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Incidence of suicide and self-harm among people with opioid use disorder and the impact of opioid agonist treatment: a retrospective data linkage study

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Abstract

Background: Rates of suicide and self-harm are elevated among people with opioid use disorder (OUD). This study examined incidence of self-harm and suicide among people who have entered OAT and assessed the impact of different OAT exposure periods on these events.

Method: We conducted a retrospective population-based cohort study of all OAT recipients (N=45,664) in New South Wales, Australia (2002–2017), using linked administrative data. Incidence rates of self-harm hospitalisations and suicide deaths were estimated per 1000 person-years (PY). The first 28 days of an OAT episode, ≥29 days on OAT, the first 28 days off OAT, and ≥29 days off OAT (maximum four years post-OAT) were exposure periods. Poisson regression models with generalised estimating equations estimated the adjusted incidence rate ratios (ARR) of self-harm and suicide by OAT exposure periods, adjusting for covariates.

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Contributors

Samantha Colledge-Frisby: Conceptualisation, Methodology, Visualisation, Writing-Original draft preparation, Writing-Reviewing and Editing. **Nicola Jones**: Data curation, Methodology, Formal analysis, Writing-Reviewing and Editing. **Prianka Padmanathan:** Writing-Reviewing and Editing. **Thomas Santo Jr:** Writing-Reviewing and Editing. **Matthew Hickman:** Writing-Reviewing and Editing. **Michael Farrell:** Writing-Reviewing and Editing. **Louisa Degenhardt:** Conceptualisation, Methodology, Writing-Reviewing and Editing, Supervision, Funding acquisition. **Natasa Gisev**: Conceptualisation, Methodology, Writing-Reviewing and Editing, Supervision.

[7].Conflict of interest

All other authors have no conflicts of interest to declare.

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Results: There were 7482 hospitalisations (4148 individuals) for self-harm and 556 suicides, equating to incidence rates of 19.2 (95% confidence intervals [CI]=18.8–19.7) and 1.0 (95%CI=0.9–1.1) per 1000 PY, respectively. Opioid overdose was implicated in 9.6% of suicides and 28% of self-harm hospitalisations. Compared to ≥29 days on OAT, the incidence rate of suicide was elevated in the 28 days following OAT cessation (ARR=17.4 [95%CI=11.7–25.9]), and the rate of self-harm hospitalisations was elevated during the first 28 days of OAT (ARR=2.2 [95%CI=1.9–2.6]) and the 28 days after leaving OAT (ARR=2.7 [95%CI=2.3–3.2]).

Conclusions: OAT may reduce suicide and self-harm risk among people with OUD; however, OAT initiation and cessation are critical periods for targeting self-harm and suicide prevention interventions.

Keywords

Suicide; self-harm; opioid agonist treatment; opioid use disorder

1. Introduction

Globally, it is estimated that over 700,000 people die by suicide each year[1]. The estimated global rate of suicide is approximately 9.4 deaths per 100,000 people, and is higher among men (13.3 deaths per 100,000) than women (5.7 deaths per 100,000)[2]. The World Health Organization's (WHO) Comprehensive Mental Health Action Plan aims to reduce the rate of suicide by one-third by 2030[3].

Non-fatal self-harm is the most well-established risk factor for suicide and, therefore, often a key outcome measure in suicide prevention intervention research[4,5]. Definitions of non-fatal self-harm vary in the literature, but often include non-suicidal self-injury and non-fatal suicide attempts. This is because suicidal intent can fluctuate within people who self-harm and is challenging to identify in clinical settings[2,6]. According to the Global Burden of Disease study, the incidence of non-fatal self-harm (regardless of suicidal intent) is 52.5 per 100,000 men and 75.9 per 100,000 women[7,8]. It is likely that the true burden of suicide and self-harm is higher due to under-reporting and miscoding[2].

Suicide and self-harm are substantially elevated among people with opioid use disorder (OUD). A global systematic review found that people who use extra-medical opioids (i.e. heroin, illicitly manufactured opioids, or pharmaceutical opioids outside the bounds of a medical prescription) die by suicide at nearly eight times the expected rate, with suicide accounting for around one in 12 deaths among this group[9]. An Australian study found that people with OUD were over 23 times, (risk ratio [RR]=23.6; 95% confidence intervals [CI]=17.9–31.1), as likely to present to hospital with self-harm compared to matched controls without OUD[10]. A similar study conducted in Hong Kong observed that relative to other substance use disorders, OUD was associated with the highest risk of suicide and self-harm (hazards ratio [HR]=27.3; 95%CI=19.5–38.3) compared to matched controls[11]. Targeting self-harm and suicide prevention to people with OUD is therefore critical, yet, globally, national suicide prevention policies rarely include substance-use specific policies[12,13].

Although there is evidence that some psychosocial (e.g. cognitive behavioural therapy and dialectical behavioural therapy) and pharmacological (e.g. anti-depressants and ketamine) interventions can reduce suicidal thoughts and behaviour in the general population, people with OUD are often excluded from these trials and few clinical interventions have been demonstrated to reduce fatal suicide risk[5,14]. There is, however, evidence that opioid agonist treatment (OAT) with methadone or buprenorphine is associated with a reduced risk of suicide among people with OUD[15]. Some form of OAT is available in 90 countries, however there is low coverage of access globally[16]. In Australia, OAT is administered within a framework including medical, social and psychological treatment and coverage is deemed high according to the WHO, with nearly 50,000 people accessing treatment in 2020[17].

The beneficial effects of OAT in reducing morbidity and mortality among people with OUD are well-documented [18,19]. More recently, a systematic review found that time in OAT was associated with more than a 50% risk reduction in suicide compared to time out of OAT[18]. Although the review was able to look at specific risk periods in and out of OAT for all-cause and overdose mortality, there were insufficient data to examine the risk of suicide during specific time periods of OAT exposure[20,21].

A recent UK study examining the impact of OAT on self-harm hospitalisations and suicide found the four weeks following treatment cessation were associated with an elevated risk for both outcomes, compared to more than four weeks on OAT[21]. The risk of self-harm hospitalisations was also elevated in the remainder of time off OAT; however, there was insufficient power to detect any effect of other OAT exposure periods on mortality due to the small number of suicide deaths. Hence, further work is needed to understand the risk of self-harm and suicide in relation to specific OAT exposure periods. Moreover, no study has yet examined the incidence of self-harm and suicide by the method used to self-harm, nor whether there are differential effects of OAT exposure across different self-harm methods.

This study aims to:

- **i.** Estimate the incidence of self-harm and suicide among people with OUD, overall, and by sex and method.
- **ii.** Compare rates of self-harm and suicide during different OAT exposure time periods to stable periods on OAT.

2. Methods

We used data from the Opioid Agonist Treatment Safety (OATS) Study[22]. Full details of the study, setting, data sources, linkage approach, and prescribing practices in New South Wales (NSW) have been reported previously[22]. The findings of this study are reported in line with the Reporting of studies Conducted using Observational Routinely collected Data (RECORD) guidelines[23].

2.1. Study design, setting, and participants

This was a retrospective cohort study using linked administrative data. The cohort comprised all people initiating or maintained on OAT between 1 August 2002 and 31 December

2017 in NSW, Australia, as recorded in the NSW Electronic Reporting and Recording of Controlled Drugs (ERRCD). A substantial proportion of people with a history of illicit opioid dependence in NSW enter OAT at some point in time[24,25], therefore closely approximating the OUD population.

Methadone and buprenorphine (including buprenorphine-naloxone) are available in public, private, and carceral settings in NSW for the management of OUD. Prescribers are required to submit an "Authority to Prescribe" form to the NSW Ministry of Health for people entering treatment and are required to inform the regulatory body when OAT medication (e.g., methadone or buprenorphine), dispense setting, or treatment enrolment changes.

2.2. Data linkage

Four databases were linked to individuals in the ERRCD by the Centre for Health Record Linkage (CHeReL) using probabilistic linkage methods. Records were matched to an individual's name, sex, date of birth, and state of residence. The Admitted Patients Data Collection (APDC) database includes all hospitalisations in NSW. Primary and secondary diagnoses were recorded using the International Statistical Classification of Diseases and Related Health Problems 10th edition (modified for use in Australia; ICD-10-AM).

Information on state-wide mental health treatment and incarceration were sourced from the Mental Health and Ambulatory Data Collection (MH-AMB) and the Re-offending database (ROD), respectively. Mortality and cause of death data were drawn from the National Death Index (NDI). Further detail on the data sources are profiled elsewhere[22].

2.3. Variables

2.3.1. Exposure—OAT status was defined by four distinct time-periods: 1–28 days on OAT, ≥29 days on OAT, 1–28 days after OAT cessation, and ≥29 days after OAT cessation[21]. Consistent with previous work[20,26–28], treatment episodes that recommenced within six days of an episode ceasing were considered a continuous episode, even if the OAT medication changed.

OAT exposure time commenced on 1 August 2002, or the date of a participant's first OAT episode. Participants exited the study on 31 December 2017, or four years after treatment cessation, or their date of death[29]. The four-year cut-off was informed by the data, it was observed that 90% of re-entry treatment episodes started within 50 months of the cessation of the last treatment episode. All OAT episodes, including first and repeat episodes, were included. Participants who moved out of NSW during the study period were unable to be followed up and were considered out of treatment unless their death was otherwise captured in the NDI.

2.3.2. Outcomes—Self-harm hospitalisations and suicide deaths were the outcomes of interest. Coding from the ICD-10-AM was used to sub-categorise self-harm and underlying cause of suicide death into overdose (opioid [T40.0-T40.4] and other substances [T40.5-T40.9]; X60-X66 and X68-X69), violent (X67, X70-X75, and X80-X82), cutting or piercing (X78), and other (e.g. self-harm by smoke or blunt object; X76-X77, X79, X82-X84) methods, and were adapted from previously defined categories[30] (Supplement; eTable 1).

2.3.3. Covariates and time-varying confounders—

Several demographic and clinical variables were examined; justifications for the inclusion and ICD-10-AM codes for each of the variables are provided in eTable 2 in the Supplement.

Demographic: Sex, Indigenous status, socio-economic disadvantage, and geographical remoteness were static variables. Participants who identified as Aboriginal or Torres Strait Islander in any dataset were considered Indigenous in the analyses. Postcode-level socio-economic disadvantage was categorised using the Socio-Economic Indexes for Areas (SEIFA) to postcode at last OAT enrolment. Geographical remoteness was determined based on the participant's latest postcode of residence (drawn from the ERRCD)[31]. Age, calendar years of observation, and past 12-month incarceration were time-varying variables.

Clinical: We derived five time-varying potential confounders to indicate receipt of drug-related or mental health treatment in the previous 12 months. Mental health treatment in an inpatient or outpatient setting that recorded anxiety or depression (without psychosis) or a severe mental disorder (i.e. schizophrenia, manic episode, bipolar affective disorder, or severe depressive episodes with psychotic symptoms) were derived. Hospital records in the previous 12 months for self-harm, overdose (unintentional), injecting-related injuries and diseases, or assault were also derived.

2.4. Data cleaning

The data cleaning process to derive the cohort is documented elsewhere[32]. A flowchart documenting participants excluded at each stage of the data cleaning process is in the Supplement (eFigure 1).

2.5. Data analysis

OAT treatment data for the cohort were transformed into discrete observation windows of defined exposure periods (see eFigure 2 for example). Within each observation period, the number of hospitalisations for self-harm and suicide were counted. The frequency of events and crude incidence rates with 95%CI for hospitalisations for any self-harm and suicide deaths were calculated and stratified by method of self-harm. Crude incidence rates of hospitalisations were calculated for each OAT exposure period, demographic characteristics, and recent hospitalisations per 1000 person-years (PY).

Poisson regression models with generalised estimating equations (GEE) were used to calculate the adjusted incidence rate ratios (ARR) of self-harm and suicide per 1000 PY with 95%CI. In addressing the second aim of the study, all variables were included as potential confounders in the adjusted model to compare the impact of different OAT exposure periods on self-harm hospitalisations and suicide mortality. GEE models adjust for multiple observations per individual by using a working correlation structure, providing robust parameter estimates. Due to a small number of events ($n<5$), the rates of suicide in the first 28 days of treatment were unable to be reported.

Tukey–Kramer adjustment was used to hold the experiment-wise error rate at 5% when comparing incidence rates by exposure level. The Score statistic was used to assess the

importance of terms in the models. Analyses were conducted in SAS V9.4 (SAS Institute Inc. Cary, NC, USA).

2.6. Sensitivity analyses

As intent of some self-harm and suicide incidents may be unclear and therefore classified as undetermined[33] (ICD-10-AM codes Y10-Y34; see eTable 1 in the Supplement), sensitivity analyses were conducted to include these events in the GEE models. Second, to account for deaths that occurred within six days of the last treatment episode (during which it is assumed that there may be some residual opioid exposure), an additional sensitivity analysis was conducted to extend the OAT exposure period by six days post-cessation in the GEE models. Finally, we tested whether adjusting the out of OAT exposure period from a maximum of four years to a cut-off of 12 months would change the interpretation of the results.

2.7. Ethics

Approval for this study was obtained from the NSW Population & Health Services Research Ethics Committee (2018/HRE0205), the NSW Corrective Services Ethics Committee and the Aboriginal Health and Medical Research Council Ethics Committee (1400/18).

3. Results

There were 45,664 individuals (67.7% male; median age 32 [IQR=26–39] years) in the cohort (Table 1). One in five were Indigenous (21.0%) and one in four were living in regional or remote communities (26.5%), and 23.6% of individuals ranked as the most socioeconomically disadvantaged.

There were 4,148 people (9.1%) with a self-harm hospitalisation (Table 1 and Supplement eTable 3), equating to an overall crude rate of 19.2 per 1,000 PY (95%CI=18.8–19.7); the rate was higher among women than men (risk ratio [RR]=1.4; 95%CI=1.3–1.5; Table 2). The most common method of self-harm was non-opioid overdose (52.7% of all individuals with a self-harm hospitalisation), followed by opioid overdose (39.3%) and cutting or piercing (22.5%).

Of the 4,491 deaths in the cohort, there were 556 suicide deaths (12.4%). The crude incidence rate of suicide was 1.0 per 1,000 PY (95%CI=0.9–1.1); this rate was lower among women than men (RR=0.6; 95%CI=0.5–0.8; Table 2). There was no gender disparity in rates of suicide by opioid overdose. Most suicide deaths did not involve opioids: they were predominantly classified as violent (66.0%), followed by non-opioid overdose (23.9%; eTable 3).

Table 3 presents the rates of self-harm hospitalisations by OAT exposure period; the crude incidence rate was 50.8 (95%CI=45.5–56.7) per 1000 PY in the first 28 days on OAT, 14.8 (95%CI=14.3–5.3) per 1000 PY in the remaining time on OAT, 58.2 (95%CI=52.7–64.3) per 1000 PY in the first 28 days off OAT, and 23.8 (95%CI=23.0–24.6) per 1000 PY in the remainder of time off OAT. This corresponded to similarly elevated risks of self-harm hospitalisation in the 28 days after OAT initiation and cessation (ARR=2.2; 95%CI=1.9–2.6; and ARR=2.7; 95%CI=2.3–3.2, respectively) compared to the remainder of time on OAT

(Figure 1). The remainder of time off OAT was associated with a 46% increased risk of a self-harm hospitalisation (ARR 1.5; 95%CI=1.3–1.7). The impact of OAT exposure was similar across self-harm methods (eTable 4).

For the remainder of time on OAT (≥29 days), the crude incidence rate for suicide was 0.5 (95%CI=0.4–0.6) per 1000 PY, 9.6 (95%CI=7.5–12.3) per 1000 PY in the 28 days following OAT cessation, and 1.4 (95%CI=1.2–1.6) for the remainder of time off treatment (Table 4). Compared to ≥29 days on OAT, there was a markedly elevated risk of suicide mortality in the month following OAT cessation (ARR=17.4; 95%CI=11.7–25.8) and a 2.8-fold increased risk in the remainder of time off OAT (95%CI=2.1–3.7). There was no evidence of an increased risk of suicide mortality in the first 28 days on OAT compared to the rest of time on OAT. The increased risk of suicide in the 28 days following OAT cessation appeared to be higher for overdose (ARR=21.6 [95%CI=11.4–40.8]) and lower for violent methods (ARR=14.2 [95%CI=8.6–23.5]), compared to suicide overall; however, confidence intervals overlapped (Figure 2 and eTable 4).

3.1. Sensitivity analyses

There were 2,296 people who presented to hospital with self-harm of undetermined intent, and an additional 199 deaths of undetermined intent (eTable 5). Analyses that included these events did not generate any meaningful differences in the magnitude of risk for the first 28 days and ≥29 days off OAT, compared to ≥29 days in OAT (eTable 6). The crude incidence rate of suicide in the first 28 days of OAT was marginally higher than ≥29 days on OAT; however, the confidence intervals of the ARR point estimate (1.4) ranged from 0.5 to 3.8 and so no increased risk was interpreted. After extending the OAT exposure period to include the six days following treatment cessation, the number of suicides in the first 28 days of OAT increased to 6 and the crude incidence rate was estimated to be 0.9 (95%CI=0.4–2.1; eTable 7). In the first 28 days out of OAT, the crude incidence rate of suicide reduced to 3.9 (95%CI=2.7–5.7) and the ARR for this period reduced to 5.5 (95%CI=3.2–9.5) compared to ≥29 days in OAT (eTable 7). Finally, after censoring the ≥29 days out of OAT exposure period to 12 months, we found that the ARR reduced when compared to ≥29 days in OAT as we would expect; however, the overall interpretation did not change (eTable 8).

4 Discussion

4.1. Main findings

We observed that retention on OAT for ≥29 days had a protective effect against self-harm hospitalisations and suicide in our cohort. The period immediately following OAT cessation was associated with a substantially elevated risk of self-harm and suicide; compared to stable periods on OAT, the risk of suicide was 17 times higher in the month after leaving OAT, and more than 21 times higher for suicide by overdose. The risk of self-harm was two-three times higher during periods of OAT entry and exit compared to stable periods on OAT. Conversely, there was no increased risk of suicide in the first 28 days on OAT, but there was almost three times the risk for continuous periods off OAT.

Opioid poisoning is the leading cause of premature death among people with OUD in Australia and globally[9,34]. Despite likely access to large quantities of opioids among this cohort, opioids were implicated in a small minority of suicides. Consistent with patterns in the general population[35], men were more likely to die by suicide and violent methods were the most common method used. Opioid overdose was implicated in only 9.6% of suicide deaths and 28% of self-harm hospitalisations. In contrast, overdoses on drugs other than opioids were recorded for 23.9% of deaths and over half of self-harm hospitalisations. This may reflect the significant challenge of discerning intent for opioid overdoses among people with OUD in clinical settings, particularly when suicidal ideation is common and can fluctuate within individuals[36–38]. However, this finding is consistent with self-report data from people with OUD, who rarely report using opioids in suicide attempts[39,40].

4.2. Implications

Our findings emphasise the importance of OAT provision for people with OUD and support previous findings that longer-term OAT reduces a broad spectrum of mental, physical, and social health harms among this population[18,19, 41]. Engagement in OAT has important benefits in improving both physical and psychological quality of life, mood stabilisation, and facilitating access to structured physical and mental health service programs[42–46].

Consistent with previous evidence[18,20,21], our findings highlight important risk periods for self-harm and suicidal behaviour. The immediate period post-treatment cessation represents a significant risk period for people receiving OAT. The risk of overdose mortality is widely recognised[18], but there is a need for greater attention on the risks of self-harm and suicide in this period. Further work developing interventions to prevent self-harm and suicide in people immediately following OAT cessation is warranted.

There was also an elevated risk for non-fatal self-harm in the period immediately following OAT initiation. On entry to OAT, individuals often require a period of treatment stabilisation, and will receive a full clinical assessment, including a clinical review of their mental health[47]. The factors underlying the increased risk of self-harm in the month following treatment initiation are not entirely clear. Health and social factors (e.g., housing instability, interpersonal violence, untreated physical and mental health comorbidities, etc.), which may be prevalent among people entering drug treatment[48], can contribute to acute psychological crises[49]. As such, therapeutic suicide risk assessments and management, as well as safety planning, may be valuable during this period[50].

4.3. Limitations

Our study is likely to have only captured more severe cases of non-fatal self-harm requiring urgent treatment (i.e., hospitalisation). Further work is needed to examine whether OAT engagement is associated with a reduced risk of self-harm in community settings, and whether similar risk periods for community-level self-harm exist.

Miscoding intentional suicide and self-harm as ‘undetermined intent’ may represent an underestimate in our results. Including these events in the sensitivity analyses did not change the interpretation of our findings; however, the ARR of suicide in the first month of OAT increased to 1.4 (95%CI=0.5–3.8). Although the confidence intervals are not

considered statistically significant, it is impossible to ascertain which result most accurately reflects the risk of suicide. Further, miscoding may extend to diagnoses classified as accidental overdoses. There may be fear of stigma or involuntary detainment for people who are hospitalised with self-harm[51]; however, there is also reportedly ambivalence towards mortality for people who have a history of overdose[52].

There may be some misclassification of the exposure groups due to administration errors that cannot be detected in the data available; however, this is likely non-differential and therefore may dilute the detected effects of OAT exposure periods. It was also not possible to stratify by planned and unplanned treatment cessation as the data were deemed unreliable. It is, therefore, not possible to determine the differential risk for those who planned to leave treatment and might have a lower risk of suicide and self-harm[53].

Finally, a limitation of the design of this analysis when comparing treatment exposure periods and suicide is that a client is required to survive their OAT episode in order to reach the out of OAT period. By definition, a client cannot be in a state of “out of treatment” unless they have survived “in treatment”. This cohort is based on people who have had OAT, there is not a control group to compare those with opioid dependence who have not been in treatment with those in treatment. Therefore, when comparing out of treatment in this study it is conditional on them surviving their last treatment (and previous episodes).

5. Conclusion

OAT appears to have a protective effect against self-harm and suicide among people with OUD. Improving retention on OAT and reducing barriers to treatment access are critical components to minimise morbidity and mortality due to self-harm. Further work developing interventions to prevent self-harm and suicide immediately following treatment cessation is warranted.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights: Incidence of suicide and self-harm among people with opioid use disorder and the impact of opioid agonist treatment: a retrospective data linkage study

1. Opioid agonist treatment (OAT) is associated with a reduction in self-harm and suicide.
2. The month following OAT cessation is associated with an increase in both outcomes.
3. Self-harm increased during OAT initiation, compared to stable periods on OAT.
4. Opioid overdose was implicated in less than 10% of suicide deaths.
5. Non-opioid overdoses were recorded for almost a quarter of suicides.

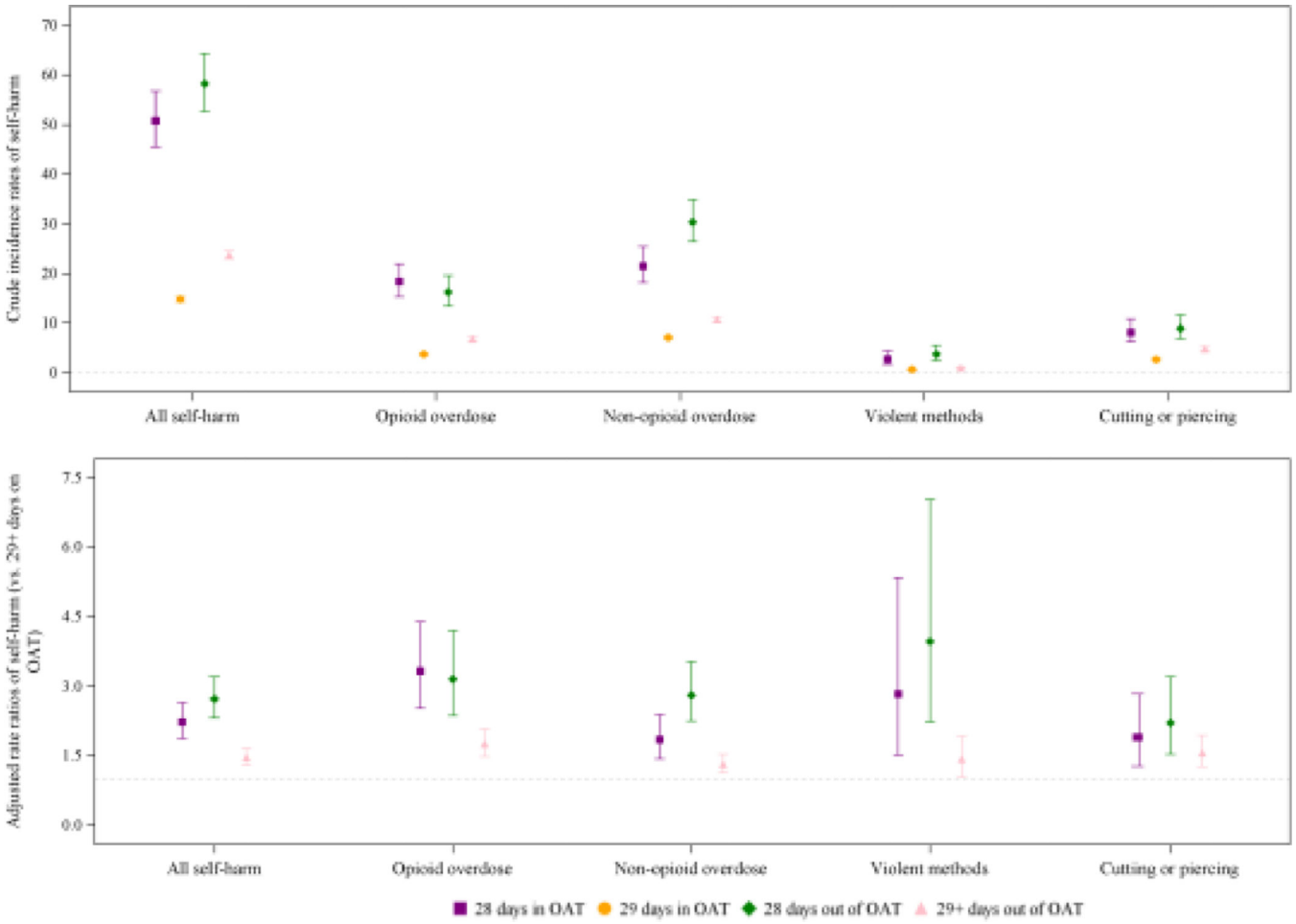

Figure 1. Crude incidence rates and adjusted rate ratios of self-harm hospitalisations by opioid agonist treatment (OAT) exposure and self-harm method.

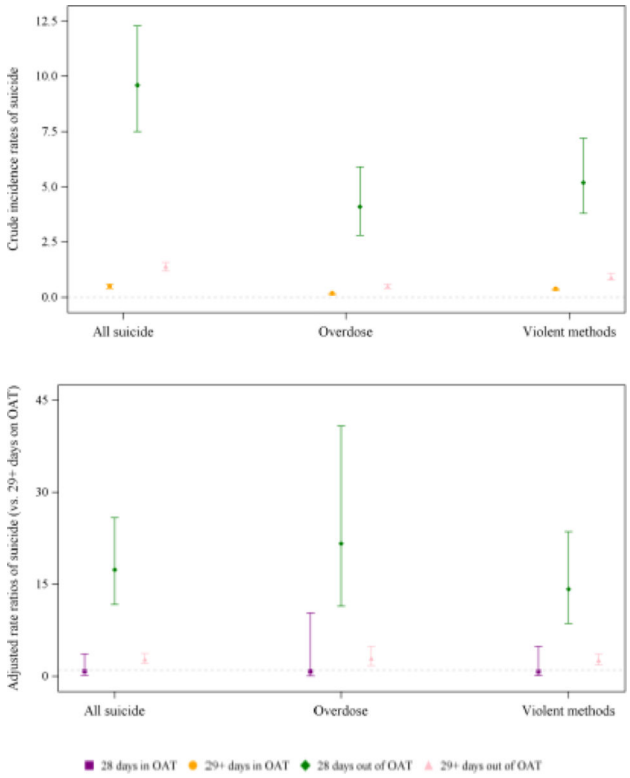

Figure 2. Crude incidence rates and adjusted rate ratios of suicide overall, by method, and by opioid agonist treatment (OAT) exposure.

Table 1.

Cohort demographic information

Cohort characteristics		N	%
Sex [a]	*Female*	14,770	32.3%
	Male	30,892	67.7%
Age at cohort entry	*<=24 years*	9,471	20.7%
	25–34 years	18,318	40.1%
	35–44 years	12,945	28.3%
	>=45 years	4,930	10.8%
Calendar year of entry	*2002–2005*	26,251	57.5%
	2006–2009	6,755	14.8%
	2010–2013	6,491	14.2%
	2014–2017	6,167	13.5%
Indigenous		9,580	21.0%
Geographical remoteness [b]	*Major Cities of NSW*	33,345	73.0%
	Regional/Remote NSW	12,084	26.5%
Socioeconomic status [c]	*Most Disadvantaged*	10,769	23.6%
	2nd Quintile	8,536	18.7%
	3rd Quintile	11,954	26.2%
	4th Quintile	8,358	18.3%
	Least Disadvantaged	5,556	12.2%
Hospitalisation	*Self-harm*	4,148	9.1%
Deaths	*Total*	4,491	9.8%
	Suicide	556	1.2%

[a] Sex information was unavailable for 2 participants.

[b] Geographical information was unavailable for 235 participants.

[c] Socioeconomic status information was unavailable for 491 participants.

Table 2.

Crude incidence rates and risk ratios of intentional self-harm hospitalisations and suicide by method and sex.

	Crude incidence rate per 1000 person-years (95% CI[a])			Risk ratio
Outcome	**All individuals**	**Females**	**Males**	**(ref[b]=Males)**
Self-harm hospitalisations	**19.2 (18.8–19.7)**	**23.7 (22.9–24.6)**	**17.0 (16.5–17.5)**	**1.40 (1.34–1.46)**
Overdose	14.4 (14.1–14.8)	18.1 (17.4–18.8)	12.6 (12.2–13.0)	1.44 (1.37–1.51)
Opioid overdose	*5.3 (5.1–5.6)*	*6.2 (5.8–6.6)*	*4.9 (4.7–5.2)*	*1.25 (1.15–1.37)*
Non-opioid overdose	*9.1 (8.8–9.4)*	*11.9 (11.3–12.5)*	*7.6 (7.3–8.0)*	*1.56 (1.46–1.66)*
Violent self-harm method	0.9 (0.8–1.0)	0.9 (0.8–1.1)	0.9 (0.8–1.0)	1.01 (0.82–1.26)
Cutting or piercing	3.7 (3.5–3.9)	4.7 (4.3–5.1)	3.0 (3.0–3.4)	1.49 (1.34–1.64)
Other method	0.9 (0.8–1.0)	0.9 (0.7–1.0)	0.9 (0.8–1.0)	0.97 (0.78–1.21)
Suicide	**1.0 (0.9–1.1)**	**0.7 (0.5–0.8)**	**1.1 (1.0–1.2)**	**0.61 (0.48–0.77)**
Overdose	0.3 (0.3–0.4)	0.3 (0.2–0.4)	0.4 (0.3–0.4)	0.83 (0.58–1.19)
Opioid overdose	*0.1 (0.1–0.1)*	*0.1 (0.0–0.1)*	*0.1 (0.1–0.2)*	*0.66 (0.33–1.30)*
Non-opioid overdose	*0.2 (0.2–0.3)*	*0.2 (0.2–0.3)*	*0.2 (0.2–0.3)*	*0.91 (0.59–1.40)*
Violent suicide method	0.6 (0.6–0.7)	0.4 (0.3–0.5)	0.7 (0.7–0.9)	0.56 (0.42–0.75)

[a] CI=Confidence intervals

[b] Referent category

Table 3.

Crude incidence rates per 1000 person-years and crude and adjusted rate ratios of self-harm hospitalisations by opioid agonist treatment (OAT) exposure, demographic, and clinical information.

	Person-years	Self-harm hospitalisations n	Crude incidence rate (95%CI)	Crude rate ratio (95%CI)	Adjusted rate ratio (95%CI)	Score (p-value)
Exposure						
In OAT (1–28 days)			50.8 (45.5–56.7)	3.44 (3.07–3.85)	2.23 (1.87–2.65)	183.27 (<.001)
Out of OAT (1–28 days)			58.2 (52.7–64.3)	3.94 (3.55–4.37)	2.73 (2.32–3.21)	
In OAT (29+ days)			14.8 (14.3–15.3)	Ref.	Ref.	
Out of OAT (29+ days)			23.8 (23.0–24.6)	1.61 (1.54–1.68)	1.46 (1.29–1.66)	
Calendar year						
2014–2017	111399	2342	21.0 (20.1–21.8)	1.18 (1.10–1.26)	1.21 (1.10–1.32)	16.92 (<.001)
2010–2013	112339	1956	17.4 (16.6–18.2)	0.98 (0.91–1.04)	1.03 (0.94–1.13)	
2006–2009	111117	2264	20.3 (19.5–21.2)	1.14 (1.07–1.22)	1.09 (0.97–1.23)	
2002–2005	84948	1519	17.8 (16.9–18.7)	Ref.	Ref.	
Sex						
Female	138239	3288	23.7 (22.9–24.6)	1.40 (1.34–1.46)	1.23 (1.10–1.37)	5.84 (0.016)
Male	281563	4793	17.0 (16.5–17.5)	Ref.	Ref.	
Age						
<=24 years	27542	784	28.4 (26.5–30.5)	2.06 (1.89–2.24)	1.74 (1.50–2.01)	16.66 (<.001)
25–34 years	132594	3063	23.0 (22.2–23.9)	1.67 (1.57–1.78)	1.49 (1.32–1.69)	
35–44 years	144993	2652	18.2 (17.5–18.9)	1.32 (1.24–1.41)	1.21 (1.11–1.33)	
>=45 years	114672	1582	13.8 (13.1–14.5)	Ref.	Ref.	
Indigenous						
Yes	87097	2569	29.4 (28.3–30.6)	1.78 (1.70–1.87)	1.53 (1.34–1.74)	8.77 (<.001)
No	332704	5512	16.5 (16.1–17.0)	Ref.	Ref.	
Socio-economic status						
Least Disadvantaged	50661	1044	20.5 (19.3–21.8)	1.27 (1.18–1.38)	1.18 (1.04–1.33)	5.78 (0.216)
4th Quintile	78991	1646	20.7 (19.8–21.8)	1.29 (1.20–1.38)	1.15 (1.03–1.29)	
3rd Quintile	109540	2214	20.2 (19.4–21.1)	1.25 (1.18–1.34)	1.14 (1.03–1.27)	
2nd Quintile	79899	1551	19.3 (18.4–20.3)	1.20 (1.12–1.29)	1.14 (0.93–1.40)	
Most Disadvantaged	100711	1626	16.1 (15.3–16.9)	Ref.	Ref.	
Geographical remoteness						
Major Cities of NSW	316885	6267	19.7 (19.2–20.2)	1.12 (1.06–1.18)	1.12 (1.01–1.24)	2.88 (0.089)
Regional/Rem ote NSW	102916	1814	17.6 (16.8–18.4)	Ref.	Ref.	

	Person-years	Self-harm hospitalisations n	Crude incidence rate (95%CI)	Crude rate ratio (95%CI)	Adjusted rate ratio (95%CI)	Score (p-value)
Recent[a] incarceration						
Yes	52015	1886	36.2 (34.6–37.8)	2.15 (2.04–2.27)	1.61 (1.45–1.78)	16.67 (<.001)
No	367787	6195	16.8 (16.4–17.2)	Ref.	Ref.	
Received treatment in the past 12 months for...						
Self-harm						
Yes	5960	2654	444.5 (427.9–461.8)	34.02 (32.47–35.64)	19.01 (16.69–21.67)	111.06 (<.001)
No	413841	5427	13.1 (12.7–13.4)	Ref.	Ref.	
Severe mental health						
Yes	23349	2846	121.2 (116.8–125.8)	9.20 (8.78–9.63)	4.62 (4.15–5.15)	108.35 (<.001)
No	396453	5235	13.2 (12.8–13.5)	Ref.	Ref.	
Depression/anxiety						
Yes	16281	4156	254.7 (247.1–262.6)	26.29 (25.17–27.46)	17.13 (15.88–18.46)	694.23 (<.001)
No	403521	3925	9.7 (9.4–10.0)	Ref.	Ref.	
Overdose						
Yes	7557	2295	303.0 (290.8–315.6)	21.65 (20.63–22.73)	9.08 (7.85–10.51)	36.62 (<.001)
No	412244	5786	14.0 (13.6–14.4)	Ref.	Ref.	
Injecting-related disease						
Yes	12662	1052	83.1 (78.2–88.2)	4.83 (4.52–5.15)	2.72 (2.28–3.25)	19.72 (<.001)
No	407140	7029	17.2 (16.8–17.6)	Ref.	Ref.	
Assault						
Yes	4803	523	108.7 (99.7–118.4)	5.98 (5.48–6.54)	2.71 (2.37–3.09)	22.02 (<.001)
No	414999	7558	18.2 (17.8–18.6)	Ref.	Ref.	

[a]Past 12 months.

CI: Confidence intervals

Table 4.

Crude incidence rates per 1000 person-years and crude and adjusted rate ratios of suicide mortality by opioid agonist treatment (OAT) exposure, demographic, and clinical information.

	Person-years	Suicides n	Crude incidence rate (95%CI)	Crude rate ratio (95%CI)	Adjusted rate ratio (95%CI)	Score (p-value)
Exposure						
In OAT (1–28 days)		-	0.5 (0.2–1.5)	0.93 (0.30–2.92)	0.82 (0.18–3.67)	119.1 (<.001)
Out of OAT (1–28 days)		-	9.6 (7.5–12.3)	18.83 (13.97–25.38)	17.39 (11.69–25.85)	
In OAT (29+ days)		-	0.5 (0.4–0.6)	Ref.	Ref.	
Out of OAT (29+ days)		-	1.4 (1.2–1.6)	2.71 (2.18–3.38)	2.81 (2.10–3.75)	
Calendar year						
2014–2017						8.3
	111399	131	1.2 (1.0–1.4)	1.49 (1.11–2.00)	1.73 (1.28–2.34)	(0.041)
2010–2013	112339	111	1.0 (0.8–1.2)	1.25 (0.93–1.70)	1.43 (1.05–1.94)	
2006–2009	111117	96	0.9 (0.7–1.1)	1.10 (0.80–1.50)	1.15 (0.83–1.58)	
2002–2005	84948	67	0.8 (0.6–1.0)	Ref.	Ref.	
Sex						
Female	138239	93	0.7 (0.5–0.8)	0.61 (0.48–0.77)	0.58 (0.46–0.74)	20.7 (<.001)
Male	281563	312	1.1 (1.0–1.2)	Ref.	Ref.	
Age						
<=24 years	27542	21	0.8 (0.5–1.2)	0.75 (0.47–1.20)	0.59 (0.37–0.95)	7.0 (0.072)
25–34 years	132594	124	0.9 (0.8–1.1)	0.92 (0.72–1.19)	0.80 (0.62–1.05)	
35–44 years	144993	144	1.0 (0.8–1.2)	0.98 (0.77–1.25)	0.90 (0.70–1.15)	
>=45 years	114672	116	1.0 (0.8–1.2)	Ref.	Ref.	
Indigenous						
Yes	87097	68	0.8 (0.6–1.0)	0.77 (0.59–1.00)	0.70 (0.54–0.92)	8.5(0.003)
No	332704	337	1.0 (0.9–1.1)	Ref.	Ref.	
Socio-economic status						
Least Disadvantaged	50661	58	1.1 (0.9–1.5)	1.52 (1.08–2.14)	1.52 (1.08–2.13)	5.5(0.243)
2nd Quintile	79899	76	1.0 (0.8–1.2)	1.26 (0.92–1.73)	1.26 (0.90–1.75)	
3rd Quintile	109540	110	1.0 (0.8–1.2)	1.33 (0.99–1.78)	1.31 (0.97–1.75)	
4th Quintile	78991	85	1.1 (0.9–1.3)	1.43 (1.05–1.94)	1.40 (1.03–1.92)	
Most Disadvantaged	100711	76	0.8 (0.6–0.9)	Ref.	Ref.	
Geographical remoteness						
Major Cities of NSW	316885	319	1.0 (0.9–1.1)	1.20 (0.95–1.53)	1.21 (0.95–1.54)	0.7 (0.411)
Regional/Remote	102916	86	0.8 (0.7–1.0)	Ref.	Ref.	
NSW						
Recent[a] incarceration						
Yes	52015	67	1.3 (1.0–1.6)	1.40 (1.08–1.82)	1.13 (0.87–1.49)	0.3 (0.584)

	Person-years	Suicides n	Crude incidence rate (95%CI)	Crude rate ratio (95%CI)	Adjusted rate ratio (95%CI)	Score (p-value)
No	367787	338	0.9 (0.8–1.0)	Ref.	Ref.	
Received treatment in the past 12 months for...						
Self-harm						
Yes	5960	42	7.0 (5.2–9.5)	8.03 (5.84–11.06)	5.19 (3.56–7.57)	1.3 (0.260)
No	413841	363	0.9 (0.8–1.0)	Ref.	Ref.	
Severe mental health						
Yes	23349	66	2.8 (2.2–3.6)	3.31 (2.54–4.30)	2.45 (1.83–3.28)	8.2 (0.004)
No	396453	339	0.9 (0.8–1.0)	Ref.	Ref.	
Depression/anxiety						
Yes	16281	86	5.3 (4.3–6.5)	6.68 (5.27–8.48)	5.39 (4.09–7.09)	34.4 (<.001)
No	403521	319	0.8 (0.7–0.9)	Ref.	Ref.	
Overdose						
Yes	7557	49	6.5 (4.9–8.6)	7.51 (5.57–10.12)	5.12 (3.57–7.36)	8.3 (0.004)
No	412244	356	0.9 (0.8–1.0)	Ref.	Ref.	
Injecting-related disease						
Yes	12662	31	2.4 (1.7–3.5)	2.66 (1.85–3.84)	2.08 (1.43–3.03)	3.9 (0.049)
No	407140	374	0.9 (0.8–1.0)	Ref.	Ref.	
Assault						
Yes	4803	9	1.9 (1.0–3.6)	1.96 (1.01–3.80)	1.37 (0.69–2.69)	0.0 (0.974)
No	414999	396	1.0 (0.9–1.1)	Ref.	Ref.	

[a]Past 12 months.

CI: Confidence intervals