdisciplinary specialized services for substance abusers' in reference to the combination of expertise from medicine, psychology and social work involved. Treatment is provided directly by hospitals, through psychiatric units at hospitals, by dedicated substance use treatment centres and through privately practising specialists and non-profit organisations contracted to provide publicly funded treatment. Services include emergency care, detoxification, outpatient treatment and inpatient treatment. Our analysis focuses on inpatient treatment.

We now describe the sequence of events from referral to inpatient treatment to post-treatment outcomes. The timeline begins on the date of referral to inpatient treatment. Patients are typically referred by a specialist providing them with another substance use service (e.g., outpatient treatment), although direct referrals from a general practitioner are also possible.[4] Patients can nominate a preferred treatment facility at the point of referral, which may be based on a recommendation from the referring doctor. Referrals for inpatient treatment routinely exceed available resources. This excess demand is rationed by waiting time. In practice, referrals are sent to a priority-setting unit, which forwards the referral to a discipline specific assessment panel. Due to the Patients' Rights Act (1999), the right to equal access implies that prioritization for treatment should be based solely on clinical need. Assessments do not take into account gender, ethnicity, socioeconomic status, past harmful behaviour or productivity, nor do they depend on the available capacity at the facility chosen by the patient nor 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, and resource constraints.

After completing their wait, patients are admitted to a residential facility. In some cases this is within a psychiatric unit at a hospital (known as VOP) but most patients attend dedicated addiction units (known as TSB). Some facilities specialize in particular substances (e.g., alcohol), patient gender (e.g., female only) and patient age (e.g., young adults only, typically using a cut off of ≤ 26 years old) but most treat a mixture of patient types. Facilities also offer different therapies (e.g., horse-assisted therapy), which may improve patient outcomes, including mortality (Gatti *et al.* , 2020). Because of this heterogeneity in the types of patients catered for and therapies offered, patients do not necessarily attend their nearest facility, nor even a facility in the same county. Moreover, rural patients can live in counties which do not have a treatment facility, in

[4]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.

which case they must attend one in another county.

Before receiving treatment, patients first undergo detoxification. The aim of detoxification is to clear substances from the body whilst managing withdrawal symptoms. This takes one week for alcohol and cannabis, and two weeks for other illicit drugs. After detoxification, a typical treatment plan comprises individual therapy (cognitive behavioural therapy and motivational interviews), group therapy, environmental therapy, education and physical activity. During the course of their treatment, patients interact routinely with 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 with treatment or if their treatment proves to be ineffective.

3 Data

We access linked administrative data from Norway. The cohort for which linking was undertaken includes all individuals who were in the specialist healthcare sector with a primary diagnosis of a SUD (ICD10 codes F10-F16 and F18-F19) between 1 January 2009 and 31 December 2010. These individuals were identified in the Norwegian Patient Registry (NPR), which covers all patients in the specialist healthcare system. The NPR patient cohort was linked by Statistics Norway (using unique individual identifiers) to the Norwegian Cause of Death Registry and to the Norwegian Population Registry which contains demographic and socioeconomic information, including county of residence on January 1 2010.[5] For this cohort, all episodes in specialist healthcare recorded up to 31 December 2013 were extracted from the NPR. Death registry data are for the period 1 January 2009 through to 31 December 2013.

The NPR cohort data are well suited to study the effects of peers acquired during an episode of inpatient treatment for a SUD because they cover the universe of inpatient episodes that include at least one day in 2009 and 2010. This allows us to observe all patients, and, importantly, for patients whose episodes began and ended in 2009-2010, all other patients who may be their peers. For each inpatient episode, the NPR provides information on the diagnosed illness (ICD10 codes) and type of care (e.g., emergency care or treatment) for which the referral for specialist healthcare is made.

The NPR data also contains information on the start and end dates for the episode, the sector (specialized interdisciplinary (TSB) or psychiatric (VOP)), and for treatment episodes in public

[5]County of residence is not recorded at the time of inpatient admission. 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.

facilities, the geographic location (Health Trust) in which the episode is provided. Combining this information with that of patient age and sex, and supplementary data on the gender, age range and substance type treated by each facility, we can match patients treated at a public facility 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 attended, in which case we keep track of the list of facilities that they could have attended. We can evaulate the quality of our match between patients and facilities by comparing the number of patients matched to each facility on each day during our sample window to their capacity (measured by number of beds).[6] Since the aggregate demand for care routinely exceeds available beds, random matching of patients to facilities would to lead to a significant fraction of facilities operating above capacity. Under our matching, the TSB facilities would have operated at or below capacity on 95% of facility-days between January 1 2009 and December 31 2010.[7] This suggests that we are able to match patients to facilities reasonably well given the data available to us.

3.1 Sample

The sample is constructed from all inpatient episodes for treatment for a SUD that occur in a public facility (either TSB or VOP) and which include at least one day of inpatient treatment during the period 1 January 2009 to 31 December 2010. As inpatient episodes entail a detoxification period prior to the start of treatment, and because patients do not interact during detoxification, we shift the date of entry into specialist care forward by the period of detoxification to obtain the start date for receipt of treatment. Accounting for detoxification ensures that we only consider the portion of the episode during which patients experienc treatment and interact with one another. This leads to a sample size of 4,856 patient-treatment episodes, 81% of which were in the TSB sector and 19% in the VOP sector. 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%), and stimulants other than cocaine (15%). Around 12% of patients have a cannabis use disorder.

From this sample, we define the analysis sample as the first inpatient episode that starts between 1 January 2009 and 31 December 2010, and ends by 31 December 2010 (N=4,257). We exclude a further 369 observations for patients who attend TSB facilities in the health region of Central Norway, as this health region does not report specialist TSB 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 based on the information available to us,

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

[7]We do not observe the number of beds for VOP facilities, so cannot perform this exercise for VOP facilities.

and 132 due to missing control variables, resulting in a sample size of N=3,838. For TSB patients, we can uniquely identify the facility attended for 57% of patients. There are two potential facilities for 15% of TSB patients, three potential facilities for 21% of patients, and four possible facilities for 6% of TSB patients.

In a placebo analysis we examine inpatient emergency care episodes. 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 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 for analysis of N=3,553.

3.2 Outcomes

Our main outcomes of interest relate to death and the underlying cause of death. The Norwegian Cause of Death Registry contains the date of death, and for 98% of deaths in the sample, the ICD10 code for the underlying cause of death and the ICD10 codes from the death notification. Death registry information for the cohort are available for 1 January 2009 until 31 December 2013.

Our primary outcomes of interest 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 of death are accidents (including accidental poisonings), intentional self harm (including suicide by intentional poisonings), assault or homicide, and other external causes. Poisonings (accidental and intentional) include deaths in which an illicit drug overdose is an underlying cause of death. Internal causes of death are infection or parasites, cancer, blood or immune system disorders, endocrine or metabolic system disorders, mental and behavioural disorders, nervous system disorders, circulatory system disorders, respiratory system disorders, digestive system disorders, skin disorders, musculoskeletal disorders, genitourinary disorders, congenital malformations, and other abnormalities.

We also consider drug induced deaths. Drug induced deaths are defined as deaths where the underlying cause was a mental-behavioural disorder due to psychoactive substance use (opioids, cannabinoids, cocaine, other stimulants, hallucinogens, multiple drug use) or poisoning (accidental, intentional or undetermined intent) by opium, heroin, other opioids, methadone, other synthetic narcotics, cocaine, other and unspecified narcotics, cannabis, lysergide, other and unspecified psy-

Table 1: Descriptive statistics - outcomes and peer exposure

	Time since left inpatient treatment within 1 year		within 3 years	
	Mean	Std. dev.	Mean	St Dev.
Death				
all-causes	0.022	0.146	0.067	0.250
internal COD	0.010	0.102	0.032	0.177
external COD	0.012	0.107	0.033	0.178
drug induced	0.004	0.064	0.013	0.113
Hospital episode				
drug induced intoxication	0.032	0.176	0.083	0.275
drug induced poisoning	0.027	0.162	0.057	0.232
Peers				
num. overlapping inpatients	36.761	49.200		
num. overlapping inpatients from same county	17.382	27.478		
peer exposure	0.542	0.316		
drug peer exposure	0.373	0.272		
alcohol peer exposure	0.169	0.184		

Notes: The table shows descriptive statistics for the N=3,838 individuals who entered a public inpatient facility for treatment for a SUD in 2009 or 2010.

chodysleptics, or psychostimulants.[8]

Table 1 reports descriptive statistics for the death related outcomes we study. 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 these deaths are due to external causes and around a fifth are drug induced. Among drug induced deaths, 86% are attributed to accidental poisoning (overdose).

In our analysis of mechanisms we draw on information from hospital episodes.[9] We consider episodes for which the first two ICD10 codes include intoxication (ICD10 F11-F19) or poisoning (ICD10 T4N) related to illicit drug use. The second panel of Table 1 shows that in the year following inpatient treatment, around 3.2% of our sample experienced a hospital episode related to intoxication and around 2.7% of our sample experienced a hospital episode related to poisoning. Over a three year time horizon these figures respectively rise to 8.3% and 5.7%.

[8]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.

[9]This information is available for calendar years only, and we construct indicators for admissions one year after leaving inpatient treatment for patients who received inpatient treatment for a SUD in 2009 if they were recorded with an admission into a somatic hospital in 2010, and for patients who received inpatient treatment for a SUD in 2010 if they were recorded with a somatic hospital episode in 2011.

3.3 Measuring peer exposure

To study the impact of peers on post-treatment outcomes outside the clinical environment, we aim to construct a measure of exposure to peers during inpatient treatment who might plausibly remain peers after exiting inpatient treatment. It is thus crucial that lasting social ties can be formed among individuals sharing a spell of inpatient treatment for a SUD. To motivate this we draw on a survey of past-four-week opioid and stimulant users, which was collected across seven Norwegian cities in September to October 2023 by the Norwegian Institute of Public Health (Gjersing, 2023). Surveyed individuals were not undergoing inpatient treatment at the time of survey. Of the 975 individuals surveyed, 698 (72%) had previously undertaken inpatient treatment. Among these individuals, 53% reported that they had subsequently been in contact with another individual they met during their last spell of inpatient treatment, suggesting that the formation of long lasting social ties during inpatient treatment is plausible and occurs reasonably frequently.

We define peers as inpatients who receive treatment for a SUD at the same facility, and whose inpatient episode overlaps with the focal patient's episode, and who reside in the same geographic area as the focal patient. We restrict peers to those who reside in the same area as we view these individuals as the most likely to maintain social ties after exiting treatment. Since the existence and intensity of social ties strongly depend on geographic proximity (Kim *et al.* , 2023), individuals exposed to more individuals resident in the same geographic area would, all else equal, be expected to have stronger and more persistent social ties after exiting treatment. Peers from the same geographic area have previously been shown to impact drug, alcohol use and crime among youths (Case & Katz, 1991; Christakis & Fowler, 2007; Kling *et al.* , 2007), entry into prescription opioid misuse (Adamopoulou *et al.* , 2024), and criminal recidivism among convicted offenders (Billings & Schnepel, 2022). We thus have strong reason to suspect that geographically proximate peers may play a role in recovery from SUDs outside the clinical setting during the post-treatment period.

The geographic unit on which our peer measure is based is the county. This is partly due to data availability, but also ensures a sufficiently large number of individuals with a SUD reside in each geographic unit so as to generate identifying variation. In 2009-2013, Norway comprised 19 counties. In 2011, the county level populations ranged from 73,417 (Finnmark) to 599,230 (Oslo) with a median of 235,493. In the same year, the 12 month prevalence of drug use disorder was at most 0.9%. The corresponding figure for alcohol use disorder was 6.5% (Torvik & Rognmo, 2011). In the median sized county, the number of individuals with a drug use disorder would then be around $0.009 \times 235,493 = 320$. For alcohol use disorder the corresponding figure is $2,307$. Thus, though counties are large, the population of individuals in a given county with an SUD is not. Using county of residence ensures a reasonably high incidence of overlapping treatment spells of

individuals whose residence is geographically proximate.

Following the literature on criminal recidivism (e.g., Bayer *et al.* (2009); Damm & Gorinas (2020)) we use the proportion of peers rather than the count. This is because the peer count is strongly positively associated with the length of stay in the treatment facility, which reflects both underlying SUD severity and the quantity of treatment received. Moreover, during times of high demand for treatment, facilities are more likely to operate close to capacity, which may impact the quality of treatment received whilst also increasing the peer count. Due to these issues, the peer count may partially capture unobservable drivers of post-treatment mortality risk. Using the proportion of peers mitigates such concerns.

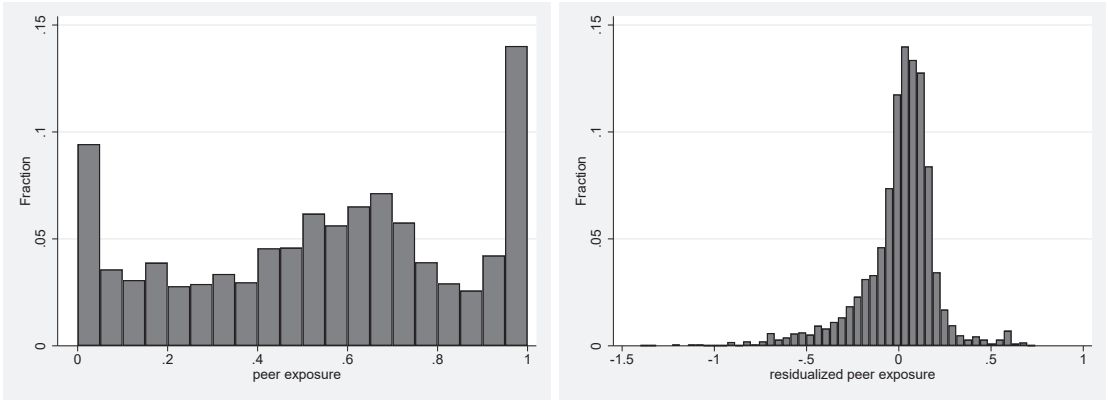
Our measure of peer exposure is the proportion of all inpatients with whom the focal patient's treatment episode overlaps who reside in the same county as the focal patient. In constructing the proportion of peers, each patient (irrespective of their county of residence) with an overlapping episode of treatment is weighted by the number of days their episode overlaps with the episode of the focal patient. For example, if an inpatient overlaps with two other inpatients for the same amount of time, only one of whom is resident in the same county, then our peer measure takes the value 1/2. If the duration of overlap with the inpatient from the same county is twice that of the other inpatient then our peer measure takes the value 2/3. Use of the day-weighted proportion implicitly implies that the impact of an additional day with a home-county peer (the numerator) is inversely proportional to the total number of days overlapping with all inpatients (the denominator). This might reasonably arise when opportunities for interaction are approximately equal across all inpatients attending the facility on any given day.

Since we observe all treatment episodes which include at least one day during a window from January 1 2009 to December 31 2010, and our analysis sample comprises only inpatient episodes which start and conclude during this window, we observe all other inpatients with whom the focal patient overlaps. This means that we do not need to correct our peer variable for missing potential peers, in contrast with some other studies with a similar research design but a different sampling method.[10] However, we are not always able to uniquely match a patient uniquely to the facility they attended. If the facility that the patient could have attended based on their observables is not unique, we use all inpatients who *could* have attended one of the focal patient's potential facilities during their episode to construct the peer variable. This introduces some measurement error, which may attenuate our estimates towards zero. Measurement error is also likely to be present in studies applying a correction due to their not observing the pool of all potential peers.[11]

[10]For example, Bayer *et al.* (2009) only observe prisoners who are *released* during a given window, and so need to correct their measure of peer exposure for the potential peers that they do not observe.

[11]For example, to correct for missing potential peers Bayer *et al.* (2009) use the expected number of days overlapped

Figure 1: Distribution of peer exposure: raw and residualized with respect to fixed effects



The bottom panel of Table 1 shows that on average, 54% of patients that the focal patient interacts with during their inpatient treatment episode (weighted by the number of days overlapped) reside in the same county. The left panel of Figure 1 shows the distribution of this variable in our analysis sample. The right shows the residual variation after partialling out the fixed effects used in our empirical model, demonstrating that there is ample within variation.[12]

In our exploration of mechanisms we also examine whether the impact of peers differs by whether the peers receive treatment for an alcohol use disorder or a drug use disorder. As shown in the bottom panel of Table 1, on average 37% of patients overlapping with the focal patient reside in the same county and are admitted to treatment for a drug use disorder, and 17% reside in the same county and are admitted for an alcohol use disorder. That more than twice as many day-weighted peers receive treatment for a drug use disorder as for an alcohol use disorder reflects the fact that 57% of patients enter treatment for a drug use disorder, as well as the longer duration of treatment observed for patients with a drug use disorder (an average of approximately 37 days for drug use disorders compared to 19 days for alcohol use disorders).

3.4 Individual and contextual controls

Individual characteristics that we control for are: type of SUD (drug use or alcohol use), sex, age, education measured by an indicator for the patient having no more than the compulsory level of education (lower secondary), socioeconomic background as measured by parents' education, an

with missing potential peers, leading to measurement error when the expected number of days overlapped differs from the actual number.

[12]We show the distribution by substance group (alcohol or drugs) with and without accounting for the full set of fixed effects in Figure 4 in the appendix.

Table 2: Descriptive statistics - controls

	Variables	Mean	St Dev.
Individual controls			
	drug use disorder	0.57	0.50
	male	0.68	0.46
	age	40.95	13.39
	age^2/1000	1.86	1.15
	no more than minimum education	0.58	0.49
	both parents min. ed.	0.26	0.44
	single	0.63	0.48
	mental illness	0.19	0.39
Contextual controls			
	prop. drug use disorder	0.70	0.21
	prop. male	0.68	0.18
	prop. young (age$\leq$ 26)	0.19	0.23
	prop. no more than min. ed.	0.58	0.19
	prop. both parents min. ed.	0.23	0.16
	prop. single	0.63	0.19
	prop. mental illness	0.25	0.20

Notes: The table shows descriptive statistics for the N=3,838 individuals who entered a public inpatient facility for treatment for a SUD in 2009 or 2010.

indicator for single marital status, and an indicator for the patient having a mental illness diagnosis in addition to a SUD at referral. We also form averages of these patient characteristics over all inpatients with overlapping treatment spells (weighted by days overlapped) so as to account for contextual effects associated with the inpatient treatment episode (Manski, 1993).

Table 2 reports descriptive statistics for the individual and contextual controls. Patients are typically male (68%), single (63%), and with a low level of education (58% have no more than lower secondary education). They have an average age of 41, 19% have a mental illness diagnosis when referred for treatment for a SUD, and 26% come from as disadvantaged background as measured by both parents having no more than lower secondary education. These figures are reflected in the contextual controls, though with smaller standard deviations due to their being averages across multiple individuals.

4 Empirical Strategy

Our primary analysis evaluates the impact of home-county peers on mortality using the following specification:

$$Death_{icjt} = \beta Peer_{icjt} + \mathbf{x}_i'\gamma + \alpha_j + \delta_c + \mu_t + u_i.$$

The dependent variable $Death_{icjt}$ indicates whether individual i who resides in county c and who was treated at facility j until exiting in period t died within a fixed time period (one or three years) after leaving treatment. $Peer_{icjt}$ measures an individual's exposure to peers resident in the same county; $\mathbf{x}_i$ includes exogenous characteristics measured prior to admission, including an indicator for whether admission was for a drug use disorder, and contextual controls; α_j is a facility fixed effect; δ_c is a county fixed effect; μ_t is a month-by-year of exit fixed effect; and u_i are unobserved factors.

In the language of the seminal paper on identification of peer effects (Manski, 1993), our empirical model includes both *contextual peer effects* and *correlated effects*. Contextual peer effects arise when the outcome of the focal individual depends on the type of other individuals in their reference group (captured by our contextual controls), whereas correlated effects arise when individuals have similar outcomes due to their exposure to a common environment (captured by facility, county and month-by-year of exit fixed effects).

The parameter of interest, β, captures the net impact of exposure to home-county peers on post-treatment mortality.[13] It is important to emphasize that there are several competing channels through which peers may impact on post-treatment mortality, and we are only able to identify the net effect. If $\beta > 0$ then the net effect of peers is to increase the risk of mortality, consistent with dominance of complementarities in joint use or access effects (Bohnert *et al.* , 2009; Curtis *et al.* , 1995), whereas $\beta < 0$ is consistent with dominance of peers supporting recovery (Jason *et al.* , 2012) or promoting safer modes of use. If peers reduce mortality through promoting safer modes of use, we expect events that would have resulted in death to instead result in hospitalisations for intoxication and (non-fatal) overdoes. We pursue this issue in our exploration of mechanisms.

Consistent with the mechanisms above, the survey of current drug users in Norway conducted by Gjersing (2023) provides descriptive evidence that peers can have both a supporting and a subverting influence on recovery from a SUD. Around 18% of respondents who had undertaken a spell of inpatient treatment reported that other individuals they met during inpatient treatment

[13] Since our data do not allow us to directly observe the formation of social ties or endurance of social ties post-treatment, β captures both the propensity to form ties and the net impact of these ties.

contributed to a reduction in their substance use. However, 23% reported that other individuals they met during inpatient treatment contributed to an increase in their substance use.[14] We note that while the survey results are informative on the question of whether former inpatients interact post-treatment, and whether those interactions affect substance use, 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, our administrative sample 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.

Our empirical model includes facility fixed effects to account for nonrandom assignment of inpatients to treatment facilities, which arisines since patients may choose the facility they attend. We also include county fixed effects because we define peers as being those who reside in the same county as the focal individual. Patients residing in different counties might differ systematically from one another in terms of their mortality risk and exposure to home-county peers. For example, even conditional on the facility attended, individuals from more populous counties (e.g., predominantly urban counties) could be systematically exposed to a greater fraction of inpatients resident in the same county. At the same time, individuals residing in such counties might differ systematically from those residing in other (e.g., smaller or rural) counties, in terms of their mortality risk, or in terms of the SUD related healthcare services available to them in the post-treatment period.

4.1 Identification

Our identification strategy leverages quasi-experimental variation in peer exposure generated by the timing of patients' dates of entry into treatment. This type of variation has previously been exploited in the context of criminal recidivism (Bayer *et al.* , 2009; Billings & Schnepel, 2022; Damm & Gorinas, 2020; Tan & Zapryanova, 2022). Our identification argument is that conditional on facility and county of residence, the identities of other inpatients who have overlapping treatment spells with the focal individual are as good as randomly assigned because the date of entry into treatment is as good as randomly assigned. The latter is because the institutional environment implies that no patient can manipulate their date of entry nor the dates of entry of other patients opting to receive treatment at the same facility.

To improve power, our empirical model operationalizes conditional exogeneity through an ad-

[14]The remaining 59% reported that individuals they met during inpatient treatment did not influence their substance use.

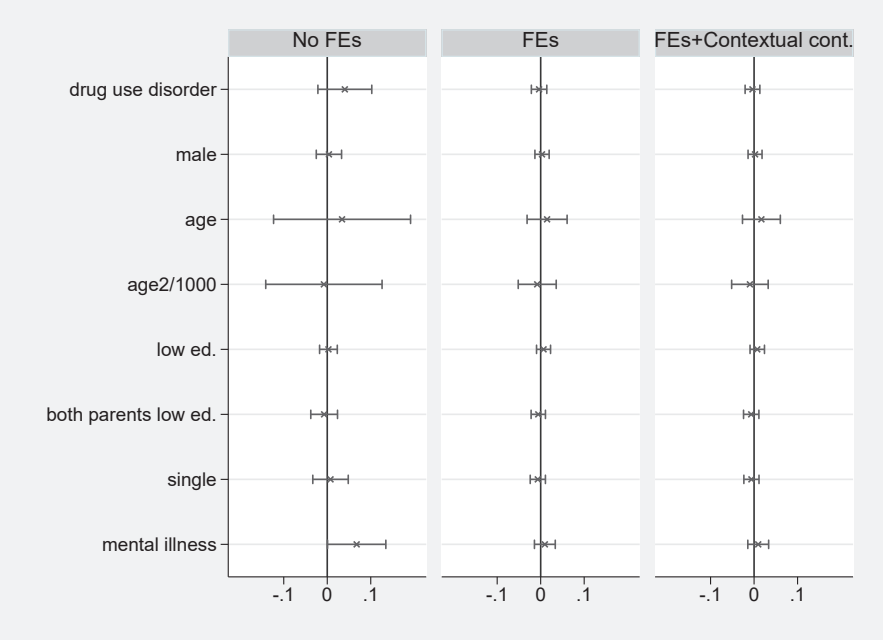
ditive separability assumption, under which the joint effect of the facility and county are assumed to comprise a facility fixed effect α_f and a county fixed effect δ_c. One may be concerned that unobservables varying at the county-by-facility level may drive both the identities of overlapping inpatients and the mortality risk of the focal individual. For example, inpatients residing in an urban county and attending a facility in that same county may have greater access to SUD related healthcare services after exiting treatment and may encounter a larger proportion of other inpatients residing in their county during their treatment. In a robustness test we address this by controlling for the average of our peer variable at the county-by-facility level (i.e., a Mundlak type regression). Whilst this does not account for all possible sources of county-by-facility unobserved heterogeneity, it is likely to account for much of the heterogeneity that is correlated with our peer variable.

Despite the institutional setting effectively guaranteeing that the date of entry into treatment is as good as randomly assigned, we may be concerned about endogenous exit from treatment, which could be more salient in our context than that of criminal recidivism. Whilst prisoners may influence their release date through their behaviour, inpatients receiving treatment for SUDs are free to leave at any time. Specifically, we might be concerned that exposure to peers drives exit from treatment. For example, it could be that inpatients elect to leave treatment once they feel they have acquired a sufficiently supportive peer group to aid their recovery outside of the clinical setting, and doing so would cut short their inpatient treatment, potentially elevating their mortality risk. Alternatively, it could be that inpatients prefer to remain in treatment once they have established social connections, hence extending their treatment period and potentially reducing their mortality risk. To address concerns around endogenous exit we conduct two robustness tests. First, we consider an alternative measure of peers, where we only use the first day of the focal individual's treatment spell to construct the peer variable. This cannot be manipulated by endogenous exit from treatment. Second, 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, as opposed to our baseline model which is built around the date of exit from treatment.

4.2 Balance tests

While we cannot establish whether exposure to home-county peers during an inpatient treatment episode is determined by unobservable factors influencing mortality risk, Figure 2 examines the relationship between peer exposure and observable pre-admission patient characteristics. It reports coefficient estimates and 0.95 confidence intervals for each individual patient characteristic from three separate regressions in which peer exposure is the dependent variable. The first model only

Figure 2: Balance test



Notes: N=3,838. The figure shows coefficient estimates and 95% confidence intervals based on standard errors clustered at the facility level. To represent all regressors on the same axis, we rescale the age variables to have standard deviation equal to one.

includes individual characteristics, the second adds the full set of fixed effects, and the third adds the contextual variables.

In all three specifications, patient characteristics 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 control for the fixed effects or contextual variables. Further, when including fixed effects, these individual characteristics are not jointly associated with peer exposure, with a p-value for the F-statistic for the test of the joint null hypothesis of 0.39 and 0.42 for specifications with fixed effects, and with fixed effects and contextual controls, respectively. These findings provide support for the assumption that, after appropriate conditioning, exposure to peers is as good as random. We consider two alternative balance tests in the appendix.

5 Results

In this section we summarise our findings, consider robustness, and explore mechanisms.

5.1 Summary

We begin by considering the impact of peers on mortality within one and three years of exit from inpatient treatment. This allows us to establish the extent to which peers impact on survival, and whether their impact is persistent or wanes over time. We find evidence that peers reduce mortality and that this arises largely in the first year after exiting inpatient treatment. Next we consider death by cause (internal or external). Death from external causes may arise suddenly as a direct and immediate consequence of substance use (e.g., overdose), whereas deaths from internal causes likely reflect a gradual deterioration in health over time. Consistent with this, we find that peers reduce deaths from external causes but not internal causes.

We then consider heterogeneity by the type of SUD (alcohol or drugs) because we expect the underlying causes of mortality to differ by substance type. Alcohol misuse is associated with a slow progression of disease (over the liver, for example) and deterioration in health, potentially leading to death from internal causes. In contrast, illicit drug use is associated with accidental poisoning (overdose), which is an external cause of death, the risk of which is elevated following an inpatient treatment episode. We show that our baseline finding that peers reduce mortality is driven by a reduction in mortality among inpatients with a drug use disorder. Whilst we find no evidence of a reduction in mortality for those with an alcohol use disorder, in a supplementary analysis in the appendix, we find that peers reduce hospital admissions for diseases associated with alcohol misuse (namely chronic hepatitis (Ganesan *et al.* , 2020) and and skin disorders (Raiker *et al.* , 2016)) among those treated for an alcohol use disorder. This suggests that peers reduce alcohol misuse, but that any impact on mortality is either too small to be detected in our data or arises over a post-treatment time frame exceeding that in our data (three years).

Finally, we consider the mechanisms underlying the impact of peers on mortality. We show that the peer-induced reduction in mortality among those with a drug use disorder is largely driven by a reduction in drug induced deaths, consistent with peers changing patterns of drug use. We then show that this is driven by peers who have themselves been treated for a drug use disorder. This suggests that peer support and/or exchange of substance specific human capital (e.g., strategies to abstain or use more safely) may drive our main results, dominating any increase in substance use that might arise from complementarities in joint substance use or access effects. That drug using peers reduce the drug induced mortality risk of drug users might either be due to peers reducing drug use or due to peers fostering safer drug use (e.g., supervised use to respond to overdoses). If the mechanism is mainly safer drug use, we might expect to find a positive impact of peers on hospital admissons for intoxication or (non-fatal) overdose, arising through peer intervention in response to severe overdoses that may otherwise have led to death. We find no such effect,

Table 3: Baseline results

	Death within 1 year of exit			Death within 3 years of exit		
	All Causes	Internal	External	All Causes	Internal	External
peer exposure	-0.025***	-0.008	-0.017**	-0.026*	-0.005	-0.019*
	(0.009)	(0.006)	(0.008)	(0.013)	(0.008)	(0.010)
Mean dep. var.	0.0219	0.0104	0.0115	0.067	0.032	0.033
+1 SD (% of mean)	-35.54	-24.00	-46.04	-12.18	-4.94	-18.60
+1 peer	-4.55	-3.04	-5.84	-1.54	-0.63	-2.36
Observations	3,837	3,837	3,837	3,837	3,837	3,837
R-squared	0.038	0.037	0.032	0.046	0.062	0.033

Notes: All models include facility, county and month-by-year of exit fixed effects. Controls are an indicator for a drug use disorder, an indicator for male, age, age^2/1000, an indicator for no more than minimum education level, an indicator for both parents having minimum education, an indicator for being single and an indicator for a mental illness comorbidity. Contextual controls (averages over all other overlapping inpatients, weighted by days overlapped) are the proportion receiving care for a drug use disorder, the proportion who are male, the proportion who are young (≤ 26), the proportion who have no more than minimum education, the proportion for which both parents have no more than minimum education and the proportion with mental illness comorbidities. Standard errors are clustered by facility.

suggesting that drug using peers primarily support drug users to reduce their consumption, rather than to consume more safely. We now present our empirical results in greater detail.

5.2 Baseline results

Table 3 provides our baseline findings. The first column presents the results from estimating a linear probability model where the outcome is death from all-causes within one year of exiting treatment; the outcome in the second column is death from internal causes; the outcome in the third column is death from external causes. The remaining columns look at death over a longer time horizon of three years. This allows us to evaluate the extent to which deaths are prevented or merely delayed. Standard errors account for clustering at the facility level. Full regression results including controls are shown in Table 11 in the appendix.

Looking at the first column of Table 3, we find that peers reduce mortality risk. The coefficient is statistically significant at the 0.01 level. The row denoted '+1 SD (% of mean)' considers the impact of a standard deviation increase in peer exposure, which leads to a 36% reduction in all-cause mortality in the year following treatment. The row denoted '+1 peer' considers the impact of an additional peer. When making this calculation we suppose that the additional peer overlapped with the entire duration of the focal individual's episode. Denoting $Peer = p/n$ where p 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 $Peer = (p + d)/(n + d)$ where

d is the number of days the focal individual received treatment. The difference between peer exposure and its counterfactual depends on p, n and d, so the impact of one additional peer is heterogeneous across individuals. Due to this we report the mean impact. Our estimates imply that, on average, being treated alongside one additional peer for the duration of the treatment episode reduces all-cause mortality in the year following treatment by around 5% relative to the mean.

Looking at the second and third columns of Table 3 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 internal causes of death. This is consistent in a reduction in mortality stemming from reduced substance use, since there is little scope for changes in substance use to impact on mortality from internal causes within a one year time frame. Looking at columns four to six of Table 3, we observe nearly identical reductions in 3-year mortality as for 1-year mortality. This suggests that peers play a substantially greater role in the first year after exiting treatment than in subsequent years. It could be that the social ties formed during inpatient treatment themselves deteriorate over time, or that the impact of social ties lessens over time. For example, if peers cause an immediate and sustained cessation in substance use, we would expect to find similar coefficients for one and three year mortality risk.

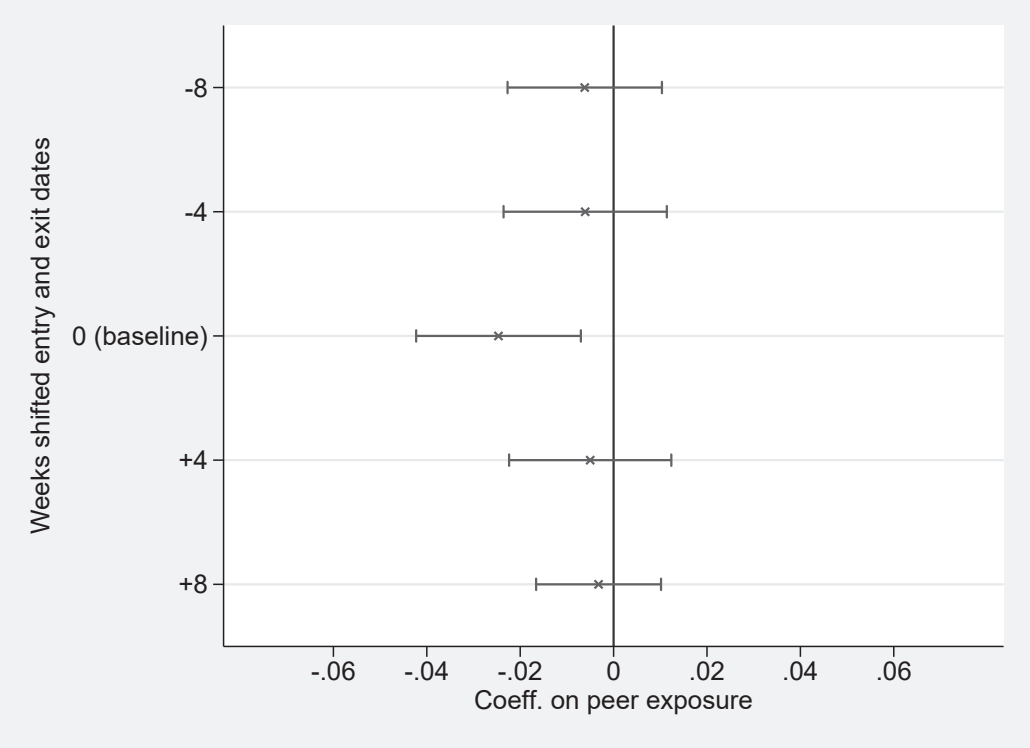
5.3 Placebo tests

Our first placebo test shifts the dates of entry and exit used to define overlapping episodes. This assigns each inpatient a placebo group of other inpatients (potential peers) who received treatment at the same facility at around the same time, but whose treatment episodes had no (or limited) overlap. We shift the entry and exit dates by four weeks and eight weeks. Shifting by four weeks implies that the shifted spell would have no overlap with the actual spell for an individual with median length of stay (9 days) and little overlap for an inpatient with mean length of stay (29 days). Shifting by eight weeks implies that the gap between the actual and shifted spell is approximately double the mean length of stay. This means that the group of potential peers will typically be entirely different in the shifted spell relative to the unshifted spell. Figure 3 reports the coefficient and 0.95 confidence intervals for the impact of peers on mortality in the first year after leaving treatment.[15] The coefficient is small in magnitude and not statistically distinguishable from zero whenever the spell is shifted. This contrasts with our baseline findings of a statistically significant negative coefficient.

Table 4 replicates our baseline results using a sample of patients admitted into inpatient treat-

[15] See the appendix for death by cause.

Figure 3: Placebo test - shifted entry and exit dates - Death within 1 year of exiting treatment



ment facilities as emergency patients (see Table 9 in the appendix for a description of the sample of 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. Inspection of Table 4 shows that the point estimates of interest are small in magnitude and do not approach statistical significance at conventional levels for any cause of death or time horizon. Our baseline results for treatment patients found a negative coefficient that was statistically significant at the 0.01 level and implies that an additional home-county peer reduced mortality in the first year after exit by 5% relative to the mean. In contrast, 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 (+0.6%) is an order of magnitude smaller than our baseline findings. Together, both placebo tests provide evidence to support the claim that our baseline findings capture a genuine influence of peers on mortality as opposed to reflecting some other artifact of inpatient admission.

Table 4: Placebo test - sample of emergency care patients

	Death within 1 year of exit			Death within 3 years of exit		
	All Causes	Internal	External	All Causes	Internal	External
peer exposure	0.003	-0.000	0.000	-0.009	-0.007	-0.005
	(0.009)	(0.007)	(0.016)	(0.005)	(0.011)	(0.012)
Mean dep. var.	0.020	0.007	0.012	0.059	0.027	0.030
+1 SD (% of mean)	4.63	-6.19	0.88	-4.78	-7.90	-5.63
+1 peer	0.56	-0.75	0.11	-0.59	-0.97	-0.69
Observations	3,553	3,553	3,553	3,553	3,553	3,553
R-squared	0.045	0.046	0.033	0.058	0.080	0.027

Notes: All models include facility, county and month-by-year of exit fixed effects. Controls are an indicator for a drug use disorder, an indicator for male, age, age^2/1000, an indicator for no more than minimum education level, an indicator for both parents having minimum education, an indicator for being single and an indicator for a mental illness comorbidity. Contextual controls (averages over all other overlapping inpatients, weighted by days overlapped) are the proportion receiving care for a drug use disorder, the proportion who are male, the proportion who are young ($\leq$ 26), the proportion who have no more than minimum education, the proportion for which both parents have no more than minimum education and the proportion with mental illness comorbidities. Standard errors are clustered by facility.

5.4 Robustness tests

Despite the institutional setting effectively guaranteeing that the date of entry into treatment is as good as randomly assigned, we may be concerned about endogenous exit from treatment. The first panel of Table 12 in the appendix addresses this by repeating our baseline analysis, but using only other inpatients encountered on the first day of the treatment spell to construct the peer variable. The estimates are nearly identical to our baseline results. The second panel of Table 12 in the appendix reports results measuring death from the 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.

Another concern is that there might exist county-by-facility level observables which drive both the mortality risk of the focal inpatient and their exposure to peers. Table 13 in the appendix addresses this by including the county-by-facility average of the peer variable in the list of controls. The reported results are similar to our baseline results, though sometimes larger in magnitude by a factor of around two.

A final concern relates to 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 timeframe. Though the linear probability model is the workhorse empirical model in a setting

Table 5: Descriptive statistics by patient SUD type - outcomes and peer variables

	Within 1 year of exit				Within 3 years of exit			
	Alcohol use disorder		Drug use disorder		Alcohol use disorder		Drug use disorder	
	Mean	St Dev.	Mean	St Dev.	Mean	St Dev.	Mean	St Dev.
Death								
all-causes	0.023	0.151	0.021	0.142	0.085	0.279	0.053	0.224
internal COD	0.017	0.131	0.005	0.071	0.056	0.231	0.014	0.117
external COD	0.006	0.077	0.016	0.124	0.025	0.157	0.038	0.192
drug induced	0.001	0.025	0.007	0.083	0.003	0.055	0.021	0.142
Hospital episode								
drug induced intoxication	0.014	0.117	0.046	0.209	0.041	0.198	0.115	0.319
drug induced poisoning	0.025	0.157	0.029	0.167	0.051	0.220	0.062	0.241
Peers								
peer exposure	0.536	0.301	0.546	0.327				
drug peer exposure	0.359	0.256	0.383	0.283				
alcohol peer exposure	0.177	0.190	0.163	0.18				

where identification rests on fixed effects, alternatives that are arguably better suited to modeling rare events are the complementary log-log model and the penalized Logit model proposed by Firth (1993). Table 14 in the appendix reports the results for these models. For ease of comparison with the linear probability model, we report average marginal effects. For all-cause mortality we find average marginal effects of -0.032 for the complementary log-log model and -0.041 for the penalized Logit model, both of which are statistically significant at the 0.01 level. These are larger in magnitude than for the linear probability model (-0.025). We also find larger average marginal effects for death from external causes (-0.029 and -0.025) than for the linear probability model (-0.017), though only the penalized Logit estimate is statistically significant at the 0.05 level.

5.5 Heterogeneity by SUD type

Our baseline results considered a sample containing individuals admitted for either alcohol use disorder or drug use disorder. We now consider each group separately. This is because their underlying causes of mortality may differ. Individuals with drug use disorders are more likely to die from external causes (e.g., overdose) whereas those with alcohol use disorders are more likely to die from internal causes (e.g., liver disease), as shown in Table 5. Unsurprisingly, we also observe that drug induced deaths are more prevalent among inpatients receiving treatment for a drug use disorder.

Table 6 repeats our baseline mortality analysis separately for those receiving treatment for alcohol use disorders and drug use disorders. The first two columns consider one year all-cause mortality. Whilst the coefficient on peer exposure is negative in both columns, it is larger and

Table 6: Heterogeneity by SUD type

	Death within 1 year of exit		Death within 3 years of exit	
	Alcohol use disorder	Drug use disorder	Alcohol use disorder	Drug use disorder
peer exposure	-0.021	-0.030***	-0.022	-0.037***
	(0.023)	(0.011)	(0.028)	(0.012)
Mean dep. var.	0.023	0.021	0.085	0.053
+1 SD (% of mean)	-27.60	-47.23	-7.63	-23.04
+1 peer	-2.95	-6.67	-0.82	-3.25
Observations	1,664	2,172	1,664	2,172
R-squared	0.065	0.058	0.070	0.064

Notes: All models include facility, county and month-by-year of exit fixed effects. Controls are an indicator for a drug use disorder, an indicator for male, age, age^2/1000, an indicator for no more than minimum education level, an indicator for both parents having minimum education, an indicator for being single and an indicator for a mental illness comorbidity. Contextual controls (averages over all other overlapping inpatients, weighted by days overlapped) are the proportion receiving care for a drug use disorder, the proportion who are male, the proportion who are young (≤ 26), the proportion who have no more than minimum education, the proportion for which both parents have no more than minimum education and the proportion with mental illness comorbidities. Standard errors are clustered by facility.

statistically significant at the 0.01 level for the sample of individuals with a drug use disorder. We find a similar pattern in columns three and four, which consider three year mortality. Overall our baseline findings appear to be driven by those receiving treatment for a drug use disorder. This likely reflects the fact that alcohol misuse may result in a slow deterioration in overall health, which might not be reflected in mortality over a three year window. In contrast, drug use may lead to an increased risk of external-cause mortality (e.g., overdose) over a shorter time horizon.

5.6 Mechanisms

To dig into the mechanisms behind the peer-induced reduction in mortality of inpatients treated for a drug use disorder, we look for evidence of a change in the patterns of drug use among inpatients treated for a drug use disorder. We first look at drug induced deaths. We consider a three year post-treatment time frame because drug induced deaths in the first year are relatively uncommon, equating to 0.7% of sample members (see Table 5). This rises to 2.1% within three years. The first column of Table 7 shows that peers reduce drug induced deaths within three years of exiting inpatient treatment. The coefficient is negative and statistically significant at the 0.01 level, with a standard deviation increase in the proportion of peers resident in the same county leading to a 36% reduction in mortality risk relative to the mean. That peers reduce mortality for causes explicitly linked to drug use suggests that peers change the patterns of drug use.

We now divide peers into two types: those with a drug use disorder and those with an alcohol use disorder.[16] Looking at the results in column two of Table 7, whilst both coefficients are negative, the coefficient on peers receiving treatment for a drug use disorder is larger and is statistically significant at the 0.05 level. This suggests that drug using peers play a greater role in reducing drug induced deaths among inpatients admitted for a drug use disorder. This might arise for a number of reasons. First, our results are consistent with common experience of a particular type of SUD facilitating peers to support one another in recovery. Second, they are consistent with peers promoting the use of effective strategies to use more safely. The efficacy of such strategies may be substance specific. For example, not using alone might deliver greater benefits when using illicit drugs relative to alcohol, due to the difference in overdose risk. The fact that we find a negative coefficient suggests that any peer influences that increase drug use are more than offset by peer influences that support recovery and facilitate strategies to mitigate risk.[17]

Thus far we have presented evidence that peers influence the patterns of use among inpatients treated for a drug use disorder. Peers could reduce mortality either through altering the quantity or safety of drug use. If peers primarily reduce mortality by facilitating safer drug use we might expect peers to increase hospital episodes for intoxication or poisoning. This would arise if peers contact emergency healthcare services in response to particularly severe overdoses that might otherwise lead to death. Table 8 presents our findings. The first two columns consider hospital episodes for drug induced intoxication and poisoning in the year following exit from inpatient treatment. The coefficients are statistically insignificant and small in magnitude, as are the implied effect sizes. Column three considers an indicator for either intoxication or poisoning within one year of exiting treatment. The coefficient remains small in magnitude and insignificant. Columns four to six consider a three year horizon. All coefficients are positive but all are insignificant. Overall we find no evidence that peers increase hospital episodes for intoxication or (non-fatal) poisoning among drug users. This suggests that the peer-induced reduction in mortality risk is mainly driven by a reduction in consumption, rather than by safer use.

[16] Heterogeneous peer effects due to the 'match' between the focal individual and their peers (in our case, by type of SUD) have previously been studied by Bayer *et al.* (2009) and Damm & Gorinas (2020). For example, Damm & Gorinas (2020) show that sharing a spell of incarceration with another individual convicted of the same type of offence increases the probability of criminal recidivism for that type of offence.

[17] A second explanation for the findings in column two of Table 7 is that similar individuals may be more likely to form lasting ties than less similar individuals (i.e., there could be homophily), and substance type might be an important dimension of similarity. Since our data do not allow us to observe whether social ties are actually formed (we only observe those that could plausibly be formed), it also could be that the type of individual with whom a social tie is formed does not influence patterns of drug use, but that individuals with a drug use disorder are more likely to connect with one another.

Table 7: Mechanisms - Drug induced deaths among inpatients with a drug use disorder

	Death within 3 years of exit	
	Drug induced death	Drug induced death
peer exposure	-0.023***	
	(0.007)	
drug peer exposure		-0.025**
		(0.010)
alcohol peer exposure		-0.016
		(0.017)
Mean dep. var.	0.021	0.021
+1 SD (% of mean)	-36.41	
+1 SD (drug)		-33.69
+1 SD (alcohol)		-13.71
Observations	2,172	2,172
R-squared	0.042	0.042

Notes: All models include facility, county and month-by-year of exit fixed effects. Controls are an indicator for male, age, age^2/1000, an indicator for no more than minimum education level, an indicator for both parents having minimum education, an indicator for being single and an indicator for a mental illness comorbidity. Contextual controls (averages over all other overlapping inpatients, weighted by days overlapped) are the proportion receiving care for a drug use disorder, the proportion who are male, the proportion who are young ($\leq$ 26), the proportion who have no more than minimum education, the proportion for which both parents have no more than minimum education and the proportion with mental illness comorbidities. Standard errors are clustered by facility.

Table 8: Drug induced somatic hospital episodes among inpatients for a drug use disorder

	Episode within 1 year of exit			Episode within 3 years of exit		
	Intoxication	Poisoning	Either	Intoxication	Poisoning	Either
peer exposure	-0.004	-0.004	-0.003	0.038	0.010	0.031
	(0.012)	(0.018)	(0.023)	(0.026)	(0.022)	(0.027)
Mean dep. var.	0.046	0.029	0.071	0.12	0.06	0.16
+1 SD (% of mean)	-2.84	-4.51	-1.24	10.36	5.45	6.34
+1 peer	-0.42	-0.65	-0.20	1.53	0.77	0.89
Observations	2,172	2,172	2,172	2,172	2,172	2,172
R-squared	0.091	0.058	0.087	0.084	0.064	0.086

Notes: All models include facility, county and month-by-year of exit fixed effects. Controls are an indicator for male, age, age^2/1000, an indicator for no more than minimum education level, an indicator for both parents having minimum education, an indicator for being single and an indicator for a mental illness comorbidity. Contextual controls (averages over all other overlapping inpatients, weighted by days overlapped) are the proportion receiving care for a drug use disorder, the proportion who are male, the proportion who are young ($\leq$ 26), the proportion who have no more than minimum education, the proportion for which both parents have no more than minimum education and the proportion with mental illness comorbidities. Standard errors are clustered by facility.

6 Conclusion

This paper provides the first empirical evidence on the role of peers in recovery from substance use disorders. Our analysis uses high quality linked administrative data from the Norwegian Patient Registry (NPR) and Cause of Death Registry. The NPR data cover the universe of patients admitted for inpatient treatment with an ICD10 code for SUDs. Exposure to peers likely to influence post-treatment recovery is measured by the proportion of overlapping inpatients at the same facility who are resident in the same county as the focal patient. We argue that due to institutional features determining the timing of entry into treatment, peer exposure is as good as randomly assigned in a model with facility and county fixed effects. While we have no direct measure of substance use, we measure it indirectly using indicators for using death, and death from specific causes, including drug induced deaths. These are particularly salient measures of relapse following inpatient treatment because the risk of death among people with problematic drug use is elevated after a period of abstinence from drug use, such as discharge from an inpatient treatment facility or release from prison (Binswanger *et al.* , 2007; Gossop *et al.* , 2002; Ravndal & Amundsen, 2010). And while there has been some attention to drug induced deaths following release from prison, much less in known about the factors contributing to the increased risk of mortality following inpatient treatment episodes.

Our analysis shows that greater exposure to home-county peers reduces deaths following inpatient treatment for a SUD. This effect is concentrated among individuals who seek treatment for drug use disorders (rather than alcohol use disorders), who benefit from a large reduction in drug induced mortality. Examining which peers matter, we find that peers who are themselves seeking treatment for drug use disorders drive the reduction in drug induced deaths among individuals who sought treatment for drug use disorders. This suggests two channels through which peer effects may be working; (1) reducing the use of illicit drugs, or (2) encouraging safer modes when using illicit drugs. To explore this we examine (non-fatal) drug induced hospital episodes following inpatient treatment. If peers reduce the probability of a drug induced death by reducing the probability of using alone, for example, then we might expect to find that peers increase drug induced hospital episodes for overdose and intoxication, in addition to reducing drug induced deaths. We find no evidence that peers increase drug induced hospital episodes, suggesting that peers reduce mortality mainly through reducing drug use rather than fostering safer drug use. All in all, the evidence points to a net effect of peers acquired during inpatient treatment for drug use disorders supporting recovery in the period following treatment.

Deaths related to substance use are preventable, yet they are the leading cause of mortality among substance users. This suggests significant scope for policy intervention. Our findings suggest

that deaths following inpatient treatment episodes may be reduced by leveraging the bonds formed among inpatients while in treatment. This could entail, for example, facilitating former patients establishing mutual self-help recovery programs, to which patients could be connected with upon discharge. If these types of mutual-help programs accommodate multiple pathways to recovery, they would fill important post-treatment gaps in support for 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 even specialization of treatment facilities by substance (e.g., facilities dedicated to drug use disorders).

References

Adamopoulou, Effrosyni, Greenwood, Jeremy, Guner, Nezih, & Kopecky, Karen. 2024. *The role of friends in the opioid epidemic*. Tech. rept. National Bureau of Economic Research.

Ajilore, O., Amialchuk, A., & Egan, K. 2016. Alcohol consumption by youth: Peers, parents, or prices? *Economics and Human Biology*, **23**, 76–83.

Aldridge, Robert W, Story, Alistair, Hwang, Stephen W, Nordentoft, Merete, Luchenski, Serena A, Hartwell, Greg, Tweed, Emily J, Lewer, Dan, Katikireddi, Srinivasa Vittal, & Hayward, Andrew C. 2018. Morbidity and mortality in homeless individuals, prisoners, sex workers, and individuals with substance use disorders in high-income countries: a systematic review and meta-analysis. *The Lancet*, **391**(10117), 241–250.

Alpert, Abby, Powell, David, & Pacula, Rosalie Liccardo. 2018. Supply-side drug policy in the presence of substitutes: Evidence from the introduction of abuse-deterrent opioids. *American Economic Journal: Economic Policy*, **10**(4), 1–35.

Alpert, Abby, Evans, William N, Lieber, Ethan MJ, & Powell, David. 2022. Origins of the opioid crisis and its enduring impacts. *The Quarterly Journal of Economics*, **137**(2), 1139–1179.

Arduini, T., Bisin, A., Özgür, O., & Patacchini, E. 2019. *Dynamic Social Interactions and Health Risk Behavior*.

Bayer, Patrick, Hjalmarsson, Randi, & Pozen, David. 2009. Building criminal capital behind bars: Peer effects in juvenile corrections. *The Quarterly Journal of Economics*, **124**(1), 105–147.

Billings, Stephen B, & Schnepel, Kevin T. 2022. Hanging Out with the Usual Suspects Neighborhood Peer Effects and Recidivism. *Journal of Human Resources*, **57**(5), 1758–1788.

Binswanger, I. A., Stern, M. F., Deyo, R. A., Heagerty, P. J., Cheadle, A., Elmore, J. G., & Koepsell, T. D. 2007. Release from prison—A high risk of death for former inmates. *New England Journal of Medicine*, **356**(2), 157–165.

Bohnert, Amy SB, Bradshaw, Catherine P, & Latkin, Carl A. 2009. A social network perspective on heroin and cocaine use among adults: evidence of bidirectional influences. *Addiction*, **104**(7), 1210–1218.

Card, David, & Giuliano, Laura. 2013. Peer effects and multiple equilibria in the risky behavior of friends. *Review of Economics and Statistics*, **95**(4), 1130–1149.

Case, Anne, & Katz, Lawrence F. 1991. *The company you keep: The effects of family and neighborhood on disadvantaged youths*.

Christakis, Nicholas A, & Fowler, James H. 2007. The spread of obesity in a large social network over 32 years. *New England journal of medicine*, **357**(4), 370–379.

Christakis, Nicholas A, & Fowler, James H. 2008. The collective dynamics of smoking in a large social network. *New England Journal of Medicine*, **358**(21), 2249–2258.

Conover, Emily, & Scrimgeour, David. 2013. Health consequences of easier access to alcohol: New Zealand evidence. *Journal of Health Economics*, **32**(3), 570–585.

Curtis, Richard, Friedman, Samuel R, Neaigus, Alan, Jose, Benny, Goldstein, Marjorie, & Ildefonso, Gilbert. 1995. Street-level drug markets: Network structure and HIV risk. *Social networks*, **17**(3-4), 229–249.

Damm, Anna Piil, & Gorinas, Cédric. 2020. Prison as a criminal school: Peer effects and criminal learning behind bars. *The Journal of Law and Economics*, **63**(1), 149–180.

Díaz, Carlos, Patacchini, Eleonora, Verdier, Thierry, & Zenou, Yves. 2021. Leaders in juvenile crime. *Journal of Economic Behavior and Organization*, **192**, 638–667.

Eddie, David, Hoffman, Lauren, Vilsaint, Corrie, Abry, Alexandra, Bergman, Brandon, Hoeppner, Bettina, Weinstein, Charles, & Kelly, John F. 2019. Lived experience in new models of care for substance use disorder: a systematic review of peer recovery support services and recovery coaching. *Frontiers in psychology*, **10**, 1052.

Eisenberg, D., Golberstein, E., & Whitlock, J.L. 2014. Peer effects on risky behaviors: New evidence from college roommate assignments. *Journal of Health Economics*, **33**, 126–138.

Evans, William N, Lieber, Ethan MJ, & Power, Patrick. 2019. How the reformulation of OxyContin ignited the heroin epidemic. *Review of Economics and Statistics*, **101**(1), 1–15.

Firth, David. 1993. Bias reduction of maximum likelihood estimates. *Biometrika*, **80**(1), 27–38.

Fletcher, J.M., & Ross, S.L. 2018. Estimating the effects of friends on health behaviors of adolescents. *Health economics*, **27**(10), 1450–1483.

Ganesan, Murali, Eikenberry, Allison, Poluektova, Larisa Y, Kharbanda, Kusum K, & Osna, Natalia A. 2020. Role of alcohol in pathogenesis of hepatitis B virus infection. *World Journal of Gastroenterology*, **26**(9), 883.

Gatti, Francesca, Walderhaug, Espen, Kern-Godal, Ann, Lysell, Jeanette, & Arnevik, Espen Ajo. 2020. Complementary horse-assisted therapy for substance use disorders: a randomized controlled trial. *Addiction science & clinical practice*, **15**(1), 1–11.

Gaviria, A., & Raphael, S. 2001. School-based peer effects and juvenile behavior. *Review of Economics and Statistics*, **83**(2), 257–268.

Gjersing, Linn. 2023. Rusmiddelbruk blant personer rekruttert fra lavterskeltiltak og gateplan-Et øyeblikksbilde og en sammenligning med rusmiddelbruk blant deltakere rekruttert i 2013 og 2017.

Gjersing, Linn, & Bretteville-Jensen, Anne Line. 2014. Gender differences in mortality and risk factors in a 13-year cohort study of street-recruited injecting drug users. *BMC Public Health*, **14**, 1–11.

Glei, Dana A, & Preston, Samuel H. 2020. Estimating the impact of drug use on US mortality, 1999-2016. *PLoS One*, **15**(1), e0226732.

Godøy, Anna, & Huitfeldt, Ingrid. 2020. Regional variation in health care utilization and mortality. *Journal of Health Economics*, **71**, 102254.

Gossop, Michael, Stewart, Duncan, Treacy, Samantha, & Marsden, John. 2002. A prospective study of mortality among drug misusers during a 4-year period after seeking treatment. *Addiction*, **97**(1), 39–47.

Hollingsworth, Alex, Ruhm, Christopher J, & Simon, Kosali. 2017. Macroeconomic conditions and opioid abuse. *Journal of health economics*, **56**, 222–233.

Hsieh, C.S., & Van Kippersluis, H. 2018. Smoking initiation: Peers and personality. *Quantitative Economics*, **9**(2), 825–863.

Jason, Leonard, Stevens, Ed, Ferrari, Joseph R, Thompson, Erin, & Legler, Ray. 2012. Social networks among residents in recovery homes. *Advances in Psychology Study*, **1**(3), 4.

Jones, Andrew M. 1994. Health, addiction, social interaction and the decision to quit smoking. *Journal of health economics*, **13**(1), 93–110.

Kaskutas, Lee Ann. 2009. Alcoholics Anonymous effectiveness: Faith meets science. *Journal of addictive diseases*, **28**(2), 145–157.

Kawaguchi, D. 2004. Peer effects on substance use among American teenagers. *Journal of Population Economics*, **17**(2), 351–367.

Kim, Jun Sung, Patacchini, Eleonora, Picard, Pierre M, & Zenou, Yves. 2023. Spatial interactions. *Quantitative Economics*, **14**(4), 1295–1335.

Kling, Jeffrey R, Liebman, Jeffrey B, & Katz, Lawrence F. 2007. Experimental analysis of neighborhood effects. *Econometrica*, **75**(1), 83–119.

Kremer, M., & Levy, D. 2008. Peer effects and alcohol use among college students. *Journal of Economic Perspectives*, **22**(3), 189–206.

Larney, Sarah, Tran, Lucy Thi, Leung, Janni, Santo, Thomas, Santomauro, Damian, Hickman, Matt, Peacock, Amy, Stockings, Emily, & Degenhardt, Louisa. 2020. All-cause and cause-specific mortality among people using extramedical opioids: a systematic review and meta-analysis. *JAMA psychiatry*, **77**(5), 493–502.

Li, Y., & Guo, G. 2020. Heterogeneous peer effects on marijuana use: Evidence from a natural experiment. *Social Science and Medicine*, **252**, 112907.

Lundborg, P. 2006. Having the wrong friends? Peer effects in adolescent substance use. *Journal of Health Economics*, **25**(2), 214–233.

Manski, Charles F. 1993. Identification of endogenous social effects: The reflection problem. *The review of economic studies*, **60**(3), 531–542.

Palali, Ali, & Van Ours, Jan C. 2017. Love conquers all but nicotine: spousal peer effects on the decision to quit smoking. *Health economics*, **26**(12), 1710–1727.

Patacchini, Eleonora, & Zenou, Yves. 2012. Juvenile delinquency and conformism. *The Journal of Law, Economics, and Organization*, **28**(1), 1–31.

Raiker, Nisha, Aouthmany, Mouhammad, & Ezra, Navid. 2016. Dermatologic signs and symptoms of substance abuse. *J Clin Exp Dermatol Res*, **7**(2), 337.

Ravndal, Edle, & Amundsen, Ellen J. 2010. Mortality among drug users after discharge from inpatient treatment: an 8-year prospective study. *Drug and alcohol dependence*, **108**(1-2), 65—69.

Room, Robin. 2005. Stigma, social inequality and alcohol and drug use. *Drug and alcohol review*, **24**(2), 143–155.

Swensen, Isaac D. 2015. Substance-abuse treatment and mortality. *Journal of Public Economics*, **122**, 13–30.

Tan, Kegon Teng Kok, & Zapryanova, Mariyana. 2022. Peer Effects and Recidivism: The Role of Race and Age. *The Journal of Law, Economics, and Organization*, **38**(3), 721–740.

Torvik, Fartein Ask, & Rognmo, Kamilla. 2011. Children of parents with mental disorders or alcohol abuse. Scope and consequences. *report National Institute of Public Health.*

White, William, Galanter, Marc, Humphreys, Keith, & Kelly, John. 2020. "We Do Recover" Scientific Studies on Narcotics Anonymous. *Cochrane Database of Systematic Reviews*, **3**, CD012880.

Appendix

Figure 4: Variation in peer exposure by SUD type (alcohol or drugs)

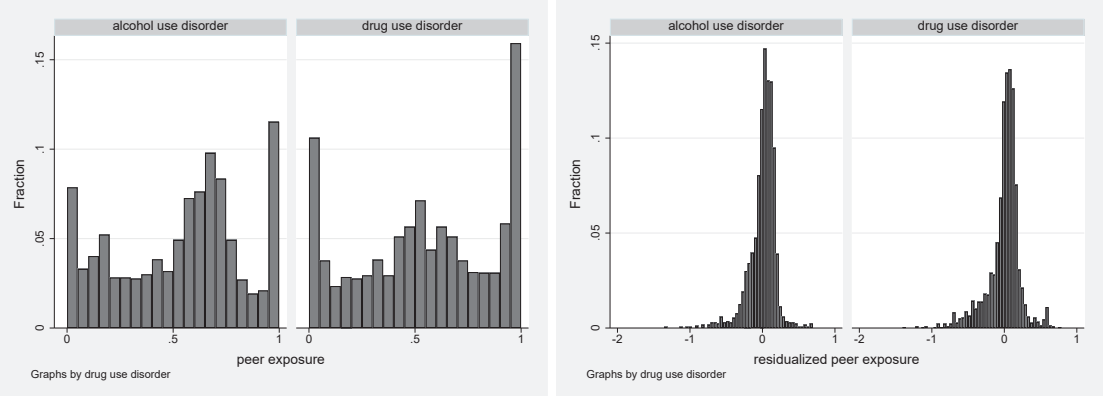
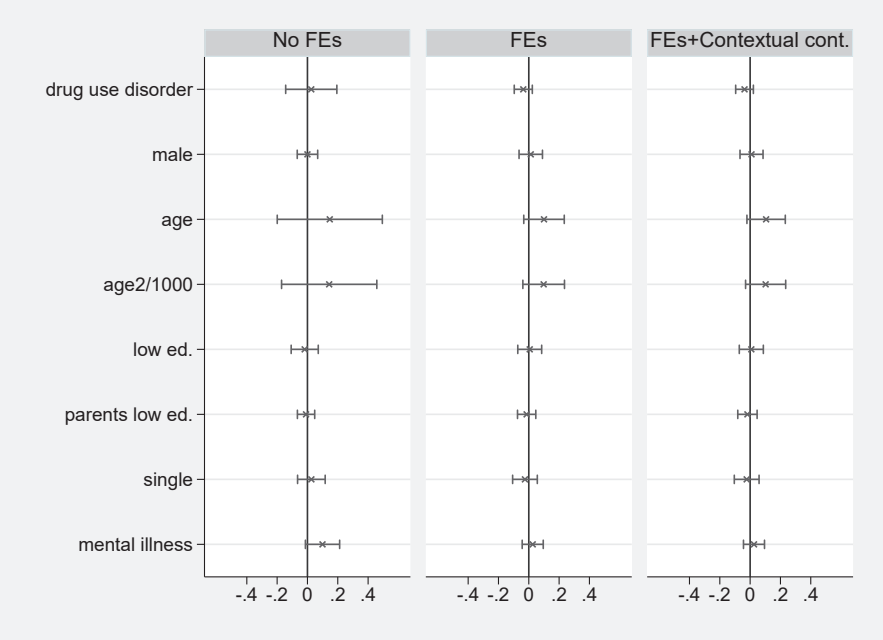


Table 9: Descriptive statistics for emergency patients - outcomes and peer exposure

	Time since left inpatient treatment			
	within 1 year		within 3 years	
	Mean	St Dev.	Mean	St Dev.
Death				
all-causes	0.020	0.140	0.059	0.236
internal COD	0.007	0.084	0.027	0.161
external COD	0.012	0.084	0.030	0.170
Peers				
num. overlapping inpatients	15.363	20.957		
num. overlapping inpatients from same county	10.262	15.155		
peer exposure	0.697	0.323		

Figure 5: Balance test - one-by-one



Notes: N=3,838. 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.

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 5 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 10 reports the results of this exercise. In models with neither fixed effects nor contextual controls we find an associated with the peers variable that is statistically significant at the 0.1 level. However, with fixed effects and contextual controls the magnitude of the coefficient on the peers variable is reduced by a factor of

Table 10: Balance test - fitted values

	Death within 1 year of exit (fitted values)			Death within 3 years of exit (fitted values)		
peer exposure	0.00291*	0.00107	0.00113	0.00291*	0.00107	0.00113
	(0.00154)	(0.000815)	(0.000773)	(0.00154)	(0.000815)	(0.000773)
Mean dep. var.	0.0219	0.0219	0.0219	0.0670	0.0670	0.0670
Observations	3,837	3,837	3,837	3,837	3,837	3,837
FEs	N	Y	Y	N	Y	Y
Contextual controls	N	N	Y	N	N	Y

Notes: Outcomes are fitted values using an indicator for a drug use disorder, an indicator for male, age, age^2/1000, an indicator for no more than minimum education level, an indicator for both parents having minimum education, an indicator for being single and an indicator for a mental illness comorbidity. Contextual controls (averages over all other overlapping inpatients, weighted by days overlapped) are the proportion receiving care for a drug use disorder, the proportion who are male, the proportion who are young (≤ 26), the proportion who have no more than minimum education, the proportion for which both parents have no more than minimum education and the proportion with mental illness comorbidities. Standard errors are clustered by facility.

three, and it is not statistically significant.

Table 11: Full baseline results

	Death within 1 year of exit			Death within 3 years of exit		
	All Causes	Internal	External	All Causes	Internal	External
peer exposure	-0.025***	-0.008	-0.017**	-0.026*	-0.005	-0.019*
	(0.009)	(0.006)	(0.008)	(0.013)	(0.008)	(0.010)
drug use disorder	0.013***	-0.002	0.014***	0.008	-0.009	0.019**
	(0.005)	(0.005)	(0.004)	(0.010)	(0.010)	(0.007)
male	0.002	0.000	0.002	0.026***	0.011**	0.014**
	(0.004)	(0.003)	(0.004)	(0.008)	(0.005)	(0.006)
age	0.000	-0.000	0.000	-0.001	-0.002	0.000
	(0.002)	(0.001)	(0.001)	(0.002)	(0.002)	(0.001)
age^2/1000	0.008	0.011	-0.003	0.046*	0.049*	-0.006
	(0.020)	(0.013)	(0.012)	(0.027)	(0.026)	(0.011)
low ed.	-0.001	-0.000	-0.001	-0.000	0.005	-0.008
	(0.005)	(0.004)	(0.003)	(0.010)	(0.006)	(0.008)
single	0.000	0.003	-0.003	0.005	0.012***	-0.004
	(0.005)	(0.003)	(0.004)	(0.006)	(0.003)	(0.005)
mental illness	0.007	-0.002	0.008*	0.009	-0.000	0.011
	(0.006)	(0.003)	(0.005)	(0.011)	(0.007)	(0.007)
both parents low ed.	-0.004	-0.000	-0.004	-0.013**	-0.003	-0.007
	(0.004)	(0.003)	(0.003)	(0.006)	(0.007)	(0.006)
prop. drug use disorder	-0.004	0.005	-0.008	-0.018	-0.009	-0.014
	(0.012)	(0.010)	(0.009)	(0.023)	(0.014)	(0.015)
prop. male	0.022*	0.014	0.009	0.020	-0.000	0.019
	(0.013)	(0.009)	(0.009)	(0.027)	(0.015)	(0.021)
prop. age$\leq$26	0.015	0.012	0.003	0.023	0.025*	0.001
	(0.015)	(0.011)	(0.008)	(0.029)	(0.014)	(0.024)
prop. low ed.	-0.019	-0.015	-0.004	-0.014	-0.012	0.005
	(0.016)	(0.014)	(0.008)	(0.027)	(0.019)	(0.021)
prop. single	0.018	0.016**	0.002	0.024	0.026**	0.004
	(0.013)	(0.007)	(0.011)	(0.024)	(0.012)	(0.016)
prop. mental illness	0.018	0.005	0.013	0.014	-0.024	0.037
	(0.021)	(0.012)	(0.012)	(0.035)	(0.019)	(0.030)
prop. both parents low ed.	0.019	0.029*	-0.010	0.006	0.008	-0.011
	(0.018)	(0.014)	(0.014)	(0.027)	(0.023)	(0.023)
Mean dep. var.	0.0219	0.0104	0.0115	0.067	0.032	0.033
Observations	3,837	3,837	3,837	3,837	3,837	3,837
R-squared	0.038	0.037	0.032	0.046	0.062	0.033

Notes: All models include facility, county and month-by-year of exit fixed effects. Standard errors are clustered by facility.

Figure 6: Placebo test - shifted entry and exit dates - internal (left) and external (right) COD within 1 year

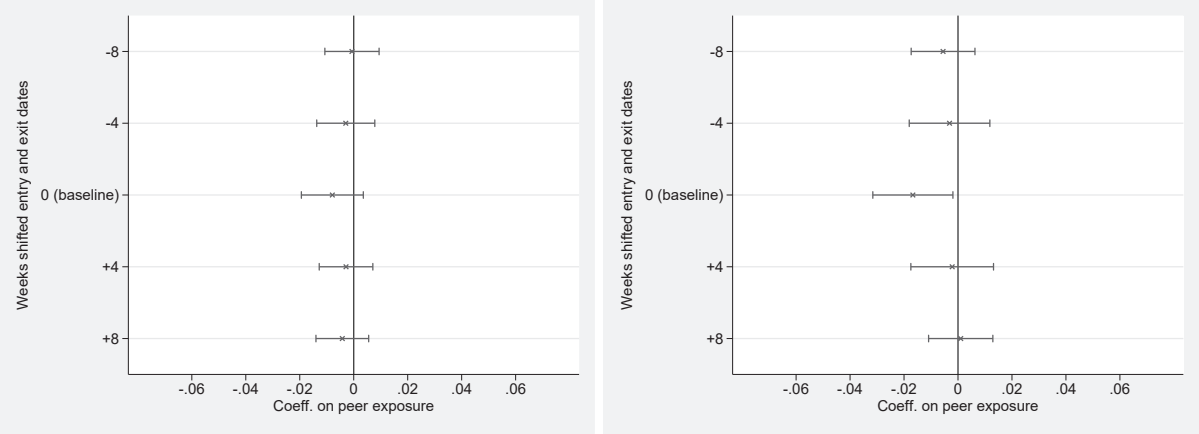


Table 12: Robustness - endogenous exit from inpatient treatment

	First day peers only					
	Death within 1 year of exit			Death within 3 years of exit		
	All Causes	Internal	External	All Causes	Internal	External
peer exposure	-0.027**	-0.007	-0.020*	-0.029**	-0.005	-0.023*
	(0.010)	(0.006)	(0.010)	(0.013)	(0.008)	(0.012)
Observations	3,770	3,770	3,770	3,770	3,770	3,770
R-squared	0.038	0.039	0.033	0.048	0.065	0.035
	Model based on entry date					
	Death within 1 year of entry			Death within 3 years of entry		
	All Causes	Internal	External	All Causes	Internal	External
peer exposure	-0.021***	-0.006	-0.015**	-0.024*	-0.002	-0.020*
	(0.008)	(0.005)	(0.007)	(0.014)	(0.008)	(0.008)
Observations	3,837	3,837	3,837	3,837	3,837	3,837
R-squared	0.038	0.037	0.034	0.047	0.064	0.030
	First day + entry date					
	Death within 1 year of entry			Death within 3 years of entry		
	All Causes	Internal	External	All Causes	Internal	External
peer exposure	-0.023**	-0.004	-0.019**	-0.026*	-0.002	-0.023*
	(0.009)	(0.005)	(0.009)	(0.014)	(0.007)	(0.013)
Observations	3,837	3,837	3,837	3,837	3,837	3,837
R-squared	0.037	0.039	0.035	0.049	0.067	0.033

Notes: Controls are an indicator for a drug use disorder, an indicator for male, age, age^2/1000, an indicator for no more than minimum education level, an indicator for both parents having minimum education, an indicator for being single and an indicator for a mental illness comorbidity. Contextual controls (averages over all other overlapping inpatients, weighted by days overlapped) are the proportion receiving care for a drug use disorder, the proportion who are male, the proportion who are young (≤ 26), the proportion who have no more than minimum education, the proportion for which both parents have no more than minimum education and the proportion with mental illness comorbidities. Standard errors are clustered by facility.

Table 13: Robustness - Controlling for county-by-facility mean of peers variable

	Death within 1 year of exit			Death within 3 years of exit		
	All Causes	Internal	External	All Causes	Internal	External
peer exposure	-0.052**	-0.008	-0.044**	-0.068**	-0.006	-0.056**
	(0.021)	(0.010)	(0.018)	(0.024)	(0.015)	(0.022)
Observations	3,837	3,837	3,837	3,837	3,837	3,837
R-squared	0.039	0.037	0.035	0.047	0.062	0.034

Notes: All models include facility, county and month-by-year of exit fixed effects. Controls are an indicator for a drug use disorder, an indicator for male, age, age^2/1000, an indicator for no more than minimum education level, an indicator for both parents having minimum education, an indicator for being single and an indicator for a mental illness comorbidity. Contextual controls (averages over all other overlapping inpatients, weighted by days overlapped) are the proportion receiving care for a drug use disorder, the proportion who are male, the proportion who are young ($\leq$ 26), the proportion who have no more than minimum education, the proportion for which both parents have no more than minimum education and the proportion with mental illness comorbidities. Standard errors are clustered by facility.

Table 14: Robustness - Alternative models for rare events

	Death within 1 year of exit					
	Complementary log-log			Penalized Logit		
	All Causes	Internal	External	All Causes	Internal	External
peer exposure	-0.032***	-0.017	-0.029	-0.041***	-0.025*	-0.031**
	(0.011)	(0.012)	(0.018)	(0.015)	(0.013)	(0.015)
Observations	3,837	3,837	3,837	3,837	3,837	3,837

Notes: Average marginal effects are reported. All models include facility, county and month-by-year of exit fixed effects. Controls are an indicator for a drug use disorder, an indicator for male, age, age^2/1000, an indicator for no more than minimum education level, an indicator for both parents having minimum education, an indicator for being single and an indicator for a mental illness comorbidity. Contextual controls (averages over all other overlapping inpatients, weighted by days overlapped) are the proportion receiving care for a drug use disorder, the proportion who are male, the proportion who are young ($\leq$ 26), the proportion who have no more than minimum education, the proportion for which both parents have no more than minimum education and the proportion with mental illness comorbidities. Standard errors are clustered by facility.

Table 15: Somatic hospital episodes for alcohol related diseases among patients with alcohol use disorders

	Episode within 1 year of exit		Episode within 3 years of exit	
	Chronic hepatitis	Skin disorder	Chronic hepatitis	Skin disorder
peer exposure	-0.015*	-0.078***	-0.038**	-0.063
	(0.008)	(0.026)	(0.017)	(0.040)
Mean dep. var.	0.008	0.035	0.018	0.076
+1 SD (% of mean)	-56.44	-67.08	-63.54	-24.95
+1 peer	-6.30	-7.09	-6.78	-2.65
Observations	1,664	1,664	1,664	1,664
R-squared	0.043	0.068	0.051	0.072

Notes: All models include facility, county and month-by-year of exit fixed effects. Controls are an indicator for a drug use disorder, an indicator for male, age, age^2/1000, an indicator for no more than minimum education level, an indicator for both parents having minimum education, an indicator for being single and an indicator for a mental illness comorbidity. Contextual controls (averages over all other overlapping inpatients, weighted by days overlapped) are the proportion receiving care for a drug use disorder, the proportion who are male, the proportion who are young ($\leq$ 26), the proportion who have no more than minimum education, the proportion for which both parents have no more than minimum education and the proportion with mental illness comorbidities. Standard errors are clustered by facility.