Peer Effects, Mortality and Recovery from Substance Use Disorders

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August 13, 2026

Abstract

Using linked administrative data on inpatients, we study the impact of peers on mortality following treatment for substance use disorders. Peers are defined as individuals who overlap in treatment and reside in the same geographic area, and their impact is identified using quasi-random variation in the timing of treatment entry arising from waitlists. We find peers reduce all-cause and substance-induced mortality post treatment. Supplementary analysis shows that peers also reduce inpatient readmissions and hospital admissions for substance-related disease, consistent with peers supporting recovery after inpatient care. These findings suggest that peers could be leveraged to save lives outside clinical settings.

Key words: Peer effects, substance use, mortality, morbidity
JEL codes: I12, D85

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[§]The authors thank Asle Enger, Department of Substance Abuse and Addiction Treatment, University of Oslo Hospital; Davide Dragone; Elaine Kelly and other participants of the Essen Health Conference; Miriam Wüst, Torben Heien Nielsen and other seminar participants at the University of Copenhagen Center for Economic Behavior and Inequality; and participants at the Risky Behaviors Workshop, the Australasian and European Workshops on Econometrics and Health Economics, and the University of York Center for Health Economics seminar. We also thank the Applied Micro group at the University of Melbourne for their valuable insights and suggestions.

1 Introduction

High-risk substance use is widespread and imposes substantial economic and societal costs in many developed countries. In the United States, where the opioid epidemic has been particularly severe, 75 million individuals aged 12 or older reported using illicit drugs in 2024, 58 million reported engaging in harmful drinking, and over 48 million had a substance use disorder (SAMHSA, 2025). The annual cost of substance use disorders to the US economy now exceeds one trillion dollars, including costs attributed to healthcare use, criminal justice involvement, lost productivity, and premature mortality (CDC, 2024; NCDAS, 2025). Opioid overdose deaths, in particular, have been identified as an important contributor to decreasing US life expectancy (Hébert & Hill, 2024). With 178,000 deaths due to alcohol and 105,000 deaths due to drug overdose in the US in 2023 alone, addressing the public health crisis of addiction, and the opioid epidemic in particular, remains a policy imperative (CDC, 2024; Garnett & Miniño, 2024).

An area of growing policy interest is the use of peers to support recovery and reduce mortality and other harms associated with problematic substance use. Social context has long been recognized as central to substance use behaviors (Strickland & Acuff, 2023). Informal peers, working through mutual support groups such as Alcoholics Anonymous, are a well-established part of the recovery landscape. Evidence shows that participation in Alcoholics Anonymous, in particular, can reduce drinking and increase abstinence (Kelly *et al.* , 2020).[1] Recently, however, interest has focused on formal peers operating within Peer Recovery Support Services (PRSS). In contrast to informal peers, peers assigned through PRSS are integrated into formal health and social service systems and aim to ensure continuity of care through peer-driven mentoring, education, and support. Federal agencies have worked to conceptualize, implement, and fund PRSS, with a significant expansion occurring under the 2018 SUPPORT Act.[2] While evaluations of PRSS find improvements in treatment engagement and retention, their effects on substance use and downstream outcomes such as overdose remain unclear (see Eddie *et al.* (2025) for a recent review). This raises important policy questions about the circumstances and mechanisms through which peer support affects substance use and related outcomes, including mortality, among people with substance use problems.

In this paper we provide evidence on how a particular type of peer impacts mortality. Specifically, we study individuals who (voluntarily seek and) undergo inpatient treatment for a substance use disorder ("rehab") and examine whether those encountered during inpatient treatment influence mortality in the post-treatment period, once patients return to the community. This type of informal peer effect is particularly interesting from a policy perspective for several reasons. First, unlike peer effects in risky behaviors more broadly, including adolescent initiation into substance

[1] This is especially the case when treatment programs link people with alcohol use disorders to Alcoholics Anonymous using Twelve Step Facilitation strategies (Kelly *et al.* , 2020).

[2] Recovery Support became a strategic initiative of SAMHSA in 2010. The Building Communities of Recovery program was established under the Comprehensive Addiction and Recovery Act (CARA) and further expanded under the Substance Use-Disorder Prevention that Promotes Opioid Recovery and Treatment for Patients and Communities (SUPPORT) Act of 2018. Annual appropriations for peer recovery support and related community recovery programs grew from about $1 million per year to $5 million per year for each fiscal year from 2019 through 2023 (NASEM, 2020).

use, the effect of treatment-acquired peers on mortality is theoretically ambiguous. Depending on how individuals interact with one another, peers from inpatient treatment could plausibly increase or decrease the risk of death. Second, if treatment peers facilitate recovery in the community, then they offer a potentially scalable and cost-effective approach to reducing the impacts of addiction, including mortality. Finally, while individuals with substance use disorders face an elevated risk of mortality in general, this risk is especially high following periods of abstinence, such as after inpatient treatment (Binswanger *et al.* , 2007; Gossop *et al.* , 2002; Ravndal & Amundsen, 2010). The combination of the potential to reduce the high post-treatment mortality risk and to do so in a cost-effective and scalable way makes causal evidence on the impact of treatment peers particularly valuable.

We address the data and identification challenges inherent to studies of peer effects using linked administrative data from Norway. In the context we study, peers are defined as those whose treatment overlapped, and who live in the same geographic area, as these are individuals who are most likely to interact in the post-treatment period.[3] Norway provides an ideal setting for our study for three reasons. First, the institutional context provides plausibly exogenous variation in exposure to peers. Specifically, excess demand for treatment and prioritization based on clinical need ensure that the date of entry into treatment is determined solely by the interaction of a prioritized waitlist and the dates that others leave treatment (i.e., that beds become available). Consequently, exposure to others through overlapping treatment episodes is as good as (conditionally) randomly assigned. We exploit this exogenous variation in a fixed effects framework that compares the mortality of individuals who attend the same inpatient facility and live in the same location, whose exposure to peers differs because they enter treatment on different dates.

A second strength of the Norwegian setting is that inpatient care is provided through the national specialist healthcare sector. This enables us to identify overlapping inpatients since the administrative data we use contains the universe of individuals receiving treatment. Finally, Norway's administrative registries can be linked. For this study we link the specialist healthcare registry to the death registry over several years, and track individuals both during treatment and after treatment. This permits us to study mortality and substance use related healthcare utilization over both the short term (one year post-treatment) and the medium term (three years post-treatment), providing a comprehensive analysis of peer effects from inpatient treatment on mortality, and of mechanisms through which these effects operate.

Our results provide evidence that the net effect of inpatient peers is a reduction in post-treatment mortality in general, and substance-induced mortality in particular. We estimate that exposure to one additional peer reduces mortality (from all causes) in the cohort we study by 5% of its mean in the first year following inpatient treatment, equating to around 1 life saved per 1000 patients. Over a three year horizon, one additional peer reduces mortality by 2% of its mean, indicating that the effect is both immediate and long lasting. To the extent that our findings can

[3]Similar definitions of peers are used in the context of criminal recidivism after release from prison (e.g., Billings & Schnepel (2022); Drago & Galbiati (2012)).

be applied to the US context, if the 2.6 million individuals who received inpatient treatment for substance use in 2024 (SAMHSA, 2025) were exposed to an additional peer, 2,600 lives would have been saved in the year following treatment alone.

We demonstrate that our empirical strategy is credible through various balance tests and two placebo tests, and we provide extensive evidence that our findings are robust using a series of tests designed to address concerns around measurement of peer exposure, endogenous exit from treatment, confounding from time-varying treatment environments, the impact of treatment group composition on treatment efficacy, econometric modeling of low probability outcomes, sensitivity to outliers, and sensitivity to the specification of controls and fixed effects.

While knowledge that treatment peers decrease post-treatment mortality is of direct policy relevance, illuminating the mechanisms through which they do so is critical for assessing the broader policy implications. This is because a peer-induced reduction in mortality could either be explained by peers supporting recovery (i.e., reducing substance use) or by peers engaging in harm reduction strategies such as using substances together to reduce overdose risk. To distinguish between potential channels we extend the model of addiction and cue-triggered decision processes of Bernheim & Rangel (2004) to incorporate peer effects. We use this extended model to show that, while these two channels operate in the same direction for mortality, they have different implications for substance use related healthcare utilization, such as readmission to an inpatient facility, hospital admissions for substance use–related disease, and hospital admissions for substance use–related poisonings. Examining these additional outcomes enables us to empirically determine which of these two channels, support recovery or harm reduction, is dominant in explaining the peer induced reduction in post-treatment mortality that we identify.

Our estimates indicate that peers reduce overall readmissions to inpatient treatment and, in particular, emergency readmissions, which typically arise as an immediate and direct consequence of intensive substance use. Further, we find that peers reduce hospital admissions for substance-related (liver) disease, which reflect harm from sustained substance use. These findings are consistent with peers supporting recovery through reduced substance use, and are inconsistent with peers reducing mortality solely through harm reduction (i.e., safer use, but no less of it). Overall, the empirical evidence suggests that peers predominantly support recovery rather than reduce harm.

Our work contributes to several distinct literatures. First, we advance the literature on the determinants of mortality and morbidity among individuals with substance use disorders. These individuals face substantially higher rates of mortality and morbidity than the general population. Mortality has been shown to vary with the type, amount of substance used and route of administration (Degenhardt *et al.* , 2011), macroeconomic conditions (Hollingsworth *et al.* , 2017), and individual characteristics such as sex (Gjersing & Bretteville-Jensen, 2014; Larney *et al.* , 2020). Focusing on studies based on quasi-experimental variation, mortality has been shown to depend on the availability of prescription opioids (Powell *et al.* , 2020), abuse-deterrent opioids (Alpert *et al.* , 2018, 2022; Evans *et al.* , 2019; Powell & Pacula, 2021), heroin supply (Moore & Schnepel, 2026), physician prescribing behavior (Eichmeyer & Zhang, 2022), treatment facilities (Swensen,

2015), naloxone availability (Doleac & Mukherjee, 2022; Rees *et al.* , 2019) and syringe exchange programs (Packham, 2022) (see Maclean *et al.* (2022) for a review). These studies mostly rely on state- or county-level variation, with individual-level designs remaining rare (Eichmeyer & Zhang, 2022; Moore & Schnepel, 2026). Focusing on informal peers, Mäckle & Ruenzi (2022) and Cutler & Donahoe (2024) examine cross-county spillovers in opioid use and mortality that could reflect peer effects. To our knowledge, ours is the first study to use individual-level data and a quasi-experimental design to study the causal impact of peers on mortality in this population.

Second, we contribute to the literature on peer effects in substance use. Extensive research examines adolescent initiation (e.g., Arduini *et al.* (2019); Clark & Lohéac (2007); Cutler & Glaeser (2005); Eisenberg *et al.* (2014); Fletcher & Ross (2018); Gaviria & Raphael (2001); Hsieh & Van Kippersluis (2018); Lundborg (2006)) and a growing body of work considers adult initiation into prescription opioid misuse (Adamopoulou *et al.* , 2024; Seamans *et al.* , 2018). In contrast, relatively little is known about *exit* from substance use. Randomized controlled trials provide evidence that Alcoholics Anonymous reduces alcohol use, especially when paired with Twelve Step Facilitated treatment programs (Bøg *et al.* , 2017; Surgeon General, 2016). Studies of formal peer based programs, such as Peer Recovery Support Services, show they can improve engagement with treatment (Bassuk *et al.* , 2016; Beaudoin *et al.* , 2022; Eddie *et al.* , 2019, 2025), but randomized controlled trials find no impact on downstream outcomes such as emergency department visits or overdose mortality (Chambers *et al.* , 2024; Watson *et al.* , 2024). Our study adds to this nascent literature on the causal effect of peers in recovery from substance use disorders by focusing on *informal* peers acquired during an episode of inpatient treatment, and who are contemporaneously experiencing and attempting to address a substance use disorder.[4]

Finally, we contribute to the broader literature on peer effects, and specifically to a body of work showing that they are shaped by the context in which interactions occur. For example, in education both the sign and magnitude of peer effects have been found to vary substantially across institutional settings, stage of education, outcomes, and group compositions, suggesting that the impact of a given peer is not invariant but shaped by the environment (Feld & Zölitz, 2017; Sacerdote, 2011). In the domain of addictive behavior, lifecycle models highlight state dependence (Becker & Murphy, 1988; Bernheim & Rangel, 2004), implying that social interactions may both facilitate initiation at early stages while also supporting cessation once individuals are motivated to recover. Consistent with this, in the context of obesity, peers have been found empirically to be detrimental for initiation (Fortin & Yazbeck, 2015) yet beneficial for recovery (Ahern *et al.* , 2017). Building on this literature, we provide empirical evidence that peers can be beneficial in recovery from substance use disorders among adults, even though there is also substantial evidence that they can encourage initiation earlier in the life cycle.

The rest of the paper is organized as follows. Section 2 describes the Norwegian institutional

[4]Previous work has examined associations between smoking cessation and social networks (Christakis & Fowler, 2008), spouses (Palali & Van Ours, 2017), or households (Jones, 1994), and studies by Jason *et al.* (2012) and Bohnert *et al.* (2009) consider quitting harmful substance use in relation to network size and composition, including cocaine and heroin use.

setting. Section 3 presents our empirical approach, including the empirical model, data and identification strategy. Section 4 presents our main mortality results and summarizes our placebo and robustness tests. Section 5 examines mechanisms, presenting our theoretical framework and empirical evidence on the channels through which peers reduce mortality. Section 6 concludes.

2 Institutional setting

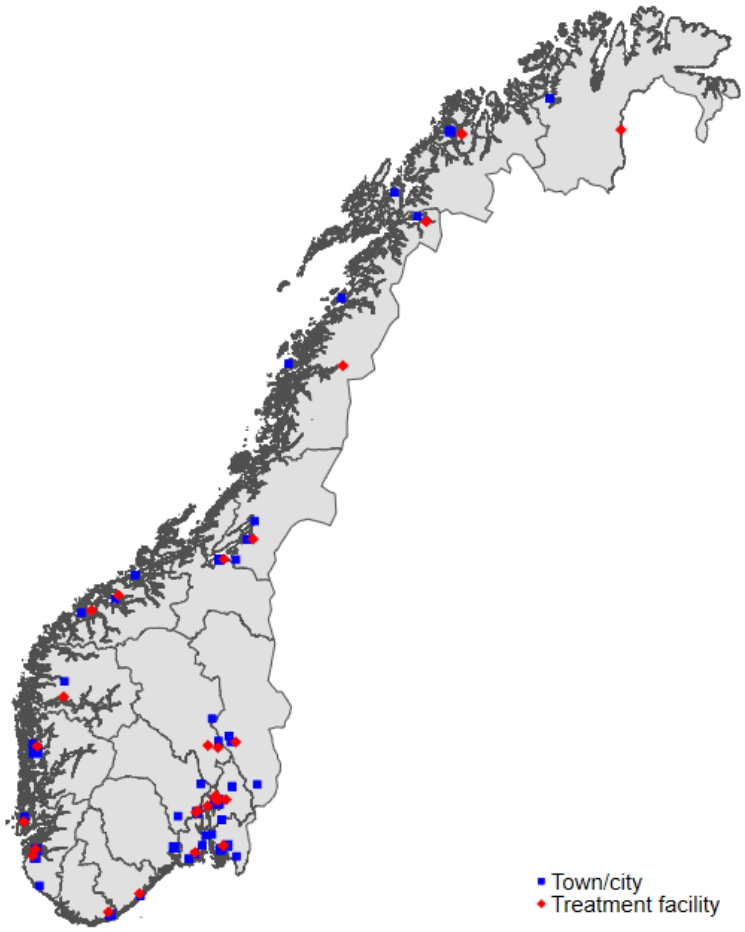
Norway has a universal public healthcare system funded by general taxation. The legislative framework is established by Parliament, while the Ministry of Health and Care Services is responsible for national policy development and oversight. Healthcare provision is decentralized to four Regional Health Authorities (Northern Norway, Central Norway, Western Norway, and South-Eastern Norway), which oversee 20 Health Trusts responsible for service delivery.

Services related to substance use are provided directly by hospitals, through psychiatric units at hospitals, by dedicated substance use treatment centers and through privately practicing specialists and non-profit organizations contracted to provide publicly funded treatment. Patients may access these services at close to zero direct cost. Services include emergency care, detoxification, inpatient treatment and outpatient treatment. We focus on inpatient treatment. Most inpatient treatment occurs at dedicated addiction units but it can also occur within psychiatric units of hospitals. During the period of our data, facilities that provide inpatient treatment ranged in size from 8 to 55 beds, with a median of 19. They often specialize in particular substances (e.g., alcohol), patient sex (e.g., female only) and patient age (e.g., young adults only, using a cutoff of ≤ 26 years old). Facilities also offer different therapies to one another, which may improve patient outcomes, including mortality (Gatti *et al.* , 2020). As patients can choose the facility where treatment is received, and because of differences in the types of patients catered to and therapies offered across facilities, patients often do not attend their nearest facility. Figure 1 depicts the locations of the treatment facilities in our data. They are often, but not always, located in urban areas, especially in the densely populated south east. However, there are also facilities located in sparsely populated regions in the far north.

The process of admission to specialized inpatient treatment for a substance use disorder (henceforth SUD) begins with the patient requesting a referral. Patients are often referred to inpatient treatment by a specialist providing them with another service (e.g., outpatient treatment) but can also be referred directly by their general practitioner.[5] The referral may be to a specific treatment facility that the patient is permitted to nominate. Referrals are sent to a priority-setting unit for the nominated facility, which forwards them to a discipline specific assessment panel. Due to the Patients' Rights Act (1999), prioritization for treatment is based solely on clinical need. Assessments do not take into account factors such as sex, ethnicity, socioeconomic status, past

[5]In Norway, referrals to tverrfaglig spesialisert rusbehandling (TSB) can be made by social services (NAV), child welfare services, private and public physicians, and other healthcare professionals with referral rights. 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.

Figure 1: Treatment facilities, towns/cities and counties of Norway in 2010



Notes: This figure depicts treatment facilities, towns/cities and county boundaries in Norway during the period of our data.

harmful behavior, productivity, the available capacity at the facility chosen by the patient, or in the specialist care sector more broadly.

The date of admission into treatment is determined by a waitlist and is not predictable. Because of prioritization, a patient's position on the waitlist—and thus their wait time—changes over time depending on the severity of illness among patients assessed both before and after them. For example, a patient may be pushed back in the waitlist when later-assessed patients are deemed more severe. Wait time also depends on the discharge dates of patients already in treatment, which determine when beds become available. Moreover, because no real-time information on wait times was available during the study period, neither patients nor referring physicians could strategically time referrals to manipulate the date of entry into inpatient treatment.

Once admitted to a treatment facility and prior to starting treatment, patients undergo medically managed detoxification. Detoxification clears substances from the body and manages with-

drawal symptoms. It takes around one week for alcohol and cannabis, and two weeks for other substances. After detoxification, a typical treatment plan comprises therapy (cognitive behavioral therapy and motivational interviews), group therapy, environmental therapy, education (including harm minimization and overdose management) and physical activity. During treatment, patients interact routinely with one another, both informally as residents of the facility and formally as part of their treatment.

A patient may be discharged on completion of their treatment or if they no longer wish to continue or if treatment is ineffective. At discharge, patients are expected to attend a meeting to plan follow-up in the community, ensure suitable housing, and receive information on community services for continued recovery and future treatment.

Due to the institutional environment, patients cannot influence their admission date, which is determined instead by the interaction of their referral date and clinical need, the referral dates and clinical needs of others, local resource constraints and the discharge dates of those already in treatment. The interaction of these factors, and the fact that no information on expected wait time is available to the patient or their referring physician, implies that patients do not know when they will be admitted or which other patients they will overlap with once admitted.[6] These institutional features are key to our identification strategy.

3 Empirical strategy

Our baseline model examines the mortality impact of exposure to peers during inpatient treatment for a SUD using the following reduced form linear probability model:

$$Y_{icjt} = \beta PeerExposure_{icjt} + \mathbf{x}'_{icjt}\gamma + \mu_t + \alpha_{jc} + u_{icjt}, \tag{1}$$

where Y_{icjt} is an indicator for death of individual i who resides in county c and who was treated at facility j until exiting on date t within a fixed time period after leaving treatment (one or three years); $PeerExposure_{icjt}$ is our proxy for the probability of peer contact in the post-treatment period; $\mathbf{x}_{icjt}$ includes individual and contextual controls (averages of the individual level controls taken over all other overlapping inpatients, defined below); μ_t is a fixed effect for month-by-year of exit; α_{jc} is a facility-by-county-of-residence fixed effect; and u_{icjt} is an error term.[7] The coefficient of interest is β.

Our model accounts for both *contextual effects* and *correlated effects* (Manski, 1993). Contextual effects arise when the outcome depends on the type of other individuals in the reference group. In our setting, contextual effects reflect the impacts of treatment group composition on mortality,

[6] There is no mechanism through which groups of individuals can seek care together. In particular, couples cannot be jointly referred or admitted as a unit, although partners may be referred separately and, in some cases, offered supplementary couple or family sessions as part of individual treatment plans.

[7] As explained below, we cannot always pin down the treatment facility attended, and for this reason j indexes the set of facilities that could have been attended. When the facility is known this set is a singleton. Our model therefore includes fixed effects for the set of possible facilities. For simplicity of exposition, from this point forwards we refer to j simply as the facility.

including through its influence on treatment efficacy. They are accounted for by the contextual controls in $\mathbf{x}_{icjt}$. Correlated effects arise when individuals in the same reference group have similar outcomes due to their exposure to a common environment. They are accounted for by the fixed effects, α_{jc} and μ_t. Accounting for both contextual and correlated effects, identification of the coefficient of interest β as the causal impact of peers on mortality relies on the assumption that the peer exposure variable is conditionally independent of unobservable determinants of mortality. We examine this assumption in Section 3.2.

3.1 Data

We access Norwegian administrative data linking inpatient treatment records from the Norwegian Patient Registry (NPR) with the Norwegian Cause of Death Registry (NCDR) and the National Population Registry. The cohort we study includes all individuals who entered inpatient treatment in the specialist healthcare sector with a primary diagnosis of a SUD (ICD-10 codes F10-F16 and F18-F19) between 1 January 2009 and 31 December 2010. For each patient in our analysis cohort, all episodes in specialist healthcare recorded up to 31 December 2013 were extracted from the NPR, and all records up to 31 December 2013 were extracted from the NCDR. Demographic and socioeconomic information, including county of residence, are from the National Population Registry.

3.1.1 Sample

The sample used to construct the peer exposure variable uses all inpatient episodes for substance use treatment that occurred in one of 58 publicly funded facilities and which include at least one day of inpatient treatment during the period 1 January 2009 to 31 December 2010 (N=4,856). The analysis sample uses the first inpatient episode for each individual that starts between 1 January 2009 and 31 December 2010, and ends by 31 December 2010. These are the patients for whom we are able to observe all other patients with an overlapping treatment episode. We omit observations on patients with missing controls and those which constitute singletons in our fixed effects analysis (N=3,755). Further details on construction of the sample are in Appendix A.

3.1.2 Outcomes

Our primary outcomes of interest are related to death.[8] The outcomes we consider are indicators for death due to all causes, death due to external causes, death due to causes other than external causes, which we refer to as internal causes for simplicity, and substance-induced deaths. External causes of death are deaths resulting from injury or poisoning due to events outside the body (e.g., overdoses, accidents, suicide, and homicide). Internal causes of death (often called natural causes) are deaths resulting from disease or physiological processes originating within the body. Substance-induced deaths are deaths that can be directly attributed to drug or alcohol use. These outcomes

[8] Our administrative data do not record substance use or treatment adherence (e.g., medication).

Table 1: Descriptive statistics: Outcomes and peer exposure

	Mean			
	within 1 year	within 3 years		
Death (0/1)				
all-causes	0.022	0.067		
internal	0.011	0.033		
external	0.012	0.032		
substance induced	0.008	0.023		
Any inpatient readmission for substance use (0/1)				
all	0.385	0.416		
emergency	0.107	0.125		
treatment	0.337	0.366		
Any hospital admission (0/1)				
liver disease	0.038	0.078		
poisoning	0.071	0.156		
	Mean	Std. dev.	Min	Max
Peers				
days in treatment	28.47	50.66	1	619
num. overlapping inpatients	36.71	49.18	1	700
num. overlapping inpatients from same county	17.76	27.66	0	403
peer exposure	0.55	0.31	0	1
Observations	3,755			

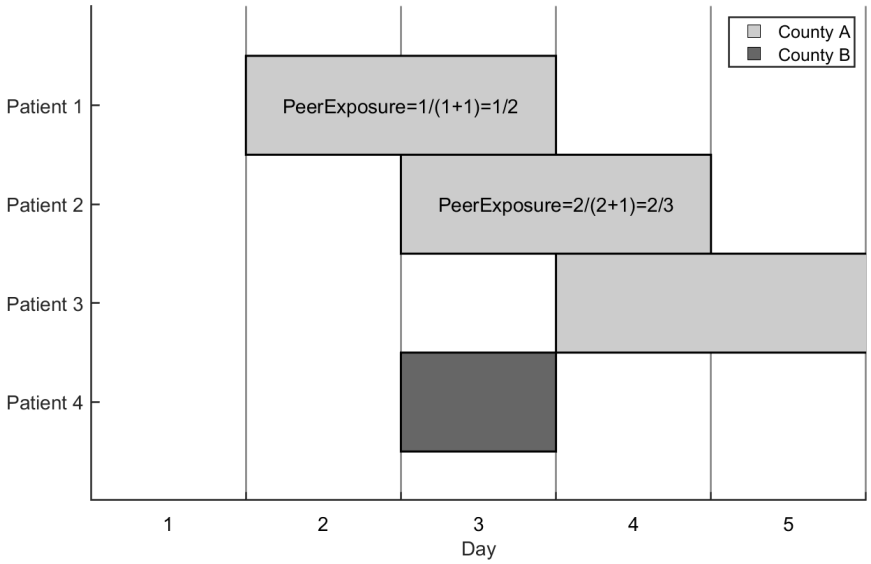
are constructed using data from the NCDR, which contains the date of death and one or more ICD-10 codes for the underlying cause. Further details on the exact definition of these variables is provided in Appendix A. Table 1 reports descriptive statistics for our death related outcomes. It shows that 2.2% of our analysis sample die within a year of leaving inpatient treatment and 6.7% die within three years. Approximately half of deaths are due to external causes and around a third are substance-induced.

To examine the channels through which peers impact mortality, we also examine outcomes related to readmission and two kinds of (non-fatal) hospital admissions. For the former we use an indicator for any readmission into specialized inpatient care for a SUD, which we further decompose into readmission for treatment and emergency readmission. Emergency readmissions occur in response to acute substance-related harm and are intended to prevent further harm to the individual or others, making them a definitive indicator of harmful substance use. Table 1 shows that 39% of our sample are readmitted into inpatient care within one year of exit, rising to 42% within three years. The figures for emergency readmission are respectively 11% and 13%.

For hospital admissions we use those due to substance-related disease and substance-related poisoning.[9] For the former we use chronic liver disease, which is associated with both drug use and

[9]In contrast to mortality and readmission for inpatient substance use treatment, we only observe the calendar year

Figure 2: Measuring peer exposure using overlapping inpatient episodes



Notes: In this example Patient 1 has 2 patient-days in total (1 with Patient 2 and 1 with Patient 4). So the denominator in our measure of peer exposure is 2. Only one of these patient-days is with another patient from the same county (Patient 2), so the numerator is 1. Hence Patient 1's peer exposure is 1/2. For Patient 2, the denominator is 3 (1 day with Patient 1, 1 day with Patient 2 and 1 day with Patient 4) and the numerator is 2 (only Patients 1 and 3 are from the same county as Patient 2). Hence Patient 2's peer exposure is 2/3.

alcohol use (Pateria *et al.* , 2013). We use an indicator for any hospital admission for which the ICD-10 code indicates either toxic liver disease, alcoholic liver disease or chronic hepatitis. Table 1 shows that 4% of our sample are admitted for liver disease within one year of exiting treatment, rising to 8% within three years. To measure poisoning admissions, we use an indicator for any hospital admission for which the ICD-10 code indicates either poisoning or acute intoxication induced by drug or alcohol use.[10] Table 1 shows that 7% of our sample are admitted for poisoning within one year of exiting treatment, rising to 16% within three years.

3.1.3 Peer exposure

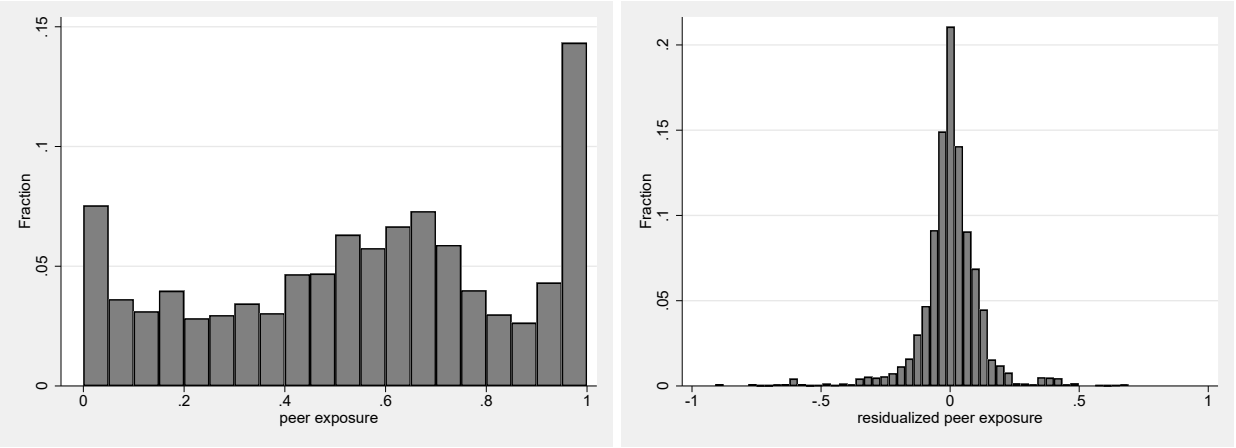
We measure peer exposure of individual i who resides in county c and who was treated in facility j as the proportion of total patient-days that overlap with patient i's treatment episode in facility j that are contributed by patients who also reside in county c.[11] An example of how we compute this is provided in Figure 2. Our measure reflects the importance of geographic proximity in peer effects

of hospital admission. For a patient leaving treatment in year y we therefore construct indicators for any hospital admission occurring in years $y+1$ to $y+x$, and refer to this as an admission within x years of leaving treatment.

[10] While hospital admissions may not be the most sensitive measure of non-fatal overdose, alternatives such as ambulance call outs are not available in our data.

[11] For a minority of patients we cannot uniquely identify the treatment facility they attended. In this case, when constructing our measure of peer exposure, we use all inpatients who *could* have overlapped in the same facility. Appendix A provides further details. In Section 4.2 we show that our baseline estimates are robust to using only those facilities for which we know all patients with certainty.

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



with respect to risky behaviors documented in the literature (Adamopoulou *et al.* , 2024; Billings & Schnepel, 2022; Case & Katz, 1991; Dustmann & Landersø, 2021; Kling *et al.* , 2005, 2007; Ludwig *et al.* , 2001; Stevenson, 2017). To give a concrete example in the context of criminal recidivism, motivated by the potential for post-release interactions, Drago & Galbiati (2012) define peers as those overlapping in the same prison and residing in the same region of Italy (or nationality, if non-Italian).

The geographic unit on which our peer measure is based is the county. During the period of our study, Norway comprised 19 counties, the boundaries of which are illustrated in Figure 1. Due to the sensitive nature of our sample, this is the most granular information that was made available to us. The ideal geographic unit is the one at which co-residence best predicts sustained social ties. Evidence from mobile phone and geocoded friendship data indicates that social interaction decays with travel time rather than raw distance, so that ties are maintained across a functional urban region connected by transport rather than confined to a small local area (Bailey *et al.* , 2020; Büchel & Ehrlich, 2020; Kim *et al.* , 2023). Because most Norwegian counties in this period contained a single population center and its surrounding catchment, and because those with a SUD tend to live in urban areas, the county approximates the functional region for most of the country,[12] with individuals residing in the same county typically being substantially closer to one another than to residents of neighboring counties. Importantly, analysis at the county level also ensures a large enough number of individuals receiving inpatient treatment residing in each geographic area so as to generate sufficient variation from overlapping treatment episodes to support our analysis. A more complete and quantitatively supported justification of the county-based peer measure is

[12]Østfold, Vestfold and Telemark, each have multiple population centers that lie within short travel distances of one another, so these counties remain well approximated by a single functional region. Rogaland is an exception, as Haugesund is separated from the Stavanger area by a fjord crossing, so that co-residence may overstate geographic proximity for some pairs. The converse arises in the capital region, where the functional region extends beyond Oslo's county boundary into Akershus, so that co-residence may understate proximity for some pairs.

Table 2: Descriptive statistics: Controls

	Variables	Mean	St Dev.	Min	Max
Individual controls					
	drug (0/1)	0.56			
	male (0/1)	0.69			
	minimum ed. (0/1)	0.58			
	both parents min. ed. (0/1)	0.26			
	single (0/1)	0.63			
	mental illness (0/1)	0.19			
	age	41.07	13.37	17	85
	$age^2/1000$	1.87	1.16	0.29	7.23
Contextual controls					
	drug	0.70	0.21	0	1
	male	0.69	0.19	0	1
	minimum ed.	0.58	0.19	0	1
	both parents min. ed.	0.23	0.16	0	1
	single	0.63	0.19	0	1
	mental illness	0.26	0.20	0	1
	young (age$\leq$ 26)	0.19	0.24	0	1
Observations		3,755			

provided in Appendix B. Alternative peer definitions based on common sex, age, substance and urban residence are considered in Appendix E.

Table 1 reports summary statistics related to our measure of peer exposure (the proportion of overlapping patient-days contributed by patients who live in the same county). The bottom panel shows that the mean number of days in treatment is 28.47 (the median is 9). During that time, the mean number of other patients with overlapping episodes is 36.71, of which 17.76 reside in the same county. The mean exposure to peers in our sample is 55%. Figure 3 shows that there is ample variation in exposure to peers, even after partialing out the fixed effects that we employ.

3.1.4 Controls

To absorb residual variation we use the following pre-admission patient characteristics: the type of SUD (drug use or alcohol use), sex, age, education (an indicator for lower secondary only), parents' education (an indicator for both having lower secondary only), an indicator for single marital status, and an indicator for a mental health comorbidity at the point of referral. We also form (patient-day) averages of these variables over all others with overlapping treatment episodes to account for contextual effects (Manski, 1993). These contextual controls account for the composition of the group of patients experiencing treatment together and any impact it may have on treatment efficacy.

Table 2 reports descriptive statistics. Around 56% of patients receive treatment for a drug

use disorder, with the remaining 44% treated for an alcohol use disorder. Patients are typically male (69%), have a low level of education (58% have only lower secondary) and are single (63%). They have an average age of 41, 19% have a mental illness, and 26% come from a disadvantaged background, as measured by both parents having only lower secondary education. The contextual controls have similar means but smaller standard deviations reflecting the fact that they are weighted averages.

3.2 Identification

Identification of the causal effect of peer exposure relies on the assumption that, conditional on the county-by-facility fixed effects, variation in peer exposure is independent of unobservables that cause mortality. By conditioning on the facility-by-county fixed effects (α_{jc} in equation (1)), we ensure that the identifying variation comes exclusively from the comparison of inpatients attending the same facility and who live in the same county, whose peer exposure varies only due to the timing of their episodes.[13] [14]

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

The conditional independence assumption needed for identification requires that patients cannot manipulate their entry and exit dates. As discussed in Section 2, the institutional environment implies that the date of entry is unpredictable and unlikely to be manipulated by the patient or their referring physician. Wait times until entry are determined by the interaction of a prioritized waitlist (which in turn depends on patients assessed both before and after the focal individual) and the discharge dates of those receiving inpatient treatment prior to the focal patient (i.e., when beds become available). Moreover, wait times are substantial. Although we are not able to determine the full time between referral and admission (because our data does not contain the date of referral), the mean and median wait times from assessment of the referral to admission are 83.35 and 30 days respectively. Around a third of inpatients are admitted within seven days of assessment and around a third are not admitted within 60 days of assessment. The significant expected wait time to enter treatment along with the date of admission being partly driven by others' discharge dates make systematic manipulation of entry into inpatient treatment unlikely.

[13] This type of variation has previously been used in the context of criminal recidivism (Bayer *et al.* , 2009; Billings & Schnepel, 2022; Damm & Gorinas, 2020; Johnsen & Khoury, 2024; Ouss, 2011; Tan & Zapryanova, 2022).

[14] Note that, due to our fixed effects strategy, our findings are not driven by selection into treatment facilities. Absent county-by-facility fixed effects we might worry, for example, that patients select into treatment facilities located outside their county of residence based on unobservable drivers of mortality, and, so may encounter fewer other inpatients resident in their county, and hence have lower peer exposure.

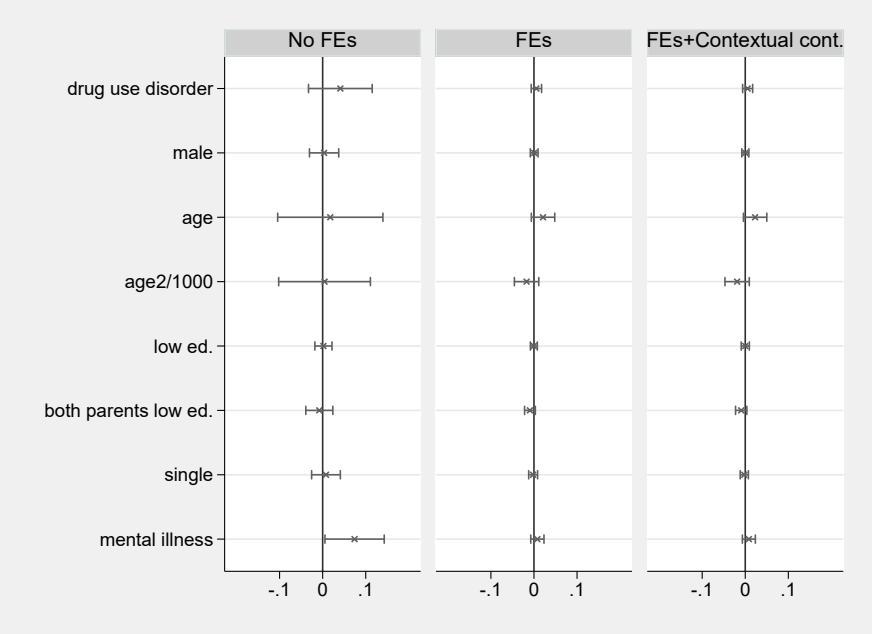
Despite this, one might worry that patients may attempt to manipulate their entry date in order to commence treatment alongside others already known to them. This threatens the conditional independence assumption, for example, if unobservably sicker patients are more likely to engage in this behavior. To investigate this, we examine the variation in admission dates among inpatients residing in the same county, attending the same facility, and assessed in the same week, by residualizing the number of days from assessment to admission with respect to a fixed effect for county-by-facility-by-week of assessment for our estimation sample. The standard deviation of this residual is 47.67 days. This is larger than the mean number of days in treatment, which is 28.47.[15] These statistics imply that individuals seeking to enter treatment together would tend to be unsuccessful, which is likely to be because they cannot manipulate the dates that beds become available. For this reason, we do not think that patients jointly seeking treatment poses a credible threat to the conditional independence assumption.

While the date of entry is plausibly exogenous, since patients can leave treatment at any time, the date of exit may not be. This threatens the conditional independence assumption, for example, if inpatients cut short their treatment once they have acquired a sufficiently supportive peer group. In this example our estimates would confound the impact of peers with the duration of treatment received. In Section 4.2 we discuss several robustness tests which jointly show that our main findings are unlikely to be driven by endogenous exit from treatment.

Given that our sample includes only those who eventually receive treatment, a final concern relates to selective attrition from the waitlist. While patients may well select out of the waitlist based on unobserved determinants of mortality (for example, deteriorating health status), it is implausible that these unobservables are systematically related to peer exposure since this is not known (or determined) at the time that the attrition decision is made. Patients could nonetheless choose to exit the waitlist based on an expectation of peer exposure, for example formed from their prospective treatment facility and county of residence. To the extent that this arises, our use of county by facility fixed effects means that it is unlikely to bias our estimates.

We cannot directly assess whether attrition is related to patient characteristics, observable or unobservable, since patients who drop off the waitlist are not observed in our data. In the next section we therefore follow precedent in the literature and provide evidence consistent with the conditional independence assumption through a set of balance tests (Aizer & Doyle, 2015; Godøy *et al.* , 2024; Prudon, 2024; Williams *et al.* , 2026). Specifically, we test for correlation between peer exposure and observable patient characteristics, including measures of severity, within our sample of patients who enter treatment. Such correlation would be expected to be present if there were selective attrition from the waitlist.

Figure 4: Balance test



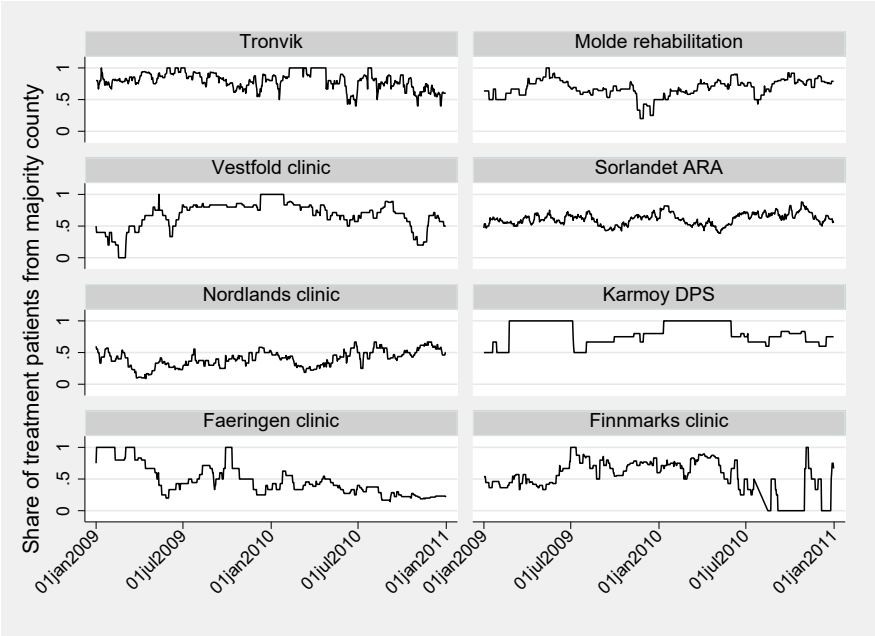
Notes: Coefficient estimates and 95% confidence intervals based on standard errors clustered at the facility level from regression in which the dependent variable is peer exposure. To represent all regressors on the same axis, we rescale the age variables to have standard deviation equal to one. All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

3.2.1 Balance tests

While the conditional independence assumption cannot be directly tested, we provide evidence in support of it by way of balance tests. Figure 4 examines the relationship between peer exposure and observable pre-admission patient characteristics. It reports coefficient estimates and 95% confidence intervals from three separate regressions in which peer exposure is the dependent variable. The first includes individual controls, the second adds the full set of fixed effects, and the third also adds contextual controls. In all three specifications, patient characteristics, including those measuring severity of illness (drug use disorder and mental illness) are not individually significantly associated with peer exposure, with the exception of the coefficient on the indicator for mental illness in the specification that does not include fixed effects or contextual controls. Further, when including fixed effects, these individual characteristics are not jointly associated with peer exposure at any conventional significance level. The p-value from the conventional joint F-test is 0.21. This test is known to over-reject the null, particularly with clustered standard errors (Kerwin *et al.* , 2024). Using the corrected randomization-inference based F-test yields a p-value of 0.42. Though we do not report it here, we also find no evidence that peer exposure is associated with any of the contextual controls, including those measuring the severity of illness of other inpatients. We consider two

[15]Since the distributions of wait time and treatment duration are both skewed to the right, we also compare quantile based measures. The interquartile range of the residualized wait time is 28.8 days whereas the median number of days in treatment is 9 days.

Figure 5: Concentration of patients by county for specific treatment facilities



Notes: This figure depicts the concentration ratio over time at different facilities in our data. For each facility, the ratio on a given date is defined as the share of the stock of patients receiving treatment at the facility who reside in its single largest source county. For these eight facilities the largest source county is the county in which the facility is located.

alternative balance tests in Appendix C, both of which yield the same conclusion.

3.2.2 Identifying variation

In addition to conditional independence, our empirical strategy requires sufficient variation in peer exposure to identify its effect on mortality. Variation in within county-by-facility exposure to peers arises from random fluctuations in the counties of residence of the patients receiving treatment in a given facility over time. These fluctuations are driven by entry of patients on the waitlist and exit of patients already receiving treatment. Figure 5 illustrates this variation for eight facilities in our data. For each facility on each date, we report the share of the stock of patients receiving treatment who reside in its single largest source county.[16] We observe that there can be large fluctuations over the course of just a few days. This demonstrates that otherwise similar individuals (attending the same facility and residing in the same county) who enter within a few days of one another can experience large differences in exposure to peers.

[16]For these eight facilities the largest source county is the county in which the facility is located.

Table 3: Mortality

	Death within 1 year of exit			Death within 3 years of exit			
	All	Internal	External	All	Internal	External	Substance induced
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
peer exposure	-0.061**	-0.011	-0.050**	-0.080***	-0.012	-0.060**	-0.027**
	(0.024)	(0.011)	(0.020)	(0.025)	(0.017)	(0.023)	(0.013)
	[0.08]	[0.39]	[0.08]	[0.03]	[0.40]	[0.08]	[0.05]
Mean dep. var.	0.022	0.011	0.012	0.067	0.033	0.033	0.023
+1 peer (%)	-4.66	-1.70	-7.37	-2.04	-0.63	-3.16	-1.98
Observations	3,755	3,755	3,755	3,755	3,755	3,755	3,755
R-squared	0.060	0.057	0.056	0.064	0.079	0.051	0.040

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1. Multiple-testing adjusted p-values are in brackets.

4 Results

4.1 Baseline results

Table 3 reports our findings for the impact of exposure to peers on mortality. Models (1) to (3) respectively consider all deaths, deaths from internal causes, and deaths from external causes in the first year after exiting inpatient treatment. Models (4) to (6) consider the same outcomes but over a three year horizon, allowing us to evaluate the extent to which deaths are prevented or merely delayed. Model (7) considers deaths that are directly attributed to substance use.[17] Standard errors account for clustering at the facility level. In brackets we provide multiple-testing adjusted p-values for statistical significance (Romano & Wolf, 2005).

Looking at model (1), we find that exposure to peers reduces mortality in the first year after exiting treatment. Without adjusting for multiple testing, the coefficient is statistically significant at the 0.05 level. The multiple-testing adjusted p-value is 0.08. The row denoted '+1 peer (%)' considers the impact of an additional peer.[18] Being treated alongside one additional peer for the full duration of the treatment episode reduces all-cause mortality in the year following treatment by around 5% of the mean. Scaling this up by the size of our sample, one additional peer for all sample

[17] We focus on a three year post-treatment time-frame since substance-induced deaths are reasonably rare, equating to only 0.8% of sample members in the first year post-treatment but rising to 2.3% by the third.

[18] We suppose that the additional peer overlapped with the entire duration of the focal individual's episode. Denoting $PeerExposure = m/n$ where m is the total number of days overlapped with peers and n is the total number of days overlapped with all inpatients, the counterfactual peer exposure we consider is $PeerExposure = (m+d)/(n+d)$ where d is the number of days the focal individual received treatment. The difference between peer exposure and its counterfactual varies across individuals, so to provide a typical effect size we use the median. Note that because peer exposure is a proportion, the implied effect of an additional peer diminishes mechanically with the number of other patients present: an additional peer raises exposure substantially when few patients overlap with the focal individual's episode, and only marginally when many do. Per-peer magnitudes should therefore not be extrapolated linearly to large changes in the number of peers.

members would be expected to have saved around 4 lives in the first year following treatment, or equivalently, approximately 1 life per 1000 person-years.

Models (2) and (3) show 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 on internally caused deaths. This likely reflects the fact that externally caused deaths are more responsive to short-term changes in substance use than internally caused deaths. For example, we might expect the risk of fatal overdose (an external cause) to adjust nearly instantaneously, but we would not expect this to be true for mortality due to substance related disease (an internal cause).

Looking at models (4) to (6) of Table 3, we find only marginally larger point estimates for peer-induced reductions in 3-year mortality compared to 1-year mortality, which suggests that peers have the greatest impact in the first post-treatment year. This is consistent with evidence that the risk of mortality is elevated following a period of abstinence (Binswanger *et al.* , 2007; Gossop *et al.* , 2002; Ravndal & Amundsen, 2010), implying that there is more scope for peers to reduce mortality in the first post-treatment year. Alternatively, it could be that social ties formed among inpatients are stronger in the first post-treatment year and decay over time. The finding that peers reduce 3-year mortality suggests that deaths are prevented rather than delayed, at least within the time horizon available in our data.

Model (7) shows that exposure to peers reduces substance-induced mortality in the 3 years following treatment. Since these deaths are directly and intrinsically related to substance use, this provides definitive evidence that exposure to peers alters substance use behavior. Further, these findings rule out subversion of recovery, whereby peers from inpatient treatment induce use that would not otherwise occur (thereby increasing mortality), as the dominant channel through which peer exposure affects mortality following inpatient treatment.

4.2 Placebo and robustness tests

In the appendix we provide evidence that our mortality estimates are credible through two placebo tests and several robustness tests. Appendix D reports results for the placebo tests. The first test shifts the start and end dates of the focal individual's inpatient treatment episode when constructing their measure of peer exposure, assigning them a placebo group of potential peers who received treatment in the same facility at around the same time, but had no (or limited) overlap. Consistent with our identification strategy, we find no impact on mortality. The second placebo test uses a sample of emergency patients. Emergency patients are admitted because they pose a risk to themselves or others. They do not receive treatment and therefore do not share treatment interactions, so should not experience any peer effects. Again, we find no impact on mortality. Together, these placebo tests provide broad empirical evidence that our baseline findings are unlikely to be driven by some artifact of inpatient admission.

Our placebo and robustness tests also address concerns about confounding from time-varying treatment environments that are correlated with the composition of patients in residence. The most direct evidence comes from the emergency patient placebo (Appendix D). Emergency patients are

present in the same facilities at the same times as treatment patients, and are hence exposed to the same fluctuations in staffing and other features of the facility environment. If our estimates reflected the environment rather than the peers, similar effects should arise for emergency patients, yet we find no such effects. Confounding would therefore require an environmental factor that affects treatment patients but not emergency patients in the same facility at the same time. Two further tests restrict what such a factor could be. Augmenting our baseline specification with county-by-facility linear trends (Appendix E), leaves our estimates essentially unchanged, limiting the impact of confounders that evolve gradually. And the shifted-episode placebo, which assigns each individual a group of patients treated at the same facility four or eight weeks earlier or later, yields null effects, essentially ruling out confounders that persist over intervals of even a few weeks. Any remaining environmental confounder would therefore need to affect only treatment patients, vary substantially within a few weeks, and move in step with the composition of patients. To the extent that features of the environment respond this closely to the patients in residence, they may be better understood as consequences of group composition than as confounders (i.e., as a mechanism rather than a threat to identification). We consider this interpretation in Section 5.

In Appendix E we also conduct several robustness tests to demonstrate that our baseline mortality estimates are unlikely to be driven by endogenous exit from treatment. First, we show that our findings are robust to the use of an alternative measure of peers based only on the first day of the focal individual's treatment episode, and to using this measure of peer exposure on the first day as an instrument for peer exposure throughout the episode. These tests rely on variation arising due to quasi-random fluctuations in timing of entry into treatment. They do not use variation that may arise from endogenous exit. Second, we show that treatment duration does not depend on our primary measure of peer exposure or that based only on the first day of the episode. This demonstrates that the decision to exit treatment does not depend on exposure to peers in a systematic way.

Finally, Appendix E shows that our mortality estimates are qualitatively robust to reasonable alternative measures of peer exposure; are not driven by outliers; are not sensitive to the controls that we employ;[19] do not depend on the choice of econometric model for the rare outcomes we consider; and are larger in magnitude for subsamples in which peer exposure is likely to be a stronger predictor of post-treatment interactions.[20]

[19] We do find that the magnitude (but not the sign) of the effect of peers depends on the specification of the fixed effects. The effect size is largest when the specification fully accounts for selection into facilities (i.e., isolates variation from timing, our baseline specification). Other specifications of the fixed effects which do not fully account for selection yield smaller effect sizes. This is consistent with unobservably sicker patients having a higher propensity to sort into facilities with peers from the same county.

[20] We find larger effect sizes when using an urban sample for which the county-based peer measure is likely to be less coarse, as well as when defining peers as sharing other common traits associated with the formation of social ties via homophily (sex, age and substance).

5 Mechanisms

5.1 Post-treatment versus in-treatment peer effects

Our finding that exposure to peers during inpatient treatment reduces all-cause, external cause, and substance-induced post-treatment mortality could either be explained by peer effects in the post-treatment period or by treatment group composition influencing outcomes during treatment itself, either directly, through interactions among patients, or indirectly, because staffing and treatment intensity respond to the mix of patients in residence. Because these channels operate downstream of peer exposure, they do not threaten identification, but distinguishing between them is important for policy. For example, if the mortality reductions arise primarily through treatment group composition rather than post-treatment peer interactions, policies aimed at fostering post-treatment social connections would be unlikely to reduce mortality.[21]

While we cannot completely rule out the possibility that treatment group composition affects treatment effectiveness,[22] we think this mechanism is unlikely to fully explain our mortality results for three reasons. First, our analysis includes contextual controls based on patient-day averages of pre-treatment characteristics among all other overlapping inpatients (see Section 3.1.4). These controls account for the effects of observable group characteristics, including those that may operate through treatment efficacy. While these controls cannot address the role of unobserved group characteristics, they substantially reduce the scope for treatment composition effects based on observed patient attributes.

Second, if treatment group composition had a meaningful effect on treatment efficacy and subsequent mortality, we would expect similar effects when defining peers based on other dimensions along which patients may be similar. In Appendix E, we show that this is not the case. Defining peers based on common age and sex produces estimates close to zero and statistically insignificant. The same pattern holds for other observed patient characteristics, including substance type, education, parental education, marital status, and mental illness, although we do not report all specifications for brevity. We find effects on mortality only when peers are defined based on common county of residence. While we cannot rule out that shared county of residence affects treatment efficacy through some specific mechanism, we view this explanation as unlikely given the limited linguistic and cultural differences across Norwegian counties.[23]

Third, if treatment group composition affects treatment efficacy, we would expect this to be reflected in treatment duration, since the institutional setting implies that patients who recover more quickly should be discharged earlier. However, Appendix E shows that treatment duration is unrelated to peer exposure. Together, these findings suggest that our results are unlikely to

[21] Confounding from time-varying treatment environments that are merely correlated with, rather than caused by, group composition is a distinct concern, which is addressed by the placebo tests and the robustness of our estimates to county-by-facility linear trends.

[22] Note that this is also true for other papers using research designs similar to ours: Bayer *et al.* (2009); Billings & Schnepel (2022); Damm & Gorinas (2020); Johnsen & Khoury (2024); Ouss (2011); Tan & Zapryanova (2022). These papers' findings may reflect peer effects or the impact of group composition on rehabilitation.

[23] Although Norway has several regional dialects, these rarely inhibit communication.

exclusively reflect treatment group composition and are instead at least partly driven by post-treatment peer effects.

5.2 Empirical evidence on post-treatment peer interactions

Having argued that our findings are unlikely to be driven solely by treatment group composition, we next provide empirical evidence of post-treatment peer interactions. This evidence comes from a recent survey of past-four-week opioid and stimulant users in seven Norwegian cities (Gjersing, 2023). Among 975 respondents, 72% had previously undertaken inpatient treatment, and of these, 53% reported having been in contact with individuals from their most recent treatment episode. This suggests that enduring social ties formed during inpatient treatment are both plausible and reasonably common.[24]

5.3 A model of post-treatment peer interactions

The existence of persistent peer connections after treatment raises the question of how such interactions influence substance use behaviors and subsequent mortality. Importantly, peer interactions need not have uniformly beneficial effects. While we find empirically that peer exposure reduces post-treatment mortality, this likely reflects a net effect. Ex ante, interactions with peers have the potential to either increase or decrease substance use. The survey evidence is consistent with this ambiguity. Among respondents who remained in contact with individuals from their most recent inpatient stay, 18% reported that these individuals had reduced their substance use after discharge, while 23% reported that they had increased it.[25]

We formalize these competing possibilities by extending the model of addiction and cue-triggered decision processes of Bernheim & Rangel (2004) to incorporate peer interactions. In the context we study, peers are defined as individuals with whom the focal individual shares an inpatient treatment episode and may interact in the post-treatment period. In the main text, we focus on the model's implications for interpreting the empirical results. Further details and derivations are provided in Appendix F.

The extended model shows that peers can affect the post-treatment risk of mortality through three channels: peers may *subvert recovery* by contagiously inducing use, *support recovery* by helping the focal individual manage cues and abstain or reduce use, or engage in *harm reduction* by

[24]The survey asked: "We would like to ask you about people who you met during your most recent inpatient substance use disorder treatment. Since being discharged, have you been in contact with any of the people you met during your treatment?" Response options were Yes, No, and Don't know.

[25]The remaining 59% reported no influence. The difference between the proportions reporting increased versus decreased use should not be over-interpreted: the survey sampled individuals who had used drugs in the past four weeks, whereas our administrative data include all individuals receiving inpatient SUD treatment (including alcohol use disorder), irrespective of whether they continue using afterwards. The same sampling difference applies to the persistence figures we cite: persistence of social ties could in principle differ for individuals who ceased use after treatment, though there is no obvious reason that ties formed in treatment would dissolve upon recovery.

using together when the alternative is using alone. Formally, the model yields the decomposition

$$\frac{\partial E(Death)}{\partial p} = \underbrace{\left[\left(\frac{1-q}{2}\right)s\right]\Delta_{\text{subvert}}}_{\text{Subvert recovery mechanism}} + \underbrace{\left[\left(\frac{1-q}{2}\right)(1-s)\right]\Delta_{\text{support}}}_{\text{Support recovery mechanism}} + \underbrace{\left[q+\left(\frac{1-q}{2}\right)s\right]\Delta_{\text{harm reduction}}}_{\text{Harm reduction mechanism}}, \quad (2)$$

where p is the probability of peer contact, q is the probability that both individuals receive a cue to use, and s is the probability that an individual who receives a cue induces use by an individual who has not. The bracketed terms give the probability that each mechanism arises and the Δ terms capture its impact on mortality when it does.

The mechanisms have distinct expected signs. Subverting recovery is the canonical "peer effect" emphasized in the literature on initiation into substance use: interaction with a peer whose own cue precipitates use causes the focal individual to use when they would otherwise have abstained. Under the assumption that the risk of death is increasing with use, $\Delta_{\text{subvert}} > 0$. Supporting recovery underpins established programs such as Peer Recovery Support Services and mutual self-help groups such as Alcoholics Anonymous and Narcotics Anonymous. By helping the focal individual manage cues and avoid or reduce use, it lowers mortality risk, so $\Delta_{\text{support}} < 0$.

The harm reduction mechanism lowers the risk attached to each instance of use. Using together rather than alone is explicitly encouraged by public health agencies in Norway because using alone substantially increases the risk of fatal overdose (NIPH, 2019), so, holding the extent of use constant, harm reduction reduces mortality risk. We consider it likely that this reduction in risk dominates any potentially harmful consequences of using together (e.g., needle sharing), and we discuss this issue further in Appendix F.

The overall sign of the harm reduction channel is nevertheless ambiguous once behavioral responses are taken into account. By reducing the immediate risks associated with each instance of use, peers who facilitate safer use may lower the perceived cost of using and thereby increase consumption. This moral hazard attenuates, and may reverse, the mortality benefit of harm reduction. Accordingly, $\Delta_{\text{harm reduction}}$ is anticipated to be negative in the absence of moral hazard and unsigned otherwise. The first column of Table 4 summarizes the anticipated impact of each mechanism on mortality, treating harm reduction with and without moral hazard separately.

The empirical counterpart of equation (2) is the coefficient on peer exposure in our empirical model, which identifies the net impact of exposure to inpatient peers on mortality. As our estimates provide robust evidence that peers decrease the probability of death, the theoretical model's predictions summarized in Table 4 rule out subversion as the dominant mechanism. This is not sufficient, however, to distinguish peers supporting recovery from peers engaging in harm reduction, with or without moral hazard.

To separate these channels we draw on three measures of healthcare utilization: readmissions into treatment, hospital admissions for substance related disease, and hospital admissions for substance related poisonings. As Table 4 shows, each mechanism has different implications for healthcare utilization. If peers reduce mortality primarily by supporting recovery (i.e., reducing use), all

Table 4: Anticipated sign of effect of each mechanism on empirical outcomes

	Death	Readmission	Disease admission	Poisoning admission
Subvert recovery	+	+	+	+
Support recovery	−	−	−	−
Harm reduction	−	0	0	?
Harm reduction w/moral hazard	?	+	+	?

three measures of utilization should decline (row 2 of Table 4).

In contrast, if peers reduce mortality through harm reduction without moral hazard (row 3 of Table 4), readmissions and disease admissions should be unaffected (because the extent of substance use is unchanged). The prediction for poisoning admissions, however, is ambiguous, because peers displace two types of overdose. Overdoses that would have been fatal may instead result in hospital admission, or, if a peer intervenes early enough, in no admission at all. At the same time, poisonings that would have led to hospital admission may instead be resolved at the scene or in the emergency room. Poisoning admissions increase due to the first type of displacement but decrease due to the second, so the net effect is unsigned.

Finally, if the dominant mechanism is harm reduction with moral hazard (final row of Table 4), readmissions and disease admissions should rise (because moral hazard increases use), while the prediction for poisoning admissions remains ambiguous.[26] Comparing the empirical findings for healthcare utilization against these predictions therefore allows us to discern the dominant mechanism.[27]

5.4 Empirical evidence on mechanisms

We now investigate the channels through which peers reduce post-treatment mortality, drawing on the secondary outcomes and the predictions summarized in Table 4. If peers primarily support recovery, readmissions and both types of hospital admission should decline. But if peers primarily engage in harm reduction (with or without moral hazard), readmissions and disease admissions

[26] In addition to moral hazard, it is also possible that the impact of harm reduction is influenced by complementarities in using together and/or additional risks such as needle sharing. Complementarities in using together would increase the intensity of use, and hence all four outcomes we consider. Needle sharing would increase disease admissions and possibly mortality, though we expect this to be negligible given the wide availability and usage of needle exchanges in Norway during the period of our data (Hordvin, 2012) and the fact that harm reduction education formed a core component of inpatient treatment programs, and using together rather than alone was promoted as a harm reduction strategy (NIPH, 2019). In the presence of these features, the overall impact of the harm reduction mechanism is in the same direction as for moral hazard, provided in the final row of Table 4.

[27] Throughout, by dominant we mean the mechanism whose contribution to the net effect in equation (2) is largest. The empirical evidence speaks to the sign of the net effect on each outcome, and hence to dominance rather than to the exclusive operation of any single mechanism.

Table 5: Readmission into inpatient care

	Readmission within 1 year of exit			Readmission within 3 years of exit		
	All	Emergency	Treatment	All	Emergency	Treatment
	(1)	(2)	(3)	(4)	(5)	(6)
peer exposure	-0.152***	-0.092*	-0.142***	-0.159***	-0.082*	-0.149***
	(0.059)	(0.047)	(0.046)	(0.051)	(0.049)	(0.041)
	[0.02]	[0.06]	[0.01]	[0.01]	[0.06]	[0.00]
Mean dep. var.	0.385	0.107	0.337	0.416	0.125	0.366
+1 peer (%)	-0.68	-1.48	-0.72	-0.66	-1.13	-0.70
Observations	3,755	3,755	3,755	3,755	3,755	3,755
R-squared	0.154	0.108	0.158	0.181	0.115	0.184

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. ∗∗∗: p<0.01. ∗∗: p<0.05. ∗: p<0.1. Multiple-testing adjusted p-values are in brackets.

should not decline, while the impact on poisoning admissions is unclear.

Table 5 reports results for readmission into inpatient treatment facilities. Model (1) considers any readmission within one year of exiting treatment, models (2) and (3) decompose readmissions into emergency readmissions and readmissions to treatment, and models (4) to (6) consider the same outcomes over a three year horizon. At both horizons, exposure to peers reduces both types of readmission, consistent with peers supporting recovery. At the one year horizon, the implied effect of an additional peer is larger for emergency readmissions (a 1.5% reduction relative to the mean) than for treatment readmissions (0.7%). This is telling evidence of the support mechanism, since emergency admissions arise as a direct and immediate consequence of intensive substance use.[28]

Table 6 reports results for hospital admissions. Models (1) and (2) examine admissions for liver disease and substance related poisoning within one year of exiting treatment, and models (3) and (4) examine the same outcomes within three years. At both horizons, peers reduce admissions for liver disease, with the effect statistically significant at conventional levels. This is consistent with studies showing that admissions for liver disease respond to changes in substance use within a one year time-frame (Andreasson *et al.* , 2006; Norström & Ramstedt, 2018). For poisoning admissions, the estimated effects are small in magnitude and statistically insignificant at both horizons.

Comparing these findings to Table 4, harm reduction cannot generate the observed declines in readmissions and disease admissions. Absent moral hazard it predicts no change in either outcome, and with moral hazard it predicts increases in both. Only the support mechanism predicts the pattern we observe. We therefore conclude that peer support for recovery is the dominant mechanism

[28] In further analysis (available on request) we studied the impact of peer exposure on receipt of outpatient treatment. Receipt of outpatient treatment in the years following inpatient treatment is a relatively uncommon patient pathway. While 42% of our sample were readmitted into inpatient treatment within three years, only around 4% received outpatient treatment during that time. We found no evidence of an impact of exposure to peers on receipt of outpatient treatment.

Table 6: Substance related hospital admission

	Any admission within 1 year of exit		Any admission within 3 years of exit	
	Liver disease	Poisoning	Liver disease	Poisoning
	(1)	(2)	(3)	(4)
peer exposure	-0.039*	0.015	-0.071**	-0.011
	(0.020)	(0.032)	(0.031)	(0.036)
	[0.05]	[0.71]	[0.05]	[0.72]
Mean dep. var.	0.038	0.071	0.078	0.156
+1 peer (%)	-1.80	0.37	-1.57	-0.12
Observations	3,755	3,755	3,755	3,755
R-squared	0.037	0.076	0.047	0.086

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. ∗∗∗: p<0.01. ∗∗: p<0.05. ∗: p<0.1. Multiple-testing adjusted p-values are in brackets.

through which peers reduce post-treatment mortality.[29]

6 Conclusion

This paper examines how peers acquired during inpatient treatment for a substance use disorder impact mortality following treatment. Recognizing the importance of proximity for maintaining contact after treatment, we define peers as individuals who share an overlapping treatment episode and live in the same county as the focal individual. Our analysis links data on the universe of inpatients who (voluntarily sought and) entered treatment for a substance use disorder in Norway's specialist healthcare system over a two year period, who we track for three years after exiting treatment, with administrative records on deaths. We identify the impact of peers on post-treatment mortality by exploiting quasi-random variation in the timing of entry into treatment that arises from the use of prioritized waitlists, where prioritization is based on severity of illness.

Our headline finding is that peer exposure during inpatient treatment reduces mortality after patients exit treatment. Peers reduce mortality not only in the first year after exiting, when risk is elevated, but across the full three years over which we follow patients, suggesting that deaths are averted rather than merely delayed. To explore the mechanisms underlying these findings, we introduce peer interactions into Bernheim and Rangel's (2004) model of addiction and cue-triggered decision processes. In the extended model, peers shape post-treatment substance use,

[29]The empirical evidence does not preclude harm reduction operating alongside support, and the null effect on poisoning admissions is consistent with this. Indeed, poisoning admissions are the one outcome for which support and harm reduction may push in opposite directions, support reducing admissions through reduced use and harm reduction displacing prevented deaths into admissions, in which case a net effect that is statistically indistinguishable from zero is consistent with both channels playing a role.

and through it the risk of mortality, via three potential channels: subverting recovery, supporting recovery, or harm reduction. The empirical finding of a reduction in mortality rules out subversion as the dominant channel through which peers shape post-treatment substance use. Supplemental analysis shows that peer exposure decreases readmissions to inpatient treatment facilities (notably, for emergency inpatient care) and hospital admissions for substance-related disease at one and three years after treatment. These findings are consistent with peers supporting recovery (by helping to navigate cues so as to avoid use), and rule out harm minimization as the dominant mechanism through which peer exposure reduces post-treatment mortality

Our findings stand in contrast to evaluations of formal peer based programs such as PRSS. While earlier reviews report tentatively favorable evidence (Bassuk *et al.* , 2016; Eddie *et al.* , 2019), recent randomized controlled trials have shown limited or mixed evidence on clinical outcomes. Watson *et al.* (2024) report no significant effects of PRSS relative to standard care on any outcome studied, including overdose related emergency department visits and linkage to medications for opioid use disorder. In the Navigator trial, a peer delivered behavioral intervention performed no better than the same intervention delivered by a clinical social worker, both on treatment engagement at 30 days (Beaudoin *et al.* , 2022) and on non-fatal overdose over 18 months (Chambers *et al.* , 2024).[30]

One possible explanation for the difference between our findings and these trials is that we consider informal peer relationships among patients receiving inpatient treatment together, which are more likely to reflect genuine and organic social ties than those induced through PRSS. Inpatient peers may foster deeper, more resilient ties, which may persist even when the individual loses contact with formal support services. This explanation is consistent with evidence from other settings showing that peer effects arising from naturally occurring social ties do not necessarily survive deliberate attempts to engineer them: when peer groups are designed by policymakers, individuals reshape their social networks and the anticipated benefits can fail to materialize or even reverse (Carrell *et al.* , 2013).

Beyond PRSS, it is useful to situate our findings within the broader literature on interventions affecting mortality among people who use substances. This literature is now sizeable, but its findings are mixed (Maclean *et al.* , 2022). On the supply side, large-scale reductions in opioid supply can produce immediate and sustained declines in mortality (Moore & Schnepel, 2026). Conversely, expanding opioid availability increases overdose mortality through greater access to prescription opioids (Powell *et al.* , 2020), more intensive physician prescribing (Eichmeyer & Zhang, 2022), and, historically, the introduction and marketing of OxyContin (Alpert *et al.* , 2022). Evidence on expanding access to treatment is favorable, with greater availability of treatment facilities reducing drug-induced mortality (Swensen, 2015). By contrast, evidence on interventions intended to reduce the harms associated with substance use is more mixed. Broader access to naloxone has been found both to reduce opioid-related deaths (Rees *et al.* , 2019) and to have no detectable effect (Doleac & Mukherjee, 2022), while syringe exchange programs reduce HIV transmission but have also been

[30] In contrast with our study, most randomized controlled trials focus on intermediate outcomes, such as engagement with treatment, rather than on mortality, readmission, and substance related hospital admissions.

found to increase opioid-related mortality in some settings (Packham, 2022). Similarly, the later reformulation of OxyContin reduced prescription opioid deaths, but these gains were largely offset by substitution toward heroin (Alpert *et al.* , 2018; Evans *et al.* , 2019).

Two features of these studies bear on the interpretation of our estimates. First, most identify effects on mortality in the general population, within which people who use substances heavily are a small minority, so estimated effects are diluted across a population that is largely not at risk. Our estimates instead apply to a clinical population that has sought inpatient treatment for a substance use disorder, whose baseline all-cause mortality of 22 deaths per 1000 patients per year is an order of magnitude above that of the general population. The closest comparator is Moore & Schnepel (2026), who use individual level data and study identified opioid users, a high risk population that is comparable to ours. They estimate that a large and sustained reduction in the supply of heroin saved the lives of around one in 48 opioid users over eight years, equivalent to roughly 2.6 deaths averted per 1000 people per year. Our estimates of the impact of an additional inpatient peer imply 1 death averted per 1000 inpatients per year, which is of a similar magnitude, despite reflecting the marginal contribution of a single individual to the social environment of treatment rather than a major national supply disruption. To the extent that our findings are applicable in the US context, where 2.6 million individuals receive inpatient treatment each year, they imply that one additional peer per patient would avert 2,600 deaths in the first year following treatment alone.

The second notable feature of the studies discussed above is that interventions based on harm reduction may have a limited impact on mortality because substitution toward more dangerous substances and other behavioral responses to reduced risk can offset the intended gains. Our results indicate that peers reduce mortality by reducing use rather than by making use safer. This may explain why the effect we identify is not similarly offset.[31]

In a setting where quasi-experimental estimates remain scarce, our findings offer a meaningful contribution to the evidence base on policies and programs that are effective at reducing substance use related mortality. The need for such evidence is pressing. Substance-induced deaths such as overdoses are preventable, yet they remain a leading cause of mortality. In the United States, deaths from opioid overdose quadrupled over the last two decades, rising from 8.2 per 100,000 in 2002 to 32.6 per 100,000 in 2022 (Spencer *et al.* , 2024). At the same time, a longstanding shortage of addiction services workers (Nguyen *et al.* , 2021) limits the scalability of clinician-delivered interventions. Our results suggest that treatment providers should consider the social environment of inpatient care as a resource in its own right. Decisions that influence the timing and structure of residential placements, as well as opportunities for interaction within treatment facilities, can affect the likelihood that supportive peer relationships emerge during treatment. Importantly, this does not require engineering social ties directly; rather, providers can create a conducive environment

[31]The clinical literature on opioid agonist treatment provides a further point of reference. A systematic review of 15 randomized trials finds no statistically significant effect on all-cause mortality (Santo Jr *et al.* , 2021), though these trials are typically small and short, and mortality is a rare outcome, so they are likely underpowered. Meta-analyses of cohort studies imply that retention in methadone treatment is associated with around 25 fewer deaths per 1000 patients per year (Ma *et al.* , 2019; Sordo *et al.* , 2017), but these are observational associations and likely overstate the causal effect, since patients who remain in treatment differ systematically from those who drop out.

for the naturally occurring interactions through which supportive relationships emerge. In this sense, the social environment created during inpatient treatment represents a potentially valuable and scalable component of care. Patient relationships formed during treatment can have lasting consequences for recovery and survival, and these benefits arise at essentially zero marginal cost as a byproduct of care that is already being delivered.

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.

Ahern, Amy L., *et al.* . 2017. Extended and Standard Duration Weight-Loss Programme Referrals for Adults in Primary Care. *Lancet Public Health*. Group behavioural weight-loss programme yields greater weight loss than brief intervention.

Aizer, A., & Doyle, J.J. 2015. Juvenile Incarceration, Human Capital and Future Crime: Evidence from Randomly-Assigned Judges. *The Quarterly Journal of Economics*, **130**(2), 759—803.

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.

Andreasson, Sven, Holder, Harold D, Norström, Thor, Österberg, Esa, & Rossow, Ingeborg. 2006. Estimates of harm associated with changes in Swedish alcohol policy: results from past and present estimates. *Addiction*, **101**(8), 1096–1105.

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

Bailey, Michael, Farrell, Patrick, Kuchler, Theresa, & Stroebel, Johannes. 2020. Social connectedness in urban areas. *Journal of Urban Economics*, **118**, 103264.

Bassuk, Ellen L, Hanson, Jennifer, Greene, Ross N, Richard, Michael, & Laudet, Alexandre. 2016. Peer-delivered recovery support services for addictions in the United States: A systematic review. *Journal of Substance Abuse Treatment*, **63**, 1–9.

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.

Beaudoin, Francesca L, Jacka, Brendan P, Li, Yu, Samuels, Elizabeth A, Hallowell, Benjamin D, Peachey, Alyssa M, Newman, Roxxanne A, Daly, Mackenzie M, Langdon, Kirsten J, & Marshall, Brandon DL. 2022. Effect of a peer-led behavioral intervention for emergency department patients at high risk of fatal opioid overdose: a randomized clinical trial. *JAMA network open*, **5**(8), e2225582–e2225582.

Becker, Gary S., & Murphy, Kevin M. 1988. A Theory of Rational Addiction. *Journal of Political Economy*, **96**(4), 675–700.

Bernheim, B Douglas, & Rangel, Antonio. 2004. Addiction and cue-triggered decision processes. *American economic review*, **94**(5), 1558–1590.

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.

Büchel, Konstantin, & Ehrlich, Maximilian V. 2020. Cities and the structure of social interactions: Evidence from mobile phone data. *Journal of urban economics*, **119**, 103276.

Bøg, Martin, Filges, Trine, Brännström, Lars, Jørgensen, Anne-Marie Klint, & Fredrikksson, Maja Karrman. 2017. 12-step programs for reducing illicit drug use. *Campbell Systematic Reviews*, **13**(1), 1–149.

Carrell, Scott E., Sacerdote, Bruce I., & West, James E. 2013. From Natural Variation to Optimal Policy? The Importance of Endogenous Peer Group Formation. *Econometrica*, **81**(3), 855–882.

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

Caulkins, Jonathan P, Pardo, Bryce, & Kilmer, Beau. 2019. Supervised consumption sites: a nuanced assessment of the causal evidence. *Addiction*, **114**(12), 2109–2115.

CDC. 2024. *Excessive Drinking Data and Statistics*. https://www.cdc.gov/alcohol/excessive-drinking-data/index.html. Accessed: 2026-01-16.

Chambers, Laura C, Li, Yu, Hallowell, Benjamin D, Langdon, Kirsten J, Samuels, Elizabeth A, Mahoney, Linda A, Beaudoin, Francesca L, & Marshall, Brandon DL. 2024. Effect of a peer-led emergency department behavioral intervention on non-fatal opioid overdose: 18-month outcome in the Navigator randomized controlled trial. *Addiction*, **119**(12), 2116–2128.

Chimbar, Lisa, & Moleta, Yvette. 2018. Naloxone effectiveness: a systematic review. *Journal of addictions nursing*, **29**(3), 167–171.

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.

Clark, Andrew E, & Lohéac, Youenn. 2007. "It wasn't me, it was them!" Social influence in risky behavior by adolescents. *Journal of health economics*, **26**(4), 763–784.

Cutler, David M, & Donahoe, J Travis. 2024. *Addiction, Thick Market Externalities and the Persistence of the Opioid Epidemic*. Tech. rept. National Bureau of Economic Research.

Cutler, David M, & Glaeser, Edward. 2005. What explains differences in smoking, drinking, and other health-related behaviors? *American Economic Review*, **95**(2), 238–242.

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.

Degenhardt, Louisa, Bucello, Chiara, Mathers, Bradley, Briegleb, Christina, Ali, Hammad, Hickman, Matt, & McLaren, Jennifer. 2011. Mortality among regular or dependent users of heroin and other opioids: a systematic review and meta-analysis of cohort studies. *Addiction*, **106**(1), 32–51.

Doleac, Jennifer L., & Mukherjee, Anita. 2022. The Effects of Naloxone Access Laws on Opioid Abuse, Mortality, and Crime. *The Journal of Law and Economics*, **65**(2), 211–238.

Drago, Francesco, & Galbiati, Roberto. 2012. Indirect effects of a policy altering criminal behavior: Evidence from the Italian prison experiment. *American Economic Journal: Applied Economics*, **4**(2), 199–218.

Dustmann, Christian, & Landersø, Rasmus. 2021. Child's gender, young fathers' crime, and spillover effects in criminal behavior. *Journal of Political Economy*, **129**(12), 3261–3301.

Eddie, David, Hoffman, Lesley, Vilsaint, Corrie, Abry, Alexis, Bergman, Brandon G, Hoeppner, Bettina B, & 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.

Eddie, David, O'Connor, Jenny B, George, Shane S, Klein, Morgan R, Lam, Tracy CS, Abry, Alexandra, Hoffman, Lauren A, Hennessy, Emily A, Vilsaint, Corrie L, & Kelly, John F. 2025. Peer Recovery Support Services and Recovery Coaching for Substance Use Disorder: A Systematic Review. *Current Addiction Reports*, **12**(1), 40.

Eichmeyer, Sarah, & Zhang, Jonathan. 2022. Pathways into opioid dependence: Evidence from practice variation in emergency departments. *American Economic Journal: Applied Economics*, **14**(4), 271–300.

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.

Feld, Jan, & Zölitz, Ulf. 2017. Understanding peer effects: On the nature, estimation, and channels of peer effects. *Journal of Labor Economics*, **35**(2), 387–428.

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.

Fortin, Bernard, & Yazbeck, Myra. 2015. Peer Effects, Fast Food Consumption and Adolescent Weight Gain. *Journal of Health Economics*, **42**, 125–138.

Garnett, M. F., & Miniño, A. M. 2024. *Drug Overdose Deaths in the United States, 2003–2023*. NCHS Data Brief 522. National Center for Health Statistics, Hyattsville, MD. Accessed: 2026-01-16.

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.

Godøy, Anna, Haaland, Venke F., Huitfeldt, Ingrid, & Votruba, Mark. 2024. Hospital Queues, Patient Health, and Labor Supply. *American Economic Journal: Economic Policy*, **16**(2), 150–81.

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.

Hébert, Anne H, & Hill, Alison L. 2024. Impact of opioid overdoses on US life expectancy and years of life lost, by demographic group and stimulant co-involvement: a mortality data analysis from 2019 to 2022. *The Lancet Regional Health–Americas*, **36**.

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

Hordvin, O. 2012. *The Drug Situation in Norway 2012. Annual Report to the EMCDDA*.

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

Jackson, Matthew O, Rogers, Brian W, & Zenou, Yves. 2017. The economic consequences of social-network structure. *Journal of economic literature*, **55**(1), 49–95.

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.

Johnsen, Julian, & Khoury, Laura. 2024. Peer Effects in Prison.

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

Kahneman, Daniel. 2011. Thinking Fast and Slow. *Farrar, Strauss and Giroux*.

Kelly, J.F., Humphreys, K., & Ferri, M. 2020. Alcoholics Anonymous and other 12-step programs for alcohol use disorder. *Cochrane Database of Systematic Reviews*.

Kerwin, Jason T., Rostom, Alex, & Sterck, Olivier. 2024. *Striking the Right Balance: Why Standard Balance Tests Over-Reject the Null, and How to Fix It*. IZA Discussion Paper 17464. IZA Institute of Labor Economics. Revised 2025.

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

Kling, Jeffrey R, Ludwig, Jens, & Katz, Lawrence F. 2005. Neighborhood effects on crime for female and male youth: Evidence from a randomized housing voucher experiment. *The Quarterly Journal of Economics*, **120**(1), 87–130.

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

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.

Levengood, Timothy W., Yoon, Grace H., Davoust, Melissa J., Ogden, Shannon N., Marshall, Brandon D. L., Cahill, Sean R., & Bazzi, Angela R. 2021. Supervised injection facilities as harm reduction: a systematic review. *American Journal of Preventive Medicine*, **61**(5), 738–749.

Ludwig, Jens, Duncan, Greg J, & Hirschfield, Paul. 2001. Urban poverty and juvenile crime: Evidence from a randomized housing-mobility experiment. *The Quarterly Journal of Economics*, **116**(2), 655–679.

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

Ma, Jun, Bao, Yan-Ping, Wang, Ru-Jia, Su, Meng-Fan, Liu, Mo-Xuan, Li, Jin-Qiao, Degenhardt, Louisa, Farrell, Michael, Blow, Frederic C., Ilgen, Mark, Shi, Jie, & Lu, Lin. 2019. Effects of medication-assisted treatment on mortality among opioids users: a systematic review and meta-analysis. *Molecular Psychiatry*, **24**(12), 1868–1883.

Mäckle, Kai, & Ruenzi, Stefan. 2022. The Social Transmission of Non-Infectious Diseases: Evidence from the Opioid Epidemic. *Available at SSRN 4104796*.

Maclean, Johanna Catherine, Mallatt, Justine, Ruhm, Christopher J, & Simon, Kosali. 2022. The opioid crisis, health, healthcare, and crime: A review of quasi-experimental economic studies. *The ANNALS of the American Academy of Political and Social Science*, **703**(1), 15–49.

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

Mastrobuoni, Giovanni, & Rialland, Pierre. 2020. Partners in crime: Evidence from recidivating inmates. *Italian Economic Journal*, **6**(2), 255–273.

Moore, Timothy J, & Schnepel, Kevin T. 2026. Opioid use, mortality risks, and crime: Insights from a rapid reduction in heroin supply. *Review of Economics and Statistics*, **108**(4), 948–966.

NASEM. 2020. *Measuring Success in Substance Use Grant Programs: Outcomes and Metrics for Improvement*. https://www.ncbi.nlm.nih.gov/books/NBK566487/.

NCDAS. 2025. *Drug Abuse Statistics*. https://drugabusestatistics.org/. Accessed: 2026-01-16.

Nguyen, Thuy, Cantor, Jonathan, Andraka-Christou, Barbara, Bradford, W. David, & Simon, Kosali. 2021. Where did the specialty behavioral health workforce grow between 2011 and 2019? Evidence from census data. *Journal of Substance Abuse Treatment*, **130**, 108482.

NIPH. 2019. *National Overdose Strategy 2019-2022*. Tech. rept.

Norström, Thor, & Ramstedt, Mats. 2018. The link between per capita alcohol consumption and alcohol-related harm in Sweden, 1987–2015. *Journal of studies on alcohol and drugs*, **79**(4), 578–584.

Ouss, Aurelie. 2011. Prison as a school of crime: Evidence from cell-level interactions. *Available at SSRN 1989803*.

Packham, Analisa. 2022. Syringe exchange programs and harm reduction: New evidence in the wake of the opioid epidemic. *Journal of Public Economics*, **215**, 104733.

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.

Pateria, Puraskar, De Boer, Bastiaan, & MacQuillan, Gerry. 2013. Liver abnormalities in drug and substance abusers. *Best practice & research Clinical gastroenterology*, **27**(4), 577–596.

Powell, David, & Pacula, Rosalie Liccardo. 2021. The evolving consequences of oxycontin reformulation on drug overdoses. *American Journal of Health Economics*, **7**(1), 41–67.

Powell, David, Pacula, Rosalie Liccardo, & Taylor, Erin. 2020. How increasing medical access to opioids contributes to the opioid epidemic: Evidence from Medicare Part D. *Journal of health economics*, **71**, 102286.

Prudon, Roger. 2024. *Is delayed mental health treatment detrimental to employment?* Tech. rept.

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.

Rees, Daniel I., Sabia, Joseph J., Argys, Laura M., Dave, Dhaval, & Latshaw, Joshua. 2019. With a Little Help from My Friends: The Effects of Good Samaritan and Naloxone Access Laws on Opioid-Related Deaths. *The Journal of Law and Economics*, **62**(1), 1–27.

Romano, Joseph P, & Wolf, Michael. 2005. Stepwise multiple testing as formalized data snooping. *Econometrica*, **73**(4), 1237–1282.

Sacerdote, Bruce. 2011. Peer Effects in Education: How Might They Work, How Big Are They and How Much Do We Know Thus Far? *Handbook of the Economics of Education*, **3**, 249–277.

SAMHSA. 2022. *Results from the 2021 National Survey on Drug Use and Health: Summary of national findings*. Tech. rept. NSDUH Series H-57, HHS Publication No. (SMA) PEP22-07-01-005. Center for Behavioral Health Statistics and Quality.

SAMHSA. 2025. *2024 National Survey on Drug Use and Health: Annual National Report*. https://www.samhsa.gov/data/sites/default/files/reports/rpt56287/2024-nsduh-annual-national-report.pdf. Accessed: 2026-01-16.

Santo Jr, Thomas, Clark, Brodie, Hickman, Matthew, Grebely, Jason, Campbell, Gabrielle, Sordo, Luis, Chen, Angela, Tran, Lucy T., Bharat, Chrianna, Padmanathan, Prianka, Cousins, Gail, Dupouy, Julie, Kelty, Erin, Muhammad, Tasrif, & Degenhardt, Louisa. 2021. Association of Opioid Agonist Treatment With All-Cause Mortality and Specific Causes of Death Among People With Opioid Dependence: A Systematic Review and Meta-analysis. *JAMA Psychiatry*, **78**(9), 979–993.

Seamans, Marissa J, Carey, Timothy S, Westreich, Daniel J, Cole, Stephen R, Wheeler, Stephanie B, Alexander, G Caleb, Pate, Virginia, & Brookhart, M Alan. 2018. Association of household opioid availability and prescription opioid initiation among household members. *JAMA internal medicine*, **178**(1), 102–109.

SIRUS. 2011. *The Drug Situation in Norway 2011: Annual report to the European Monitoring Centre for Drugs and Drug Addiction – EMCDDA*. Tech. rept.

Sordo, Luis, Barrio, Gregorio, Bravo, Maria J., Indave, Blanca I., Degenhardt, Louisa, Wiessing, Lucas, Ferri, Marica, & Pastor-Barriuso, Roberto. 2017. Mortality risk during and after opioid substitution treatment: systematic review and meta-analysis of cohort studies. *BMJ*, **357**, j1550.

Spencer, MR, Garnett, MF, & Miniño, AM. 2024. *"Drug overdose deaths in the United States, 2002–2022*. Tech. rept. NCHS Data Brief, no 491.

Stevenson, Megan. 2017. Breaking bad: Mechanisms of social influence and the path to criminality in juvenile jails. *Review of Economics and Statistics*, **99**(5), 824–838.

Strickland, Justin C., & Acuff, Samuel F. 2023. Role of social context in addiction etiology and recovery. *Pharmacology Biochemistry and Behavior*, **229**, 173603.

Surgeon General. 2016. *Facing Addiction in America: The Surgeon General's Report on Alcohol, Drugs, and Health. Washington*. U.S. Department of Health and Human Services.

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.

Timoneda, Joan C. 2021. Estimating group fixed effects in panel data with a binary dependent variable: How the LPM outperforms logistic regression in rare events data. *Social Science Research*, **93**, 102486.

Watson, Dennis P, Tillson, Martha, Taylor, Lisa, Xu, Huiping, Ouyang, Fangqian, Beaudoin, Francesca L, O'Donnell, Daniel, & McGuire, Alan B. 2024. Results from the POINT pragmatic randomized trial: an emergency department-based peer support specialist intervention to increase opioid use disorder treatment linkage and reduce recurrent overdose. *Substance use & addiction journal*, **45**(3), 378–389.

WHO. 2018. *Norway - Factsheet 2016*. Tech. rept.

Williams, Jenny, Bretteville-Jensen, Anne Line, & Rose, Christiern. 2026. The Benefits of Timely Access to Treatment: Substance Use Disorders, Healthcare Utilization and Employment. *Journal of Health Economics*, **108**, 103162.

Appendix

A Data

Measuring outcomes

Our main outcomes of interest are indicators for death within a fixed period of exiting treatment. We break deaths down into those due to external causes, internal causes, and those induced by substance use.

External causes of death include accidents (e.g., accidental poisonings), intentional self harm (including suicide by intentional poisoning) and homicide. Poisonings (accidental and intentional) include deaths in which an illicit drug overdose is the cause of death. Internal causes of death include those due to infection or parasites, cancer, blood or immune system disorders, endocrine or metabolic system disorders, nervous system disorders, circulatory system disorders, respiratory system disorders, digestive system disorders, skin disorders, musculoskeletal disorders, genitourinary disorders, congenital malformations, and other abnormalities.

Substance-induced deaths are either drug-induced or alcohol-induced. Drug-induced deaths are those caused by a mental-behavioral disorder due to psychoactive substance use (opioids, cannabinoids, cocaine, other stimulants, hallucinogens, multiple drug use) or poisoning (accidental, intentional or of undetermined intent) by opium, heroin, other opioids, methadone, other synthetic narcotics, cocaine, other and unspecified narcotics, cannabis, lysergide, other and unspecified psychodysleptics, or psychostimulants. 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. Alcohol-induced deaths are deaths from alcohol abuse, degeneration or damage to the nervous system due to alcohol, death due to alcoholic cardiomyopathy, gastritis, liver disease, or pancreatic disorders, and poisoning by alcohol (accidental, intentional, undetermined intent).

Identifying the treatment facility

To identify the treatment facility attended we merge patient level data with supplementary facility level data on the health trust, sector, and the types of patients catered to (sex, age range and substance). We can uniquely identify the facility attended for 57% of patients. There are two potential facilities for 15% of patients, three potential facilities for 21% of patients, and four possible facilities for 6% of patients. To assess the match quality we compare the implied occupancy with the number of beds. If a patient could have attended more than one facility, for this exercise we distribute them fractionally across each possible facility. Since aggregate demand routinely exceeds capacity, low quality matching would lead to a significant fraction of facilities appearing to operate above capacity. Using our matching algorithm, capacity was exceeded on fewer than 5% of facility-days. This suggests that we are able to match patients to facilities reasonably well given the data available to us.

If the facility that the patient could have attended is not unique, when constructing any measure

of peer exposure, we use all inpatients who *could* have overlapped at the same facility as the focal individual. This introduces some measurement error, which may attenuate our estimates toward zero. Measurement error is also a feature of other papers using a similar research design. For example, Bayer *et al.* (2009) only observe prisoners who are *released* during a given window, and correct for the potential peers that they do not observe using the expected number of days of overlap.

Sample

The sample is constructed from all inpatient episodes for substance use treatment that occurred in one of 58 public facilities and which include at least one day of inpatient treatment during the period 1 January 2009 to 31 December 2010. Since patients do not interact during detoxification, we shift the date of admission forward by the period of detoxification to obtain the start date of treatment. This leads to a sample size of 4,856 treatment episodes. Of these, 41% of episodes are for alcohol use disorders and 59% are for drug use disorders, of which the most common is opioid use (37%), followed by multiple drug use (28%), stimulants other than cocaine (15%) and cannabis use (12%). This sample is used to construct the peer exposure variable (described below).

The analysis sample uses only the first inpatient episode for each individual that starts between 1 January 2009 and 31 December 2010, and ends by 31 December 2010 (N=4,257). These are the patients for which we are able to observe all other patients with an overlapping treatment episode. We exclude a further 369 observations for patients who attend facilities in Central Norway, as this health region does not report specialist inpatient treatment at the health trust level (which implies that we cannot match patients to facilities), 16 observations for which there are more than four facilities that they may have attended, and 132 due to missing controls (N=3,838). Of these, we lose another 83 observations from our fixed effects analysis because they are singletons (N=3,755).

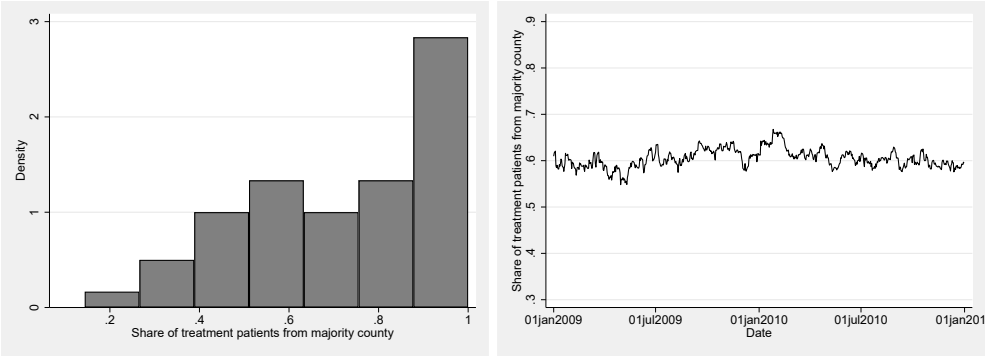
B Measuring peer exposure

As stated in the main text, we measure the peer exposure of individual i who resides in county c and who was treated in facility j as the proportion of total patient-days that overlap with the patient i's treatment episode in facility j that are for patients who also reside in county c. Here we provide further justification for our county-based measure of peers and document its variation.

Further justification of county-based measure

Due to the sensitive nature of our sample, the county of residence is the most granular geographic information that was made available to us. This is less of a limitation than it may first appear, because the county is close to the ideal unit for our purposes rather than merely the finest one available. As explained in the main text, the object of interest is the geographic unit at which co-residence best predicts the maintenance of social ties, which the literature locates at the level of the functional urban region defined by travel time. We argue below that the Norwegian county

Figure A1: Concentration ratio of patients by county



Notes: The left panel shows a facility-level histogram of concentration ratios. For each facility, the ratio is defined as the share of patients coming from its single largest source county (which is typically, though not always, the county where the facility is located). The right hand panel is a patient-level time series plot. For any given date it reports the share of the stock of patients residing in the single largest source county of their facility (i.e., the patient-level mean of an indicator for residing in the largest source county of their facility).

approximates this unit well. Analysis at the county level also ensures a large enough number of individuals receiving inpatient treatment residing in each geographic area so as to generate sufficient variation from overlapping treatment episodes.

During the period of our data there were 19 Norwegian counties, as depicted in Figure 1 in the main text. Reflecting population sparsity, counties varied considerably in area, ranging from 454 km^2 (Oslo) to 48,631 km^2 (Finnmark), but to a lesser extent in population, from 73,417 (Finnmark) to 599,230 (Oslo). In terms of substance use prevalence, a median sized county had around 519 injecting drug users and 13,903 problematic alcohol users.[32] In our estimation sample of those receiving inpatient treatment, the number of inpatients residing in each county ranges from 19 (Nord-Trøndelag) to 815 (Oslo) with a median of 133. These figures demonstrate that the pool of potential peers in each county is not particularly large, which reinforces the interpretation of the county as a meaningful social unit: individuals drawn from the same modestly sized, largely urban pool are plausibly likely to encounter one another after treatment.

We now present evidence that county of residence closely tracks the functional urban region. During the period of our data, around 80% of the Norwegian population resided in an urban settlement (known as a *tettsted*), and 60% resided in a city (known as a *by*). Those with a SUD are more concentrated in urban centers than the general population (Gjersing, 2023; SAMHSA, 2022; Swensen, 2015). In our analysis sample, 70% of individuals live in a city.[33] At the time of our data

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

[33]We do not observe the fraction living in an urban settlement, but it is likely to be notably higher than the 80%

the median county had one city, up to a maximum of three.[34] Because most counties therefore contained a single urban center, members of our sample recorded as residing in the same county are likely to also reside in the same urban area. Even when they do not, the mean distance to the *furthest* city in the same county is 40.32km (a 43 minute drive), whereas the mean distance to the *nearest* city in another county is 172.51km (a 2h5m drive). Same-county pairs are therefore likely to fall within a single functional region, and cross-county pairs outside one, which is precisely the alignment that makes the county an effective proxy. For this reason, we expect our county-based measure of peer exposure to be a reliable proxy for co-location within the functional region in which post-treatment interaction occurs.

Corroborating quantitative evidence

The argument so far has rested on the institutional geography of Norway. Because most counties contain a single urban center and cross-county travel times are large, same-county pairs of individuals tend to fall within a single functional region. We now provide quantitative support for the same conclusion. Doing so also lets us address a question the institutional argument leaves open. That argument establishes that the county is a reasonable proxy, but not whether it is the best available one. A finer unit such as the municipality, or a coarser one such as a multi-county region, might measure social ties more accurately.

Framed as a measurement problem, we wish to classify pairs of individuals according to whether they maintain a social connection formed during inpatient treatment, and the county is only one of many geography-based indicators that could be used. An indicator that is too fine, say, the immediate neighborhood, may code genuinely connected pairs who live a short journey apart as unconnected. One that is too coarse, say, a multi-county region, is likely to code vast numbers of unconnected pairs as connected. Either form of misclassification introduces measurement error into the peer variable. The ideal unit is the one that best predicts the existence of a social tie, and the exercise that follows locates the county on that spectrum.

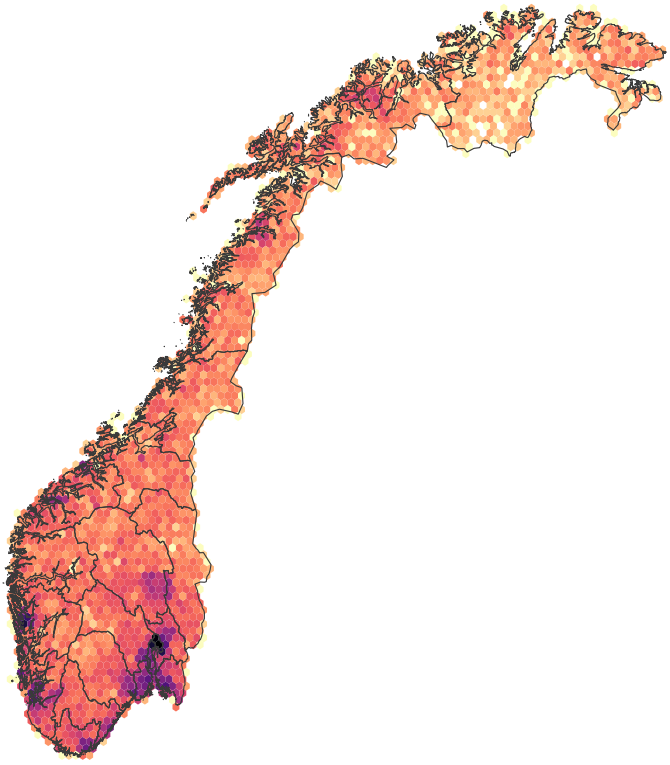
Because we do not observe social ties directly, we proceed by simulation. We construct a synthetic population on the true geography of Norway, generate social ties from a data generating process calibrated to the empirical relationship between geographic proximity and social connectedness (Bailey *et al.* , 2020), and then ask how well various geography-based indicators recover those ties. We consider both partitions of the country (grids, municipalities, counties, regions) and dyadic indicators of the form "within K kilometers" or "within T minutes" of travel, which would be available only with exact location data. The county is evaluated against all of these.

We place a synthetic population on the real geography of Norway as it was structured during the period of our data (in 2010), comprising 19 counties and the 357 municipalities nested within them. The distribution of the population is shown in Figure A2. It is concentrated around Oslo, the

of the general population who live in one.

[34] This figure is based on *by* with a population exceeding 30,000 in 2011, as city status is sometimes conferred on the basis of factors such as historical importance or administrative function, rather than population size.

Figure A2: Heatmap of simulated population density



western coastal cities, and Trondheim, thinning sharply to the north. For each pair of individuals i and j we compute travel time τ_{ij} from the great-circle distance between them, scaled by a detour factor to reflect the indirectness of the Norwegian road network and converted to minutes at a representative travel speed. We generate social ties from a specification that mirrors the gravity model of Bailey *et al.* (2020), who estimate the elasticity of social connectedness with respect to travel time using geolocated social network data. In their model, the probability that i and j are connected by a social tie is

$$\Pr(i \sim j) \;=\; \exp\big(\phi_i + \phi_j\big)\, \tau_{ij}^{\beta} \tag{3}$$

where we take $\beta = -1.42$ as the travel-time elasticity estimated by Bailey *et al.* (2020) and ϕ_i is an individual fixed effect. We choose the fixed effects in the data generating process so that the implied degree distribution (distribution of the number of social ties) is realistic, however our findings do not rest on this. For example all of our conclusions are unchanged when $\phi_i = \phi$.

Two features of the simulated network can be checked against what is known about real social networks. Figures A3 and A4 show summary statistics for a single replication of the data generating process. First, the degree distribution (Figure A3) is right-skewed with a mean near 18 ties and a coefficient of variation near 0.5, both in the range observed empirically, and it differs only mildly between urban and rural residents. Second, the geography of realized ties is sensible (Figure A4).

Figure A3: Degree distribution of the simulated network

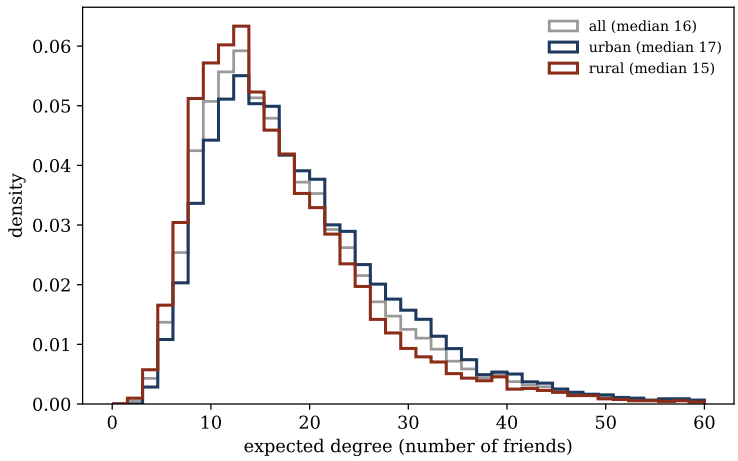
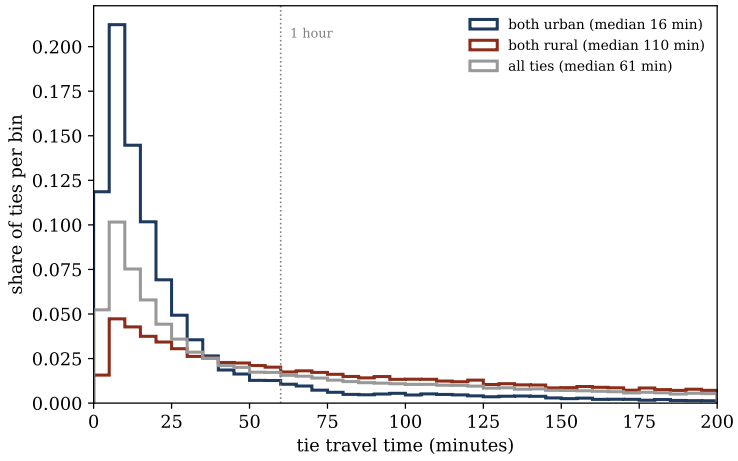


Figure A4: Distribution of travel time between connected pairs



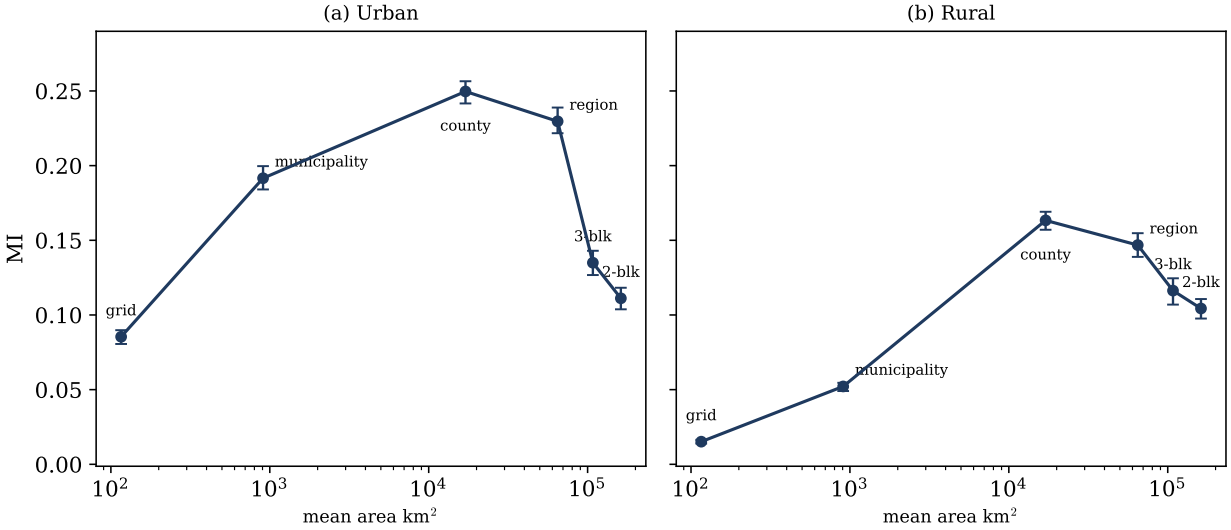
Among ties between two urban residents (the configuration most relevant to our sample) the median tie spans about 16 minutes of travel and more than 80% fall within an hour, consistent with the intuition that friendships are predominantly locally maintained. As expected, ties involving two rural residents involve longer travel times.

For a given binary indicator: "same county," "within K km," and so on, we measure how well it classifies the simulated ties using the normalized mutual information (MI).[35] Normalized MI is zero when the geography-based indicator is uninformative about realized ties and one when it perfectly determines the realized ties. Figure A5 plots MI against mean area for a nested sequence of partitions, from a fine sub-municipal grid, through municipalities and counties, to regions and coarser multi-region blocks.[36] Each point represents the mean MI across 200 simulations. The 95%

[35]Mutual information is the appropriate criterion here because social ties are rare. The overwhelming majority of pairs are unconnected, and a metric such as classification accuracy or the area under the ROC curve rewards coarse indicators simply for labeling more pairs as connected, without penalizing the resulting false positives.

[36]The municipality (357 units) and county (19 units) are real administrative partitions. The region (5 units)

Figure A5: Classification quality of geographic partitions



confidence bar shows the 2.5 and 97.5 percentiles of the MI distribution across the 200 simulations. Panels (a) and (b) respectively consider the cases where individual i resides in an urban and rural location. In both cases we observe that classification quality is inverted-U-shaped in unit area, and the county sits at the peak. This result is intuitive: a unit finer than the county severs genuine social ties, while a coarser unit pools together individuals who never interact.[37]

Figures A6 and A7 provide analagous results for the dyadic indicators "within K km" and "within T minutes," which would require exact location data. They plot the mean MI for the full sample over 200 simulations and the associated 95% confidence intervals. In both figures we observe an inverted U in the threshold peaking modestly above the county-based indicator (shown as a horizontal reference line). These figure show that the best attainable MI from an exact-distance or exact-travel-time indicator exceeds the county's by only a small margin: the county captures roughly 85% of the information available to an optimal dyadic indicator constructed from precise coordinates. In other words, exact location data, even used optimally, would improve only marginally on the county we actually use.

Overall, across a realistic synthetic population on the true geography of Norway, with social ties generated from a process that reproduces the (Bailey *et al.* , 2020) travel-time elasticity, the county emerges as close to the ideal geography-based binary classifier of social ties. It dominates all finer and coarser partitions, and captures the large majority of the information available even

corresponds to the standard Norwegian macro-regions. The three- and two-block partitions are coarser groupings included only to trace the full shape of the curve, and do not correspond to administrative units in use.

[37] For urban residents the region performs better than the municipality, and almost as well as the county. Whilst this may be surprising at first glance, it reflects geographic partitions in the greater Oslo area. Oslo county and its constituent municipalities do not cover the metro area, which includes additional counties, notably Akershus. For this reason, an indicator for both residing in the Eastern Norway region is a better classifier of social ties than an indicator for both residing in the same municipality.

Figure A6: Classification quality of the dyadic "within K km" indicator

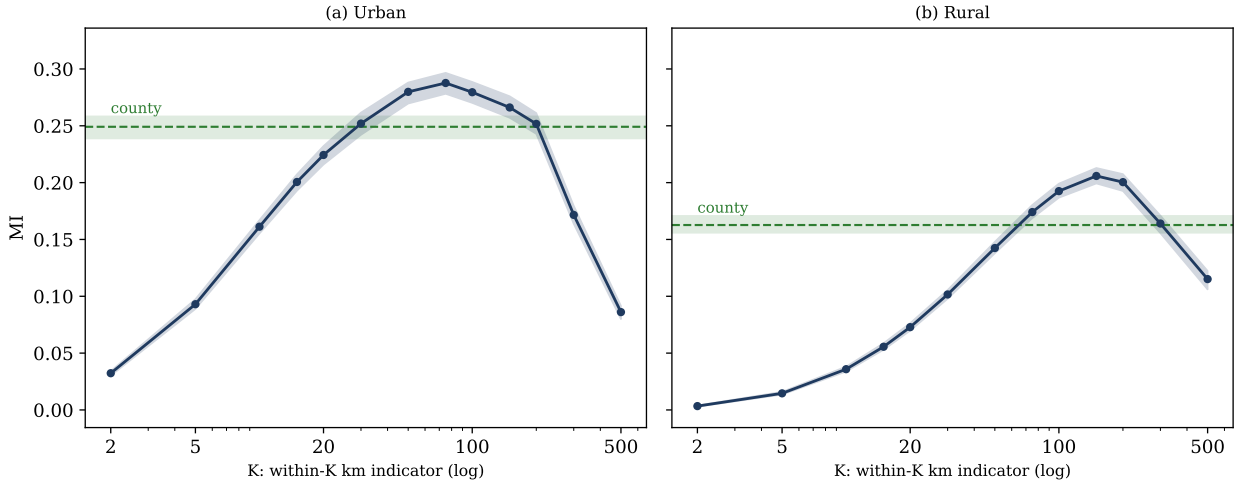
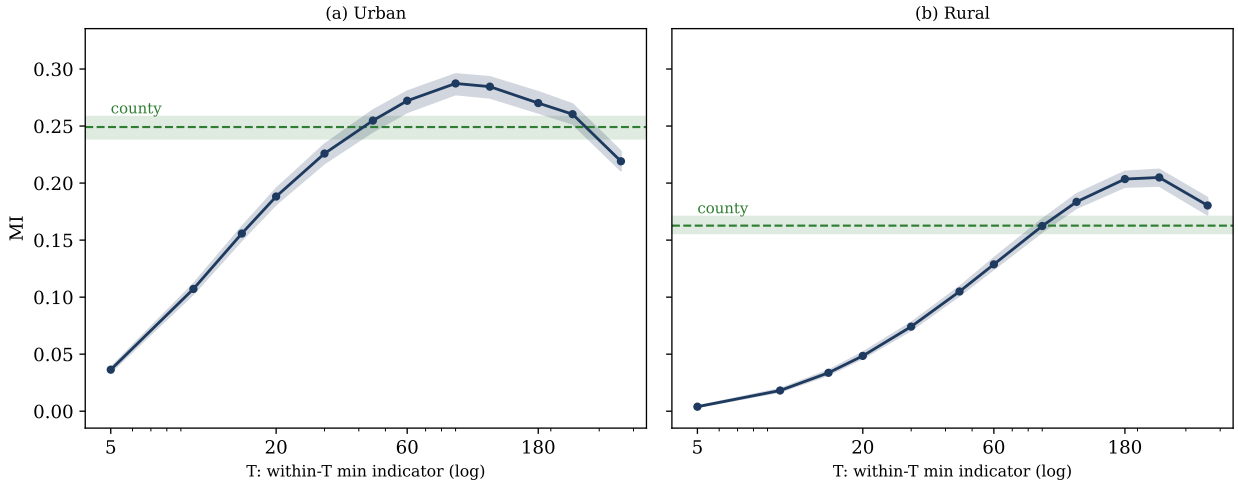


Figure A7: Classification quality of the dyadic "within T minutes" indicator



to dyadic indicators built from exact location data. These conclusions are robust across urban and rural subpopulations and across the specification of the individual fixed effects.[38] This exercise supports the use of the county as the geographic basis for our peer measure.

Measurement error

Even though the county is close to the optimal geographic unit we might still be concerned about measurement error. Specifically, given the bounded nature of our measure of peer exposure between 0 and 1, we may be concerned about non-classical measurement error. What matters for identification is the extent of measurement error that remains after partialing out the fixed effects

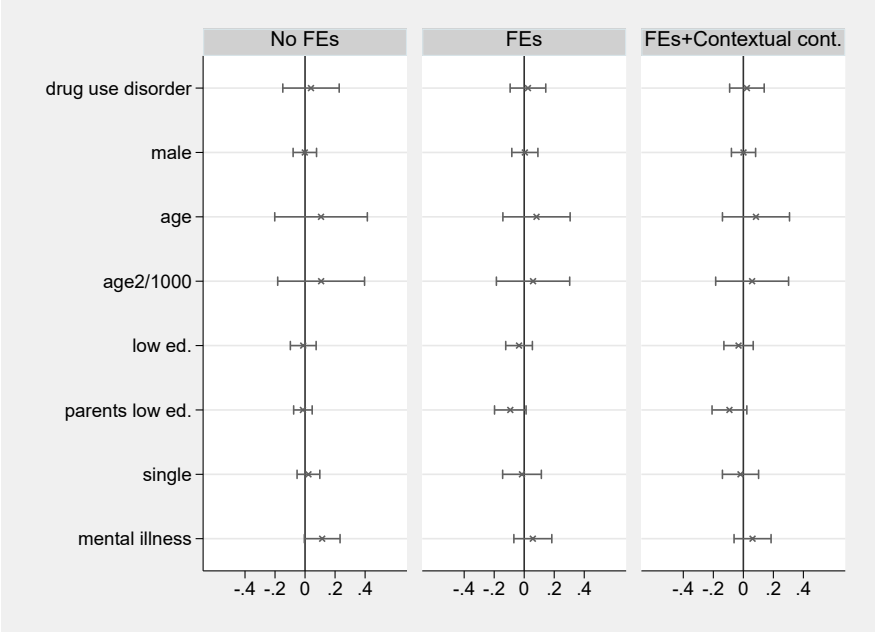
[38] Results of this sensitivity analysis are available on request.

(right-hand side of Figure 3). Reassuringly, we do not observe significant mass at the upper and lower bounds of the residualized distribution, implying that the mass points of individuals with peer exposure equal to one or zero are largely explained by county-by-facility variation. Given this, the most obvious source of non-classical measurement error in a bounded [0,1] regressor — spurious point masses at the boundaries — is unlikely to be driving our results. To the extent that remaining measurement error in the residualized variable is approximately classical, our estimates of the impact of peers can be interpreted as a lower bound on the true effect.

Variation

As illustrated by Figure A1, variation in exposure to peers arises both due to differences between treatment facilities and random fluctuations over time. The figure depicts concentration ratios of patients by county in our data. The left panel shows a facility-level histogram. For each facility, the concentration ratio is defined as the share of patients coming from its single largest source county (which is typically, though not always, the county where the facility is located). As might be expected, the mode of this distribution is close to one (i.e., the modal facility sources most of its patients from the county in which it is located). In such facilities, patients have high exposure to peers. However, there is considerable heterogeneity. Some (rural) facilities only acquire around 20% of their patients from their single largest source county, implying that their patients have low exposure to peers. The right hand panel of Figure A1 shows how the concentration of patients

Figure A8: Balance test: Controls as dependent variable



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

Table A1: Balance test: Fitted values

	Death within 1 year of exit (fitted values)			Death within 3 years of exit (fitted values)		
peer exposure	0.003*	0.002	0.002	0.006	0.004	0.004
	(0.001)	(0.001)	(0.001)	(0.005)	(0.005)	(0.005)
Mean dep. var.	0.022	0.022	0.022	0.067	0.067	0.067
Observations	3,755	3,755	3,755	3,755	3,755	3,755
FEs	N	Y	Y	N	Y	Y
Contextual controls	N	N	Y	N	N	Y

Notes: Outcomes are fitted values using all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1.

across counties fluctuates over time. For any given date it reports the share of the national stock of patients receiving inpatient treatment who reside in the single largest source county of their facility (i.e., the patient-level mean of an indicator for residing in the largest source county of their facility). This fluctuates between 55% and 67%, illustrating that the timing of treatment also influences exposure to peers. As explained in the main text, our identification strategy is based around within county-by-facility fluctuations in exposure to peers over time, depicted in Figure 5.

C Additional balance tests

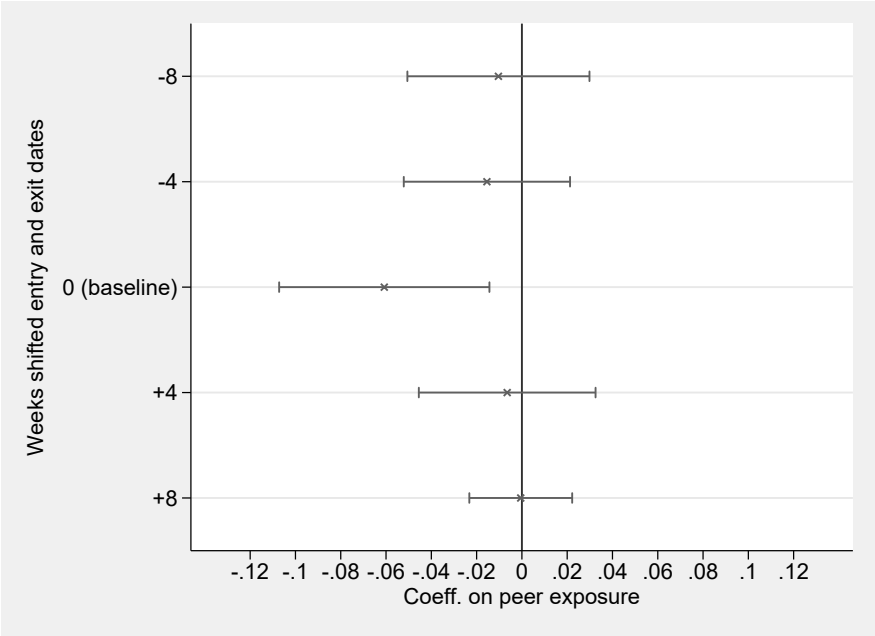
In the main text we presented the results of a balance test in which the dependent variable was peer exposure and the independent variables comprised patient characteristics, contextual controls, and fixed effects. An alternative is to consider regressions where the dependent variable is a patient characteristic and the independent variables comprise the peer exposure measure, contextual controls and fixed effects. Figure A8 reports the coefficient on the peer exposure measure and its 95% confidence interval. In all models it is close to zero and statistically insignificant.

A further balance test 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 value of the outcome (death) and the independent variables are the peer exposure variable, fixed effects and contextual controls. Table A1 reports the results of this exercise. In models with neither fixed effects nor contextual controls we find an association with the peers variable that is statistically significant at the 0.1 level. However, with fixed effects and contextual controls the magnitude of the coefficient on the peers variable is reduced, and it is not statistically significant.

D Placebo tests

To provide evidence that our baseline findings are driven by genuine peer effects, rather than some other artifact of inpatient admission, we conduct two placebo tests.

Figure A9: Shifted treatment episode (death within 1 year)



Shifted treatment episode

The first placebo test shifts the treatment episode of the focal individual to a different point in time when constructing all variables, including our measure of peer exposure. This assigns each inpatient a placebo group of other inpatients who received treatment at the same facility at around the same time, but whose treatment episodes had no (or limited) overlap. We shift episodes by four weeks and eight weeks. Shifting by four weeks implies that the shifted episode would have no overlap with the actual episode for an individual with median treatment duration (9 days) and little overlap for an inpatient with mean treatment duration (28 days). Shifting by eight weeks implies that the gap between the actual and shifted episode is approximately double the mean treatment duration. This means that the group of overlapping inpatients will tend to be entirely different in the shifted episode relative to the unshifted episode. Figure A9 reports the coefficient and 95% confidence intervals for the impact of peer exposure on mortality in the first year after leaving treatment. The coefficient is small in magnitude and not statistically distinguishable from zero whenever the episode is shifted. Results for deaths from internal and external causes are reported in Figure A10. Again, we find no impact of placebo exposure to peers.

Sample of emergency patients

The second placebo test replicates our baseline findings using a sample of patients admitted into inpatient treatment facilities as emergency patients. Emergency care is used when people with a SUD are a danger to themselves or others, and involves detoxification but not treatment to address

Figure A10: Internal (left) and external (right) cause deaths within 1 year

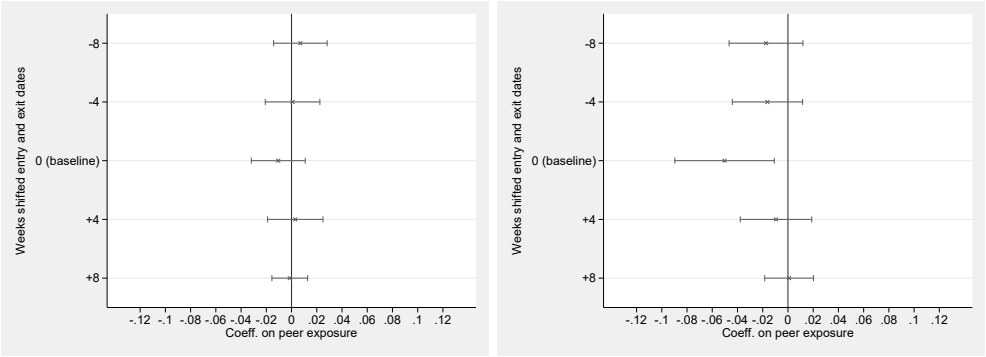


Table A2: Descriptive statistics for emergency patients: Outcomes and peer exposure

	Mean			
	within 1 year	within 3 years		
Death				
all-causes	0.020	0.060		
internal	0.007	0.027		
external	0.012	0.030		
	Mean	Std. dev.	Min	Max
Peers				
days admitted	9.65	20.88	1	349
num. overlapping inpatients	15.38	20.91	1	265
num. overlapping inpatients from same county	10.52	15.26	0	219
peer exposure	0.72	0.31	0	1
Observations	3,461			

disordered substance use. As patients admitted to inpatient facilities for emergency care do not interact with other patients through treatment, we should find no impact of overlapping patient episodes on the focal patient's mortality risk.

The sample of emergency patients is constructed as follows. We have 3,787 emergency care inpatients, of whom 42% are admitted for an alcohol use disorder and 58% for a drug use disorder. Among the latter, 55% were admitted with a polysubstance diagnosis, 14% for stimulants other 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 in a sample size of N=3,553. A further 92 observations are lost due to their being singletons in our fixed effects analysis, resulting in an analysis sample of N=3,461. Table A2 summarizes the

Table A3: Emergency patients

	Death within 1 year of exit			Death within 3 years of exit			
	All	Internal	External	All	Internal	External	Substance induced
peer exposure	0.020	-0.002	0.012	0.006	-0.006	0.005	0.022
	(0.022)	(0.013)	(0.013)	(0.026)	(0.017)	(0.016)	(0.015)
Mean dep. var.	0.020	0.007	0.012	0.060	0.027	0.030	0.020
+1 peer (%)	1.55	-0.34	1.64	0.17	-0.35	0.24	1.72
Observations	3,461	3,461	3,461	3,461	3,461	3,461	3,461

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1.

sample.

Table A3 repeats our baseline analysis of treatment patients using the sample of emergency patients. The point estimates of interest are reasonably small in magnitude and do not approach statistical significance at conventional levels for any cause of death or time horizon. Our main results for treatment patients within 1 year of exit found a negative coefficient that was statistically significant at the 0.05 level and implies that exposure to an additional peer reduced mortality in the first year after exit by 4.66% relative to the mean. For the sample of emergency patients the coefficient is close to zero, statistically insignificant (despite the similar sample size), and implies that the effect of an additional peer (+1.55%) is smaller than our baseline findings. Over three years, the coefficients for the placebo sample are even smaller in magnitude. For all cause mortality the effect size of an additional peer is (+0.17%), which is an order of magnitude smaller than for our main sample (-2.04%). While we find a larger coefficient for substance-induced deaths, it is marginally smaller in magnitude than our baseline estimates (+1.72% vs -1.98%) and, in contrast to our baseline estimate, is not statistically significant at any conventional level.

E Further robustness tests

Endogenous exit

While the use of waitlists plausibly provides quasi-experimental variation in the date of entry, we may be concerned about endogenous exit from treatment. The first panel of Table A4 addresses this by repeating our baseline analysis, but using only other inpatients encountered on the first day of the treatment episode to construct the peer variable, which cannot be driven by endogenous exit. The estimates are similar to our baseline results. The second panel of Table A4 uses the first day peer measure as an instrument for our primary measure of peer exposure (over the entire episode). The 2SLS estimates are similar to our baseline estimates, though tend to be larger in magnitude. For example, our baseline results imply that an additional peer reduces the risk of death within one year by 4.66%, whereas our 2SLS estimates suggest it is 6.07%. However, as with the reduced

Table A4: First day peers

		Death within 1 year of exit			Death within 3 years of exit			
		All	Internal	External	All	Internal	External	Substance induced
Reduced form	peer exposure (first day only)	-0.057** (0.026)	-0.007 (0.009)	-0.051** (0.025)	-0.069** (0.028)	-0.012 (0.021)	-0.054* (0.028)	-0.024* (0.013)
	Mean dep. var.	0.022	0.011	0.012	0.067	0.033	0.033	0.023
	+1 peer (%)	-4.40	-1.07	-7.42	-1.75	-0.47	-2.88	-1.75
2SLS	peer exposure	-0.079** (0.036)	-0.009 (0.012)	-0.070* (0.035)	-0.095** (0.038)	-0.016 (0.020)	-0.075* (0.038)	-0.033* (0.017)
	+1 peer (%)	-6.07	-1.48	-10.23	-2.42	-0.65	-3.97	-2.42
	Olea-Pflueger F	558	558	558	558	558	558	558
	Observations	3,755	3,755	3,755	3,755	3,755	3,755	3,755

Notes: The top panel replaces our measure of peer exposure with peer exposure on the first day of the treatment episode. The second panel uses peer exposure on the first day to instrument peer exposure during the entire episode. All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1.

Table A5: Duration of treatment

	Duration	Duration	Log duration	Log duration
peer exposure (first day only)	-2.667 (8.428)		0.192 (0.178)	
peer exposure		-2.888 (4.737)		0.173 (0.163)
Mean dep. var.	28.47	28.47	2.280	2.280
+1 peer (%)	-0.16	-0.17	0.13	0.15
Observations	3,755	3,755	3,755	3,755

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1.

form results in the first panel, all of our qualitative conclusions remain the same as for our baseline model in the main text: peers reduce all cause, external cause and substance induced mortality, and the effects are evident in the first year following treatment but persist until the third. That we draw the same qualitative conclusions from analysis in Table A4 suggests that our main findings are not driven by endogenous exposure to peers during inpatient treatment.

To further assess the extent to which endogenous exit may drive our findings, Table A5 considers the duration of treatment as the dependent variable, and estimates models in which the variable of interest is either peer exposure on the first day of the episode, or peer exposure throughout the

episode. Since the treatment duration is skewed to the right, we also estimate models where the dependent variable is log duration. If inpatients systematically extend or curtail their treatment based on the social ties that have or have not formed, we would expect the coefficients in Table A5 to be different from zero. We find no evidence of any systematic relationship between peer exposure and duration. All of the coefficients are statistically insignificant and all of the implied effect sizes are close to zero.

Measurement of peer exposure

To assess the sensitivity of our findings to measurement error arising from the existence of multiple possible treatment facilities for a minority of patients, we repeat the mortality analysis in the main text but restrict the sample to individuals who receive treatment at facilities for which all patients could not have attended another facility. As this sample is comprised of patients whose treatment facility is uniquely identified, it is not subject to such measurement error concerns. Table A6 reports our findings. The coefficients and their standard errors are similar to our baseline analysis.

We might also be concerned that including rural patients in our analysis limits the extent to which living in the same county implies geographic proximity in the post-treatment period. To address this we repeat the mortality analysis in the main text but limiting the estimation sample to those who live in a city. Our measure of peer exposure for this sample is the weighted proportion of all other overlapping inpatients (including those who do not live in a city) who live in the same county and who live in a city. Given that the median county has just one city, and Norwegian cities are small, these are the individuals for whom we can be most confident of geographic proximity in the post-treatment period.[39] Table A7 reports our findings. Consistent with our findings in the main text, we find evidence that exposure to peers reduces mortality from all causes at both the one and three year time horizons after exiting treatment, and that this is driven by mortality from external causes, rather than internal causes. The effect sizes are larger than for our main results.

[39]Note that we do not observe a city identifier, only an indicator for living in one.

Table A6: Sample with a known treatment facility

	Death within 1 year of exit			Death within 3 years of exit			
	All	Internal	External	All	Internal	External	Substance induced
peer exposure	-0.049**	-0.019	-0.030**	-0.068*	-0.030	-0.029	-0.038**
	(0.022)	(0.018)	(0.012)	(0.032)	(0.025)	(0.021)	(0.013)
Mean dep. var.	0.021	0.012	0.009	0.067	0.031	0.034	0.023
+1 peer (%)	-4.83	-3.32	-6.72	-2.19	-1.79	-2.14	-3.38
Observations	1,706	1,706	1,706	1,706	1,706	1,706	1,706

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. ∗∗∗: p<0.01. ∗∗: p<0.05. ∗: p<0.1.

Table A7: Sample of inpatients residing in a city

	Death within 1 year of exit			Death within 3 years of exit			
	All	Internal	External	All	Internal	External	Substance induced
peer exposure	-0.065*	-0.000	-0.065**	-0.096**	-0.012	-0.077**	-0.012
	(0.035)	(0.019)	(0.026)	(0.041)	(0.029)	(0.030)	(0.024)
Mean dep. var.	0.023	0.010	0.013	0.067	0.031	0.034	0.026
+1 peer (%)	-6.80	-0.05	-11.96	-3.49	-0.94	-5.42	-1.06
Observations	2,606	2,606	2,606	2,606	2,606	2,606	2,606

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1.

Here we find that exposure to one additional peer for the duration of the inpatient treatment episode reduces mortality in the first year after exiting treatment by 6.80% of the mean, compared to 4.66% for our baseline analysis. This is consistent with our hypothesis that the geographic distance between individuals is likely to be lower when residing in a city, hence strengthening the probability of interaction in the post-treatment period.

Another potential concern is that in the main text we define peers among overlapping inpatients based solely on geographic proximity. It is possible, however, that geographic proximity is not what drives the formation of long-run ties, but rather the tendency of similar individuals to form ties with one another (Jackson *et al.* , 2017), including in the context of risky behaviors (Johnsen & Khoury, 2024; Mastrobuoni & Rialland, 2020), a phenomenon known as homophily. In this case, geography may be one dimension along which homophily operates, but others such as sex, age or the substance for which treatment is received may also matter. We explore this issue by considering additional definitions of peers based on overlapping inpatients who share (a) the same sex and age (defined by an indicator for age$\leq$ 26, the threshold used in Norway to define treatment facilities specializing in young people) and (b) the same treatment substance class (alcohol use or drug use). Table A8 reports the results. We *only* find any evidence of an impact of peers on mortality when the definition of peers includes a common county of residence. These findings suggest that geographic proximity is necessary for peers to impact mortality.

A final potential concern is that in the main text we use the proportion of peers rather than the peer count. This is because the count of inpatients from the same county with overlapping treatment episodes is likely to be correlated with the duration of treatment, and hence with unobservable severity of illness. To examine the sensitivity of our findings to using the proportion rather than the count, we repeat our analysis using the number of (unique) peers exposed to during the episode. We address its endogeneity by instrumenting using the number of peers exposed to on the first day of the episode. The identifying variation then comes exclusively from the timing of entry into treatment, which we expect to be as good as randomly assigned due to the use of waitlists. Since the peer count is highly skewed we include it logarithmically in the empirical model. We also specify

Table A8: Alternative peer definitions

		Death within 1 year of exit			Death within 3 years of exit			
		All	Internal	External	All	Internal	External	Substance induced
Same sex + age	peer exposure	-0.008	-0.007	-0.001	0.012	0.015	-0.004	0.009
		(0.020)	(0.009)	(0.017)	(0.036)	(0.015)	(0.031)	(0.016)
	Mean dep. var.	0.022	0.011	0.012	0.067	0.033	0.033	0.023
	+1 peer (%)	-0.86	-1.59	-0.20	0.46	1.17	-0.27	0.96
Same sex + age	peer exposure	-0.061***	-0.024	-0.037**	-0.062	-0.007	-0.053	-0.010
		(0.021)	(0.015)	(0.018)	(0.039)	(0.024)	(0.036)	(0.019)
+ county	+1 peer (%)	-9.19	-7.63	-10.61	-3.14	-0.68	-5.58	-1.40
Same substance	peer exposure	-0.007	-0.006	-0.001	-0.031	-0.018	-0.012	-0.016
		(0.016)	(0.010)	(0.010)	(0.033)	(0.022)	(0.016)	(0.011)
	+1 peer (%)	-0.58	-1.06	-0.15	-0.85	-0.98	-0.65	-1.23
Same substance	peer exposure	-0.045**	-0.014	-0.031*	-0.079**	-0.027	-0.048**	-0.037***
		(0.019)	(0.009)	(0.018)	(0.034)	(0.021)	(0.023)	(0.011)
+ county	+1 peer (%)	-5.86	-3.80	-7.72	-3.43	-2.41	-4.30	-4.69
	Observations	3,755	3,755	3,755	3,755	3,755	3,755	3,755

Notes: In each model, to ensure that identification comes from timing alone, the fixed effects are for the dimensions along which we define peers. For example, when we define peers using sex and age only, the fixed effects are for sex-by-age-by-facility. When we also require that they reside in the same county, the fixed effects are for sex-by-age-by-county-by-facility. All models also include month-by-year of exit fixed effects and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1.

the instrument logarithmically.

Table A9 reports the results. The first panel is for least squares, the second reports the reduced form, and the third reports the 2SLS estimates. The least squares results tend to be more positive than the reduced form and 2SLS estimates. This is likely due to endogeneity of the peer count: those patients who are unobservably sicker receive more treatment, and hence acquire more peers, while also being more likely to die in the post-treatment period. The reduced form and 2SLS results are in line with our baseline findings in the main text. Greater exposure to peers reduces mortality in the first year following treatment, and this is driven by a reduction in deaths from external causes. The effect sizes and precision are both lower than for our baseline findings. For all cause mortality within a year of exiting treatment, our instrumental variables estimates imply an effect size of 3.51% of the mean, whereas in our baseline analysis we found an effect size of 4.66%. Moreover, in contrast to our analysis in the main text the 2SLS results do not provide statistical evidence that the effect persists into the third post-treatment year.

Econometric modeling of rare outcomes

Another concern relates to econometric modeling of low probability events. This applies mainly to mortality by cause within one year of exiting treatment, since only 2.2% of sample members die over

Table A9: Number of peers

		Death within 1 year of exit			Death within 3 years of exit			
		All	Internal	External	All	Internal	External	Substance induced
OLS	log(1 + n. peers)	-0.001	0.000	-0.001	-0.010	-0.005	-0.002	-0.006*
		(0.004)	(0.002)	(0.003)	(0.009)	(0.003)	(0.007)	(0.003)
	Mean dep. var.	0.022	0.011	0.012	0.067	0.033	0.033	0.023
	+1 peer (%)	-0.26	0.01	-0.50	-0.95	-0.99	-0.39	-1.58
Reduced form	log(1 + n. peers) (first day only)	-0.007	0.002	-0.009**	0.001	0.008	-0.006	-0.000
		(0.005)	(0.005)	(0.004)	(0.011)	(0.006)	(0.009)	(0.005)
	+1 peer (%)	-2.12	1.27	-5.20	0.08	1.23	-1.75	-0.10
2SLS	log(1 + n. peers)	-0.012*	0.003	-0.015*	0.001	0.014	-0.010	-0.001
		(0.006)	(0.008)	(0.007)	(0.019)	(0.010)	(0.016)	(0.009)
	+1 peer (%)	-3.51	2.10	-8.62	0.13	2.86	-2.05	-0.16
	Olea-Pflueger F	113	113	113	113	113	113	113
	Observations	3,755	3,755	3,755	3,755	3,755	3,755	3,755

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. ∗∗∗: p<0.01. ∗∗: p<0.05. ∗: p<0.1.

this time-frame. Though the linear probability model is the workhorse empirical model in a setting where identification rests on fixed effects, and has been shown to perform well when modeling rare events (Timoneda, 2021), alternatives that may be more suitable are the Complementary log-log model and the Penalized Logit model proposed by Firth (1993). Table A10 reports the results for these models. For ease of comparison with the linear probability model, we report average marginal effects. The signs of the average marginal effects are the same as those for the linear probability model in the main text. If anything, the average marginal effects for these alternative models are larger than our baseline linear probability estimates.

Outliers

We might also be concerned that our main findings are driven by outliers. Though we view this as unlikely given that (a) our dependent variable is binary and our measure of peer exposure lies between zero and one, and so does not have long tails (see Figure 3, left panel) and (b) we find no effects in two placebo tests using two similar samples, here we perform two robustness tests to ensure that our main findings are not driven by outliers. First, we repeat our main analysis using only treatment facilities with above median number of beds (> 19 beds). We do this since extreme values of peer exposure (zeros and ones) are more likely to arise in facilities with fewer beds. Table A11 reports our findings. We find similar point estimates and effect sizes to our baseline results, though the estimate for substance induced deaths loses statistical significance. Second, we repeat

Table A10: Alternative models for rare outcomes

	Death within 1 year of exit					
	Complementary log-log			Penalized Logit		
	All	Internal	External	All	Internal	External
peer exposure	-0.099***	-0.046*	-0.113***	-0.095***	-0.037	-0.085***
	(0.024)	(0.026)	(0.028)	(0.027)	(0.027)	(0.026)
Mean dep. var.	0.022	0.011	0.012	0.022	0.011	0.012
+1 peer (%)	-7.61	-7.28	-16.38	-7.30	-5.85	-12.33
Observations	3,755	3,755	3,755	3,755	3,755	3,755

Notes: Average marginal effects are reported. All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. ∗∗∗: p<0.01. ∗∗: p<0.05. ∗: p<0.1.

our analysis dropping the most extreme 0.5% of values from each tail of the distribution of peer exposure residualized on the fixed effects that we employ (see Figure 3, right panel). Table A12 reports our findings. We find the same signs and statistical significance as in our baseline analysis. However we also find some of the effect sizes are attenuated. In the first year after exiting treatment we find that an additional peer reduces mortality by 3.61% of the mean, rather than the 4.66% reported for our baseline model.

Specification of controls and fixed effects

Finally we might be concerned that our findings are sensitive to the specification of controls and/or fixed effects. To explore this we repeat our analysis varying the controls (none; individual only; individual and contextual) and fixed effects (county and facility, county-by-facility). Our baseline model uses all controls and county-by-facility fixed effects.

Table A13 reports our findings. Models (1)-(3) use county and facility fixed effects. Model

Table A11: Sample of inpatients attending above median sized facilities (> 19 beds)

	Death within 1 year of exit			Death within 3 years of exit			
	All	Internal	External	All	Internal	External	Substance induced
peer exposure	-0.081**	-0.020	-0.061**	-0.088**	-0.010	-0.067*	-0.021
	(0.032)	(0.015)	(0.029)	(0.033)	(0.019)	(0.033)	(0.015)
Mean dep. var.	0.022	0.010	0.012	0.068	0.032	0.033	0.024
+1 peer (%)	-5.86	-3.20	-8.07	-2.07	-0.48	-3.23	-1.42
Observations	3,476	3,476	3,476	3,476	3,476	3,476	3,476

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. ∗∗∗: p<0.01. ∗∗: p<0.05. ∗: p<0.1.

Table A12: Trimmed sample

	Death within 1 year of exit			Death within 3 years of exit			
	All	Internal	External	All	Internal	External	Substance induced
peer exposure	-0.046**	-0.014	-0.032**	-0.075***	-0.020	-0.045**	-0.035**
	(0.018)	(0.013)	(0.013)	(0.022)	(0.018)	(0.018)	(0.014)
Mean dep. var.	0.022	0.011	0.011	0.067	0.033	0.032	0.024
+1 peer (%)	-3.61	-2.30	-4.86	-1.92	-1.05	-2.41	-2.53
Observations	3,738	3,738	3,738	3,738	3,738	3,738	3,738

Notes: All models include county-by-facility and month-by-year of exit fixed effects, and all controls listed in Section 3.1.4. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1. Outliers are defined as observations for which the value of residualized peer exposure (with respect to the fixed effects that we employ) is below the 0.5 percentile or above the 99.5 percentile.

(1) uses no controls, model (2) uses individual controls only, and model (3) uses individual and contextual controls. Models (4)-(6) use county-by-facility fixed effects. Model (4) uses no controls, model (5) uses individual controls only and model (6) uses individual and contextual controls (our baseline specification in the main text). We draw two conclusions from Table A13. First, the choice of control variables makes little difference to our findings. The coefficient of interest does not vary much within models (1)-(3) or within models (4)-(6). We expect this to be the case since the balance tests above show that these controls are not correlated with peer exposure. Second, the choice of fixed effects does influence our findings. The coefficient of interest is smaller in models with county and facility fixed effects (models (1)-(3)) than with county-by-facility fixed effects (models (4)-(6)). This suggests that there is some selection into treatment facilities, which is not fully accounted for using county and facility fixed effects separately. The results in Table A13 suggest that unobservably sicker patients have a higher propensity to sort into facilities with others from their own county.

A remaining concern is that our facility-by-county fixed effects, while absorbing persistent differences in case mix, referral patterns, and facility quality across cells, are additive with month-of-exit fixed effects in our baseline specification and therefore do not capture confounding that evolves differently over time across facility-county cells. For example, a local overdose wave, a gradual deterioration in staffing, or changes in care delivery at a particular facility could affect both the composition of peers a patient is exposed to and their underlying mortality risk in ways that are not fully captured by common month-of-exit fixed effects.

To address this concern, we augment our baseline specification by including county-by-facility linear trends, where time is measured by month of exit. These trends allow each county-facility cell to follow its own underlying trajectory over time, absorbing smoothly evolving confounding that differs across facilities and counties while preserving identifying variation in peer exposure within each county-facility cell. Identification therefore rests on deviations from these cell-specific trends,

rather than on common time patterns alone. The estimated effects in column (7) are very similar to those in column (6). This indicates that the results are not driven by smoothly evolving changes in patient composition or treatment environments within county-facility cells over time.

Table A13: Sensitivity to specification of controls and fixed effects

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Death within 1 year	-0.032***	-0.033***	-0.033***	-0.060**	-0.062**	-0.061**	-0.054**
	(0.011)	(0.012)	(0.011)	(0.024)	(0.025)	(0.024)	(0.025)
Internal	-0.010	-0.010	-0.010	-0.012	-0.012	-0.011	-0.003
	(0.007)	(0.007)	(0.007)	(0.011)	(0.011)	(0.011)	(0.011)
External	-0.023**	-0.023**	-0.023**	-0.048**	-0.050**	-0.050**	-0.051**
	(0.009)	(0.009)	(0.009)	(0.021)	(0.021)	(0.020)	(0.020)
Death within 3 years	-0.030*	-0.034**	-0.034**	-0.076***	-0.080***	-0.080***	-0.078**
	(0.016)	(0.016)	(0.016)	(0.026)	(0.027)	(0.025)	(0.029)
Internal	-0.006	-0.009	-0.009	-0.010	-0.012	-0.012	-0.010
	(0.010)	(0.010)	(0.010)	(0.016)	(0.017)	(0.017)	(0.019)
External	-0.022	-0.022	-0.022	-0.057**	-0.060**	-0.060**	-0.063**
	(0.013)	(0.014)	(0.013)	(0.025)	(0.024)	(0.023)	(0.026)
Substance induced	-0.011	-0.012	-0.012	-0.024*	-0.026*	-0.027**	-0.030*
	(0.009)	(0.009)	(0.009)	(0.014)	(0.013)	(0.013)	(0.016)
Individual controls	N	Y	Y	N	Y	Y	Y
Contextual controls	N	N	Y	N	N	Y	Y
County and facility FEs	Y	Y	Y	N	N	N	N
County-by-facility FEs	N	N	N	Y	Y	Y	Y
Month-by-year of exit FEs	Y	Y	Y	Y	Y	Y	Y
County-by-facility linear trends	N	N	N	N	N	N	Y

Notes: This table reports the coefficient on peer exposure across a range of different model specifications. Standard errors (in parentheses) are clustered by facility. $***$: p<0.01. $**$: p<0.05. $*$: p<0.1.

F Theoretical framework

Our theoretical framework extends Bernheim & Rangel (2004) to include peer interactions. A key feature of Bernheim & Rangel's model is that it accounts for the fact that addictive substances interfere with processes in the brain involved in forecasting near-term rewards. As a consequence, when exposed to strong environmental cues, individuals enter a "hot" decision making mode in which their cue-triggered impulses dominate their cognitive processes, and they use the substance irrespective of their underlying preferences. Otherwise, individuals are in "cold" mode and make decisions in line with their preferences. Such decision making states have also been referred to as 'fast' (instinctive and emotional) and 'slow' (deliberative and logical) (e.g., Kahneman (2011)).

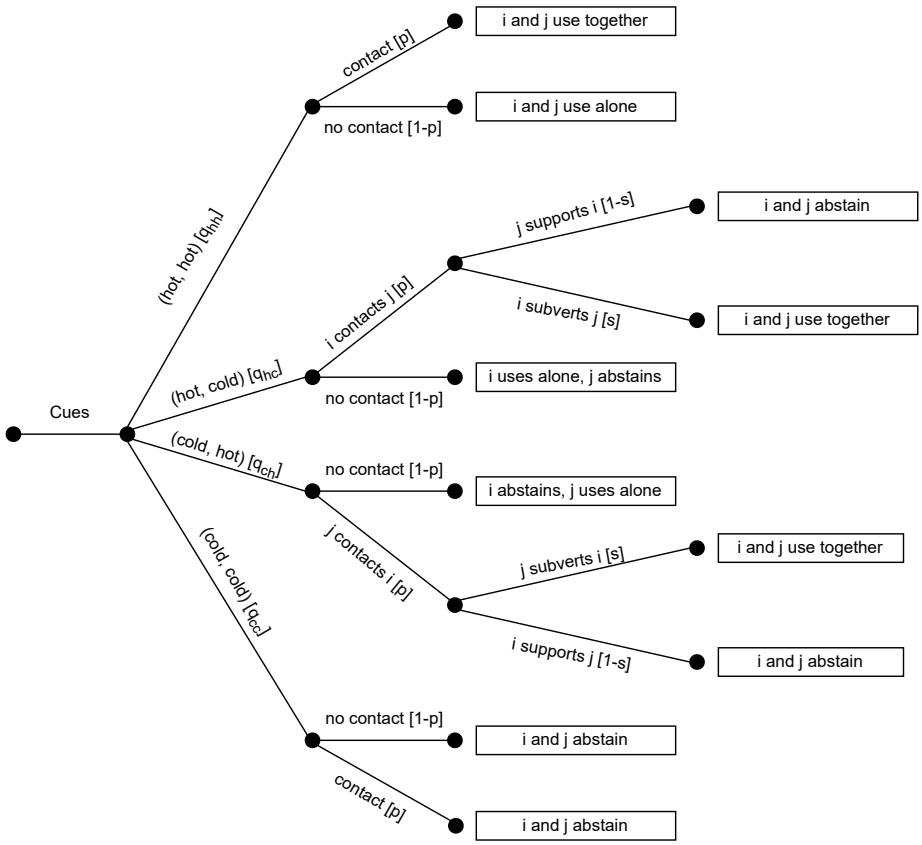
To incorporate peers into the model, we consider two representative individuals, labeled i and j. Both are exposed to environmental cues, which are random, identically distributed (for simplicity) and potentially correlated. Sufficiently strong cues result in individuals entering the hot decision making mode. Otherwise, decision making occurs in cold mode. This leads to four possible states of the world: (cold, cold), (cold, hot), (hot, cold), (hot, hot). i and j's substance use behavior depends on the state of the world and whether or not they are in contact with one another.

Figure A11 shows the set of possible interactions for i and j and the resulting substance use behavior. The first branch captures the state of the world, and subsequent branches capture interactions between i and j. The probabilities of each branch are shown in brackets. Since we study individuals who voluntarily undertake inpatient treatment, we assume that underlying preferences are for recovery. So, if both i and j are in cold mode, both abstain from using whether they are in contact or not. If at least one of them is in hot mode, an individual can influence the other's behavior in one of three ways. First, if the individual is in hot mode and they make contact with a peer who is in cold mode, they may *subvert* the recovery of their peer by causing them to use. Second, if the individual is in cold mode and they are contacted by a peer in hot mode, they may *support* the recovery of their peer by helping them manage their desire to use and avoid use. The third way that peers can influence substance use behavior arises if both i and j are in hot mode. If both are in hot mode and make contact, then they engage in *harm reduction* by using together rather than using alone.

A key determinant of the effect of peers in the post-treatment period is the probability that i and j are in contact, which we denote by p. We also denote the probability of subversion given contact by s, and the probability of support given contact by $1-s$. To simplify the analysis below, we normalize $q_{cc} = 0$ (because peers play no role in state (cold, cold)), $q_{hh} = q$, and $q_{hc} = q_{ch} = (1-q)/2$.[40] Our interest lies in the impact of a change in p on i's substance use behavior, and through this, on i's observable outcomes, denoted Y. Reflecting the empirical measures available to us in the administrative data, the outcomes we consider are death, readmission into an inpatient treatment facility, hospital admission for substance related disease, and hospital admission for substance related poisoning. While we do not observe substance use, these outcomes capture its primary

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

Figure A11: A model of cue-triggered substance use among peers



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

impacts on health and demand for healthcare, and are hence of direct policy relevance. This is particularly true for mortality.

In order to examine the impact of a change in p on i's post-treatment outcome, we start by expressing the expected outcome as:

$$E(Y) = P(\text{use alone})E(Y|\text{use alone}) + P(\text{use together})E(Y|\text{use together}) + P(\text{abstain})E(Y|\text{abstain}) \tag{4}$$

where $P(\text{use alone}) = q(1-p) + \left(\frac{1-q}{2}\right)(1-p)$ is the probability of i using alone, $P(\text{use together}) = qp + (1-q)ps$ is the probability of i using with j and $P(\text{abstain}) = 1 - P(\text{use alone}) - P(\text{use together})$ is the probability that i abstains.[41] To determine the impact of a change in p on i's post-treatment outcome, we conduct comparative statics in p, decomposing $\partial E(Y)/\partial p$ (i.e., the net impact of

[41] As j's substance use is symmetric to that of i, we omit the i subscript from Y.

peers) as follows:

$$\frac{\partial E(Y)}{\partial p} = \underbrace{\left[\left(\frac{1-q}{2}\right)s\right]\Delta_{\text{subvert}}}_{\text{Subvert recovery mechanism}} + \underbrace{\left[\left(\frac{1-q}{2}\right)(1-s)\right]\Delta_{\text{support}}}_{\text{Support recovery mechanism}} + \underbrace{\left[q+\left(\frac{1-q}{2}\right)s\right]\Delta_{\text{harm reduction}}}_{\text{Harm reduction mechanism}}. \quad (5)$$

Each term on the right hand side corresponds to one of the three mechanisms through which j changes i's substance use. The effect of each mechanism can be broken down into two parts. The leading part in brackets is the probability that it arises. The trailing part denoted by Δ captures the impact of the mechanism on the outcome when it does arise.

The subvert recovery mechanism causes i to use with j rather than abstain, hence $\Delta_{\text{subvert}} = E(Y|\text{use together}) - E(Y|\text{abstain})$. The support recovery mechanism causes i to abstain when, absent contact with j, i would have used alone, hence $\Delta_{\text{support}} = E(Y|\text{abstain}) - E(Y|\text{use alone})$. The harm reduction mechanism causes i to use with j rather than alone, hence $\Delta_{\text{harm reduction}} = E(Y|\text{use together}) - E(Y|\text{use alone})$.

To sign the impact of each mechanism (i.e., the Δs) we need to establish relative values for $E(Y|\text{abstain})$, $E(Y|\text{use alone})$ and $E(Y|\text{use together})$ for each health outcome. We assume that the probability of death, readmission and hospital admission is lowest when abstaining, and so we normalize $E(Y|\text{abstain}) = 0$.

Subverting and supporting recovery. Both mechanisms move i between abstention and use: subversion causes i to use with j rather than abstain, and support causes i to abstain rather than use alone. Since all outcomes are more likely under use than abstention, $\Delta_{\text{subvert}} = E(Y|\text{use together}) > 0$ and $\Delta_{\text{support}} = -E(Y|\text{use alone}) < 0$ for all four outcomes. This delivers the first two rows of Table 4 in the main text.

Harm reduction. The harm reduction mechanism moves i from using alone to using together, so signing $\Delta_{\text{harm reduction}}$ requires comparing $E(Y|\text{use alone})$ with $E(Y|\text{use together})$ for each outcome.

Consider first mortality. Using together, rather than alone, is a harm reduction approach, explicitly encouraged by the Norwegian public health agencies (NIPH, 2019). This is motivated by the fact that using under supervision has a lower risk of death (Caulkins *et al.* , 2019; Levengood *et al.* , 2021). Peers can supervise one another to guard against overdose or poisoning, and in the event of overdose or poisoning, they can contact emergency services and place the victim in the recovery position. They can also administer take-home anti-overdose medication such as naloxone (Chimbar & Moleta, 2018).

The harm reduction mechanism may have both direct and indirect implications for mortality and the other outcomes we study. Direct effects arise from changes in the safety of use, as discussed in the previous paragraph. Indirect effects may also arise through how an individual's choice to use responds to the safety of use.

Regarding the direct effect, absent technological differences in using together relative to alone (i.e., if ex-ante safety of use is the same in both cases), these arguments imply that the effect of

using together is driven by $E(D|\text{use alone}) > E(D|\text{use together})$, where D denotes an indicator for death, and hence $\Delta_{\text{harm reduction}} < 0$ for mortality. Next consider readmissions into inpatient care (denoted R) and hospital admissions for substance related disease (denoted H_D). Both of these outcomes should depend only on the extent of use rather than whether it takes place alone or with a peer, implying $E(R|\text{use together}) = E(R|\text{use alone})$ and $E(H_D|\text{use together}) = E(H_D|\text{use alone})$, and hence $\Delta_{\text{harm reduction}} = 0$ for both. Finally consider poisoning admissions. For poisoning admissions, two displacements operate in opposite directions. When a peer prevents a death while using together, it may result instead in a (non-fatal) hospital admission for poisoning (denoted H_P). At the same time, a peer may intervene early enough that a poisoning which would have led to hospital admission when using alone is instead resolved at the scene or in the emergency room. This means that

$$E(H_P|\text{use together}) = (1-\phi)E(H_P|\text{use alone}) + \theta\left[E(D|\text{use alone}) - E(D|\text{use together})\right]$$

where θ is the fraction of prevented deaths that instead result in hospital admission,[42] and ϕ is the fraction of poisoning admissions that would arise when using alone that are instead resolved without admission when using together. The first term captures the reduction in admissions from peers limiting the severity of poisonings, and the second captures the displacement of fatal overdoses into non-fatal admissions. Hence

$$\begin{aligned}\Delta_{\text{harm reduction}} &= E(H_P|\text{use together}) - E(H_P|\text{use alone}) \\ &= \theta\left[E(D|\text{use alone}) - E(D|\text{use together})\right] - \phi E(H_P|\text{use alone}),\end{aligned}$$

which is positive when the displacement of prevented deaths dominates and negative when the reduction in severity dominates. The sign of $\Delta_{\text{harm reduction}}$ for poisoning admissions is therefore ambiguous, as reported in Table 4. Together, these comparisons deliver the third row of Table 4, which accounts only for the direct effects of the harm reduction mechanism.

Indirect effects of harm reduction: moral hazard. The predictions for harm reduction in the final row of Table 4 account for moral hazard: by lowering the risk attached to each instance of use, harm reduction may lower the perceived cost of using and thereby increase the extent of use.[43] An increase in use raises the probability of readmission and disease admission. Its effect on the probability of death opposes the direct risk reduction from using together, so the sign of $\Delta_{\text{harm reduction}}$ for mortality is ambiguous. For poisoning admissions, increased use adds a force toward admissions without resolving the competing displacement effects above, so the sign remains ambiguous. This yields the signs in the final row of Table 4.

[42] Not all prevented deaths result in hospital admission. This is because the poisoning may be mild enough to be treated by paramedics at the scene or in the emergency room, without leading to subsequent hospital admission.

[43] We treat moral hazard as a reduced form adjustment to the harm reduction term rather than modeling the cue response explicitly; the qualitative predictions are what matter for our empirical strategy.

Complementarities and technological differences in harm reduction. Complementarities in using together increase use, and hence raise all four outcomes. Needle sharing, a technological difference among injecting drug users, would raise disease admissions and possibly mortality. On balance, we expect these features to be negligible in our setting for two reasons. First, needle exchanges were widely available, accessible, and extensively used during our sample period (Hordvin, 2012). Second, harm reduction education formed a core component of inpatient treatment programs, and using together rather than alone was promoted as a harm reduction strategy (NIPH, 2019). Government public health agencies and treatment facilities would not have promoted using together as a harm minimization strategy if there were significant complementarities that increased health risks associated with this behavior. Nonetheless, where present, the effects of complementarities and needle sharing are subsumed in the final row of Table 4 (i.e., they do not change the predictions relative to moral hazard alone).