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Opioid substitution treatment in prison and post-release: Effects on criminal recidivism and mortality

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Opioid substitution treatment in prison and post-release: Effects on criminal recidivism and mortality

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ACRONYMS

ABS	Australian Bureau of Statistics
BOCSAR	Bureau of Crime Statistics and Research
CHeReL	Centre for Health Record Linkage
CMR	crude mortality rate
HIV	human immunodeficiency virus
ICD-10	International Classification of Diseases, 10th edition
IDRS	Illicit Drug Reporting System
MIN	Master Index Number
MLK	Master Linkage Key
OIMS	Offender Integrated Management System
OST	opioid substitution treatment
PHDAS	Pharmaceutical Drugs of Addiction System
PWP-GT	Prentice-Williams-Peterson gap-time (statistical model)
RCT	randomised controlled trial
ROD	reoffending database
SMR	standardised mortality ratio
WHO	World Health Organization

EXECUTIVE SUMMARY

Opioid substitution treatment (OST) is an effective treatment for heroin dependence that is increasingly available in correctional settings globally; in 2009, at least 29 countries offered OST in at least one correctional institution (Larney & Dolan 2009). In Australia, OST is available in prisons in all jurisdictions, albeit with limitations on treatment access in some jurisdictions (AIHW 2010a). One rationale that is often given in support of prison OST is that it reduces post-release criminality; however, the evidence for this proposition is equivocal. Another rationale for prison OST is that it will reduce the risk of death by drug overdose in the post-release period. The aims of the studies presented in this report are to assess the effects of prison OST on re-incarceration, criminal convictions and mortality.

Methodology

Participants were a cohort of 375 male heroin users recruited in NSW prisons in 1996–97 (Dolan et al., 2003). The cohort was originally recruited to take part in a randomised controlled trial of prison OST. Data on participation in OST, incarceration, convictions and mortality for the cohort were obtained from administrative datasets held by state government agencies. Data from the administrative datasets were linked to the cohort baseline data for the time period 1 June 1997 to 31 December 2006. Recurrent event survival analysis was used to identify relationships between prison OST participation and criminal outcomes (re-incarceration and post-release criminal convictions). Mortality rate ratios were used to explore risk of death during OST while in prison and in the first 28 days after leaving prison.

Key findings

Effects on re-incarceration

Three hundred and seventy participants were released from prison during the follow-up, of whom 332 (90%) were re-incarcerated. Participants were re-incarcerated much faster and more frequently than the general NSW prison population, with 84 percent of participants re-incarcerated with two years of release, compared with 43–45 percent of all prisoners (SCRGSP 2010). Fifty-eight percent of all OST episodes were commenced while the participant was in prison. Being in OST at release from prison did not affect re-incarceration; however, when retention in treatment post-release was taken into account, there was a significant effect, with risk of re-incarceration reduced by an average of 20 percent while participants remained in treatment.

Effects on criminal convictions

Data linkage identified 325 participants (88%) as having been convicted of an offence during the follow-up period. Participants who were reconvicted were significantly younger than those who were not. The median number of convictions per participant was 15 (range 1–79) and theft was the most common offence type for which participants were convicted. No effects of prison OST on risk of criminal convictions were identified; however, sensitivity analyses suggested that the use of convictions (as opposed to arrests or criminal charges) may have introduced bias into the analysis.

Effects on mortality

Twenty-eight participants (7.5%) died during the follow-up period; half of these were classified as accidental drug-related deaths. Participants in this cohort died at six times the rate of their age- and sex-matched peers in New South Wales. Mortality was significantly reduced while participants were in treatment or in prison. There were some indications that the four week period post-release was a time of increased risk of mortality, particularly from drug overdose, but the comparison to mortality during all other time at liberty did not reach statistical significance.

Implications of findings

Restrictions on prison OST prevent treatment entry at a time when demand for treatment is high

Over half of the treatment episodes in this cohort were commenced in prison. Treatment exposure in prison reduces needle and syringe sharing (Dolan et al. 2003; Heimer et al. 2006; Larney 2010) and according to the data presented in this report, inmates who remain in treatment post-release have a significantly reduced risk of re-incarceration.

Three Australian jurisdictions—Queensland, Tasmania and the Northern Territory—do not permit inmates to commence OST in prison (AIHW 2010a). Restrictions such as these prevent treatment access at a time when demand for treatment is high and the potential benefits of treatment are significant.

OST in prison and post-release can potentially reduce the size of the prisoner population

Modelling studies suggest that even minor reductions in re-incarceration among groups of offenders with high re-incarceration rates can have significant impacts on the size of the overall prisoner population (Weatherburn et al. 2009); therefore, assisting released inmates to access OST in the community will not only reduce their personal risk of re-incarceration, but will also benefit the correctional system as a whole. Post-release participation in OST and positive treatment outcomes are maximised when treatment is commenced while in prison (Kinlock et al. 2007; Magura et al. 1993; Tomasino et al. 2001); hence, the maximum benefits from OST can be obtained by providing treatment while in prison, and facilitating a smooth transition to a post-release treatment provider.

OST alone may not affect reoffending

Effects of prison OST on offending were not identified, but there was some evidence that use of convictions as an outcome measure produced overly conservative results. It is also important to consider that OST in prison addresses only one of the risk factors for reoffending, namely, substance use (Andrews, Bonta & Wormith 2006). Future research should examine the extent to which reoffending is reduced by a combination of OST and therapeutic programs that address non-drug criminogenic risks/needs, such as pro-criminal attitudes and behaviours.

OST protects against death, and is likely to reduce the risk of post-release drug overdose

A sample size in the tens of thousands would be required to unequivocally state that OST in prison reduces the risk of death in the post-release period (Bird 2009). In the absence of data specific to this issue, the general protective effects of OST in relation to mortality (Degenhardt et al. 2009) can reasonably be assumed to apply to post-release inmates. Correctional authorities may carefully consider the role that prison OST may be able to play in reducing the high risk of drug overdose death that is seen in the period following release from prison.

1. INTRODUCTION

Heroin dependence is associated with significant health and social harms. Among the most prominent of social harms is criminal offending by heroin users. Purchase and use of heroin are, in themselves, offences in most countries. Heroin users also frequently engage in income-generating crimes such as theft and drug-dealing (DeBeck et al. 2007). Among samples of heroin users entering treatment, a majority report recent criminal activity (Davstad et al. 2009; Loebmann & Verthein 2008; van der Zanden et al. 2007). In Australia, 55 percent of a sample entering treatment for heroin dependence in 2001–02 reported offending in the past month, with level of heroin use positively correlated with level of criminal activity. One-fifth of this sample reported criminal activity to be their main source of income (Ross et al. 2005).

As a consequence of high levels of criminal activity, many heroin users experience periods of incarceration. In the United States, it has been conservatively estimated that up to one-third of all heroin dependent individuals pass through a correctional facility each year (Boutwell et al. 2007), while in the United Kingdom, around one-third of a cohort of treatment-seeking heroin users were incarcerated during a five year period (Oliver et al. 2010). Cross-sectional data show high levels of self-reported prior incarceration among Australian heroin users, with prevalence ranging from 41 percent (Ross et al. 2005) to 52 percent (personal communication N Sindicich national co-ordinator, Illicit Drug Reporting System, 2010). Unsurprisingly, longer duration of heroin use has been associated with increased likelihood of incarceration, with each additional year of use translating to an 11 percent increase in risk of having ever been in prison; however, risk of recent incarceration appears to decline with age (Darke et al. 2009).

Incarceration is itself associated with significant harms. In addition to general social harms such as social dislocation and reduced employability (Graffam, Shinkfield & Hardcastle 2008; Halsey 2007), injecting drug use while in prison is associated with transmission of human immunodeficiency virus (HIV) and the hepatitis C virus (Dolan et al. 2010; Dolan & Wodak 1999). Following release, inmates are at a greatly increased risk of death, particularly from drug overdose (Farrell & Marsden 2007; Kariminia et al. 2007c). Despite these significant concerns, incarceration can also be seen as an opportunity to offer treatment for heroin dependence in an effort to obviate further harms while in prison and post-release (Boutwell et al. 2007). This report presents a series of studies that examine the role of prison-based opioid substitution treatment in reducing post-release criminality and mortality.

Opioid substitution treatment

OST is a thoroughly evaluated and well-established treatment for heroin dependence that involves long-term, regular consumption of a long-acting opioid agonist[1] medication. OST was first described in the literature in the 1960s by two physicians, Dole and Nyswander. They reasoned that a heroin dependent person is unable to cease heroin use is because withdrawal from the drug causes physiological and psychological distress; in their efforts to avoid withdrawal, the individual's life is dominated by drug seeking and consumption. Provision of long-acting opioid medications at high or 'blockade' doses would relieve withdrawal symptoms and cravings, thereby reducing the need for heroin and activities associated with its procurement (Dole & Nyswander 1967, 1965).

Methadone, a synthetic opioid with effects lasting 24–36 hours, was the medication first used for substitution treatment. Other opioid agonists, particularly buprenorphine, have also been investigated for their potential in the treatment of heroin dependence (Anglin et al. 2007; Ling et al. 2010). Diamorphine, or pharmaceutical heroin, is available in a limited number of countries as a second-line substitution treatment for individuals who do not respond to treatment with methadone or buprenorphine (Lintzeris 2009). In the absence of a universally accepted term that encompasses these various medications, this report uses the term 'opioid substitution

1 An agonist is a chemical that binds to a receptor cell in the brain, activating a response.

treatment' to refer to all treatments for heroin dependence that follow the Dole and Nyswander model of provision of an opioid agonist over an extended period.

In the first clinical trial of opioid substitution treatment, long-term heroin users were provided with a daily dose of methadone and participants showed increased employment and decreased criminal behaviour (Dole & Nyswander 1965; Dole, Nyswander & Warner 1968). In the intervening decades, there have been hundreds of studies of the various forms of OST, with the evidence overwhelmingly supporting the effectiveness of this treatment approach (Ward, Hall & Mattick 2009). OST is superior to non-pharmacological treatments in retaining individuals in treatment (Mattick et al. 2009, 2008). It is associated with significant reductions in heroin use (Dolan et al. 2003; Fudala et al. 2003; Gruber et al. 2008; Kakko et al. 2003; Kinlock et al. 2009; Ling et al. 1998) and reduced injecting risk behaviours while in OST translates to a reduced rate of HIV seroconversion among heroin users in OST (Gowing et al. 2008).

Rationales for opioid substitution treatment in prison

Four main arguments have been advanced supporting the provision of OST in prison, encompassing human rights, public health and crime-reduction rationales.

Equivalence of care

Under multiple international covenants and legal instruments, incarcerated persons are entitled to health services equivalent to those available to the general community outside the prison. For example, the United Nations Basic Principles for the Treatment of Prisoners note that 'prisoners shall have access to the health services available in the country without discrimination on the grounds of their legal situation' (UNGA 1990: np) and the United Nations Committee on Economic, Social and Cultural Rights has affirmed that states are obliged to refrain from 'denying or limiting equal access to all persons, including prisoners or detainees... to preventive, curative and palliative health services' (UNCESCR 2000). Hence, countries treating heroin dependence with OST in community settings are obliged to make this treatment available to prisoners (Hall, Ward & Mattick 1994).

Prevention of HIV transmission

As noted above, OST in community settings reduces injecting-related HIV risk behaviours, which results in reduced HIV incidence among heroin users in treatment (Gowing et al. 2008; Metzger et al. 1993). It is argued that providing OST in prisons will similarly reduce risk behaviours such as injecting drug use and sharing of needles and syringes (Dolan, Hall & Wodak 1998; UNODC/WHO/UNAIDS 2006). There is evidence that this is the case, with inmates receiving OST showing significantly reduced injecting drug use and syringe sharing compared with untreated incarcerated heroin users (Dolan et al. 2003; Heimer et al. 2006; Larney 2010).

Reductions in post-release crime and re-incarceration

Upon leaving prison, inmates often rapidly recommence frequent illicit drug use (Dolan et al. 1996; Kinner 2006) and hence are likely to commit further offences and be re-incarcerated. Providing OST in prison to treat heroin dependence can potentially reduce post-release relapse to regular drug use and therefore reduce reoffending and subsequent incarceration. The evidence relating to the role of prison OST in reducing incarceration and criminal activity will be discussed in greater detail in Chapters 3 and 4 of this report.

Reductions in post-release mortality

While in prison, an inmate's opioid tolerance decreases markedly as a result of abstinence or greatly reduced heroin use (Wakeman et al. 2009). Thus, the risk of overdose is greatly increased in the event that the inmate uses heroin after release (Bird & Hutchinson 2003; Farrell & Marsden 2007). It has been proposed that providing OST in prisons may assist in reducing the risk of post-release overdose by maintaining opioid tolerance in heroin-using inmates (Christensen et al. 2006). Post-release mortality and the role of prison OST in reducing this harm will be discussed in greater detail in Chapter 5 of this report.

Arguments against opioid substitution treatment in prisons

Despite its endorsement by international bodies such as the World Health Organization (WHO 2009, 2007), there often remains a reluctance among correctional authorities to implement OST in prisons. There is some evidence that this stems from a philosophical position that methadone and buprenorphine are no different to illicit heroin and that abstinence from all opioids—even medically prescribed opioids—is the only legitimate outcome of treatment for heroin dependence (Alberti & Cowie 2001; Boucher 2003; Gjersing et al. 2007). OST is conceptualised as 'facilitating addiction', whereas prisons are perceived as sites of opportunity to achieve abstinence (Nunn et al. 2009); this is despite the evidence that many heroin users continue to use heroin while in prison (Calzavara et al. 2003; Dolan et al. 1996; Strang et al. 2006) and that incarceration is negatively associated with cessation of drug use (DeBeck et al. 2009). Opposition to prison OST does not just come from correctional authorities (Alberti & Cowie 2001; McMillan & Lapham 2005); negative attitudes towards OST and a preference for abstinence-based treatment services have also been reported among prison healthcare staff (Gjersing et al. 2007; Nunn et al. 2009).

Beyond philosophical objections, there are potential safety concerns associated with the provision of OST in prisons, just as there are in community settings. Concern generally centres around the potential for violence and 'standover' tactics to force inmates receiving OST to provide their medications to other inmates, or the voluntary diversion of medication to other inmates in exchange for money or goods (Alberti & Cowie 2001; Hume & Gorta 1988; Nunn et al. 2009). Several studies have examined this issue, finding that when programs are appropriately resourced and consumption of medications is supervised, diversion occurs infrequently (Alberti & Cowie 2001; Gorta 1987; Magura et al. 1993; Wale & Gorta 1987b). The fact that diversion may occur does not negate the potential benefits of prison OST programs; should diversion of medication be detected, it can be addressed as in community-based OST programs, for example, through discussion with the inmate to identify motivations for diversion, closer supervision of dosing and, if necessary, cessation of treatment for that individual (Alberti & Cowie 2001).

Global implementation of prison OST

OST programs based in correctional institutions were first documented in the 1980s. In 1986, prisons in New South Wales, Australia, began offering methadone maintenance treatment to inmates three to four months prior to their release from prison (Wale & Gorta 1987a) and in 1987, a heroin detoxification program that had been operating in Riker's Island Jail in New York City was expanded to allow methadone maintenance treatment (Magura, Rosenblum & Joseph 1992). An international review published in 1996 identified only five countries in which methadone maintenance treatment was provided in prisons—Australia, the United States (in New York only), Denmark (commencing in 1988), Switzerland (1989) and Spain (1992) (Dolan & Wodak 1996; Nelles et al. 1998; Stover, Hennebel & Casselman 2004).

With the recognition of prisons as potential sites for HIV transmission, the number of correctional jurisdictions offering OST to heroin-dependent inmates rapidly expanded in the late 1990s, particularly in Western Europe (Stover, Hennebel & Casselman 2004). By 2009, OST was available in prisons in at least 29 countries (Larney & Dolan 2009). However, there are at least 70 countries that provide OST in community settings (Mathers et al. 2010); hence, there are as many as 41 countries that offer OST in the community, but not in prison, in breach of the basic right of prisoners to have access to healthcare equivalent to that they would if at liberty.

Although the provision of OST in prisons is now more widespread (Dolan & Wodak 1996; Larney & Dolan 2009), there are still significant issues in relation to treatment coverage. Many prison OST programs operate as pilot programs, open only to small numbers of inmates in one or two of a country's correctional institutions (Larney & Dolan 2009). Program entry may be restricted to inmates who had been in OST immediately prior to incarceration, or to those with short sentences (Larney & Dolan 2009). There are also often jurisdictional differences within a country, such that inmates in one jurisdiction can access OST, but their peers elsewhere cannot (Nunn et al. 2009). Such restrictions and jurisdictional differences contribute to inequality in access to healthcare while in prison (Larney & Dolan 2009).

Data on the number of prisoners globally receiving OST are limited. A search of the literature and contact with relevant correctional officials revealed the figures shown in Table 1.1. Of the 21 countries with data available, OST is provided to less than 0.1 percent of inmates in five countries and less than one percent in a further five countries. It is acknowledged that it would be more appropriate to compare the number of prisoners in OST to the number of prisoners with a history of heroin use or dependence, but such data are rare and often refer to a different time period to that for which the number of prisoners in OST is available. Despite this limitation, the figures in Table 1.1 demonstrate that in many countries, it is unlikely that the level of treatment coverage is meeting demand (Larney & Dolan 2009). Furthermore, one of the rationales for prison OST is to reduce injecting drug use and hence reduce HIV transmission among prisoners (Dolan, Hall & Wodak 1998). It is unlikely that population-level results such as reduced HIV transmission can be achieved with so few inmates receiving treatment (Larney & Dolan 2009).

Table 1.1 Number of prisoners in OST, by country

Country	Number of prisoners receiving OST*	% of all prisoners receiving OST^
Ireland	472 (2006)	14.5
Spain	6,893 (2006)	12.3
Australia	3,328 (2009)	11.4
Denmark	333 (2005)	9.2
Austria	531 (2002)	7.7
France	3,653	6.6
United Kingdom	5,093 (estimate)	5.8
Iran	8,200	5.2
Portugal	533 (2006)	4.8
Italy	1,860 (2003)	3.3
Canada	592 (2006)	1.8
Albania	10	<1
Germany	500 (estimate)	<1
Moldova	15	<1
New Zealand	60	<1
Puerto Rico	24	<1
Indonesia	50 (estimate)	<0.1
India	35 (2009)#	<0.1
Poland	12 (2004)	<0.1
Taiwan SAR	4	<0.1
United States	1671–1967 (2008)*	<0.1

*2007, unless otherwise indicated

^Calculated using prison population figures from the International Centre for Prison Studies (http://www.kcl.ac.uk/depsta/law/research/icps/). The prison population for the year of the 'numbers in OST' figure was used for the calculation. #http://www.unodc.org/india/en/india_-tihar-jail-looking-beyond-the-bars.html

Source: Nunn et al. 2009; all other references available from Larney & Dolan 2009

Opioid substitution treatment in Australian prisons

Opioid substitution treatment is available in prisons in all Australian states and territories. 'Snapshot' figures of OST participation show that in 2009, there were 3,328 people receiving OST in Australian correctional facilities (AIHW 2010b). This was approximately 11 percent of all people in prison and around eight percent of all people in OST (ABS 2009; AIHW 2010b). As shown in Table 1.2, all jurisdictions permit inmates to continue OST if they were enrolled in treatment immediately prior to incarceration, except for Queensland, which only allows female inmates to continue treatment. Three jurisdictions do not permit initiation of OST in prison (see Table 1.2); these jurisdictions are also those with the smallest proportions of inmates in OST.

Table 1.2 Number of Australian prisoners receiving OST, 2009, by jurisdiction

	OST availability[1]		n receiving prison OST[2]	% of all OST clients[2]	% of all prisoners[3]
	Continuing	Initiation			
NSW	✓	✓	1,948	10.9	17.5
Vic	✓	✓	728	5.8	16.7
Qld	✓ (females only)	x	34	0.7	0.6
WA	✓	✓	305	9.6	6.9
SA	✓	✓	255	8.1	13.0
Tas	✓	x	5	0.8	0.9
ACT	✓	✓	51	6.4	25.1
NT	✓	x	2	1.7	0.2
Total			3,328	7.7	11.4

1 AIHW 2010a

2 AIHW 2010b

3 ABS 2009

The current study

As promoted by WHO and other prominent health agencies, the primary goal of OST in prison settings is to reduce injecting drug use and associated HIV risk behaviours; specifically, sharing of needles and syringes (UNODC/WHO/UNAIDS 2006; WHO 2007). In addition to potentially reducing HIV risk, there has been significant interest in the role of prison-based OST in reducing post-release reoffending and re-incarceration (Dolan et al. 2005; Gordon et al. 2008); it has also been proposed that by maintaining opioid tolerance, OST prior to release may help to reduce deaths by drug overdose (Christensen et al. 2006). Although there is good evidence that OST in prisons reduces injecting-related HIV risk behaviours (Dolan et al. 2003; Heimer et al. 2006; Larney 2010), the evidence regarding the effectiveness of prison OST on post-release criminality and deaths is less conclusive. This report summarises this research and presents three studies designed to examine whether engagement in OST while in prison improves post-release criminal justice and mortality outcomes.

2. DATA SOURCES AND LINKAGE

The studies described in this report employed data linkage to examine the effects of prison OST on post-release outcomes. Routinely collected data from state government health and criminal justice agencies were linked to baseline data for a cohort of 375 men who were recruited to a prison-based randomised controlled trial (RCT) of opioid substitution treatment in 1996–97 (Dolan et al. 2003, 2002). To participate in the trial, inmates were required to be male[2], have a history of heroin injecting, be serving a sentence of at least four months and be willing to be randomly allocated to either opioid substitution treatment or wait-list control. Inmates expressing an interest in participating in the trial were assessed using the same assessment process as all other inmates wishing to commence OST in prison. If assessed as suitable for OST and enrolled in the trial, participants assigned to the treatment condition were prescribed and dispensed methadone as patients of the NSW prison OST program. Participants assigned to the control condition could enter OST after four months (the initial follow-up period). The findings of the RCT and a four year follow-up study appear elsewhere (Dolan et al. 2005, 2003, 2002).

The baseline data for the cohort included information on participant demographics, drug use and prison histories, and participation in OST during the trial period. These data were to be linked to administrative datasets for the period 1 June 1997 to 31 December 2006[3]. A range of datasets with unit record level data were considered for linkage. In the cases of incarceration and reoffending data, unit record data are maintained in multiple databases at the jurisdictional (ie state or territory) level. With regards to mortality, although unit record data are held in a national database, the Centre for Health Record Linkage only has ready access to state-level data. The data sources selected for linkage were therefore all specific to New South Wales. The databases selected for linkage are described below.

Offender integrated management system

The Offender Integrated Management System (OIMS) is maintained by Corrective Services New South Wales. The OIMS monitors all prisoner movements—admissions, transfers and releases—in New South Wales.

Pharmaceutical drugs of addiction system

In New South Wales, prescribing of medications for opioid substitution treatment is monitored through the Department of Health's Pharmaceutical Drugs of Addiction System (PHDAS). To prescribe OST, doctors must request approval from the NSW Director-General of Health. PHDAS records provides, among other information, the date that an approval is granted (ie a treatment episode commences) and the date that the treatment episode ceases.

Reoffending database

The Reoffending Database (ROD) is maintained by the NSW Bureau of Crime Statistics and Research (BOCSAR). ROD contains records of all finalised criminal court appearances in the Children's, Local, District and Supreme Courts of New South Wales, including date and type of offence (Hua & Fitzgerald 2006).

Centre for Health Record Linkage Master Linkage Key

The Centre for Health Record Linkage (CHeReL) maintains a Master Linkage Key (MLK) containing data from 11 major data collections in New South Wales, including death registrations from the Registry of Births, Deaths and Marriages and causes of death as recorded by the Australian Bureau of Statistics (ABS).

2 Women were excluded from the trial as a pilot study showed that it was not possible to recruit enough female prisoners with sentences of sufficient length (Dolan et al. 2002).

3 It had originally been intended to collect data for the calendar years 1997–2006; however, the Corrective Services Offender Integrated Management System was implemented in May 1997; hence, data were obtained for the period June 1997 to December 2006.

Data linkage

Data linkage was undertaken with the assistance of CHeReL. CHeReL was established in 2006 specifically to support research using linked, administrative data. It acts as a third-party intermediary between data custodians and researchers, separating the processes of data linkage and analysis and thereby maintaining the confidentiality of individuals whose data are accessed (Lawrence, Dinh & Taylor 2008).

On receiving evidence of ethical approval for the project, CHeReL requested identifiers for the members of the cohort so that linkage to other datasets could commence. The original cohort contained 382 individuals. Manual inspection of this cohort revealed that six records (2% of the cohort) contained no name details and hence were unsuitable for linkage. One record was found to be a duplicate and was not included in the cohort. Thus, full names, dates of birth and date of last contact for 375 individuals were supplied to CHeReL for linkage.

The initial linkage identified fewer matches to the cohort than expected. To increase the number of matches, a request was made to the Department of Corrective Services to supply CHeReL with all known aliases for participants in the baseline dataset. These were added to the identifiers used for the first round of linkage and the linkage procedure was undertaken for a second time. The records thus matched were supplied to the researchers for analysis.

Data cleaning and preparation for analysis

Baseline dataset

Data were extracted from the baseline dataset for the 375 participants for whom data linkage had been undertaken. Data extracted were date of entry to the trial; demographic variables (eg age, Indigenous status); and self-reported age at first incarceration, number of prior custodial episodes, drug use history and use of drugs in prison.

OST data, Pharmaceutical Drugs of Addiction System

Start and end dates were obtained for all episodes of opioid substitution treatment undertaken by participants during the follow-up period. A continuous treatment episode was defined as one where there were six or fewer days between a treatment exit and entry date. When there was a gap of seven or more days between a treatment exit date and entry date, a new episode of treatment was considered to have begun (Burns et al. 2009).

Incarceration data, Offender Integrated Management System

Dates of prisoner movements and reason for movement were obtained for matched participants. Continuous custodial episodes were identified. The date of entry to prison was identified by a 'reason for movement' code of admission and a person was taken to have exited custody if they had a 'reason for movement' code of acquitted, bail, escape, parole, sentence expired or deceased.

Offending data, Reoffending Database

Offending variables obtained were date of proved offences and offence type as categorised under the Australian Standard Offence Classification system (ABS 2008). Note that proved offences rather than arrests or criminal charges were utilised in data analyses.

Mortality data, Master Linkage Key

Date and cause of death data for participants were provided for the follow-up period. One duplicate record was identified and deleted prior to use of the mortality data in analyses.

Ethical approvals

Approval for this research project was received from the NSW Health Population and Health Services Research Ethics Committee, the Justice Health Research Ethics Committee, the University of NSW Human Research Ethics Committee and the Corrective Services NSW Research Approval Committee.

3. OPIOID SUBSTITUTION TREATMENT AND RE-INCARCERATION[4]

As described in the *Introduction*, incarceration is a common experience among people who use heroin (Boutwell et al. 2007; Oliver et al. 2010; Ross et al. 2005). Once released from prison, this population is highly likely to experience further periods of incarceration. In an earlier follow-up of the cohort under study, 82 percent of participants who had been released from prison were re-incarcerated at least once during a four year period (Dolan et al. 2005). Re-incarceration is often rapid, with half of released heroin users returning to prison within six months in studies in the United States (McMillan, Lapham & Lackey 2008) and France (Marzo et al. 2009).

Drug use after release from prison increases the risk of an individual returning to custody (Kinner 2006; Kjelsberg & Friestad 2008). Of particular relevance for the current study, among a sample of NSW and Victorian prisoners, risk of re-incarceration was significantly increased among participants reporting 'worsening heroin use' in the post-release period (Baldry et al. 2006: 29). Given this, treatment for heroin dependence while in prison may contribute to reducing risk of re-incarceration.

There have been several studies examining the role of prison-based OST in reducing risk of re-incarceration. Indeed, one of the earliest studies of OST was undertaken with a small sample of prisoners; in that study, three of 12 (25%) inmates treated with OST were re-incarcerated within a year of release, compared with 15 of 16 (94%) of untreated inmates (Dole et al. 1969). In a larger, more recent randomised controlled trial, compared with untreated participants, significantly fewer treated participants had been re-incarcerated at three months post-release (Kinlock et al. 2008); however, by six months post-release, the two groups had equivalent levels of re-incarceration (Gordon et al. 2008). Recent data linkage studies in the United States and France both reported that receiving OST in prison was unrelated to risk of re-incarceration (Marzo et al. 2009; McMillan et al. 2008).

A limitation of studies examining the role of prison OST in re-incarceration is lack of analysis of the post-release treatment status of participants. In community samples, incarceration risk is only reduced while the individual remains in OST, rising during periods out of treatment (Oliver et al. 2010; Werb et al. 2008); as such, it is critical that post-release participation in OST is factored into analyses of how prison OST affects re-incarceration. The only study of prison OST to have taken retention in treatment into account in data analysis found that, if participants remained in treatment for more than eight months, their risk of re-incarceration was reduced by 70 percent (Dolan et al. 2005). As such, the present study examines the effects of both treatment status on release, and retention in treatment post-release, on risk of re-incarceration.

Aims

The aims of this study are to:

- describe the study cohort, including patterns of incarceration and OST participation between 1 June 1997 and 31 December 2006;
- assess if being in OST on day of release from prison is protective against re-incarceration; and
- assess if retention in OST after release from prison is protective against re-incarceration.

4 Although reoffending generally occurs before re-incarceration, analysis of the effect of OST on re-incarceration required linkage of four datasets (baseline, OIMS, PHDAS and MLK), while analysis of reoffending required linkage of five datasets (the abovenamed 4 and the ROD). Hence, re-incarceration data are presented first, with reoffending data analysed in Chapter 4.

Method

Data sources

The data sources used in this study were the baseline dataset (for demographic variables); the incarceration data from the Offender Integrated Management System (OIMS; for dates of entry to and exit from prison); the OST data from the Pharmaceutical Drugs of Addiction System (PHDAS; for dates of entry to and exit from treatment); and the mortality data from the Master Linkage Key (MLK; for correct censoring of observations in survival analysis).

Data structure

Each participant could have multiple periods of incarceration recorded in the OIMS. These data were manipulated to allow analysis of 'release intervals'. Each release interval began with a release date and ended at either the date of re-incarceration, date of death or 31 December 2006 (the end of the observation period) (McMillan et al. 2008). Release intervals ending on 31 December 2006 without a re-incarceration were censored in the survival models, as were release intervals that ended because the participant died. Release intervals were linked to the OST data and categorised as 'treated' if the participant was in OST at the start of the release interval and 'untreated' if not. A variable recording the number of days a participant remained in OST from the beginning of the release interval was constructed. Demographic characteristics from the baseline data were also added to the analysis database.

Cohort characteristics

Demographic variables and variables describing participant drug use and incarceration histories were extracted from the baseline data. Continuous variables were highly skewed, so medians are presented. Patterns of incarceration and OST during the follow-up period were examined by extracting data from the OIMS and PHDAS. Re-incarceration within two years of release was calculated to allow for comparisons with the re-incarceration rate of the general NSW prisoner population.

Modelling re-incarceration

Risk of re-incarceration was modelled using recurrent event survival analysis; specifically, Prentice-Williams-Peterson gap-time models (Ezell, Land & Cohen 2003; Guo, Gill & Allore 2008; Lim, Liu & Melzer-Lange 2007; Prentice, Williams & Peterson 1981). Further details regarding this model can be found in *Appendix C*.

Two variables defining OST exposure were created. The first indicated the treatment status of participant at the beginning of the release interval, as in prior research (McMillan et al. 2008). Treated release intervals were coded as 1 and untreated intervals coded as 0. The second OST variable took into account not only whether a release interval was treated, but also the length of time that a participant remained in that treatment episode following release. This was achieved by coding retention in treatment as a time-dependent variable in the model. A time-dependent variable can change in value over time; in this case, participants with treated release intervals commenced the release interval with an OST value of 1, which changed to 0 at the time of ceasing treatment.

Two multivariate models were developed. Model 1 incorporated the first OST variable (ie treatment status at release) and Model 2, the time-dependent OST variable (ie retention in treatment post-release). To identify covariates for inclusion in the models, a range of variables were tested for univariate associations with re-incarceration, with $p \leq 0.25$ selected as the cut-off for inclusion in the multivariate models (Hosmer & Lemeshow 1999). Covariates tested were age at first drug injection, age at first incarceration, age at release, Indigenous status, number of incarcerations prior to baseline incarceration, use of heroin during baseline or prior incarceration, injecting drug use during baseline or prior incarceration and number of drug classes used in the month prior to baseline incarceration.

All analyses were conducted in SAS 9.1 (SAS Institute 2003). The recurrent event models were fit using PROC PHREG.

Results

Cohort characteristics

All participants were male. The median age of participants at baseline was 26 years (range 18–46 years) and 24 percent (91/375) of participants identified as Aboriginal or Torres Strait Islander. Sixty-three percent (236/375) of participants reported that they were hepatitis C positive; no participants reported HIV infection. Drug use and imprisonment histories prior to the baseline incarceration are shown in Table 3.1.

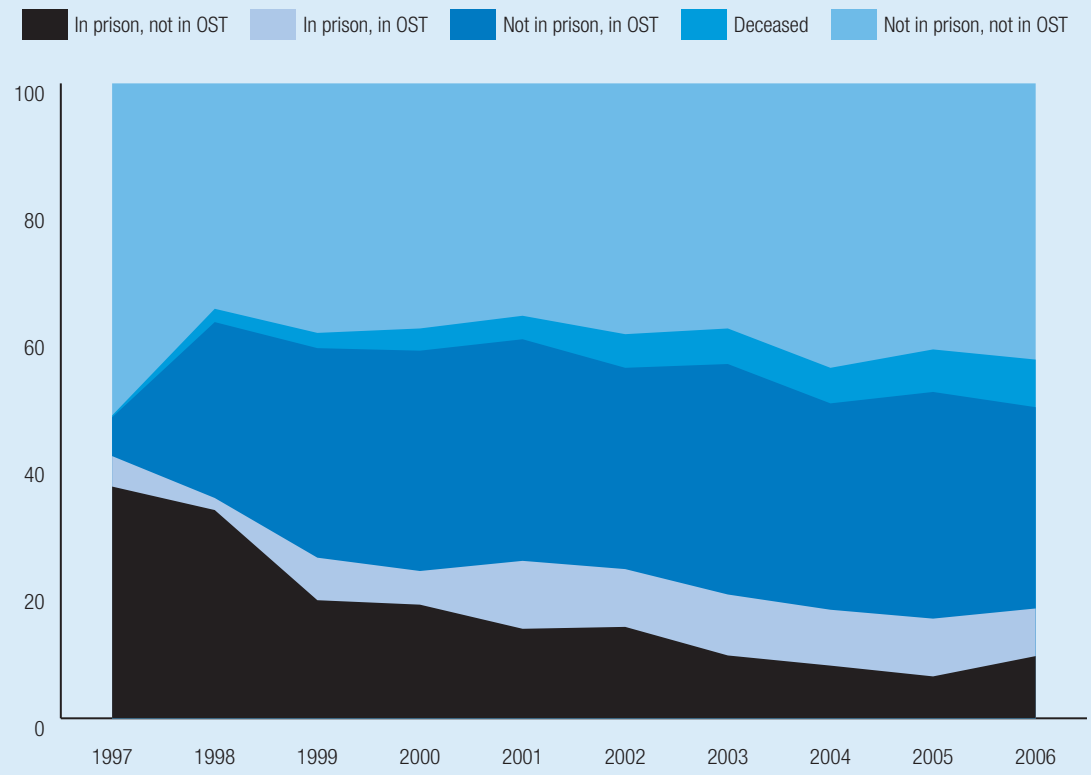
Table 3.1 Baseline demographics and drug use and imprisonment histories

	Median (range)
Age in years first injected drugs	16 (7–40)
Age in years first incarcerated	18 (15–43)
Number of prior custodial episodes	4 (1–62)
Drug use in month prior to baseline incarceration	**n (%)**
Heroin	360 (96.0)
Cannabis	312 (83.2)
Benzodiazepines	261 (69.6)
Methamphetamine	163 (43.5)
Illicitly obtained methadone	162 (43.3)
Cocaine	143 (38.1)
Prescribed methadone	30 (8.0)
Number of above drug types used	**Median (range)**
Median (range)	4 (1–6)
Drug use in baseline incarceration or prior incarceration	**n (%)**
Used heroin in prison	239 (63.7)
Injected heroin in prison	226 (60.3)
Any drug injection in prison	238 (68.4)
Shared needle/syringe in prison	195 (81.9)*

*percentage of participants reporting any drug injection in prison

Figure 3.1 provides a graphical representation of incarceration, participation in OST and deaths in the cohort at 30 June each year 1997–2006. The Figure shows that the proportion of participants in prison decreased over time, from 41 percent in 1997 to 17 percent in 2006. Participation in OST increased rapidly between 1997 and 1999, before stabilising from 1999 onwards, with around 40–45 percent of participants in OST at any point in time. From 2003, around half of those in prison were receiving OST. Twenty-eight participants, or 7.5 percent of the cohort, were deceased by 31 December 2006.

Figure 3.1 Natural history of incarceration, opioid substitution treatment and mortality, 1997–2006 (%)



Linkage between the baseline dataset and OIMS identified that all but two of the 375 participants were incarcerated for at least one day between 1 June 1997 and 31 December 2006. Three participants were in custody for the entire follow-up period. Participants commenced 2,036 custodial episodes, with a median of five (range 1–25) episodes per participant. The median length of each episode (based on 1,946 completed episodes) was 99 days (range 1–3,180 days). The median total length of time in prison over the follow-up period was 1,337 days (range 0–3,500 days), or 3.6 years.

From the linkage between the baseline dataset and PHDAS, 88 percent (331/375) of participants were in opioid substitution treatment for at least one day between 1 June 1997 and 31 December 2006. Participants commenced a total of 1,081 OST episodes, with a median of two (range 1–12) episodes per participant. Median episode length (based on 927 completed treatment episodes) was 156 days (range 1–2,957), or approximately five and a half months. The median total length of time in treatment over the follow-up period was 592 days (range 3–3,444), or 1.6 years.

Of the 1,081 episodes of OST that were commenced during the follow-up period, 58 percent (632/1,081) were commenced in prison. Eighty percent (300/375) of participants commenced an OST episode while in custody.

Risk of re-incarceration

Of the 375 participants, two were not identified in the incarceration data and three were incarcerated for the entirety of the follow-up, leaving 370 participants who were released from prison at least once during the follow-up and therefore at risk for re-incarceration. Ninety percent (332/370) of these participants were re-incarcerated at some point following their first observed release, with 84 percent (309/370) of released participants re-incarcerated within two years. There were 2,088 release intervals during the follow-up period; 40 percent (842/2,088) were treated release intervals. The median number of release intervals was four (range 1–23). The median length of release intervals (ie time to re-incarceration or censoring) was 111 days

(range 1–3,391) and the median duration of post-release retention in OST was 63 days (range 1–3,391); note that these latter two figures are not adjusted to take into account the correlation of release intervals within participants.

In univariate analyses, age at first incarceration and age at release were associated with reoffending at the p≤0.25 level (see Table 3.2). These variables were entered into multivariate models. Results are shown in Table 3.2.

Model 1: Dosing status of the release interval as the independent variable

In Model 1, controlling for age at first incarceration, there was no statistically significant association between OST status at release from prison on risk of re-incarceration. Age at release was significantly associated with re-incarceration, with each additional year of age associated with a three percent decrease in risk of re-incarceration (see Table 3.2).

Model 2: Retention in OST post-release as the independent variable

In contrast to Model 1, there was a significant effect of OST exposure on re-incarceration in Model 2. As long as participants remained in OST, their risk of re-incarceration was reduced by an average of 20 percent (see Table 3.2). As in Model 1, older age at release was associated with a small reduction in risk of re-incarceration.

Table 3.2 Recurrent event models of the effect of OST status at release from prison and retention in OST post-release on risk of re-incarceration

			Multivariate models*			
	Univariate* hazard ratio (95% CI)	p	Model 1 adjusted hazard ratio (95% CI)	p	Model 2 adjusted hazard ratio (95% CI)	p
Opioid substitution treatment variables						
Treatment status of release interval	0.96 (0.86–1.06)	0.4	0.97 (0.87–1.08)	0.6		
OST status post-release#	0.79 (0.70–0.89)	<0.0001			0.80 (0.71–0.90)	0.0002
Covariates						
Age at first drug injection	1.00 (0.99–1.00)	0.7				
Age at first incarceration	0.98 (0.97–1.00)	0.01	1.00 (0.99–1.02)	0.9	1.00 (0.98–1.02)	0.9
Age at release	0.97 (0.96–0.98)	<0.0001	0.97 (0.96–0.98)	<0.0001	0.97 (0.96–0.98)	<0.0001
Indigenous status	1.05 (0.93–1.18)	0.5				
Number of prior incarcerations	1.00 (0.99–1.01)	0.8				
Used heroin in prison	1.00 (0.90–1.12)	0.9				
Injected any drug in prison	1.04 (0.93–1.17)	0.5				
Number of drug classes used month prior to baseline	1.02 (0.97–1.07)	0.4				

CI=confidence interval

*Models stratified by release episode

#Time-dependent covariate

Discussion

Almost all participants were re-incarcerated and re-incarceration was rapid. Simply being in treatment at the time of release did not affect re-incarceration risk; however, remaining in treatment post-release resulted in a significant reduction in the average risk of re-incarceration.

Cohort characteristics

This cohort of male, heroin-using prisoners had a median age of 26 at recruitment and one-quarter were of Aboriginal or Torres Strait Islander origin. As recruitment occurred in prisons and was restricted to men only, this cohort may not be representative of all heroin users. In comparison to a group of Australian heroin users recruited to a treatment outcomes study (Ross et al. 2005), this cohort appears to be younger and to have initiated injecting earlier. Earlier onset of injecting drug use is an indicator of socioeconomic deprivation, traumatic experiences in childhood and adolescence, and poorer educational attainment (Fuller et al. 2002; Kerr et al. 2009; Seddon 2008), suggesting that the cohort under study may be a particularly disadvantaged subgroup of heroin users.

Patterns of incarceration

Virtually all participants experienced incarceration during the follow-up. When total time in prison was considered, participants spent over one-third of the follow-up period in custody. Most commonly, incarceration was experienced as a series of episodes of three to four months duration. The proportion of participants in prison decreased over time, in line with a study of Australian heroin users that found that risk of recent incarceration decreased with age (Darke et al. 2009).

A major concern raised by the observed pattern of incarceration is the heightened mortality risk that individuals are exposed to each time they are released from prison. In a study of NSW prisoners, Kariminia et al. (2007b) found that as number of releases from prison increased, so too did risk of death. Greatly increased risk of death, particularly from drug overdose, in the weeks following release from prison has been frequently observed (Bird & Hutchinson 2003; Kariminia et al. 2007c; Odegard et al. 2010). The frequent cycling in and out of prison seen in this cohort thus strongly suggests the need for pre-release overdose prevention strategies. It has been suggested that pre-release OST may reduce post-release overdose risk by maintaining opioid tolerance (Christensen et al. 2006) and this issue will be considered in greater detail in Chapter 5. Providing takeaway doses of naloxone, an overdose 'antidote', to heroin users on release from prison has also been suggested as a way to reduce mortality risk. Naloxone training and distribution programs have been successfully deployed in a number of locations (Doe-Simkins et al. 2009; Strang et al. 2008; Wagner et al. 2010), and an exploratory study of overdose prevention in the United States reported high acceptability of takeaway naloxone among recently released prisoners (Wakeman et al. 2009). At present, naloxone is only available in Australia on prescription (Lenton et al. 2009a, 2009b) and there are no formal naloxone distribution programs for prisoners nearing release.

Patterns of OST

The majority (88%) of participants entered OST at some stage during the follow-up period. Strikingly, 80 percent of participants commenced an OST episode while in custody and these episodes made up over half of all treatment entries. This reflects the fact that OST is an attractive treatment option for incarcerated heroin users because it allows inmates to avoid heroin withdrawal and alleviates opioid cravings. The appeal of OST in prison is also illustrated in a population-level study of the NSW OST program, which found that one-quarter of those starting OST for the first time did so while in a correctional facility (Burns et al. 2009); that is, individuals who had not entered OST while at liberty did so when in custody. These results suggest that restrictions on commencing OST while in prison, as exist in some Australian jurisdictions and internationally (Larney & Dolan 2009), are counterproductive as they prevent treatment entry at a time when demand for treatment is strong and the potential benefits of treatment are high.

Effects of OST on re-incarceration

Re-incarceration following first observed release from prison was the norm and occurred rapidly, with median time to re-incarceration around 4.5 months. The proportion of released prisoners who had been re-incarcerated within two years of release was much greater than that seen among NSW prisoners in general (84% vs 43–45%) (SCRGSP 2010). Studies modelling prisoner population growth show that even minor reductions in re-incarceration of groups of offenders with high levels of re-incarceration deliver significant

benefits in terms of reducing the size of the prisoner population (Weatherburn et al. 2009). Therefore, any intervention that reduces re-incarceration of heroin users would produce benefits for the correctional system as a whole.

In Model 1, with the treatment status of the release interval as the independent variable, treatment status on release from prison had no significant effect on risk of re-incarceration. This result is in keeping with two recent studies of recurrent re-incarcerations in inmates treated with OST (Marzo et al. 2009; McMillan, Lapham & Lackey 2008), which both found that OST exposure in prison neither decreased nor increased risk of re-incarceration.

The results from Model 2 contrasted with the above findings. Model 2 included retention in treatment after release from prison as a time-dependent covariate, finding that while participants remained in OST after release, their risk of re-incarceration was reduced by an average of 20 percent. Thus, it appears that it is not OST exposure in prison per se that affects re-incarceration, but whether a person remains in treatment following release. This makes sense in light of research showing that the benefits of OST are maintained only while individuals remain in treatment; in community samples, rates of criminal offending and incarceration are reduced only while in OST, rising during periods out of treatment (Davstad et al. 2009; Lind et al. 2005; Werb et al. 2008).

Although risk of re-incarceration was reduced as long as participants were retained in treatment, the median retention time was only 63 days. That is, in half of treated release intervals, treatment had ceased within two months of release. The post-release period is a highly stressful time, marked by difficulties in finding appropriate housing and employment (Baldry et al. 2006; Halsey 2007). Released inmates typically return to a drug- and crime-involved peer group and even among those who intend to avoid doing so, post-release illicit drug use and criminal activity is common (Halsey 2007; Kinner 2006). In the face of such pressures, the requirement to attend a clinic daily for pharmacotherapy may be a burden too difficult to maintain. Comprehensive pre-release planning and post-release support is needed to address not only treatment needs, but also the multitude of other difficulties that released inmates contend with. One pre-release program aiming to address these needs is the Connections Project, established in 2007 in New South Wales by Justice Health. The Connections Project aims to link inmates not only to community OST providers, but also to housing and other health and welfare services. An evaluation of this project is underway (Martire & Howard 2009).

It could be argued that, because post-release retention is the key to reducing re-incarceration, there is no need to provide OST while in prison and that resources should instead be directed to assisting inmates with a history of heroin use to enter OST on release. However, several studies have shown that post-release participation in OST is maximised when treatment is commenced while in prison (Kinlock et al. 2007; Magura et al. 1993; Tomasino et al. 2001). For example, in a randomised trial of prison OST in the United States, those who had been in treatment while in prison were significantly more likely to enter post-release treatment than participants who were simply given a post-release treatment referral (Kinlock et al. 2007). There are also other benefits to OST in prison, such as reduced drug injecting and sharing of needles and syringes (Dolan et al. 2003; Larney 2010). Hence, the maximum benefits from OST can be obtained by commencing (or continuing) treatment while in prison, and providing assistance to ensure a smooth transition to a post-release treatment provider.

Limitations

It is important to keep in mind that the cohort under study was recruited in prison and is all male. There were some indications that, in comparison to a community-recruited cohort of Australian heroin users (Ross et al. 2005), this cohort became entrenched in illicit drug use at an earlier age. Hence, these participants may not be representative of the broader population of heroin users. However, this does not invalidate the findings, as the more severe profile of this cohort would serve to produce conservative estimates of treatment effects.

A limitation of all data linkage studies is the potential for poor linkage accuracy. A sensitivity and specificity analysis (reported in *Appendix B*) suggested that the linkage to PHDAS was relatively accurate and the linkage to OIMS was conducted using a unique identifier, promoting high sensitivity and specificity of linkage.

However, it is possible that some participants may have had periods of incarceration or treatment outside of New South Wales. In a recent survey of NSW inmates, around 10 percent had been living interstate in the year prior to their current incarceration (Indig et al. 2010). Incarceration or treatment episodes occurring interstate could not be identified in the linkage, potentially leading to underestimation of re-incarceration.

Conclusion

This study has identified a pattern of repeated incarceration of heroin users, with participants spending more than twice as much time in prison as in opioid substitution treatment. It was common for participants to commence OST while in prison, demonstrating the importance of prisons as sites for engaging heroin users in treatment.

Heroin users who remained in OST after release from prison had a reduced risk of re-incarceration. Thus, in terms of reducing re-incarceration, the maximum benefits of prison-based OST can be obtained by providing treatment in prison and facilitating transfers to community-based treatment after release. In addition to the benefit to the individual, reduced re-incarceration of heroin users would benefit the correctional system as a whole, through reducing the size of the prisoner population and the costs of correctional administration.

4. OPIOID SUBSTITUTION TREATMENT AND POST-RELEASE OFFENDING

The relationship between heroin use (and illicit drug use more generally) and criminal activity has been widely studied. Although there is no objective 'true' record of how much crime an individual commits, self-reported and officially recorded offending data generally show high levels of criminal offending among people who use heroin (Bennett, Holloway & Farrington 2008; Oliver et al. 2010; Oviedo-Joekes et al. 2008; Sheerin et al. 2004; Stewart et al. 2000).

Multiple studies have examined offending among Australian heroin users. In a national study of treatment entrants, 39 percent reported committing any crime in the past month, most commonly property offences (eg theft; 22%) and drug dealing (21%) (Digiusto et al. 2006). In another treatment-seeking cohort, 55 percent reported criminal activity in the previous year, with property crimes (38%) and dealing (27%) again being the most common offences (Ross et al. 2005). More recently, just over half (52%) of heroin users interviewed nationally for the 2009 Illicit Drug Reporting System (IDRS) reported committing at least one crime in the last month. Thirty percent reported drug dealing and 23 percent had committed property offences (N Sindicich, national IDRS co-ordinator, personal communication 3 May 2010).

There is evidence that at least some, if not most, offending by heroin users is driven by the need to generate funds for drug purchases. As noted above, the crimes most commonly committed by this group are those that provide income—drug dealing and property crimes such as shoplifting and theft from individuals (Best et al. 2001; Digiusto et al. 2006; Maher et al. 2002; Manzoni et al. 2010; Ross et al. 2005). Several studies have demonstrated that as the intensity or frequency of heroin use increases, so too does the frequency of offending and that during periods of relatively less drug use, offending is reduced (Ball, Shaffer & Nurco 1983; Manzoni et al. 2010).

Although the economic imperative drives much offending by heroin users, there is also evidence that, for some individuals, offences are committed for reasons unrelated to drugs. For example, in one study, half of injecting drug users who engaged in criminal activity said they would continue to commit crimes even if they did not need money to buy drugs (DeBeck et al. 2007). Multiple studies have noted that within samples of heroin users, there are individuals who are particularly prolific offenders (Hall, Bell & Carless 1993; Nurco et al. 1988; Stewart et al. 2000). For example, among treatment entrants in one study, 75 percent of property crimes were committed by just 10 percent of the sample (Stewart et al. 2000). These individuals with higher levels of criminal activity tend to have commenced their offending careers at an earlier age, often preceding illicit drug use (Nurco 1998; Nurco et al. 1988). Nurco classified heroin users such as these as 'high-crime' users, as compared with 'low-crime' users, and showed that it was only low-crime users who reduced their offending behaviour during periods of reduced drug use; in contrast, high-crime individuals maintained a high level of offending behaviour irrespective of changes in drug use patterns (Nurco 1998; Nurco et al. 1988).

Does opioid substitution treatment reduce offending?

It is generally argued that reducing heroin use through treatment should have the flow-on effect of reducing criminal activity (Hall 1996). However, research from a forensic psychology perspective shows that offending is most effectively reduced by treatment approaches that address a suite of 'criminogenic' risks and needs (Andrews & Bonta 2010; Andrews, Bonta & Wormith 2006; Andrews & Dowden 2006). Drug use is one of these risks; others include antisocial/pro-criminal cognitions, behaviours and associates, and limited involvement in pro-social leisure activities and employment (Andrews, Bonta & Wormith 2006). In the risk/needs model, addressing drug use through OST will only reduce offending in those individuals for whom drug use is the primary reason for offending (ie Nurco's low-crime heroin users). The fact that many heroin users have motivations other than purchasing drugs when committing crimes (DeBeck et al. 2007; Nurco 1998; Nurco et al. 1988; Stewart et al. 2000) suggests that OST alone may not always be effective in reducing offending.

This limitation of OST—that it addresses only one risk factor for offending—has been borne out by research evidence suggesting that participation in OST substantially reduces criminal activity overall, but that a minority of individuals continue to commit crime while in treatment. For example, in a national evaluation of OST in Australia, the proportion of participants reporting past-month criminal activity was significantly reduced from 39 percent to 20 percent (Digiusto et al. 2006). Similarly, half of participants entering OST as part of a UK treatment evaluation study reported recent acquisitive crime at baseline; at one-year follow-up, one-quarter reported committing an acquisitive crime in the three months prior (Gossop et al. 2000). This pattern of findings—that overall offending is decreased while in treatment, but a minority continue to commit crimes—has been reported many times over (Bell et al. 1997; Hser, Anglin & Chou 1988; Lawrinson et al. 2008; Sheerin et al. 2004).

Other factors besides individual propensities for offending also affect the extent to which OST reduces offending behaviour. Retention in treatment has been shown to be critical in reducing offending, with offending rates increasing after leaving treatment (Deck et al. 2009; Lind et al. 2005), although perhaps not to the same level as pre-treatment (Hser et al. 1988). Indeed, there is evidence from recent studies that shorter episodes of OST (eg 3–4 months duration) may have zero effect on offending (Deck et al. 2009; Oliver et al. 2010). For example, a study of OST patients in a primary care clinic found that there was no difference in number of criminal convictions over a five year period between those who left treatment after one short episode, and those who engaged in multiple treatment episodes. In contrast, those who were in treatment continuously over the five year period had significantly fewer convictions than either of these groups (Oliver et al. 2010).

Prison-based OST and post-release offending

One rationale for OST in prisons is that treatment in prison will reduce criminal offending after release (Hall, Ward & Mattick 1994). As for re-incarceration, the evidence to support this assertion is not strong, with several studies reporting no effect of prison OST on either self-reported or officially recorded offending (Hume & Gorta 1989; Johnson, van de Ven & Grant 2001; Magura et al., 1993). A randomised controlled trial of prison OST reported that for the first six months post-release, treated inmates had lower levels of offending than untreated inmates (Gordon et al. 2008; Kinlock et al. 2008); however, by 12 months post-release, this difference was no longer evident (Kinlock et al. 2009). This pattern of results is potentially related to retention in treatment after release, but an analysis incorporating post-release treatment retention was not conducted.

In line with the analyses described in Chapter 3, this study examines the role of both treatment status on release from prison and retention in treatment post-release in reducing risk of reoffending.

Aims

The aims of this study are to:

- describe the number and types of criminal convictions received by participants;
- assess if being in OST on the day of release from prison reduces reoffending, as measured by criminal convictions; and
- assess if retention in OST after release from prison reduces reoffending, as measured by criminal convictions.

Method

Data sources and preparation

The data sources used in this study were the baseline dataset (for demographic variables), convictions data from the ROD (dates and types of offences for which participants were convicted), the incarceration data from OIMS (for dates of entry to and exit from prison), OST data from PHDAS (for dates of entry to and exit from treatment) and the mortality data from MLK (for correct censoring of observations in survival analysis).

Criminal convictions

Participants were grouped by whether they had a proved conviction during the follow-up period, and differences between participants with and without proved offences were tested using t-tests and chi-square tests as appropriate. For those participants with proved offences, the number and types of offences were tabulated.

Reoffending analyses

Data structure

The dataset used to assess re-incarceration in Chapter 3 was linked to the offending data from the ROD. In the data used in Chapter 3, release intervals ended with a date of re-incarceration, date of death, or 31 December 2006 (the end of the study period). For analysis of reoffending, release intervals ended at either the date of first offence after release from prison, date of re-incarceration, date of death, or 31 December 2006. Release intervals ending in re-incarceration, death or on 31 December 2006 without an offence were censored in the survival models. Participants could be re-incarcerated without an offence event, because a participant could be re-incarcerated while awaiting trial for an offence, but if the offence was not proved, it would not appear in the offending data. The use of re-incarceration as a censoring event was recognised as potentially introducing bias through informative censoring. This was addressed through a sensitivity analysis, described below. Further details on informative censoring can be found in *Appendix C*.

Model building

Time to reoffending was analysed using the same modelling strategy as in Chapter 3. Two multivariate models of factors affecting time to first proved offence following release from prison were developed—Model 1, with the treatment status of the release interval as the independent variable and Model 2, with retention in OST after release from prison as the independent variable. Covariates in the models were identified by testing for associations between a range of variables and time to first proved offence, with variables reaching $p \leq 0.25$ included in the multivariate models.

Because of the potential for informative censoring introduced by the use of re-incarceration as a censoring variable, sensitivity analyses were undertaken. Each model was re-fit under the assumption that in all release intervals that ended with re-incarceration, the participant had committed an offence that day (Allison 1995). If the results of the sensitivity analysis differ markedly from the original analysis, then it is possible that the results of the model are biased as a result of informative censoring.

Results

Criminal convictions

According to the linkage with the ROD, 325/375 (88%) participants were convicted of a new offence between 1 June 1997 and 31 December 2006. Comparisons between participants with new convictions and those without showed that the former were significantly younger than the latter (see Table 4.1). No other variables tested showed a significant difference between groups.

Table 4.1 Participant characteristics, by conviction status during follow-up

	Criminal conviction during follow-up? Mean (SD)			
	Yes	No	Student's t	p
Age in years	26.6 (6.0)	28.4 (6.6)	1.94	0.05
Age in years at first injection	17.3 (7.5)	17.8 (4.0)	0.72	0.5
Age in years at first incarceration	19.7 (3.6)	20.4 (4.5)	0.99	0.3
Prior incarcerations	4.9 (5.3)	4.4 (3.2)	.02	0.3
Drug classes used in month prior to baseline incarceration^	3.7 (1.1)	3.7 (1.4)	-0.41	0.7
	n (%)	χ^2	p	
Indigenous status				
Indigenous	79 (87)	12 (13)		
Non-Indigenous	246 (87)	38 (13)	0.002	0.9
Used heroin in prison				
Yes	203 (85)	36 (15)		
No	122 (90)	14 (10)	1.7	0.2
Injected drugs in prison^				
Yes	201 (85)	36 (15)		
No	123 (90)	14 (10)	1.9	0.2

^ Data missing for 1 participant

Participants were convicted of 5,975 offences during the follow-up period. The median number of proved offences per participant was 15 (range 1–79). As shown in Table 4.2, theft and related offences were the charges most frequently brought against participants, making up almost one-third of offences. Theft was also the most widespread offence, with 83 percent of participants being found guilty of theft or a related offence at least once (see Table 4.2).

Table 4.2 Criminal convictions of participants, 1 June 1997 to 31 December 2006

Offence type	Convictions (n=5,975) n (%)	Participants (n=325) n (%)
Theft and related offences	1,736 (29.0)	268 (82.5)
Traffic and vehicle regulatory offences	826 (13.8)	184 (56.6)
Public order offences	646 (10.8)	225 (69.2)
Unlawful entry/burglary, break and enter	628 (10.5)	159 (48.9)
Offences against justice procedures	520 (8.7)	187 (57.5)
Illicit drug offences	456 (7.6)	196 (60.3)
Acts intended to cause injury	435 (7.3)	171 (52.6)
Fraud, deception and related offences	205 (3.4)	98 (30.2)
Dangerous or negligent acts endangering persons	175 (2.9)	104 (32.0)
Property damage and environmental pollution	120 (2.0)	80 (24.4)
Miscellaneous offences	84 (1.4)	59 (18.2)
Prohibited and regulated weapons offences	75 (1.3)	52 (16.0)
Robbery, extortion and related offences	61 (1.0)	41 (12.6)
Other*	8 (<1)	8 (<1)

*Includes abduction and other offences against the person, homicide and related offences, and sexual assault and related offences

Risk of new conviction post-release

The median length of time between release from prison and a proved offence was 155 days (range 1–3,391 days) and the median duration of post-release retention in OST was 63 days (range 1–3,391 days); note that these two figures are not adjusted to take into account the correlation of release intervals within participants.

In univariate analyses, age at first incarceration, age at release, number of prior incarcerations, having injected drugs in prison and number of drug classes used prior to baseline incarceration were all associated with a new conviction at the $p \leq 0.25$ level (see Table 4.3). These variables were entered into multivariate models that were stratified by release number. Results are shown in Table 4.3.

Model 1: Treatment status of the release interval as the independent variable

Adjusted for all other variables in the model, there was no association between OST status at release from prison and time to first proved offence (see Table 4.3). There was a significant association with age at release, with each additional year of age associated with a three percent reduction in risk of new conviction post-release. Incarceration history was also significantly associated with risk of new conviction, with each additional prior incarceration contributing a two percent increase in risk.

In the sensitivity analysis, treating release intervals that ended with re-incarceration as though they were offending events had minimal effect on the hazard ratio of the OST variable (adjusted hazard ratio for OST status 0.99, 95% CI 0.89–1.10, $p=0.8$), suggesting that the results were robust to the effects of potentially informative censoring.

Model 2: Retention in OST post-release as the independent variable

Adjusted for all other variables in the model, there was no association between retention in OST after release from prison and time to first proved offence (see Table 4.3). As in Model 1, age at release and number of prior incarcerations were significantly associated with small changes in risk of new conviction.

In the sensitivity analysis, treating release intervals that ended with re-incarceration as though they were offending events led to a statistically significant finding (adjusted hazard ratio 0.90, 95% CI 0.80–1.00, $p=0.05$), suggesting that the results of the model may have been biased (see *Appendix C* for further details regarding informative censoring and bias).

Table 4.3 Recurrent event models of the effect of OST status at release from prison and OST status post-release on criminal convictions

			Multivariate models*			
	Univariate* hazard ratio (95% CI)	p	Model 1 adjusted hazard ratio (95% CI)	p	Model 2 adjusted hazard ratio (95% CI)	p
Opioid substitution treatment variables						
Treatment status of release interval	1.03 (0.91–1.18)	0.6	0.99 (0.87–1.13)	0.9		
Treatment status post-release#	0.97 (0.85–1.12)	0.7			0.88 (0.77–1.01)	0.07
Covariates						
Age at first injection	0.99 (0.99–1.00)	0.49				
Age at first incarceration	0.98 (0.96–1.00)	0.01	1.00 (0.98–1.02)	0.3	1.00 (0.98–1.02)	0.9
Age at release	0.97 (0.96–098)	<0.0001	0.97 (0.96–0.98)	<0.0001	0.97 (0.96–0.98)	<0.0001
Heroin use in prison	1.03 (0.88–1.19)	0.7				
Indigenous status	1.06 (0.88–1.26)	0.5				
Number of prior incarcerations	1.01 (1.00–1.02)	0.01	1.02 (1.01–1.03)	0.0007	1.02 (1.01–1.03)	0.001
Injected any drug in prison	1.10 (0.95–1.28)	0.2	1.12 (0.97–1.30)	0.1	1.13 (0.98–1.32)	0.1
Number of drug classes used in month prior to baseline incarceration	1.05 (0.98–1.21)	0.1	1.04 (0.98–1.10)	0.2	1.04 (0.97–1.10)	0.3

CI=confidence interval

*Models stratified by release episode

#Time-dependent covariate

Discussion

Participants were heavily criminally involved, being convicted of a median of 15 offences between June 1997 and December 2006. Being in OST at release from prison did not protect against new criminal convictions; neither did remaining in OST post-release. This somewhat contradicts the finding in the previous study that remaining in OST post-release reduces risk of re-incarceration, as it would be logical to assume that reductions in re-incarceration are a result of reduced offending. Potential explanations for this inconsistency and the impact of using convictions, rather than arrests or criminal charges, as an outcome measure, are discussed below.

As they stand, these results accord with those of other studies that defined reoffending in terms of convictions (Hume & Gorta 1989; Johnson, van de Ven & Grant 2001). They contrast with the self-reported criminal activity data that Kinlock and colleagues analysed at three- and six-months post-release in their randomised trial of prison OST. Those studies found that treated inmates reported significantly less criminal activity than untreated inmates (Gordon et al. 2008; Kinlock et al. 2008); however, when officially recorded arrests were analysed for this cohort at 12 month follow-up, treated inmates had the same risk of arrest as untreated inmates (Kinlock et al. 2009).

Setting aside the possibility that the obtained results are overly conservative, a potential explanation for the lack of a treatment effect on post-release offending is the limited effect that OST has on criminogenic risks and needs other than drug use (Andrews, Bonta & Wormith 2006). Having been recruited in prison and as shown in the previous chapter, potentially having a more severe clinical profile than other samples of Australian heroin users, it is likely that a proportion of the individuals in this cohort have motivations for offending that extend beyond acquiring funds for drug purchases. As such, OST alone will not necessarily reduce offending. This highlights the importance of therapeutic programs in prison that address the range of criminogenic risks

and needs exhibited by heroin users (and other offenders); for example, programs that address educational and vocational disadvantage, or that encourage offenders to develop pro-social attitudes and behaviours (Andrews & Bonta 2010; Andrews, Bonta & Wormith 2006). To date, there have been no evaluations of prison OST in conjunction with such programs; this is an area worthy of further research.

The finding in Chapter 3, that remaining in OST after release from prison reduced risk of re-incarceration, somewhat contradicts the findings of this study. It is possible that although OST did not reduce participants' criminal convictions, being enrolled in OST made it less likely that a participant, once convicted, would receive a custodial sentence. That is, sentencing magistrates may have taken into account that an individual was in treatment and applied a non-custodial sentence in order to avoid interrupting this treatment program. It is also possible that although participants continued to offend, the seriousness of the offences was reduced while in treatment and hence custodial sentences were not applied for this reason. Given the apparent contradiction in findings between this and the previous chapter, this is an area requiring further analysis.

Limitations

Results of the sensitivity analysis for Model 2 suggest that if arrests or criminal charges had been used as the outcome measure, rather than convictions, there may have been a statistically significant effect of retention in treatment on risk of reoffending. This finding highlights the importance of careful selection of outcome measures. In this case, convictions were selected as the most conservative proxy measure of reoffending; however, as Lind et al. (2005) notes, offences can be resolved informally, without a criminal conviction. As such, convictions may be too conservative a measure of reoffending and arrests or criminal charges may be a more appropriate outcome measure.

An additional limitation of the analysis was the inability to assess the sensitivity and specificity of the linkage between the baseline data and the ROD. Contrary to initial expectations, a number of incarceration episodes were not preceded by a conviction. This can be explained by imprisonment following arrests for offences which did not proceed to court (Lind et al. 2005), or for which the participant was acquitted by the court. It is also possible that the data linkage with the ROD may not have been as sensitive as that with the custodial data from OIMS. A unique identifier (the Master Index Number, or MIN) was used to match the baseline data to the OIMS, while matches between the baseline data and the ROD were made using names, aliases and dates of birth. The use of the MIN may have assisted in identifying more matches between the baseline and custodial data than the combination of identifiers used for matching the baseline data with the ROD.

Conclusion

This chapter has reported results suggesting that OST in prison or post-release does not assist in reducing post-release criminal convictions. However, results of the sensitivity analysis for Model 2 suggest that there may have been some bias in the analysis as a result of informative censoring and convictions may be an overly conservative indicator of reoffending. Further examination of the effects of post-release retention in OST on reoffending, measured by arrests, criminal charges or self-reported offences, is warranted. In particular, evaluations of OST combined with therapeutic programs that address non-drug criminogenic risks and needs may provide insights into how best to reduce offending.

5. OPIOID SUBSTITUTION TREATMENT, INCARCERATION AND MORTALITY

Heroin use is associated with a significantly increased risk of death (Darke, Degenhardt & Mattick 2007). In Australian cohort studies, heroin users die at around six times the rate seen in the general population (Degenhardt et al. 2009; Stoove et al. 2008). Leading causes of death are overdose, accidental injury and suicide (Degenhardt et al. 2009; Gibson et al. 2008; Stoove et al. 2008). Low HIV prevalence in Australian heroin users (National Centre in HIV Epidemiology and Clinical Research 2009) means that AIDS is not a significant contributor to excess mortality, in sharp contrast to European (Bargagli et al. 2001; Sanchez-Carbonell & Seus 2000) and North American (Colon et al. 2006; Tyndall et al. 2001) cohorts.

Mortality and imprisonment

As a group, prisoners and ex-prisoners suffer substantially increased mortality by comparison with the general population (Farrell & Marsden 2005; Kariminia et al. 2007a; Rosen, Schoenbach & Wohl 2008). In Australia, male prisoners and ex-prisoners die at 3.7 times, and females at 7.8 times, the rate of their age-matched, non-incarcerated peers (Kariminia et al. 2007a, 2007b). The most common causes of death among people with a history of incarceration are drug overdose, suicide and cardiovascular disease, but mortality rates for all causes of death are elevated compared to non-incarcerated persons (Kariminia et al. 2007a). Factors associated with increased risk of death include having been hospitalised for psychiatric illness, and multiple episodes of imprisonment (Kariminia et al. 2007b).

The increased risk of death experienced by prisoners and ex-prisoners is not constant across time. Multiple studies have demonstrated that the immediate post-release period is a time of particularly extreme mortality risk (Binswanger et al. 2007; Farrell & Marsden 2007; Hobbs et al. 2006; Kariminia et al. 2007c; Krinsky et al. 2009). In a study of US inmates, the risk of death in the two weeks after leaving prison was 13 times that of the general population (Binswanger et al. 2007). In the United Kingdom, excess mortality was even greater, with the first week post-release associated with a 29-fold increase in risk of death among men and a 69-fold increase among women (Farrell & Marsden 2007).

Up to 92 percent of deaths in the two weeks post-release are due to drug overdose, largely heroin or other opioid overdose (Binswanger et al. 2007; Bird & Hutchinson 2003; Farrell & Marsden 2007, 2005). Risk of overdose is increased at this time because of reduced tolerance following sustained abstinence from, or significantly lowered use of, heroin while in prison (Wakeman et al. 2009). In a large study of NSW inmates, the risk of overdose in the first two weeks post-release was nine times that at six months post-release (Kariminia et al. 2007c). Similarly, a study of Norwegian prisoners found that the risk of overdose death was 10 times higher in the two weeks post-release than at all other time at liberty, with risk of death remaining elevated for four weeks (Odegard et al. 2010).

Interventions to reduce post-release overdose-related mortality

Two main interventions have been suggested to counteract the increased risk of overdose death after release from prison; however, there have been limited evaluations of their effectiveness. It has been argued that prisoners with a history of heroin use would benefit from being provided with several doses of naloxone, a drug that counteracts the effects of opioid overdose, at the time of release (Wakeman et al. 2009). Evaluations of community-based naloxone training and distribution programs have shown that this approach can be effective (Doe-Simkins et al. 2009; Strang et al. 2008; Wagner et al. 2010); however, these programs operate on the premise that the person receiving the training and medication will administer these to other heroin users in the event of an overdose. For takeaway naloxone to be an effective intervention for released inmates, the inmates'

peers need to be aware of how it is used and whether the inmate is in possession of a dose. This issue has not been addressed in literature examining naloxone provision for released prisoners and to date there have been no evaluations of naloxone in relation to this population.

As described above, post-release overdoses occur largely because of reduced opioid tolerance following abstinence or reduced use of heroin while in prison. Accordingly, it has been suggested that maintaining opioid tolerance through the use of OST in prison may help to reduce overdose risk in the event of post-release heroin use (Christensen et al. 2006). OST is undoubtedly effective in reducing mortality risk in community settings; in an examination of deaths among OST clients in New South Wales over a 20 year period, the relative risk of death while in treatment was half that while out of treatment (Degenhardt et al. 2009). Significant reductions in mortality associated with OST have also been reported internationally (Brugal et al. 2005; Kimber et al. 2010; Peles, Schreiber & Adelson 2010). It is highly plausible that being in OST prior to release from prison is protective against post-release death; however, analysis of this question has been hampered by the need for a sample size in the tens of thousands (Bird 2009).

There have been two studies of prison OST that have reported on participant mortality in relation to OST status. In Kinlock (2009), there were eight deaths among 204 participants (3.9%) who were followed for 12 months post-release, while in Dolan (2005), 17 of 382 participants (4.5%) were deceased after four years of follow-up. None of these deaths occurred while participants were in treatment, supporting the assertion that prison OST may reduce post-release mortality.

Aims

The aims of this chapter are to:

- describe the causes of death in the cohort;
- assess the mortality rate of the cohort and compare to the general population; and
- compare mortality rates for the following states:
  - in and out of treatment;
  - in and out of prison; and
  - in the first 28 days post-release and all other time at liberty.

Method

Data sources

The datasets used in this chapter were the incarceration data from OIMS, the OST data from PHDAS and the mortality data from the MLK.

Data analysis

All analyses were conducted in SAS 9.1 (SAS Institute 2003) and Microsoft Excel.

Causes of death

All deaths in Australia are coded by expert clinical coders at the ABS on the basis of information contained in the death certificate. For the period 1997–2006, deaths were coded using the International Classification of Diseases, tenth edition (ICD–10) (WHO 1993).

Based on the literature (eg Degenhardt et al. 2009) and the mortality findings of the four year follow-up of this cohort (Dolan et al. 2005), it was expected that drug-related events (eg accidental overdose), violence, accidents and suicide would account for most deaths. The ICD-10 codes used to identify deaths in each of these categories are shown in *Appendix D*. Drug-related deaths were further classified into those in which opioids were specifically mentioned as the underlying or contributing cause of death. Drug-related deaths that were classified as intentional were to have been counted under suicide deaths, but there were no such deaths in the cohort.

Mortality rates and standardised mortality ratio

Crude mortality rates (CMR) were calculated by dividing the number of deaths by the number of person–years contributed by participants, and multiplying by 1,000 to obtain results in terms of deaths per 1,000 person–years of follow-up. The all-cause CMR and the opioid-related CMR were calculated.

For comparison with the general population, an indirectly standardised mortality ratio (SMR) was calculated. The SMR is the ratio of observed deaths to expected deaths, with an SMR of greater than one reflecting elevated mortality in the observed group. Data on expected deaths were calculated from age, sex and calendar-specific mortality rates in New South Wales for the years 1997–2006, available from the ABS[5].

Mortality in relation to custodial episodes and opioid substitution treatment

To assess how risk of death may vary in relation to imprisonment and OST, crude mortality rates and rate ratios (Degenhardt et al. 2009) were calculated for:

- in treatment versus out of treatment. In keeping with previous work addressing mortality in relation to OST, when calculating CMRs for periods in and out of treatment, the six days following the end of a treatment episode were classed as time in treatment (Degenhardt et al. 2009);
- periods in custody versus at liberty. Deaths in custody were defined as those participants with a final OIMS release code of 'deceased'; and
- the first 28 days post-release versus all other time at liberty. Twenty-eight days was chosen as the expected period of elevated mortality risk in line with the recommendation of Odergard (2010). For deaths in the first 28 days post-release, causes of death and OST status at time of death were noted.

Results

There were 28 deaths among the 375 participants (7.5%). The median age at death was 31 years (range 25–47).

Causes of death

Half (14/28; 50%) of the recorded deaths were drug-related; of these, 10 were opioid related. There were three suicide deaths, all by hanging. Additionally, one death was coded as an accidental hanging. This individual was in prison at the time of death and given the common use of hanging as a method of suicide in prison, it is possible that this death was in fact a suicide. Other accidents (eg motor vehicle accidents, accidental drowning) accounted for four further deaths. Two deaths were due to cancer and one death each was due to alcoholic cirrhosis of the liver, epilepsy and homicide. Finally, there was one death in which the participant died after being hit by a moving vehicle, but intent (ie whether accidental or intentional self-harm) could not be determined. Causes of death are summarised in Table 5.1.

Table 5.1 Causes of death

Cause of death	n
Accidental drug-related	14
Accidents	5
Suicide	3
Violence	1
Other	5
Total	28

5 http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3302.02007

Crude mortality rate and standardised mortality ratio

Participants were observed for 3,607.6 person–years, making for an all-cause CMR of 7.8 per 1,000 person–years (95% CI 5.3–11.1). Considering only opioid-related deaths (n=10), the CMR was 2.8 per 1,000 person–years (95% CI 1.4–4.9).

Compared with the NSW population, all-cause mortality was significantly elevated in the cohort over the length of the follow-up period, with participants dying at 6.1 times the rate of men of the same age (95% CI 4.1–8.6; $\chi^2 = 118.3$, $p<.001$) (see Table 5.2).

Table 5.2 Annual and total standardised mortality ratio, 1997–2006

	Recorded deaths	Expected deaths	SMR	95% CI
1997	1	0.51	2.0	0.1–9.7
1998	7	0.53	13.3	5.8–26.3
1999	1	0.52	1.9	0.1–9.6
2000	4	0.48	8.4	2.7–20.3
2001	1	0.48	2.2	0.1–10.8
2002	6	0.44	13.7	5.6–28.5
2003	1	0.40	2.5	0.1–12.3
2004	0	0.41	0	–
2005	4	0.44	9.1	2.9–22.0
2006	3	0.46	6.6	1.7–17.9
Total	28	4.62	6.1	4.1–8.6

SMR=Standardised mortality ratio

CI=Confidence interval

Timing of deaths in relation to imprisonment and treatment

Six participants died while in OST. Cause of death in three cases was opioid overdose; the remaining deaths were due to cancer, a motor vehicle accident and suicide. The ratio of the out-of-treatment CMR over the in-treatment CMR showed that the risk of death while out of treatment was 2.4 times that while in treatment ($p=0.04$) (see Table 5.3). There were three deaths in custody, as identified by release codes of 'deceased' in the custodial data. Cause of death in two cases was hanging; the third death was a homicide. Participants were six times more likely to die while at liberty than while in prison ($p<0.001$) (see Table 5.3).

There were four deaths within 28 days of release from custody. Causes of death were accidental opioid overdose (6, 22 and 27 days post-release) and cancer (3 days post-release). The post-release overdose deaths all occurred while participants were not in treatment. Although the CMR in the first 28 days post-release was elevated compared with that during all other time at liberty, the difference was not statistically significant see (see Table 5.3).

Table 5.3 Crude mortality rates and rate ratios

	Deaths	Person–years	CMR per 1,000 py	95% CI	Rate ratio	95% CI	p
Treatment							
Out of treatment	22	2,163.2	10.2	6.5–15.2			
In treatment	6	1,444.4	4.2	1.7–8.6	2.4	1.0–6.6	0.04
Imprisonment							
At liberty	25	2,114.5	11.8	7.8–17.2			
In prison	3	1,493.1	2.0	0.5–5.5	5.9	2.0–24.5	<0.001
Post-release^							
First 28 days post-release	4	146.2	27.4	7.4–70.0			
Remainder of time at liberty	21	1,968.3	10.7	6.7–16.3	2.6	0.8–7.0	0.1

^Deaths post-release sum to 25 as 3 participants died in custody

Discussion

The crude mortality rate and standardised mortality ratio for this cohort were similar to those seen in other cohorts of Australian heroin users (Degenhardt et al. 2009; Stoove et al. 2008). Causes of death were also similar and highlight the risks associated with illicit drug use and criminality. Half of the 28 deaths were directly attributable to accidental drug overdose; 'natural' deaths (ie not a result of substance use, accident or violence) were rare.

As in previous studies, being in OST was protective against death, with the risk of death while in treatment around 40 percent that out of treatment. Being in prison was associated with a greatly reduced risk of death. This is in line with other research on prisoners both in Australia (Kariminia et al. 2007b) and internationally (Bobrik et al. 2005). It appears that while in prison, heroin users are protected against some of the common causes of death in this group, such as overdose and accidents.

Although the crude mortality rate was higher in the first 28 days post-release than at all other time at liberty, this difference was not statistically significant. It is of note that three of the four post-release deaths were opioid overdoses, highlighting the risk of overdose death during this period and that none of the deceased were in OST prior to their deaths.

Limitations

Given the small sample size, it was not possible to conduct a meaningful analysis of predictors of mortality, including the role of prison OST in moderating risk of death in the period immediately after release from prison. Larger studies with sufficient statistical power to detect a change in mortality rates associated with prison OST may be desirable; however, it has been calculated that a study with 80 percent power to detect an effect would require a sample size of 80,000 (Bird 2009). It is highly unlikely that a single study could achieve a sample size this large. Despite the lack of unequivocal answer, it is known that OST reduces mortality and there is no apparent reason why this effect would not hold for inmates being released from prison. The safety and risks of OST in general are established and there is no reason to assume that prison OST would have hitherto unknown mortality risks. Thus, it is reasonable to assume that prison OST would reduce post-release mortality risk, either by reducing risk of heroin use, or, in the event of heroin use, reducing the risk of overdose.

Conclusions

The results of this study accord with the findings of other cohort studies in terms of overall mortality risk (Degenhardt et al. 2009; Stoove et al. 2008) and reduced risk while in treatment (Degenhardt et al. 2009). There were some indications that the post-release period was a time of elevated risk of death, particularly from drug overdose, but the difference between mortality rates in the post-release period and all other time at liberty did not reach statistical significance. Research to date has not definitively shown that OST in prison reduces the risk of post-release overdose; however, there is sufficient evidence of the benefits of OST in relation to mortality to make this a reasonable assumption.

6. CONCLUSION

Summary of findings

This report has focused on a cohort of heroin users who are heavily involved in crime and who spent more than twice as much time in prison (median 3.6 years) as in opioid substitution treatment (1.6 years). Commencing OST while incarcerated was common, with 80 percent of participants commencing at least one treatment episode in prison.

When released from prison, the participants in this study were re-incarcerated much faster and more frequently than the general NSW prisoner population (SCRGSP 2010). Risk of re-incarceration was reduced by an average of 20 percent while participants remained in OST post-release. No effect of prison or post-release OST on reoffending was identified; however, it is possible that the use of convictions, rather than arrests or criminal charges, may have produced overly conservative results. Finally, mortality was significantly elevated in this cohort compared with their age- and sex-matched peers. Risk of death was lower while in OST and while in prison. There were four deaths within 28 days of release from prison (crude mortality rate 27.4 per 1,000 person–years), of which three were opioid overdoses; none of these participants were in OST at the time of their death.

Implications of findings

Restrictions on prison OST prevent treatment entry at a time when demand for treatment is high

Although the frequency of commencing OST in prison may reflect a desire to avoid opioid withdrawal and cravings rather than the desire to cease illicit opioid use, it remains true that over half of the treatment episodes in this cohort were commenced in prison. Treatment exposure in prison reduces needle and syringe sharing (Dolan et al. 2003; Heimer et al. 2006; Larney 2010) and according to the data presented in this report, when inmates remain in treatment post-release they have a significantly reduced risk of re-incarceration.

Currently, in Queensland, Tasmania and the Northern Territory, prisoners cannot commence OST while in prison (AIHW 2010a). Preventing heroin-using inmates from accessing treatment is inequitable and ignores the facts that demand for treatment is high in prisons and the potential benefits of treatment in prison are significant.

OST in prison and post-release can potentially reduce the size of the prisoner population

Modelling studies suggest that even minor reductions in re-incarceration among groups of offenders with high re-incarceration rates can have significant impacts on the size of the overall prisoner population (Weatherburn et al. 2009). It was reported in Chapter 3 that remaining in OST post-release reduced the average risk of re-incarceration; therefore, ensuring released inmates are engaged with OST in the community will benefit not only the individual, but also the broader correctional system.

Although post-release retention in treatment is the key to reducing re-incarceration, this does not mean that there is no need to provide OST while in prison. Post-release participation in OST is maximised when treatment is commenced while in prison (Kinlock et al. 2007; Magura et al. 1993; Tomasino et al. 2001) and there are other benefits of OST in prison, such as reduced HIV risk behaviours (Dolan et al. 2003; Larney 2010). Hence, the maximum benefits from OST can be obtained by providing treatment while in prison and facilitating a smooth transition to a post-release treatment provider.

OST alone may not affect reoffending

Effects of prison OST on offending were not identified, but there was some evidence that the use of convictions as an outcome measure and the resultant use of re-incarceration as a censoring event may have biased the analysis. It is also important to consider that OST in prison addresses only one of the risk factors for reoffending (ie substance use) (Andrews et al. 2006) and it would be worthwhile evaluating the extent to which reoffending is reduced by a combination of OST and therapeutic programs that address non-drug related risk factors for offending.

OST protects against death, and is likely to reduce the risk of post-release drug overdose

Very large sample sizes (c. 80,000) would be required to unequivocally state that OST in prison reduces the risk of death in the post-release period (Bird 2009). In the absence of data specific to this issue, the general protective effects of OST in relation to mortality (Degenhardt et al. 2009) can reasonably be assumed to apply to post-release inmates. Prison OST may reduce post-release mortality by reducing the risk of relapse to heroin use, or, in the event of heroin use, by reducing the risk of overdose (Christensen et al. 2006). Correctional authorities must carefully consider the role that prison OST may be able to play in reducing the high risk of drug overdose death that is seen in the period following release from prison (Kariminia et al. 2007b).

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APPENDIX A: DATA LINKAGE THEORY AND METHODS

Longitudinal research has been crucial in establishing the chronic and relapsing nature of drug dependence and in quantifying the risks of long-term substance use (Hser, Longshore & Anglin 2007). A common approach to longitudinal research has been to establish a cohort of participants who provide baseline data and are then re-interviewed at set follow-up points. One of the longest running studies of this design consists of a cohort of heroin users originally recruited between 1962 and 1964, with follow-up interviews conducted 10, 24 and 33 years after recruitment (Hser, Anglin & Powers 1993; Hser et al. 2001; McGlothlin, Anglin & Wilson 1977). Findings from this cohort have included the identification of distinct trajectories of heroin dependence (Hser et al. 2007) and quantification of the number of years of potential life lost due to heroin dependence (Smyth et al. 2007), as well as theoretical (Hser et al. 2007) and statistical (Chou et al. 2004) developments in longitudinal research methods.

Although this approach to longitudinal research has been popular, re-contacting and personally interviewing participants can be expensive and time-consuming. Furthermore, it may be difficult at follow-up to locate sufficient numbers of participants to enable robust conclusions about changes over time to be drawn. This is particularly the case in illicit drug research, as participants often lack reliable contact details such as a stable address or telephone number. Finally, there can be issues of recall bias when participants are asked to report on past events. Day and colleagues asked heroin users to recall their drug use, drug treatment status and criminal activity over four discrete time periods during the previous two years. Participants were re-interviewed seven days later to assess the reliability of responses. They found that although recall of heroin use was generally reliable, recall of other activities was variable and worsened as participants were asked to recall more distant time periods (Day et al. 2004).

An alternative, or complementary, data source that overcomes these issues is administrative data. These are existing data that are routinely collected for management or other non-research purposes (Evans et al. 2008). Administrative data sources include hospital admissions, police arrest records and registries of births and deaths. One of the key advantages of administrative data over self-reported, retrospectively collected data is that administrative data provide an 'official' record of events as they happen; there is no reliance on participants to accurately recall past events. Although administrative databases may contain errors, these are likely to be random (eg transposing of numbers in a date field) rather than related to participant characteristics.

Data linkage

The utility of administrative data in answering research questions is greatly enhanced by linking data for individuals across different databases. Records from each source database are linked based on information common to each database, such as names or dates of birth, enabling analysis of events across services and systems. For example, Amin et al. (2006a, 2006b) and colleagues linked hepatitis B and hepatitis C notifications with the NSW Central Cancer Registry and the Australian National Death Index, allowing examination of incidence of liver cancer and causes of death among people with viral hepatitis.

Data linkage methods

There are two distinct methods used for data linkage—deterministic and probabilistic. Deterministic linkage involves matching records based on exact agreement between key variables. Consider the example data in Table A.1. In a deterministic linkage based on surname, given name, and date of birth, only record 1 from dataset A and record 3 from dataset B would be considered a matched pair.

Table A.1 Example data

	Dataset A				Dataset B		
	Surname	Given name	Date of birth		Surname	Given name	Date of birth
1	Blakely	Toni	12/04/78	1	Johnston	Anthony	11/03/76
2	Johnson	Anthony	03/11/76	2	Middleton	Bill	05/02/74
3	Middleton	William	02/05/74	3	Blakely	Toni	12/04/78

Deterministic linkage is precise; it links only those records that match exactly. In deterministic linkage, it is assumed that the data entered into each source dataset are wholly accurate. In reality, this assumption may not hold. Typing errors, variation in the spelling of names and transposing of numbers are just some sources of variation when data are entered. In the data in Table A.1, despite minor variations in names and dates of birth, there is a high probability that record A2 is a match for record B1 and that record A3 is a match for record B2. In order to identify such likely, but not exact, matches between records, it is necessary to use probabilistic data linkage.

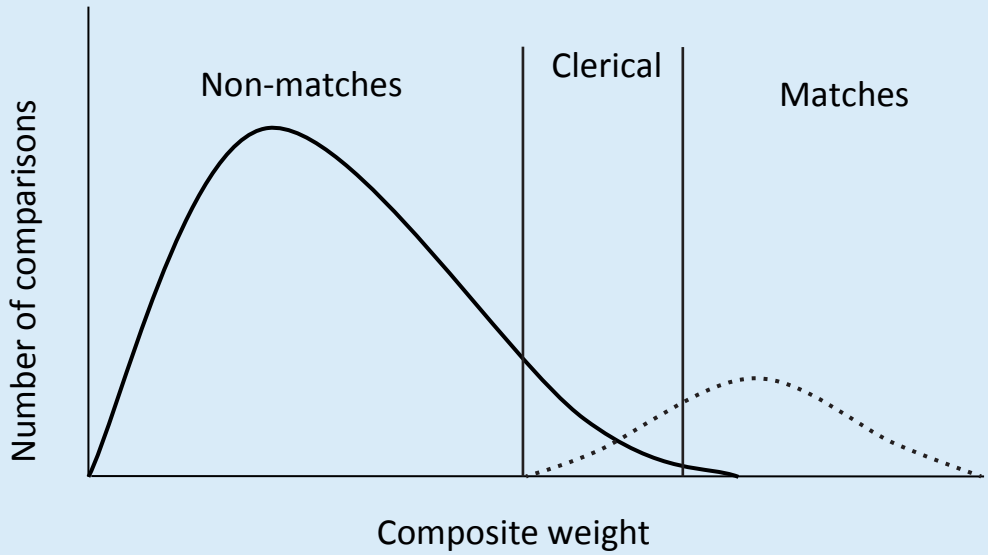
Probabilistic data linkage

In probabilistic data linkage, each pair of records (consisting of one record from dataset A and one record from dataset B) is classified as belonging to one of two sets—matched pairs or non-matched pairs. To do this, record x from dataset A is compared to each record in dataset B, and each resulting pair (x–B1, x–B2, etc.) is classified as either a match or a non-match. In determining whether two records are a match, allowances are made for variations in spelling of names (eg Melissa and Mellissa), common variants of names (eg Bob and Robert) and other sources of variation that may obscure the fact that two records belong to one individual.

Each field (eg surname, date of birth) shared by datasets provides information to assist with determining which pairs are matches and which are non-matches. However, some fields provide more information than others. For example, a postcode field is less informative than a surname field for matching purposes. To account for this, fields are weighted. To determine weights, each field is assigned two probabilities. The ‘m’ probability is the probability that a field agrees, given that the pair being examined is a matched pair. The ‘u’ probability is the probability that a field agrees, given that the pair being examined is an unmatched pair. That is, ‘u’ is effectively the probability that the field agrees at random. The weight for a field is calculated as a ratio of ‘m’ and ‘u’, with fields that provide more information in determining a match (ie fields in which ‘u’ is low) receiving higher weights (Jaro 1995).

For each pair, a composite weight, consisting of the sum of the field weights, is calculated. The distribution of weights is then used to determine an appropriate cut-off weight, above which pairs are accepted as matches, and below which pairs are taken to be non-matches. Often, a cut-off range is defined, with pairs that fall within the range subject to clerical review to determine whether the pair should be considered a match or non-match (see Figure A.1).

Figure A.1 Typical distribution of weights and cut-off points for non-matched, matched and clerical review pairs



Ultimately, linkage using probabilistic methods involves a trade-off between false positives (incorrect matches) and false negatives (incorrect non-matches). Another way to conceptualise this is to consider whether it is more important for the research at hand to prioritise sensitivity (correctly identifying matches) or specificity (correctly rejecting non-matches) (Blakely & Salmond 2002). The sensitivity and specificity of a linked dataset can be estimated if true links are known for a proportion of the sample. In the absence of known true links, it is possible to calculate, based on the weight of a comparison pair, the odds of that pair being a true match. This is known as the positive predictive value; however, this method is prone to bias and is unsuitable for use when a record from one dataset can match to multiple records in another dataset. If it is not possible to calculate the sensitivity and specificity or positive predictive value, it is generally recommended that a conservative approach be taken that minimises the number of false positives, thereby maximising specificity (Blakely & Salmond 2002; Howe 1998).

APPENDIX B: ASSESSMENT OF LINKAGE SENSITIVITY AND SPECIFICITY

Data linkage is based on the probability that two records from disparate datasets belong to the same individual. As such, there is potential for error in the linkage process, which reduces the accuracy of the linkage. There are two aspects to linkage accuracy: sensitivity and specificity. Sensitivity refers to the extent to which a linkage correctly detects matches, while specificity is the extent to which a linkage correctly rejects non-matches (Blakely & Salmond 2002). Sensitivity and specificity are calculated by comparing linkage outcomes to known matches, with known matches assumed to be 100 percent accurate. For example, in the only published study of sensitivity and specificity of a linkage between an Australian prison population and a health dataset, true mortality status was able to be determined for a subset of inmates who were either currently alive and in prison, or had died in custody. The true mortality was compared with matches that had been obtained through linkage of OIMS and the National Death Index. Sensitivity was 88.4 percent and specificity was 99.7 percent (Kariminia et al. 2005); that is, the linkage correctly identified 88.4 percent of known deaths and only 0.3 percent of individuals known to be alive were incorrectly linked to a mortality record. It was assumed that although based on a subset of the total sample, these sensitivity and specificity estimates were applicable to the entire sample.

Sensitivity and specificity of linkage processes varies between studies. In a systematic review of health data linkage studies, sensitivities ranged from 74–98 percent, while specificities were 99–100 percent (da Silveira & Artmann 2009); however, what is considered an acceptable level of sensitivity and/or specificity in one study cannot be extrapolated to other studies. Rather, the sensitivity and specificity of a linkage are used to assist in evaluating the reliability of results that are obtained from analysis of the linked data.

Often, it is not possible to conduct a sensitivity and specificity analysis, as data that contain 'known links' for at least some of the population under study cannot always be identified. Indeed, if such data existed, there may not be any need for linkage. Many health-related data linkage studies have been published without any analysis of the accuracy of the linkage process (da Silveira & Artmann 2009); however, in studies using general population samples, it is unlikely that any significant proportion of the sample has deliberately obscured their identity by using an alias when, for example, presenting to hospital. As such, it is assumed that the linked data are reasonably accurate.

It is unlikely that this assumption holds in relation to research with criminally involved populations, where participant aliases are a very real complication for data linkage. For example, in a study of cervical cancer in female inmates, a prison database was linked to Canada's Cervical Cancer Screening Program records. Half of the inmates had three or more names, raising concerns that aliases would increase the likelihood of false matches when linked to a general population database such as a cancer screening registry (Martin et al. 2004). To address this issue, inmates with five or more surnames, or four or more given names, were excluded from the analysis. However, this resulted in disproportionate exclusion of Canadian Aboriginal women and women with lower education levels (Martin et al. 2005), potentially affecting the reliability of the research findings.

In the prisoner-mortality linkage described above, each individual had an average of two aliases and all aliases were used in the linkage process (Kariminia et al. 2005). Using only the inmates' birth names for linkage may have resulted in a less sensitive linkage, as true matches may have been missed; however, using aliases may reduce specificity, through increased false matches. In light of the potential for aliases to influence the accuracy of a linkage, it becomes important to formally assess accuracy.

In the present data linkage studies, information on the date that a participant in the cohort entered treatment as part of the randomised controlled trial was recorded in the baseline dataset. By comparing these dates to the treatment entry dates recorded in PHDAS, it was possible to calculate the sensitivity and specificity of the linkage between the cohort and PHDAS.

Method

Data sources

The data sources used in this chapter were the baseline dataset and PHDAS data. Beyond the demographic variables used to describe the cohort, the main variables of interest were the dates of OST entry as recorded in each dataset.

Data analysis

For the length of the original trial (August 1997 to July 2000), the baseline dataset recorded the date on which any participant in the trial started an episode of OST. These dates were then taken as known matches and were compared to dates of OST entry that were recorded in PHDAS (ie linked matches). Data manipulation and statistical analyses were conducted in SAS 9.1.

Sensitivity was calculated as the proportion of participants with a treatment entry date in the trial dataset who were also found to have entered treatment on that date in PHDAS. This was a relatively simple process, requiring only matching of two dates. Calculating specificity was somewhat more complex, as this required matching of two 'non-dates'—the date the person did not enter treatment in the trial database, and the date they did not enter treatment in PHDAS.

It was not possible to take the date of trial entry as the date on which the person did not enter treatment and then check that the person also did not enter treatment on that date in PHDAS. This was because among those participants who did commence treatment, there was typically a delay between entering the trial and starting treatment. Although there were some participants who experienced delays of more than a month before commencing treatment, 90 percent of those who were assigned to receive treatment (and then commenced treatment) did so within 20 days of entering the trial. Therefore, it was assumed that if the person did not have a treatment entry date in the baseline dataset, they should also not have a treatment entry date in PHDAS at any time between their date of trial entry and the subsequent 20 days. Hence, specificity was calculated as the proportion of participants without a treatment entry date in the baseline dataset who were also found in PHDAS to have not entered treatment on their day of entry to the trial or within the next 20 days. Precision of the sensitivity and specificity estimates was measured using exact binomial confidence intervals.

To assess for any systematic bias in the linkage, participants with matching baseline and PHDAS records were compared with those who entered treatment but did not have a matching PHDAS record. A t-test compared matched participants to non-matched participants on age, while chi-square tests were used to compare matched participants to non-matched participants on Indigenous status and trial group (treatment vs control).

Results

The baseline dataset recorded treatment entry dates for 225 of 375 participants. Due to circumstances beyond the control of the original investigators (eg the participant was unable to be placed in a prison offering OST), 17 participants randomised to the treatment arm did not commence treatment during the study period. Fifty-five participants randomised to the control arm of the study commenced treatment after being placed on the waiting list.

Two dates were considered to be data entry errors as they occurred before the start of the trial. These two records were excluded from the analysis, leaving 223 participants with, and 150 participants without, baseline treatment entry records (see Table B.1). Treatment entry dates ranged from 15 August 1997 to 24 July 2000.

Table B.1 Distribution of treatment entry records in baseline dataset

		Treatment entry recorded in baseline dataset		
		No	Yes	Total
Study group	Treatment	17	170	187
	Control	133	53	186
	Total	150	223	373

Of the 223 participants with treatment entry dates, 192 (151 treatment and 41 control) had a matching treatment entry record in PHDAS, for a sensitivity of 86.1 percent (95% CI 80.8%–90.4%).

Of the 150 participants without a treatment entry date in the baseline dataset, none had a PHDAS-treatment entry date in the 20 days following trial recruitment, for a specificity of 100 percent (one-sided lower 97.5% CI 97.5%).

Participants with matching baseline and PHDAS records were compared with those who entered treatment but did not have a matching PHDAS record. There were no differences in age or Indigenous status, but participants who had been assigned to the control group (but still commenced treatment) were less likely than participants assigned to the treatment group to be correctly matched to a PHDAS record (see Table B.2).

Table B.2 Comparison of matched and unmatched records on age, Indigenous status and trial group

	Matched	Not matched	Significance
Age (years)	27.1	28.6	ns
Indigenous status n (%)			
Indigenous	46 (86.8)	7 (13.2)	
Non-Indigenous	146 (85.9)	24 (14.1)	ns
Trial group n (%)			
Treatment	151 (88.8)	19 (11.2)	
Control	41 (77.4)	12 (22.6)	χ^2=4.4, df=1, p=.04

ns=not significant

Discussion

Assuming the baseline dataset to be accurate, linkage to PHDAS opioid substitution treatment data correctly identified 86 percent of treatment entrants, without any incorrect matches. It is generally accepted that data linkage is unlikely to be 100 percent accurate (da Silveira & Artmann 2009) and 86 percent is an acceptable sensitivity, particularly given the common use of aliases in this population (Kariminia et al. 2005). It can be inferred from the sensitivity and specificity findings that OST episodes for this cohort have largely been accurately ascertained.

In considering those participants known to have entered treatment during the trial but not matched to PHDAS, participants originally assigned to the treatment group were more likely to be correctly matched to PHDAS than those in the control group who entered treatment after moving through the waiting list. It is possible that when a participant commenced treatment as part of the research trial, greater care was taken to ensure the participant's details were accurately recorded for PHDAS than when a participant commenced treatment external to the trial, affecting the extent to which control participants were accurately matched to PHDAS. On demographic variables, there was no difference in the age or Indigenous status of matched versus non-matched participants, suggesting the linkage was unbiased in relation to these variables. Assuming the sensitivity and specificity calculated for this time period are generalisable to the entire follow-up period, the data extracted from PHDAS are a reliable record of OST episodes for this cohort.

APPENDIX C: DETAILS OF STATISTICAL ANALYSES

Modelling recurrent time-to-event data

The data analysed in Chapters 3 and 4 were time-to-event data. Survival analysis using the Cox proportional hazards model is the standard approach to modelling such data (Cox 1972); however, this approach only permits analysis of time to a single event of interest per participant. In the data to be analysed here, participants experienced the events of interest—re-incarceration and criminal convictions—multiple times over the follow-up period. Modelling recurrent event data such as these requires statistical models that account for correlations of event times within individuals. There are several possible models to choose from and choice of model is guided by the research question that is to be answered (Guo, Gill & Allore 2008; Hosmer & Lemeshow 1999; Lim, Liu & Melzer-Lange 2007).

For the recurrent event analyses in this report, the Prentice-Williams-Peterson gap-time (PWP-GT) model (Prentice, Williams & Peterson 1981) was determined to be the most appropriate model. The PWP-GT model is an extension to the Cox model in which dependence of event times within individuals is accounted for by stratifying the analysis by event number (Ezell, Land & Cohen 2003; Guo, Gill & Allore 2008; Hosmer & Lemeshow 1999; Lim, Liu & Melzer-Lange 2007). In addition, the standard errors of the parameter estimates are adjusted using a robust sandwich variance estimator (Ezell, Land & Cohen 2003; Lin & Wei 1989).

Counting time in the model

A key aspect of the model is the manner in which time is counted. Most models for recurrent event data count time continuously, from zero to the end of the observation period; however, in gap-time models, time is 're-set' when an event occurs. Thus, each time interval begins at zero, and continues until the event of interest (Hosmer & Lemeshow 1999). This allows modelling of time between events, rather than time to each event from the beginning of the observation period (Box-Steffensmeier & Zorn 2002). This is particularly useful for analysis of criminal justice populations, as it allows modelling of multiple periods of time from prison release to an event of interest, while excluding time spent in prison.

Model stratification

The PWP-GT model is stratified by event number to control for dependence of event times within individuals. If there are a limited number of individuals with higher-order events, the estimates for these strata may be unstable and imprecise (Box-Steffensmeier & Zorn 2002; Ezell, Land & Cohen 2003). There are two methods for dealing with the problem—strata for higher-order events are combined into a single stratum for analysis, or the analysis is restricted to lower-order events (Therneau & Grambsch 2000). Rather than discard data, it was decided that strata with fewer than 10 percent of participants included would be combined into one stratum for analysis.

Model interpretation

Although often referred to as a conditional model (Box-Steffensmeier & Zorn 2002; Ezell, Land & Cohen 2003; Guo, Gill & Allore 2008; Hosmer & Lemeshow 1999), the PWP gap-time model is a marginal model in the traditional statistical sense because the parameter estimates are calculated without participant-specific effects (Ezell, Land & Cohen 2003; Therneau & Grambsch 2000). As such, results are interpreted as population average effects, rather than participant-specific effects (Ezell, Land & Cohen 2003).

Informative censoring

For the purposes of the analyses in Chapter 4, re-incarceration was defined as a censoring event. It had been assumed prior to conducting these analyses that all re-incarcerations would be preceded by an offence

date and therefore that all release intervals that did not end in death or at the end of the study period would end with an offence. Once the data were linked, it became apparent that this was not the case. Only data on proved offences were obtained for the study. Thus, it was possible for individuals to appear in the prison data following an arrest for an offence, but, if the offence was not proved, it would not appear in the offending data. As a result, there were a number of release episodes which ended with re-incarceration, but the participant had not experienced an offending event as defined for this study. In order to avoid treating time in prison the same as time at liberty in terms of risk of new conviction, these release intervals were censored on the day of re-incarceration.

The use of re-incarceration as a censoring variable potentially introduces the problem of 'informative censoring' to the analysis. Informative censoring occurs when the time to censoring is related to the outcome of interest, potentially biasing results (Clark et al. 2003). In this case, it is possible that re-incarceration was related to offending behaviour that resulted in arrest, but not conviction.

It is difficult to identify and assess the impact of informative censoring. The standard approach is to conduct a sensitivity analysis in which two models are generated, each under a different extreme assumption (Allison 1995; Clark et al. 2003; Collett 2003). If the model results under each of these assumptions are broadly similar to those of the original model, it can be assumed that the results are not sensitive to the presence of informative censoring (Collett 2003).

The first sensitivity analysis assumption is that censored observations actually experienced an event at their censoring time. This assumption is based on the hypothesis that those who are censored were actually at high risk of experiencing the event of interest. The second assumption is that censored observations, although still censored, were observed as long as the longest time to event in the sample. This assumption is based on the hypothesis that those who were censored were at low risk of the event of interest (Allison 1995; Collett 2003). Allison (1995) notes that in many cases, one of these assumption may be more plausible than the other, in which case greater attention may be given to the assumption that is more likely to affect the data at hand. In this case, the first assumption—that people censored because of re-incarceration had actually committed an offence—is more likely than the second. This second assumption could not be implemented without interfering with the recurrent event structure of the data, so this analysis was not conducted.

APPENDIX D: ICD-10 CODES FOR DRUG-RELATED, VIOLENT, ACCIDENTAL AND SUICIDE DEATHS

Table D.1 ICD-10 codes for drug-related, violent, accidental and suicide deaths

Cause of death	ICD-10 codes	Definition
Accidental drug-related	F10–F19	Mental and behavioural disorders due to psychoactive substance use
	X40–X45	Accidental poisoning by and exposure to noxious substances
	Y10–Y14	Poisoning by and exposure to noxious substance, undetermined intent
Accidental drug-related: opioids specified	F11	Mental and behavioural disorders due to use of opioids
	F19 *and* F11	Mental and behavioural disorders due to multiple drug use and use of other psychoactive substances *and* mental and behavioural disorders due to use of opioids
	X42 *and* T40.0–T40.4 or T40.6	Accidental poisoning by and exposure to narcotics and psychodysleptics, not elsewhere classified *and* poisoning by narcotics and psychodysleptics
	X44 *and* T40.0–T40.4 or T40.6	Accidental poisoning by and exposure to other and unspecified substances *and* poisoning by narcotics and psychodysleptics
	F19 *and* T40.0–T40.6 or T40.6	Mental and behavioural disorders due to multiple drug use and use of other psychoactive substances *and* poisoning by narcotics and psychodysleptics
Violence	X85–Y09	Assault
	Y87.1	Sequelae of assault
Accidents	V01–V99	Transport accidents
	W00–X39, X50–X59	Other external causes of accidental injury
Suicide	X60–X84	Intentional self-harm
	Y87.0	Sequelae of intentional self-harm

Source: Randall et al. 2009