DRUG FREE AUSTRALIA

TWO DRUG INTERVENTIONS FAILING TO DEMONSTRATE SCIENTIFIC EFFECTIVENESS

ACT's contemplated directions for injecting rooms and pill testing must be according to evidence-base science

1. **The only rigorous systematic review of injecting rooms to date, where one study found reductions in overdoses at the community level (Canada), reductions in ambulance callouts (Australia – one study) and reductions in crime (Canada – one study) relies entirely on discredited research**
    a. **The Canadian study concealed a tripling in police numbers with Insite's introduction which removed drug dealers and associated overdoses to other areas of Vancouver, and which entirely explains any reduction in overdoses**
    b. **The Sydney study recorded a 31% reduction (against control) in ambulance callouts by day, but a 70% reduction at night. This demonstrates that the reductions were not due to the injecting room which is a daytime operation**
    c. **The Canadian study recording reductions in crime concealed the tripling in police numbers at Insite's introduction. It also concealed changes in policing policy away from a philosophy of 'containment' to one of 'zero tolerance.' All reductions in crime are explained by the changed policing methods**
2. **The only studies on ecstasy deaths in Australia indicate that ecstasy itself causes almost every pill death, while pill testing does in fact promote ecstasy use – the very substance causing almost all Australian party pill deaths**

Central Issues
&
Compiled Evidence

DRUG FREE AUSTRALIA

THE SCIENCE DEMONSTRATING INEFFECTIVENESS

Executive Summary

1. **The science on injecting rooms shows no demonstrated effectiveness across all legislated outcomes**

   The only rigorous review on injecting rooms to date found reductions in

   1. **overdoses**
   2. **ambulance callouts**
   3. **crime**

   However, Drug Free Australia has irrefutably demonstrated that the Vancouver study on overdose reductions it relies on is contradicted by Vancouver's official statistics. Additionally the then Police Commander John McKay recounts a permanent tripling of police numbers with Insite's introduction and a permanent change in policing philosophy from one of 'containment' to one of 'zero tolerance' at that time. The policing displacing drug dealers, drug buyers and their overdoses to other areas of Vancouver fully explains the 35% reduction in overdoses found around Insite.

   The Australian study on reduced ambulance callouts failed to note that there were superior reductions at night when measured against the control area of the rest of NSW (70% reduction) when the injecting facility was closed compared to the hours it was open (31% reduction against control), thus discrediting its conclusions.

   The study that claimed reduced crime in Vancouver concealed the seismic changes in policing and tripling of numbers, thus falling to the same criticisms levelled against the above study on reduced overdoses.

   Thus no positive outcomes have been demonstrated for injecting rooms in rigorous scientific studies.

   The recent June 2020 review of the Melbourne MSIR shows that the facility failed against all legislated outcomes, while simultaneously increasing crime in

the North Richmond area such that there is a complaint regarding these increases on the Victorian Police Association website.

2. **The only studies on ecstasy deaths in Australia indicate that ecstasy itself causes almost every pill death, while pill testing does in fact promote ecstasy use – the very substance causing almost all deaths**

Pill testing doesn't address the causes of ecstasy deaths:

1. **It cannot identify individual vulnerabilities to ecstasy that cause deaths**
2. **It doesn't identify other co-used drugs such as alcohol or amphetamines which make ecstasy deadly**
3. **It can't identify which ecstasy user will have an ecstasy-fuelled accident (mostly car accidents)**

The evidence supporting the failure of both interventions is found in the following pages

Table of Contents

EVIDENCE DEMONSTRATING INEFFECTIVENESS - 1

INJECTING ROOMS

The science on injecting rooms shows no demonstrated effectiveness across all legislated outcomes

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2. **ambulance callouts**
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outcomes, while simultaneously increasing crime in the North Richmond area such that there is a complaint regarding these increases on the Victorian Police Association website.

The science of injecting room ineffectiveness

Reviews of scientific evaluations of SIFs (Kerr et al., 2007; McNeil and Small, 2014; Potier et al., 2014; Garcia, 2015; Kennedy, Karamouzian, and Kerr, 2017; May et al., 2018 (retracted); Kilmer et al., 2018), have reported positive outcomes across a range of evaluated criteria, **but most have used studies which methodologically fail to demonstrate the effectiveness of SIFs to alter individual or population-level outcomes**. Just two reviews, May et al. 2018 and Kilmer et al. 2018 (RAND Corporation) included only studies with a quasi-experimental design using control groups/areas, with May et al. subsequently being retracted because of "methodological weaknesses linked to the pooling of diverse outcomes into a single composite measure" (International Journal of Drug Policy, 2018) but not for its selection criteria of high-quality studies on Safe Injection Facility effectiveness.

The RAND Corporation similarly identified nine studies with quasi-experimental design, noting that four of the earlier studies had been superseded by others within the remaining five which studied the same outcomes with longer time series in the same locations. This effectively reduced the available number of reviewed studies to just five which are limited to overdose-related outcomes, discarded injecting equipment and crime. These studies examined SIFs in only three cities – Sydney, Vancouver and Barcelona, where the latter's two studies gave conflicting results.

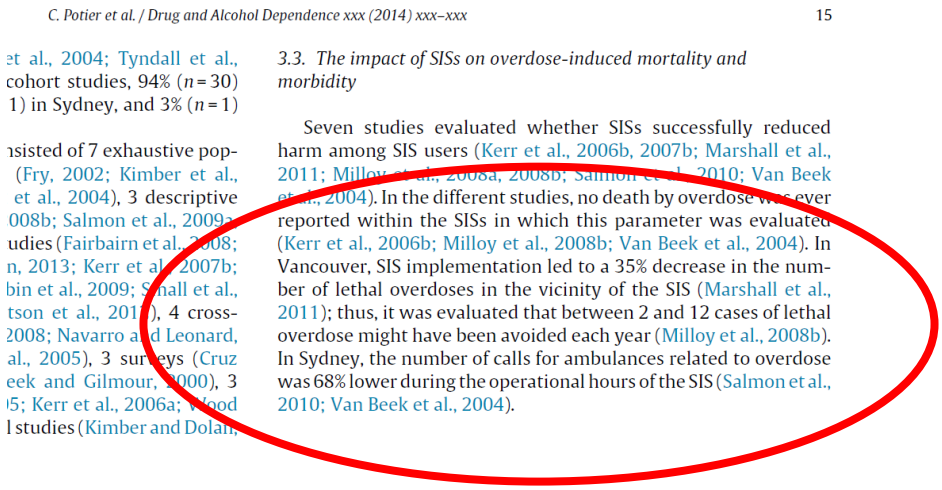
Of these five studies:

- Marshall et al. found a 35% reduction in opiate overdose fatalities in the immediate area surrounding Vancouver' s Insite
- Salmon et al. 2010 found a greater reduction in ambulance callouts for overdose in the Kings Cross postcode housing the Sydney MSIC than for the rest of New South Wales
- Donnelly and Mahoney found a null effect of the Sydney MSIC on crime in the Kings Cross neighbourhood
- Myer and Belisle found a significant reduction in property and violent crime in the area surrounding Vancouver's Insite immediately after its opening
- Espelt et al. 2017 had conflicting results regarding discarded injecting equipment

These results led to the Rand Corporation review delivering a largely positive report concerning the possibility of implementing SIFs in the United States where no such facilities currently exist. However the Rand review did not scrutinise the 'positive' studies for errors nor for silences on confounders that would destroy the conclusions made by the studies' researchers.

RAND review relied on discredited studies

Two of the studies demonstrating the supposed effectiveness of a Medically Supervised Injecting Centre in reducing overdose mortality (Marshall et al. Lancet 2011) and ambulance overdose callout reductions (Salmon et al. Addiction 2010) *both demonstrate either incompetence on the part of the researchers or possibly fraudulent intent*, and yet likewise form the centre of the other major literature review to that date (see the 2014 review by Potier, C., et al., Supervised injection services: What has been demonstrated? A systematic literature review. Drug Alcohol Depend. (2014), http://dx.doi.org/10.1016/j.drugalcdep.2014.10.012 as displayed below).



C. Potier et al. / Drug and Alcohol Dependence xxx (2014) xxx–xxx 15

et al., 2004; Tyndall et al., cohort studies, 94% ($n = 30$) 1) in Sydney, and 3% ($n = 1$)

nsisted of 7 exhaustive pop- (Fry, 2002; Kimber et al., et al., 2004), 3 descriptive 008b; Salmon et al., 2009a udies (Fairbairn et al., 2008; n, 2013; Kerr et al., 2007b; bin et al., 2009; Small et al., tson et al., 2011), 4 cross- 2008; Navarro and Leonard, al., 2005), 3 surveys (Cruz eek and Gilmour, 2000), 3 05; Kerr et al., 2006a; Wood l studies (Kimber and Dolan,

3.3. The impact of SISs on overdose-induced mortality and morbidity

Seven studies evaluated whether SISs successfully reduced harm among SIS users (Kerr et al., 2006b, 2007b; Marshall et al., 2011; Milloy et al., 2008a, 2008b; Salmon et al., 2010; Van Beek et al., 2004). In the different studies, no death by overdose was ever reported within the SISs in which this parameter was evaluated (Kerr et al., 2006b; Milloy et al., 2008b; Van Beek et al., 2004). In Vancouver, SIS implementation led to a 35% decrease in the number of lethal overdoses in the vicinity of the SIS (Marshall et al., 2011); thus, it was evaluated that between 2 and 12 cases of lethal overdose might have been avoided each year (Milloy et al., 2008b). In Sydney, the number of calls for ambulances related to overdose was 68% lower during the operational hours of the SIS (Salmon et al., 2010; Van Beek et al., 2004).

A third Canadian study used by the RAND review, which tracked changes in property and violent crime, likewise exhibits *either incompetence or fraudulent intent*, seeing as its same authors had previously covered the seismic policing changes in a 2004 CMAJ study https://www.cmaj.ca/content/170/10/1551.full which tracked the actual **displacement** of dealers to other areas within Vancouver precisely due to the tripling of police numbers and the new zero tolerance approach (see the screen shot below of the CMAJ study Abstract and researchers involved). The Police Commander of that time, John McKay, has put in writing that the police particularly targeted property and violent crime.

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Research article

Displacement of Canada's largest public illicit drug market in response to a police crackdown

Evan Wood, Patricia M. Spittal, Will Small, Thomas Kerr, Kathy Li, Robert S. Hogg, Mark W. Tyndall, Julio S.G. Montaner and Martin T. Schechter
CMAJ May 11, 2004 170 (10) 1551-1556; DOI: https://doi.org/10.1503/cmaj.1031928

Article Figures & Tables Metrics Responses PDF

Abstract

Background: Law enforcement is often used in an effort to reduce the social, community and health-related harms of illicit drug use by injection drug users (IDUs). There are, however, few data on the benefits of such enforcement or on the potential harms. A large-scale police "crackdown" to control illicit drug use in Vancouver's Downtown Eastside provided us with an opportunity to evaluate the effect.

Methods: As part of our ongoing prospective cohort study of IDUs in Vancouver, we examined data collected from 244 IDUs in the 3 months before the police crackdown and from 142 IDUs in the 3 months after the start of the crackdown, on Apr. 7, 2003. All study subjects were active drug users. We also examined external data on needle exchanges and syringe disposal.

In This Issue

CMAJ
Vol. 170, Issue 10
11 May 2004
Table of Contents
Index by author

Article Tools

It thereby emerges that the only studies which are quasi-experimental and which display sufficiently designed research are all, each and every one, exhibiting omissions or silences which leave only two conclusions – the researchers were either inept or were fraudulent because all expressly avoided locally-known information that would destroy the findings of their study. This points to a strong possibility of bias controlling the published results.

The table below summarises the issues which remove confidence in the conclusions of each of these studies on injecting room effectiveness.

Studies ostensibly demonstrating effectiveness	**Issues with these studies**
Marshall et al. found a 35% reduction in opiate overdose around Vancouver's Insite	Statistics from the British Columbia Coroner show that deaths increased in Vancouver after Insite opened, as well as in the local DTES area. Any reductions in deaths immediately around Insite were due to the tripling of police numbers driving drug dealers away from the area, as per Drug Free Australia's letter to Lancet which was published in January 2012 by that journal
Salmon et al. 2010 found a greater reduction in ambulance callouts for overdose	Data from this study shows a 31% decrease in ambulance callouts in Kings Cross when compared to the rest of NSW. However, at night when the Centre was closed, reductions were 71% better than the rest of NSW. This points to the success of sniffer dog policing introduced 1 month after the MSIC opened, which was more active at night than be day, and which alone explains any decreases in ambulance callouts

Donnelly and Mahoney found a null effect of the Sydney MSIC on crime in the Kings Cross neighbourhood	These studies failed to mention the introduction of sniffer dog policing 1 month after the MSIC opened. This policing drove drug dealers and buyers and their overdoses (which normally occur within close proximity to where drugs are purchased) into neighbouring Darlinghurst, where overdoses increased at a comparable rate to decreases in Kings Cross
Myer and Belisle found a significant reduction in property and violent crime in the area surrounding Insite immediately after its opening	This study did not test for the effect of a tripling in police numbers and a newly adopted zero tolerance approach at Insite's introduction, which specifically targeted property and violent crime as well as drug dealing

The silences and omissions in the two Vancouver studies

The 2011 Marshall et al. Lancet study so central to these positive reviews spuriously claimed that Insite likely reduced overdoses in Vancouver by 9% despite official BC Coroners' stats showing clear increases in ODs for Vancouver after Insite's 2003 opening as per screenshot of the BC Coroner's document immediately below. Drug Free Australia corrected Lancet on these statistics in a full page letter printed by Lancet in its January 2012 issue (See Appendix A).

BRITISH COLUMBIA

BC Coroners Service
Illicit Drug Deaths 1997 to 2007
Ministry of Public Safety and Solicitor General

Age

	2007	2006	2005	2004	2003	2002	2001	2000	1999	1998	1997
20 and under	5	9	7	8	7	5	7	7	10	12	6
21 - 30	37	47	40	43	34	39	52	38	49	73	61
31 - 40	55	54	64	58	49	56	97	101	111	174	141
41 - 50	66	77	75	57	72	56	65	73	82	121	81
51 - 60	32	38	28	26	22	12	19	28	24	35	17
61 and over	5	3	4	2	5	2	6	1	2	2	4
Total	**200**	**228**	**218**	**194**	**189**	**170**	**246**	**248**	**278**	**417**	**310**

Gender

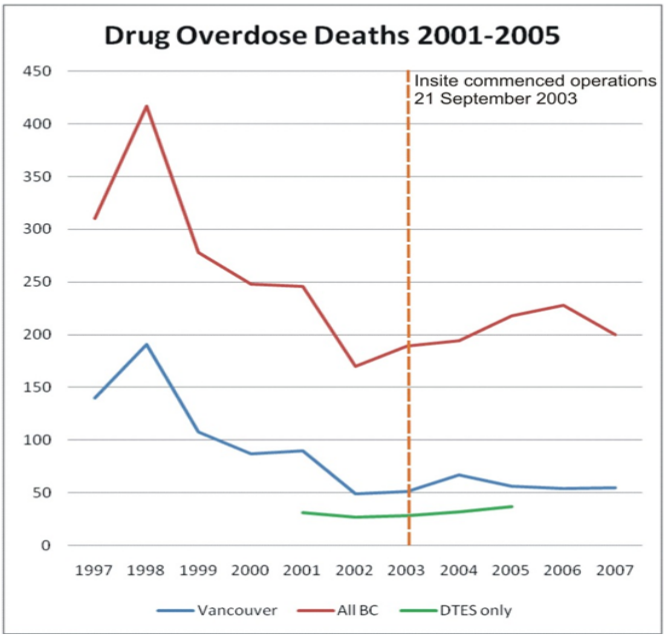
	2007	2006	2005	2004	2003	2002	2001	2000	1999	1998	1997

Town / City

	2007	2006	2005	2004	2003	2002	2001	2000	1999	1998	1997
100 Mile House	0	0	1	0	0	1	1	0	0	0	0
108 Mile Ranch	0	0	0	0	0	0	0	0	0	0	0
Abbotsford	4	8	8	5	5	1	11	14	6	6	8
Agassiz	0	1	2	1	1	1	0	0	0	2	1
Alexis Creek	0	0	0	1	0	0	0	0	0	0	0
Armstrong (BC)	0	0	1	0	0	0	0	0	0	0	0
Black Creek	0	0	0	0	0	0	1	0	0	0	0
Bowser	0	1	0	0	0	0	0	0	0	0	0
Brentwood Bay	0	1	0	0	0	0	0	1	0	1	0
Bridge Lake	0	1	0	0	0	0	0	0	0	0	0
Burnaby	9	6	7	3	8	2	11	6	13	20	13
Campbell River	3	4	5	3	4	1	1	2	2	9	2
Castlegar	[illegible]								0	3	1
Trail	2	1	1	0	0	1	0	0	0	1	0
Ucluelet	0	0	0	0	0	0	0	0	0	0	1
Vancouver	56	54	55	67	51	49	90	87	108	191	140
Vanderhoof	0	0	0	0	1	0	0	0	0	0	0
Vernon	3	4	1	4	7	3	2	4	1	[illegible]	2
Victoria	[illegible]						25	20	31	26	17
Warfield	0	0	0	0	0	0	0	0	0	1	0
West Vancouver	1	0	1	0	0	1	0	1	1	0	0
Westbank	1	1	2	0	0	1	0	0	0	0	0
Whistler	0	0	0	0	0	0	1	0	0	0	0
White Rock	1	2	1	0	1	1	1	0	0	1	0
Williams Lake	3	0	2	0	0	0	1	1	0	1	1
Winfield (BC)	0	1	0	0	1	0	0	0	0	0	0
Wycliffe	0	0	0	0	1	0	0	0	0	0	0
Ymir	0	0	0	0	1	0	0	0	0	0	0
Unknown	1	0	0	0	0	0	0	0	0	0	0
Total	**200**	**228**	**218**	**194**	**189**	**170**	**246**	**248**	**278**	**417**	**310**

The previous screen shot was originally on the BC Coroner's website at: http://www.pssg.gov.bc.ca/coroners/publications/docs/stats-illicitdrugdeaths-1997-2007.pdf
Now found at:
https://web.archive.org/web/20120321162004/http://www.pssg.gov.bc.ca/coroners/publications/docs/stats-illicitdrugdeaths-1997-2007.pdf

	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007
Vancouver	140	191	108	87	90	49	51	67	56	54	55
All BC	310	417	278	248	246	170	189	194	218	228	200
DTES only					31	27	28	32	37		



The same study also claimed overdose reductions by 35% in the area immediately surrounding Vancouver's Insite. Drug Free Australia's Australian/Canadian team of epidemiologists and addiction specialists demonstrated in 2012 that Marshall et al. **had concealed the tripling of police numbers around Insite in 2003**,[1] particularly in light of the Lancet authors falsely counter-claiming (Appendix B) that this was a temporary policing arrangement finishing once Insite opened when in fact it was permanent,[2] as attested by the DTES Area Commander at that time, John McKay (See Appendix C) who in 2011 said that the approach was still in place. It is also attested by the very same document cited by the Lancet researchers in their Lancet correspondence, addressing the Drug Free Australia letter. They claimed that the document 'Confident Policing' had recorded that the policing crackdown commencing 6 months before Insite opened ceased weeks after Insite opened. However 'Confident Policing' clearly agrees with the Police Commander.

[1] https://drugfree.org.au/images/13Books-FP/pdf/Lancet_2011_Insite_Analysis.pdf, https://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736(12)60054-3.pdf?code=lancet-site
[2] https://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736(12)60055-5.pdf

> The initiative was first designed as a three-month project and was later extended for an additional three months after an early internal evaluation of the initiative had been conducted. In fact, as of August 2004, the initiative is still ongoing, albeit in a slightly modified form.
>
> The focus of the CET initiative was clearly the DTES, but it was initially anticipated that the Task Force thus created would be able to redeploy part of its complement to address issues of crime and disorder displacement to other areas of the District and the City. This is why the terms "city-wide" were selected to describe the initiative. However, it soon became clear that the best that the Task Force could do to address displacement issues was to pass on information for action by the Drug Squad and other units.
>
> https://www.vancouveragreement.ca/wp-content/uploads/ConfidentPolicing2004sm.pdf

Such policing served to disperse drug dealers away from the area around Insite, reducing crime and loitering, and of course ODs as users purchased their drugs elsewhere. Policing alone was shown to be demonstrably capable of reducing overdoses around Insite by 35%.[3] **This then additionally collapses the Vancouver study describing reduced crime around Insite, the result of tripled policing which changed from a philosophy of containment to one of zero tolerance 6 months before Insite opened.**

The Vancouver study claiming decreases in crime with the introduction of Insite falls to the same omissions and silences, given that the authors demonstrably knew of the policing changes given their coverage of the displacement effect it created in 2003 https://www.cmaj.ca/content/170/10/1551.full.

The error of the Kings Cross ambulance study

The 2010 Salmon et al. Addiction study, which claimed a **31%** greater reduction in overdose ambulance callouts for Kings Cross (80%) than for the rest of NSW (61%) when Australia's heroin drought ensued, failed to note that there were significantly greater reductions in ambulance callouts during nighttime hours, where Kings Cross, where reductions in Kings Cross (71% at night) was **a full 70% better** than the rest of NSW (42% reduction at night) when the injecting room *was closed*.[4] This can be clearly seen in the ringed cells on the spreadsheet below and the screenshot of the study's actual data beneath it.

[3] https://drugfree.org.au/images/13Books-FP/pdf/Lancet_2011_Insite_Analysis.pdf
[4] https://www.drugfree.org.au/images/13Books-FP/pdf/2017InjectingRoom.pdf

	AMBULANCE CALLOUTS BEFORE MSIC OVER 36 MONTHS					
	During Op hours	Average per month	Outside Op hours	Average per month	Total all hours	Average per month
Postcode 2011 - Kings Cross	626	17.4	922	25.6	1548	43.0
Postcode 2010 - Darlinghurst	338	9.4	311	8.6	649	18.0
Rest of NSW	6779	188.3	2901	80.6	9680	268.9
	AMBULANCE CALLOUTS AFTER MSIC OVER 60 MONTHS					
	During Op hours	Average per month	Outside Op hours	Average per month	Total all hours	Average per month
Postcode 2011 - Kings Cross	210	3.5	440	7.3	650	10.8
Postcode 2010 - Darlinghurst	311	5.2	383	6.4	694	11.6
Rest of NSW	4382	73.0	2806	46.8	7188	119.8
	PERCENTAGE REDUCTION IN AMBULANCE CALLOUTS					
		During Op hours		Outside Op hours		Total all hours
Postcode 2011 - Kings Cross		80%		71%		75%
Postcode 2010 - Darlinghurst		45%		26%		36%
Rest of NSW		61%		42%		55%

680 *Allison M. Salmon* et al.

Table 2 Ambulance attendances at opioid-related overdoses by location and operating hours: May 1998–2006.

Geographic location	*Ambulance attendances Average per month* *Pre-MSIC: May 1998 –April 2001*	*Post MSIC: May 2001– April 2006*	*Ratio of change Post-MSIC/pre-MSIC (95% CI)*	*P-value*	*Interaction—geographic location and period*
Immediate MSIC area (inside o/h[a])	626 (17)	210 (4)	0.20 (0.17–0.24)	<0.001	
Immediate MSIC area (outside o/h)	922 (26)	440 (7)	0.29 (0.26–0.32)	<0.001	$\chi^2_{(1)} = 12.79$; *P*-value < 0.001
Neighbouring MSIC area (inside o/h)	338 (9)	311 (5)	0.55 (0.47–0.64)	<0.001	
Neighbouring MSIC area (outside o/h)	311 (9)	383 (6)	0.74 (0.64–0.86)	<0.001	$\chi^2_{(1)} = 7.08$; *P*-value = 0.001
Rest of NSW (inside o/h)	6779 (188)	4382 (73)	0.39 (0.37–0.40)	<0.001	
Rest of NSW (outside o/h)	2901 (81)	2806 (46)	0.58 (0.55–0.61)	<0.001	$\chi^2_{(1)} = 150.85$; *P*-value < 0.001

This irrefutably indicates reductions were not due to the MSIC, and suggests it was rather due to sniffer dog policing introduced one month after the MSIC opened, where sniffer dogs were used extensively at night https://www.zdnet.com/article/sniffer-dog-avoidance-a-wireless-app-with-bite/ and in the early morning hours when the MSIC was closed https://www.parliament.nsw.gov.au/Hansard/Pages/HansardResult.aspx#/docid/HANSARD-1323879322-25542/link/11.

The Kings Cross crime study omissions

Any null effect of the MSIC on crime in the area, found in three of the RAND review studies they cited, can be slated to changed policing, just as was the case for Vancouver's Insite.

Thus four studies on SIS/SIF/MSIR impacts on crime in their immediate area are voided due to the effect of increased police operations.[5] The

[5] Wood et al. 2004; Fitzgerald et al. 2010; Milloy et al. 2009; Wood et al. 2006[a]; Freeman et al. 2005

upshot is that there is no science which supports injecting room causing decreased crime.

Summary of shortcomings of the quasi-experimental studies

Every one of the studies that purport to show injecting room effectiveness exclude or minimise very significant changes in policing, where all policing sought to remove drug dealers from the immediate area around injecting room sites. Because opiate users tend to overdose in the immediate vicinity of their drug purchase, and most often within a short period of time after the purchase, overdoses and ambulance callouts for overdose are inevitably removed from the injecting room area as a result of heightened policing, which likewise discourages crime in the area.

Latest MSIR review well-illustrates the failure

The recently released review of the North Richmond Medically Supervised Injecting Room (MSIR) evaluated the performance of the facility against its six legislated objectives, with the review's own data and comments demonstrating failure on five of the six objectives, despite rosier media reports indicating otherwise. The facility has also been associated with increases in drug-related crime.

The review records the following regarding its six objectives (please note the verbatim comments by the MSIR reviewers within the quotation marks):

1. **Reduce discarded needles on streets** - "Local people record no difference in seeing discarded injecting equipment" (p 76 of the review)

2. **Improve public amenity** - "significantly fewer residents and business respondents reported feeling safe walking alone during the day and after dark due to concerns about violence and crime . . . " (p 85)

3. **Reduce the spread of blood-borne viruses** - "There is not a significant difference between MSIR service users and other people who inject drugs in reporting that they had injected with someone's used needle/syringe in the previous month." (p 100)

4. **Referrals to treatment and other services** - "in the first year of operation (the MSIR) has not demonstrated higher levels of service take-up for MSIR users as compared with other people who use drugs." (p 48).

5. **Reduce heroin deaths** - Figure 17 on p 45 of the review shows that there were 12 heroin deaths within 1 km of the MSIR the year before it opened, and 13 the year after. Figure 19 on p 47 shows that for the top 5 Local Government Areas for heroin deaths in Melbourne there was a cumulative 65 deaths before the MSIR opened and 67 in its first year.

Clearly there is no observable reduction in heroin deaths in Melbourne or North Richmond in its first year of operation. Furthermore, had the 112,831 heroin injections in the MSIR over 18 months happened on the *streets* of North Richmond, there would, according to Australian statistics, have been only *one* death to be expected, indicating that the MSIR spent $6 million to save only one life, an extremely expensive failure.

6. **Reduce ambulance and hospital attendances** - On the streets of Melbourne, 112,831 opiate injections would have produced 26 overdoses, (25 non-fatal and 1 fatal) according to an important Australian study (see p 59). Of these 19 would likely have been attended by an ambulance. Comparing 18 months before and after, the MSIR would therefore have reduced ambulance callouts by just 5%. Yet the review egregiously claims reductions of 36%, which were clearly due to heightened police operations arresting drug dealers in the vicinity of the MSIR, sending drug dealers elsewhere to ply their trade. Because users most often overdose near where they bought their drugs (p 83), ambulance callouts were clearly the result of policing, which nullifies (see footnote on p 67) the review's spurious claims regarding callouts. Additionally, analysis of heroin OD presentations at nearby St Vincent's Hospital "found that the number of heroin overdose cases did not change significantly after the facility opened." (p 74)

Adding to the failure against objectives listed above, police complained of increasing crime around the MSIR, and residents of a honey-pot effect where drug dealers were drawn to the streets outside the MSIR.

EVIDENCE DEMONSTRATING INEFFECTIVENESS - 2

PILL TESTING

The only studies on ecstasy deaths in Australia indicate that ecstasy itself caused almost every pill death, while pill testing does in fact promote ecstasy use – the very substance causing almost all deaths

Pill testing doesn't address the causes of ecstasy deaths:

1. **It cannot identify individual vulnerabilities to ecstasy that cause deaths**
2. **It doesn't identify other co-used drugs such as alcohol or amphetamines which make ecstasy deadly**
3. **It can't identify which ecstasy user will have an ecstasy-fuelled accident (mostly car accidents)**

Two Australian studies show ecstasy itself causal of most deaths

In January 2020 data on 392 ecstasy-related deaths between July 2000 and November 2018 was published in the International Journal of Drug Policy (see Appendix D). This study extended the data beyond the MDMA-related deaths from July 2000 and December 2005 examined in the only other Australian study https://pubmed.ncbi.nlm.nih.gov/19604654/ of ecstasy deaths.

There were three main causes of deaths. 14% of deaths were caused by ecstasy alone, often due to individual vulnerabilities to the drug. Anna Wood took an ecstasy pill from the same batch as four friends, but only she died, no doubt from an individual vulnerability. It was not an overdose because the science clearly shows that ecstasy overdose is in fact rare. 48% of deaths were from ecstasy being co-consumed with other legal or illegal drugs such as alcohol, amphetamines or cocaine which create deadly synergies. A further 29% were from accidents due to ecstasy/other drug intoxication, mostly car accidents.

Very few deaths from adulterant drugs mixed with ecstasy

No more than 5% of Australian ecstasy-related deaths, according to the above study, were from other exotic drugs mixed into ecstasy pills. Obviously, it is not

clear at autopsy whether these other exotic drugs caused the death, or whether it was the ecstasy in the pill.

Very few deaths from party drugs other than ecstasy

Drug Free Australia has identified a handful of MDMA-related deaths that lie outside of the years 2000 to 2018, with 6 PMA deaths in South Australia in the mid-1990s.

Again there are a handful of deaths from party drugs other than ecstasy, with a number of NBOMe deaths identified by Google search between 2012 and 2016, where evidence indicates the deceased users knew what they were taking. Notably, three Melbourne deaths in January 2017 were caused by pills containing NBOMe and 4-FA but it is questionable whether these drugs would have been delineated by the Bruker Alphas used for the Canberra pill testing trials simply because this mobile equipment often fails in identification where there are multiple drugs in a pill (Written advice from toxicologist Dr Andrew Leibie as contained in DFA document “Why-have-pill-testing-when-most-ecstasy-deaths-are-from-normal-doses-of-MDMA).

Pill testing does not address the real causes of MDMA deaths

With at least 95% of Australian deaths caused or co-caused by ecstasy itself, pill testing fails to address the causes of most MDMA-related deaths.

Causes of MDMA-related deaths	Pill testing applicability
Individual vulnerabilities to MDMA	Pill testing cannot test for individual vulnerabilities
MDMA used with alcohol, cocaine etc	Pill testing tests pills, not user blood samples
Accidents, mostly car accidents	Pill testing will not stop MDMA-related accidents

Pill testing might prevent that 5% of deaths, but very good evidence from the second Canberra pill-testing trial indicates that it would do nothing to stop the other 95% of deaths. Worse, pill testing increases the likelihood that the drug responsible for almost all Australian party pill deaths will be taken by those who have purchased it.

Pill testing can't advise an appropriate dose

Pill Testing Australia is now calling for governments to buy them new equipment that can measure the purity and dose in an MDMA pill, saying they need to advise users on how to more safely moderate their doses.

Given that every person metabolises the MDMA in their ecstasy pill differently there will be blood concentrations which will differ tenfold for roughly the same amount of MDMA taken. The graph below from this South Australian study shows the blood MDMA concentrations for 49 ecstasy users, NONE of which died in the study, against the amount of carefully measured MDMA they ingested.

The light blue shaded area in the graph below shows the blood concentration range for 196 of the 392 MDMA-related Australian deaths (the lower 50%) between 2001 and 2018 (30 - 450 ng/ml – see this and the Roxburgh study previously detailed above for the range). As can be clearly seen, even small doses of MDMA (80-90 mgs) yield blood concentrations well ABOVE the levels which caused 50% of our Australian ecstasy deaths. Notice that ingestion of just 100-115 mg of ecstasy gives blood levels ranging tenfold from 120 – 1040 ng/ml. When it is considered that of 125 – 150 mg of ecstasy can be routinely used for experimental PTSD research with no ethics approval problems, such individual differences against toxic levels makes advice on dose absurd.

Festivals do not need pill testers advising on dose. All that is needed is a large photo of a decedent at each festival captioned – "this ecstasy user died after taking ¼ of a pill". Messages on what to look for when someone is hyperthermic or toxically affected by ecstasy can be delivered via all sorts of social media and screens at festivals. No need for pill testing at all.

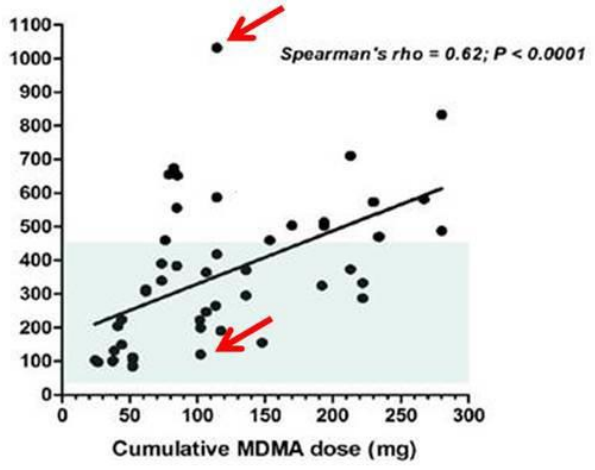


Figure 4 The relationship between maximum plasma 3,4-methylendioxy-methamphetamine (MDMA) concentration (C_{max}) and cumulative MDMA dose consumed by the time of maximum plasma concentration ($n = 49$). Correlation coefficient (Spearman's rho), *P*-value and line of best fit are shown ($n = 49$ participants where MDMA detectable in plasma)

The clincher - users MORE likely to take ecstasy after pill testing

The Australian National University evaluation of the 2019 Canberra pill testing trial confirms that the methods used by Pill Testing Australia to classify substances they identify is actually increasing the likelihood the user will take that substance.

When pill testing identifies a substance to be what the user thought they had purchased, the substance is given an "all-clear" white card which is displayed on a noticeboard in the pill testing tent, declaring it to not contain substances "associated with increased harm / multiple overdoses / death" (see p 11). If a 'dangerous' drug is identified, it is given a red card.

Yet while the evaluation stated that "most of the patrons had a generally accurate perception of the contents" of their pills before testing, it also states that "**those who received a test result confirming the substance to be what they thought it was were likely to take as much or more than originally intended**" and "**concordance between expectation and identification is associated with stable or increased intention to take a substance**."

When it is considered that 90% of the 158 pills presented in the trial contained ecstasy, the drug found in Dr Amanda Roxburgh's study to be responsible for almost all of the 392 MDMA-related deaths in Australia between 2000 and 2018, the symbolics of a white card rather than the red card it deserves makes it clear why a user would be more likely to use it after the pill has been tested.

Pill testing clearly sends all the wrong messages which will only increase party drug deaths in Australia.

Pill testing counselling failed to deter use

The same evaluation as described above also confirms that only seven pills were discarded by users after pills were tested, each containing N-ethylpentylone, which would likely come from a batch or batches of 200 or more pills each somewhere in Canberra or Australia which has caused no hospitalisations or deaths.

Pill Testing Australia claims that they tell users of the dangers of ecstasy but there was no evidence of counsellors dissuading any user from taking their tested pill, with not one ecstasy user recorded discarding their pills, evidencing zero behaviour change.

Drug Free Australia asserts that it is too late to be telling ecstasy users that their substance is dangerous saying the horse has bolted once they have spent $100 purchasing it, and the real need is government-funded social media campaigns telling the truth about ecstasy before they make the cash outlay.

Pill testing a failure in England/Wales

Statistics from England and Wales show that the introduction of pill testing did not produce any reduction in deaths as promised, nor did it appear to change the behaviour of users by getting some to quit using ecstasy, as also forecast by its advocates. While European countries have poor to non-existent statistics on ecstasy deaths, the UK keeps up-to-date figures. Pill testing operated by "the Loop" began in 2013 and by 2016 began expanding into 12 music festivals with government assent. In 2013 ecstasy was used by 1.2% of the population, rising significantly to 1.7% by 2017/18 (see Table 1.02). In 2013 there were 43 ecstasy deaths, more than doubling to 92 deaths in 2018.
Harm Reduction Australia's specious campaign to establish an intervention that provides little to no protective effect for ecstasy users will continue to mislead young Australians, broaden the pool of novice users and lead to more needless deaths.

Drug Free Australia notes that any ACT move towards pill testing at festivals or a permanent site in Canberra will not in any way address the problem of pill tests in the ACT.

Only public education campaigns will alert ecstasy users to the dangers of the substance they are using.

APPENDICES

Appendix A	Letter published in 2012 Lancet disproving the 2011 study
Appendix B	Reply by Lancet authors falsely claiming crackdown ended Also text from the same policing report which shows that they were untruthful in their claim that police crackdowns ceased as of September 2003
Appendix C	Letter to Lancet by Police Commander John McKay
Appendix D	Roxburgh study on 392 ecstasy-related deaths in Australia

APPENDIX A

Overdose deaths and Vancouver's supervised injection facility

The report by Brandon Marshall and colleagues (April 23, p 1429),[1] in which it is claimed that the opening of a supervised injection facility on Sept 21, 2003, in Vancouver, BC, Canada, was associated with a 35% decrease in overdose deaths in its immediate surrounding, contains serious errors.

The claim that all overdose deaths in Vancouver declined between 2001 and 2005 is strongly affected by the highly questionable inclusion of the year 2001—a year of much higher heroin availability and overdose fatalities than all subsequent years. A study period starting from 2002 in fact shows an increasing trend of overdose deaths both for Vancouver and for the Downtown Eastside area in which the facility, Insite, is situated (figure),[2] the control areas compared in Marshall and colleagues' study.

Curiously, the higher availability of heroin up until 2001, which declined by 2002 and which has remained low since that year, was specifically tracked in two previous articles[3,4] by three of the current paper's researchers and therein treated as extraordinary. In their latter 2007 study,[4] the aforesaid three researchers noted that, in a large cohort of Vancouver drug users, 21% had reported non-fatal overdoses in the previous 12 months in 1997, dropping to 12% at the beginning of 2001 and to 5% by the end of 2001, rising to 6% in 2004. They clearly point to reduced heroin supply as the reason, and yet in the *Lancet* paper specifically state that "we have no evidence that significant changes in drug supply or purity occurred during the study period", which of course was 2001 to 2005.

Of even greater concern is the statement in the *Lancet* paper that "we know of no changes in policing policy that could have confounded our results". Again, three of the researchers were so well appraised of major policing changes in the area immediately around Insite during 2003, the same year it opened, that they wrote a 2004 article tracking the "displacement" of drug users out of the policed area around Insite and into other areas of Vancouver.[5] In that article they record counts of discarded needles reducing by 46% in the policed areas whereas needle counts in other areas of Vancouver increased by similar proportions. Most of the overdoses that were the subject of the questionable 35% reduction immediately around Insite lay specifically in the 12 city blocks patrolled by 48–66 police added in 2003 and operative to this day (personal communication). This major change in policing around Insite is clearly the most likely cause of any real reductions in overdoses that might be found in the immediate vicinity of the injection facility.

Finally, Marshall and colleagues do not declare that 41% of British Columbia's overdose mortality is non-injection-related.[6] This being the case, the researchers had the obligation of declaring the specific proportion of deaths that were non-injection-related in the vicinity of Insite, compared with the rest of Vancouver.

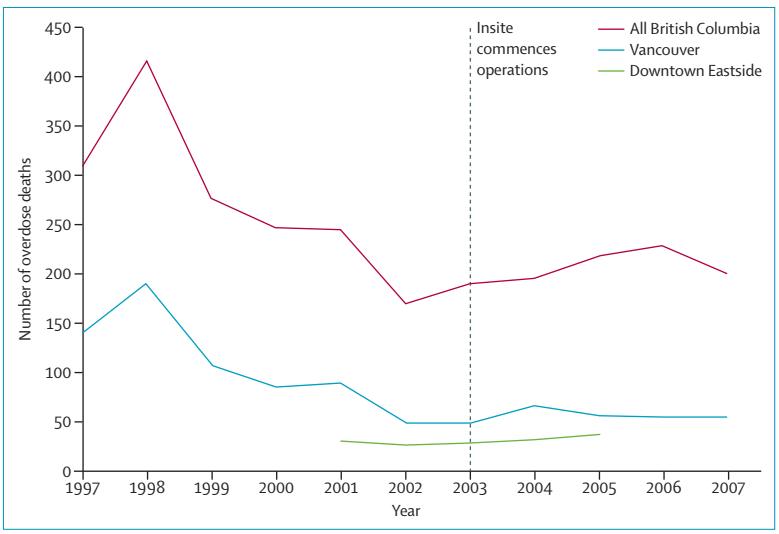


Figure: **Drug overdose deaths 2001–05**

An extended analysis is available online. We declare that we have no conflicts of interest.

For the **extended analysis** see http://www.drugfree.org.au/fileadmin/Media/Global/Lancet_2011_Insite_Analysis.pdf

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1 Marshall BDL, Milloy M-J, Wood E, Montaner JSG, Kerr T. Reduction in overdose mortality after the opening of North America's first medically supervised safer injecting facility: a retrospective population-based study. *Lancet* 2011; **377:** 1429–37.
2 Ministry of Public Safety and Solicitor General. Illicit drug deaths 1997 to 2007. http://www.pssg.gov.bc.ca/coroners/publications/docs/stats-illicitdrugdeaths-1997-2007.pdf (accessed Dec 7, 2011).
3 Wood E, Stoltz JA, Li K, Montaner JS, Kerr T. Changes in Canadian heroin supply coinciding with the Australian heroin shortage. *Addiction* 2004; **101:** 689–95.
4 Kerr T, Fairbairn N, Tyndall M, et al. Predictors of non-fatal overdose among a cohort of polysubstance-using injection drug users. *Drug Alcohol Depend* 2007; **87:** 39–45.
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6 Milloy MJ, Wood E, Reading C, Kane D, Montaner J, Kerr T. Elevated overdose mortality rates among First Nations individuals in a Canadian setting: a population-based analysis. *Addiction* 2010; **105:** 1962–70.

APPENDIX B

Authors' reply

Gary Christian and colleagues raise various concerns in reference to our paper that showed a 35% reduction in overdose mortality within the vicinity of Vancouver's supervised injecting facility. They refer to publicly available data from the British Columbia Vital Statistics Agency to argue that overdose deaths increased rather than decreased in the geographic area of interest between 2001 and 2005. This apparent discrepancy can be explained by several problematic assumptions that underlie Christian and colleagues' critique.

First, our study focused on an a-priori-defined area in close proximity to the supervised injecting facility that included 41 city blocks, the centroid of each being within 500 m of the facility. The data considered by Christian and colleagues refer to a much larger region (ie, the entire local health area) that includes about 400 city blocks (figure). As shown clearly in figure 3 of our paper,[1] the reduction in overdose mortality was only noted in close proximity to the supervised injecting facility, with the effect diminishing strikingly beyond this area.

Second, although we restricted our analysis to deaths deemed by the coroner to be caused by an accidental illicit drug overdose, the data referred to by Christian and colleagues include all drug-induced deaths (eg, suicides and adverse effects of drugs in therapeutic use).[2] Finally, we examined mortality rates as opposed to absolute death counts to account for changes in the population at risk.

Christian and colleagues further claim that the noted reduction in overdose mortality was due to increased heroin availability in 2001; however, we have previously published data to show that daily heroin use remained stable between 2001 and 2005.[3,4] These data were referenced in our original report. Additionally, publicly available assessments of the police crackdown to which Christian and colleagues refer show that this operation ended within weeks of the opening of the supervised injecting facility and was not ongoing as they claim;[5] therefore, any brief displacement of drug users would have probably resulted in a conservative bias by differentially reducing overdose mortality in the area of interest before the facility's opening.

Finally, regarding mode of drug use, we note that coroners' records do not indicate whether deaths were injection-related or not. However, if we restrict our analysis to records in which injection drug use was indirectly suggested, including for example discarded injection paraphernalia surrounding the decedent (ie, 85% of the original 89 deaths occurring within 500 m of the supervised injecting facility), our estimate for the reduction in overdose mortality is slightly greater at 36%.

The results of our study show that Vancouver's supervised injecting facility had a localised yet significant effect on overdose mortality. These facilities can and should be a central component of evidence-based responses to reducing drug-related harms in communities with a high burden of overdose related to injection drug use.

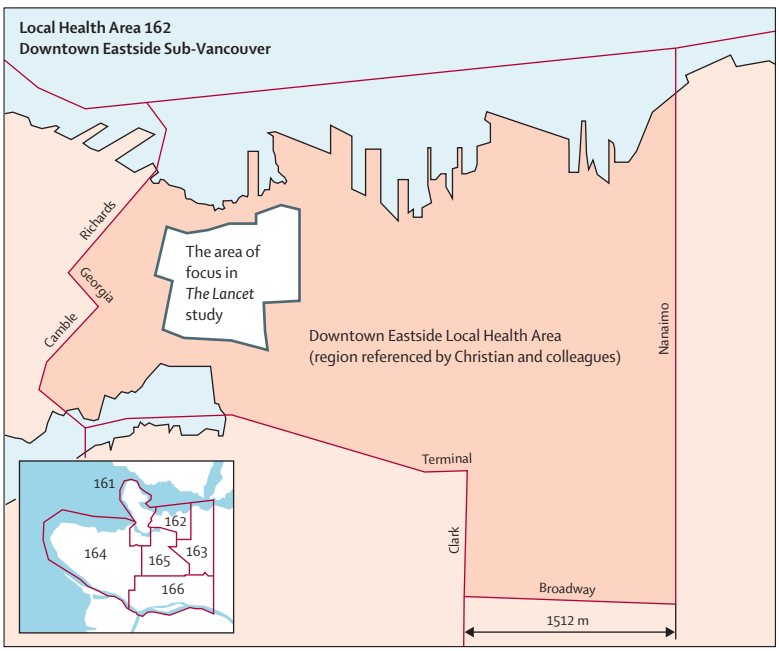

Figure: **Comparison of geographic regions defined as the area of interest in our paper versus that referred to by Christian and colleagues**
Figure modified and reproduced from publicly available documentation maintained by BC Stats. For this documentation see http://www.bcstats.gov.bc.ca/data/pop/maps/LHApdf/hamap162.pdf.

JSGM as received educational grants from and served as an ad-hoc adviser to or speaker at various events sponsored by Abbott Laboratories, Agouron Pharmaceuticals, Boehringer Ingelheim, Borean Pharma, Bristol-Myers Squibb, DuPont Pharma, Gilead Sciences, GlaxoSmithKline, Hoffmann-La Roche, Immune Response Corporation, Incyte, Janssen-Ortho, Kucera, Merck Frosst Laboratories, Pfizer Canada, Sanofi Pasteur, Shire Biochem, Tibotec, and Trimeris. All other authors declare that they have no conflicts of interest.

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1 Marshall BDL, Milloy M-J, Wood E, Montaner JSG, Kerr T. Reduction in overdose mortality after the opening of North America's first medically supervised safer injecting facility: a retrospective population-based study. *Lancet* 2011; **377:** 1429–37.
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Pelvic floor muscle training after prostate surgery

The conclusion of the scientifically robust study by Cathryn Glazener and colleagues (July 23, p 328),[1] that pelvic-floor muscle exercise taught by a continence health professional after prostate surgery is unlikely to be effective or cost effective, is misleading and possibly erroneous.

Glazener and colleagues should have considered that their *intervention* was not effective and provided a more critical appraisal of its failure. The intervention was weak on several counts but particularly lacked a plausible biological rationale, since it did not address the importance of control of the urethral sphincter, which did not rate a mention anywhere. Men were only—and repeatedly—instructed to "contract the pelvic floor as if holding on to wind" and assessed at each of four visits per anum. This not only provided inappropriate sensory feedback but also taught and reinforced inappropriate motor control.

Lacking was the action of flow-stopping, which produces an antero-cranial movement of the urethra at the bladder base, owing to activation of the puboperineales, and activation of the external *urethral* sphincter.[2] The ability to lift the urethra more than 2 mm with this action has been correlated with early recovery of urinary control.[2] Examination per anum might be a reproducible test of pelvic floor muscle function in men,[3] but that does not make it a valid test of urethral sphincter function. Information from the internet[4] might have reinforced such erroneous concepts for the 50% of men in the control group doing exercises, contributing to the lack of between-group difference and poor results overall.

We declare that we have no conflicts of interest.

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1 Glazener C, Boachie C, Buckley B, et al. Urinary incontinence in men after formal one-to-one pelvic-floor muscle training following radical prostatectomy or transurethral resection of the prostate (MAPS): two parallel randomised controlled trials. *Lancet* 2011; **378:** 328–37.
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In the MAPS trial, Cathryn Glazener and colleagues[1] noted similar high rates of incontinence at 12 months after radical prostatectomy or trans-urethral resection of the prostate in patients randomised to therapist-guided pelvic-floor muscle training or to standard care. We did a similar trial after radical prostatectomy,[2] which Glazener and colleagues state had unexplained differential dropout from the control group. We wish to add some comments on this matter as well as on other aspects of Glazener and colleagues' Article.

First, the relatively high dropout rate (13 of 53) in the control group of our trial[2] did not jeopardise the randomisation efficacy of the two groups. Additionally, the heterogeneity of a Cochrane meta-analysis, to which Glazener and colleagues suggest that our trial added, is due to variability in several features, such as patient selection, surgeon technique and volume, definition of urinary incontinence, duration and frequency of training, and choice of control.

Second, in our trial, long-term physician-guided pelvic-floor muscle training until urinary continence was achieved or for up to 12 months proved to be more effective than no training. This effect is supported by the results of a randomised trial by Overgård and colleagues,[3] which showed that patients who received long-term physiotherapist-guided pelvic-floor muscle training compared with those training on their own had a significantly lower incontinence rate at 12 months (3 of 36 *vs* 11 of 39), despite a similar continence rate at 3 months.

Third, although in the MAPS trial[1] a pad test was not used because of practical difficulties and the apparently more important role of subjective incontinence measures, we consider it important to discriminate the degree of incontinence, since in our[2] and others'[4] experience pelvic-floor muscle training seems to be more effective for mild and moderate incontinence.

We declare that we have no conflicts of interest.

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1 Glazener C, Boachie C, Buckley B, et al. Urinary incontinence in men after formal one-to-one pelvic-floor muscle training following radical prostatectomy or transurethral resection of the prostate (MAPS): two parallel randomised controlled trials. *Lancet* 2011; **378:** 328–37.
2 Manassero F, Traversi C, Ales V, et al. Contribution of early intensive prolonged pelvic floor exercises on urinary continence recovery after bladder neck-sparing radical prostatectomy: results of a prospective controlled randomized trial. *Neurourol Urodyn* 2007; **26:** 985–89.

The VPD also brought to the attention of crown counsel that the police often deal with individuals in one jurisdiction who are already under charge in another. Furthermore, many of those who are under charge and who have an area restriction in one jurisdiction openly admit to moving to another jurisdiction in order to continue participate in the drug trade. To combat this, the police requested that the Crown ask for "joint area restrictions" for persons charge with trafficking and posse for the purposes of trafficking. This included the provision for residents of Burnaby that the y "not to attend within "3 blocks of any Sky Train station in Burnaby" and for non-residents not to "attend with the entire city of Burnaby". Similar area restriction orders were being requested for offenders within the City of New Westminster.

Finally, the initiative was supported by an intelligence analysis capacity whose role was to:

1. monitor changes in patterns of criminal activity and recommend strategic changes;
2. monitor potential crime displacement; and,
3. identify core groups of criminals (high volume offenders) and issue bulletins identifying the criminals – including a regularly up-dated list of the top ten offenders active in the DTES.

The initiative was first designed as a three-month project and was later extended for an additional three months after an early internal evaluation of the initiative had been conducted. In fact, as of August 2004, the initiative is still ongoing, albeit in a slightly modified form.

The focus of the CET initiative was clearly the DTES, but it was initially anticipated that the Task Force thus created would be able to redeploy part of its complement to address issues of crime and disorder displacement to other areas of the District and the City. This is why the terms "city-wide" were selected to describe the initiative. However, it soon became clear that the best that the Task Force could do to address displacement issues was to pass on information for action by the Drug Squad and other units.

Staffing the CET

To assemble the sixty police positions required, 20 were assigned from District Two (the "Core" officers) in which the DTES is situated and a total of forty additional positions were taken, through secondment,

APPENDIX C

A second letter was sent to Lancet on 6 April 2012, a letter which Lancet chose not to publish. We note that the Chief Editor of Lancet is a co-Board member of a drug law reform organisation of which two of the authors of the erroneous Lancet study which we have here addressed are also members as per http://www.icsdp.org/network/scientific_board.aspx.

Gary Christian
DFA Research Coordinator

The Lancet Editor

We have read the authors' response and respectfully repeat our request for retraction of the study on the grounds that the authors' conclusions are based on demonstrable fallacies.

The central fallacy which invalidates the study is the claim that the authors knew of no changes in policing that could otherwise explain their findings. We have previously demonstrated that there was a police crackdown commencing at the mid-point of the study period so effective that drug use indicators were reduced by 46%. This occurred precisely in the Vancouver city blocks where the highest concentrations of overdose mortality studied by the authors had previously occurred. These policing changes readily explain the 35% decrease in overdose mortality around Insite claimed by the authors.

The authors' response also incorrectly claims that the April 2003 crackdown ceased after 6 months, when Insite opened in September 2003. To support that claim the authors cite a City of Vancouver evaluation of the crackdown. However, if read in its entirety, this document clearly states, "as of August 2004, the initiative is still ongoing, albeit in a slightly modified form."[i][i] At best, the authors' response lacks the appropriate rigour.

Furthermore, we have forwarded a written statement by the Vancouver Police commander directing the ongoing crackdown throughout the second half of the Lancet article's study period ending 2005. This statement unambiguously contradicts the authors' response that the crackdown ceased in September 2003. There was, in fact, only a change of operational name for the policing crackdown (CET became BET) with no significant change in operational approach, personnel or strategy. The continuation of the crackdown to this day is beyond conjecture. On these grounds alone, the authors' central claim about the impact of Insite is rendered invalid. There are, however, other substantive errors in the authors' response.

Plummeting heroin use between 1998 and 2002, which the authors continue to deny in their response, is verified in another study of Vancouver's VIDUS cohort by the same authors. It states, "As indicated in Fig. 1, the proportion of participants reporting a non-fatal overdose has declined steadily since enrolment, with 21% of individuals reporting a non-fatal overdose in 1997 compared with just 6% in 2004. The most substantial decline occurred during 2001, with the proportion of participants reporting a non-fatal overdose declining from 12% to 5% during this year."[ii][ii]

Consistent with this, Vancouver experienced a 74% decrease in heroin mortality between 1998 and 2002, with non-fatal overdoses decreasing in the VIDUS cohort between 1997 and 2001 (as would be expected) by 76%, as per quote above. Yet the authors' response cites largely irrelevant VIDUS cohort *daily heroin use* figures rather than overdose percentages, in a study focusing on overdose mortality. Where Canadian heroin users were estimated to inject on average four times daily, daily use figures will remain relatively unchanged even

though the average number of daily injections declines along with a 75% reduction in heroin supply and a 75% reduction in overdoses.[iii][iii] Tracking non-fatal overdoses and overdose mortality is a more accurate measure of fluctuations in supply, as is done by these same researchers in two previously studies quoted in our analysis, and by Australian researchers correlating overdose mortality with a heroin drought.[iv][iv] Elevated heroin supply and elevated overdoses ended with 2001, making that year invalid for inclusion in the study period. Its inclusion creates the illusion of a subsequent decline in overdose mortality. In fact there is a trend towards an increase in overdose mortality from 2002 onwards, starting the year before Insite opened.

We also note that the authors' response claims there are flaws in our analysis. We refute these as follows.

1. Contrary to the authors' assertion, Vital Statistics coroner's data are never used in our analysis to infer any increases in overdose deaths in the 41 block area where the claimed 35% decline occurred. Rather, BC Coroner's data is used to show that there was an increasing trend in overdose deaths for the CONTROL AREA of the City of Vancouver, and the Vital Statistics coroner's dataset was used to show that the same increasing trend was true for the 400+ block area around Insite from 2002-2005.
2. Contrary to the authors' assertion, we did exclude the 5 of 155 Vital Statistics deaths, leaving the same 150 DTES non-intentional overdoses on which the authors deliberated. We thereby demonstrated increases in DTES area deaths for 400+ city blocks from 2002 to 2005 even after these 5 intentional/other deaths were excluded.
3. The authors are also incorrect in their statement that we failed to do an in-depth analysis of the 41 block area where the 35% decrease was alleged to have occurred. Rather, our analysis contains a map with the exact location of all 89 deaths within the 41 block area. We further demonstrated that two-thirds of these deaths fall within the 12 block area patrolled by the 48-66 extra police deployed since April 2003. This suggests that the majority of these deaths likely happened in the pre-Insite comparison period when these blocks were an 'open drug scene'.
4. We have noted elsewhere that, "When . . . increases in overdose deaths are compared against population growth in both Vancouver and the DTES the increases in deaths well overwhelm any changes in population. The Lancet study, at Table 2, calculates a 3% change in Vancouver's population between 2001 and 2005, yet drug deaths increased by a much greater 14% from 2002. The Lancet study calculated an 8% increase in population for the DTES, yet drug deaths increased by 37% from 2002. In the scenario where all 5 intentional/other deaths, as discussed previously, occurred in the DTES in 2005 alone, the increase in drug deaths would still be 18%, well beyond the 8% population increase for that area of Vancouver."[v][v]

In summary, in their response to our analysis, the authors have failed to satisfactorily address any of our criticisms. The Lancet Insite article therefore remains seriously flawed on multiple grounds. It should be retracted.

[i][i] Dandurand Y et al., Confident Policing in an Troubled Community – Evaluation of the Vancouver Police Department's City-wide Enforcement Team Initiative p 49 http://www.vancouveragreement.ca/wp-content/uploads/ConfidentPolicing2004sm.pdf
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From: John McKay [mailto:john_mckay@shaw.ca]
Sent: Friday, 23 March 2012 8:28 AM
To: 'Gary Christian'
Subject: Fw: Statement to Lancet

STATEMENT TO LANCET

Beat Enforcement Team (BET) - Vancouver Police Department 2003 - 2006

John Mc-Kay - then Officer in Charge (BET)

Downtown East Side Vancouver - Policing Rationale

The inception of what eventually became known as the Beat Enforcement Team (BET) occurred in early 2003. At that time the Vancouver Police Department recognized that the Vancouver Agreement between 3 levels of government with the so called " 4 Pillars approach" was going to have a major effect on the VPD's ability to successfully police the Down Town East Side (DTES) of Vancouver. This was largely due to the harm reduction pillar which emphasized the value of the Supervised Injection Site which was going to be located in the heart of the DTES in the 100 block of East Hastings.

While the VPD could not at the time argue against the 4 Pillars approach – harm reductionists using statistics and opinion on European Model success – they believed that there had to be some control over the situation in the DTES because of the impact on the community once the dealers figured out that their clients were not being charged and indeed allowed to be in possession of the drugs. VPD feared that there would be a free for all and open warfare between dealers who wanted a greater share of the clientele. As well, the harm reduction philosophy might bring "drug tourists "into the area which would add to the policing problem.

Closely associated to the drug use in the DTES was the movement of stolen property into the local pawnshops of which there were 49 in the immediate area. Selling stolen property was a method of obtaining hard cash for the purpose of buying drugs.

In order to maintain some control over the potential outcomes of the new harm reduction philosophy the VPD began what was known at the Beat Enforcement Team. This unit was made up of 4 squads of police, administration staff, and a police Inspector totaling 65 personnel.

The unit consisting of 65 officers was originally named CET for Citywide Enforcement Team. The name was used because other parts of the city also wanted more beat cops so the effort in the DTES was disguised as a unit that could go anywhere to patrol, hence the name "Citywide Enforcement Team." The original concept under Inspector Doug Lepard, the OIC CET, and DCC, Bob Rich, was to have members stand on the corner and intercept drugs and stolen property. They had a high profile and there was some success with the mandate which was to disrupt the flow of stolen property etc.

The mission of BET was to interrupt the flow of stolen property and disrupt the trafficking of drugs in the area. As the officer in charge of the unit from September 2003 – September 2006 it was my role to achieve these goals.

In order to achieve these goals I spent as much time on the street as possible learning and from several good civilian contacts who had been working in the area for years I was able to glean a lot of background knowledge about the people and the issues around addiction. I implemented a combination of surveillance, undercover work, high presence uniform police and intelligence driven tactics. In a nutshell we shut down all but 7 pawnshops for failure to comply with the law on property and due to specifically targeted undercover operations we gained a lot of success in getting rid of the dealers. Many of these operations such as Operation Lucille, New Boy, became high profile media covered events.

It is my understanding that the effect of 65 police officers in the DTES is negated in the Lancet analysis produced by the harm reduction proponents. That attitude is much too convenient for them because the truth of the matter is that the police were integral to the lowered death rates by being on the street and in and out of the various Single Residence Occupancy hotels in which the addicts reside. The projects and contacts that police made in SROS and on the street with the mentally ill also helped to lower death rates because of the positive nature for the most part of the officers assigned to that beat.

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APPENDIX D

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Research Paper

MDMA-related deaths in Australia 2000 to 2018

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ABSTRACT

Background: MDMA markets have undergone substantial changes internationally, with increasing manufacture of high purity MDMA recorded. This study examined trends in MDMA-related deaths in Australia, investigating characteristics, circumstances and toxicology of these deaths.

Methods: Analysis of MDMA-related deaths in Australia between 2001 and 2018, extracted from the National Coronial Information System (NCIS). Deaths were categorized into (1) drug toxicity deaths, where MDMA (with and without other drug) toxicity was considered by the coroner to be the underlying cause of death; and (2) other cause deaths, with MDMA (with and without other drug) intoxication/toxicity considered contributory to death.

Results: 392 deaths were identified, with a median age of 26 years. 81% were male. Females were significantly younger than males (24 vs. 27 years). Two-thirds (62%) of deaths were attributed to drug toxicity (48% multiple drug toxicity and 14% MDMA toxicity alone), and one third (38%) to other causes (predominantly motor vehicle accidents) with MDMA recorded as a contributory factor. Death rates increased significantly between 2001 and 2007, declined between 2008 and 2010, and increased again between 2011 and 2016. Median MDMA concentration was 0.45 mg/L, and was significantly higher amongst females than males (0.70 vs. 0.42 mg/L). Deaths attributable to MDMA toxicity alone had a significantly higher blood MDMA concentration than multiple drug toxicity deaths (1.20 vs. 0.43 mg/L).

Conclusions: Deaths occurred predominantly among males in their mid-twenties, with females likely to be significantly younger. Three marked periods of trends in death rates (increases and declines) were observed, consistent with international supply trends. While most deaths were due to multiple drug toxicity, a notable proportion were attributed solely to MDMA toxicity.

Introduction

There are an estimated 22 million users of 3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy'), with an estimated 0.4% of the global population reporting recent use (United Nations Office on Drugs & Crime, 2018). Over the past decades MDMA markets across Europe, where the majority of MDMA is manufactured (European Monitoring Centre for Drugs & Drug Addiction, 2016), have undergone substantial changes with implications for use and harm. An international shortage in 2008 of the precursor safrole, used to manufacture MDMA, impacted on the availability and use of MDMA globally up until 2010 (European Monitoring Centre for Drugs & Drug Addiction, 2016; Mounteney et al., 2018). Since then, however, MDMA manufacture, use, seizures and purity have been increasing in Europe, the United States, the United Kingdom, central America and Australia (European Monitoring Centre for Drugs & Drug Addiction, 2018; Home Office Statistics, 2018; Mounteney et al., 2016; 2015; Substance Abuse & Mental Health Services Administration, 2015,2018; United Nations Office on Drugs & Crime, 2018). Moreover, during this period the purity of MDMA appears to have increased, with the use of high purity crystalline forms becoming more prevalent (Mounteney et al., 2016).

Increased manufacture, purity and prevalence of MDMA use is of concern as the drug is associated with a range of harms. MDMA is a psychostimulant and shares effects with methamphetamine and amphetamine such as increased arousal and alertness. This can result in adverse effects such as muscle tension, jaw clenching and tooth grinding (Kalant, 2001). Elevated heart rate and blood pressure are also common and can fluctuate for days after MDMA consumption (Kalant, 2001). As a result of the pharmacokinetic actions related to MDMA use, the more serious adverse effects include hypertension, hyperthermia, serotonin syndrome, seizures, stroke, hyponatremia and

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cardiac arrest, as well as an elevated risk for traumatic injury and suicide (Darke, Duflou, Kaye, Farrell & Lappin, 2019; Darke, Lappin & Farrell, 2019; Elliott, 2005; Kaye, Darke & Duflou, 2009; Schifano, 2004). The mechanisms of MDMA-related death are complex and some, including hyperthermia, involve multiple factors including not only the pharmacologic action of MDMA (which impedes the temperature-regulating center in the brain) (Green, O'Shea & Colado, 2004) but also other factors such as environment (dancing in high temperatures in crowded spaces) (Darke, Lappin et al., 2019) . Hyponatremia, (low sodium concentration in the blood) may lead to seizures and coma, and has been documented in the context of over-hydrating in a hot environment following MDMA consumption (Darke, Duflou et al., 2019). Serotonin syndrome, characterised by marked increases of the neurotransmitter serotonin being released following consumption (resulting in seizures and coma), cardiac arrest, and intracranial hemorrhage have also been documented in MDMA-related deaths (Schifano, 2004).

The relationship between MDMA consumption and the experience of adverse effects is also complex, with some research suggesting that the probability of these experiences increases rapidly with MDMA doses exceeding 120 mg (Brunt, Koeter, Niesink & van den Brink, 2012), while other research suggests that adverse effects may in part be driven by individual differences in the metabolic processing of MDMA (Kalant, 2001).

Previous work has examined the number of MDMA-related deaths in Australia between 2000 and 2005 (Kaye, Darke & Duflou, 2009), however, given the substantial changes over the past decade in MDMA markets internationally, more contemporary investigation of these deaths is crucial. This study extends previous work, reporting median concentrations of MDMA reported in postmortem toxicology. Analysis of coronial data provides a unique opportunity to differentiate MDMA-related deaths from amphetamine-related deaths (using objective toxicology data and coroner attributed medical cause of death fields), which is not possible using deaths data that are coded under the current ICD-10 coding system (World Health Organization, 2010).

Aims

1 Describe trends in MDMA-related death rates in Australia 2001 to 2016;
2 Describe the characteristics and circumstances of MDMA-related death; and
3 Describe the toxicology of MDMA-related deaths.

Methods

National coronial information system (NCIS)

The NCIS is an online database containing information relating to all deaths that are reportable to the coroner. Cause of death is ascertained by a forensic pathologist and documented on the autopsy and coroner's report. The forensic pathologist may report on: i. the direct cause of death, ii. the antecedent cause, and iii. other significant conditions associated with the death. Although it varies from one jurisdiction to another in Australia, a death is generally reportable to a coroner where: the person died unexpectedly and the cause of death is unknown; the person died in a violent and unnatural manner; the person died during or as a result of anaesthesia and/or various medical and surgical procedures; the person was 'held in care' or in custody immediately before they died; a medical practitioner has been unable to issue a death certificate stating the cause of death; or the identity of the decedent is unknown.

Categorization of deaths

Only deaths where MDMA was considered by the coroner to be the underlying cause of death (with or without other drug toxicity) and deaths where MDMA toxicity or intoxication were considered contributory to the death were included. Deaths were selected if the medical cause of death (underlying and contributory) was noted as MDMA toxicity. In the case of multiple drug toxicity deaths, if MDMA was noted by the coroner as one of the drugs, these deaths were included. Deaths where MDMA was detected in toxicology but the coroner attributed the death to causes unrelated to MDMA and where MDMA toxicity was not noted as the medical cause of death, were excluded. MDMA related deaths were identified from the NCIS for the period July 2000 to November 2018. Trends over time are only shown for deaths occurring between 2001 and 2016, given that data for 2000 only represent a 6-month period, and that 2017 and 2018 data are likely to be incomplete.

Circumstances of death

Investigative reports, including police narratives, autopsy and coroners' findings, and toxicology reports are attached to most death cases in the NCIS. These reports were searched for the location of where the death occurred to determine the proportion of deaths that occurred in public locations such as music festivals or events, and in private locations. Coroners findings and toxicology reports were used to assess the presence and contribution of other drugs to MDMA-related deaths.

Toxicology

Toxicological data were reported for MDMA, other psychostimulants, hypnosedatives, alcohol, opioids, cannabis (Δ-9-THC), GHB, ketamine, antidepressants and antipsychotics. In cases of hospitalization prior to death, antemortem blood samples taken on or near admission to hospital were reported, and drugs administered by hospital and medical staff excluded.

Statistical analyses

MDMA death rates per 100,000 population aged 15–64 were calculated using estimates of the resident population of Australia at June of each year (Australian Bureau of Statistics, 2016). Rates were modelled using negative binomial regression where there was over-dispersion, and Poisson regression where there was not (Coxe, West & Aiken, 2009). Trends were analysed within three distinct time periods: Time period 1 (2001–2007) prior to the global shortage of the precursor safrole, Time period 2 (2008–2010) during which the shortage occurred and reduced MDMA use and availability was recorded, and Time period 3 (2011–2016), during which an increase in international MDMA availability and use was recorded, as well as increases in drug purity. For dichotomous categorical variables, odds ratios (OR) and 95% confidence intervals (95% CI) were reported. Where distributions were highly skewed, medians were reported, and differences assessed using the Mann–Whitney *U* test. Analyses for trends in rates of deaths were made using SAS 9.4 (Inc., 2013). All other analyses were conducted in SPSS 23.0 (IBM inc., 2016).

Ethics approval

Ethics approval to access the NCIS was granted by the Victorian Department of Justice and Community Safety and the University of New South Wales Human Research Ethics Committee.

Results

392 MDMA-related deaths were identified during the period 2000–2018, 62% of which were due to drug toxicity. MDMA-related death rates fluctuated between 2001 and 2016, with three distinct trends apparent (Fig. 1). MDMA-related death rates increased between

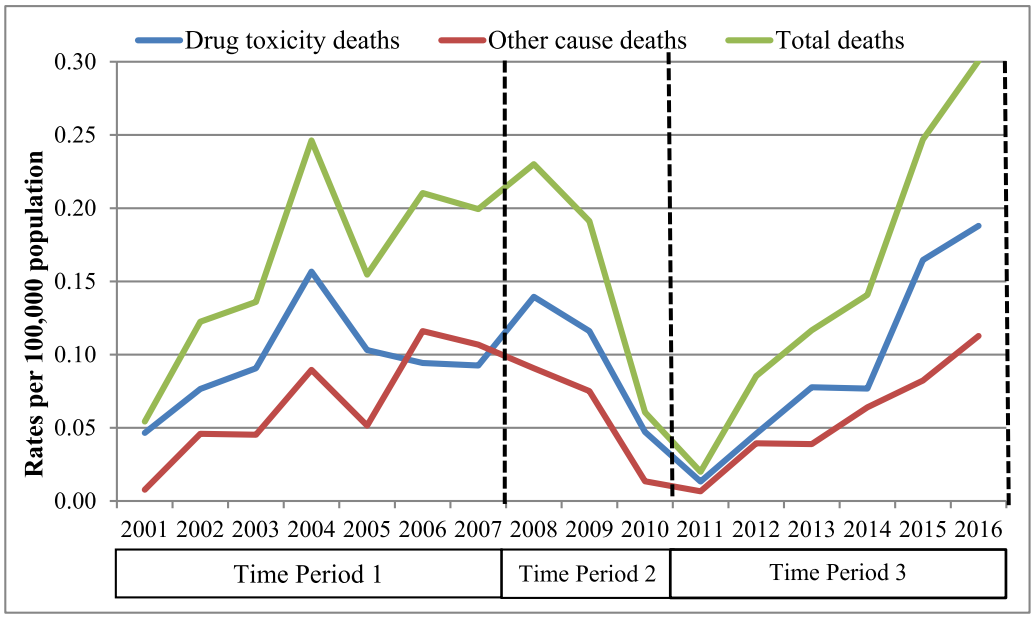

Fig. 1. Rates of MDMA-related deaths per 100,000 population, Australia 2001 to 2016
Time Period 1: 2001–2007, prior to the global shortage of the precursor safrole (used to manufacture MDMA)
Time Period 2: 2008–2010, during the precursor shortage and a period of reduced MDMA use and availability
Time Period 3: 2011–2016, after the precursor shortage, and a period of increased MDMA use and availability.

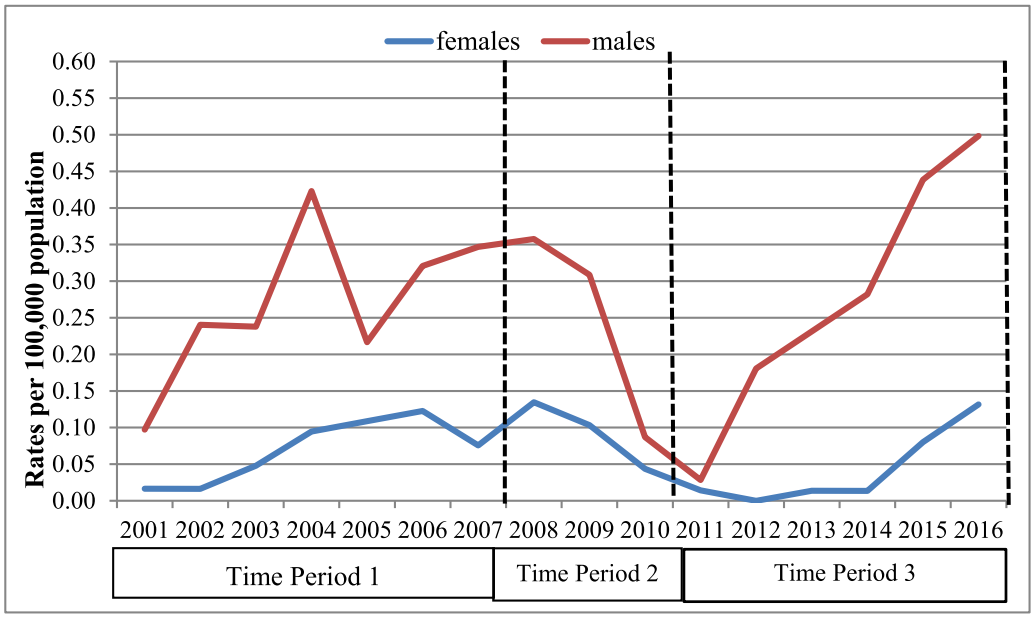

Fig. 2. Rates of MDMA-related deaths per 100,000 population by gender, Australia 2001 to 2016
Time Period 1: 2001–2007, prior to the global shortage of the precursor safrole (used to manufacture MDMA)
Time Period 2: 2008–2010, during the precursor shortage and a period of reduced MDMA use and availability
Time Period 3: 2011–2016, after the precursor shortage, and a period of increased MDMA use and availability.

2001 and 2007 ($p<0.01$), declined significantly between 2008 and 2010 ($p<0.0001$), and increased significantly from 2011 to 2016 ($p<0.0001$). Increases during this period were driven in part by multiple drug toxicity deaths ($p<0.0001$). The number of deaths increased from 7 in 2001 to 33 in 2008, declined to 9 in 2010, and increased to 48 in 2016. These trends were evident for both drug toxicity and other cause deaths (Fig. 1). Rates of MDMA-related deaths were higher among males than females, and increases in these deaths were largely driven by males (Fig. 2).

Characteristics

Deaths occurred predominantly among males (81%), with a median age of 26 (range 15–58) (Table 1). Females were significantly younger than males (24 v 27 years, $U = 9036.5$, $p<0.01$). Most decedents (62%) were employed, and a fifth were married or in a defacto relationship. More than half (56%) of all incidents occurred in a private location, as did three-quarters of the toxicity incidents. Significantly higher proportions of drug toxicity incidents occurred in private locations (73%) compared to other cause incidents (29%) (OR 2.6, 95% CI 2.3–4.4, $p<0.0001$) (Table 1).The most common public location was streets/roadways, followed by outdoor areas (parks, beaches and

Table 1
Characteristics of MDMA-related deaths by cause of death, Australia.

	Total deaths n = 392 % (n)	Drug toxicity n = 244 % (n)	Other cause n = 148 % (n)
Demographics			
Median age (range)	26 (15–58)	26 (15–56)	25 (16–58)
Male	81 (318)	77 (188)	88 (130)
Employed	62 (242)	59 (145)	66 (97)
Married/Defacto	20 (77)	20 (48)	20 (29)
Location			
Private location	56 (220)	73 (177)	29 (43)
Public location	44 (172)	27 (67)	71 (105)

Table 2
Cause and intent of MDMA-related deaths, Australia.

All deaths	Total *n* = 392 % (*n*)	Female *n* = 74 % (*n*)	Male *n* = 318 % (*n*)
Drug toxicity	62 (244)	76 (56)	59 (188)
Multiple drug toxicity	48 (189)	43 (32)	49 (157)
MDMA toxicity only	14 (55)	33 (24)	10 (31)
Other cause	38 (148)	24 (18)	41 (130)
Traumatic accident	29 (115)	19 (14)	32 (101)
Violent suicide	6 (23)	3 (<5)	7 (21)
Disease	3 (10)	3 (<5)	3 (8)

countryside). Only 7% ($n = 17$) of drug toxicity incidents occurred at music festivals or dance parties.

Cause and intent of death

Two thirds (62%) of deaths were attributed to drug toxicity (Table 2). Approximately half of all deaths were attributed to multiple drug toxicity, and one seventh to MDMA toxicity alone. The remaining deaths (38%) were due to other causes, with MDMA considered as contributing to death (Table 2).

The overwhelming majority (84%, $n = 206$) of drug toxicity deaths were accidental, with 13 (5%) attributed to deliberate self-poisoning (i.e. suicide), and intent undetermined among the remaining 25 (10%) drug toxicity deaths.

One third (29%) of other cause deaths were due to traumatic accidents (predominantly motor vehicle accidents), with MDMA considered as contributing to the death. Small proportions of other cause deaths were due to violent suicide (6%), and disease (3%).

Gender differences were apparent in relation to the cause of death, with deaths among females significantly more likely to be attributed to drug toxicity than among males (OR 1.3, 95% CI 1.1–1.5). Specifically, women were significantly more likely to die as a result of MDMA toxicity alone than men (OR 3.3, 95% CI 2.0–5.3). Investigating other causes of death, women were significantly less likely to die as a result of a traumatic accident than men (OR 0.5, 95% CI 0.2–0.9).

Among females, where death was attributed to drug toxicity, the median age was 5.5 years younger than among males (22 vs. 27.5 years, $U = 3496.5$, $p < 0.001$). There was no difference in the age of females and males where death was attributed to other causes (25.5 vs. 25 years) (Table 2).

Toxicology

Toxicology data were available in 342 of the deaths (87%) (50 cases did not have data available), with MDMA detected in all deaths. The median blood MDMA concentration was significantly higher amongst females than males among all deaths ($U = 7081.5$, $p < 0.05$) and drug toxicity deaths ($U = 3236.0$, $p < 0.05$) (Table 3). Deaths attributable to MDMA toxicity alone had a significantly higher blood MDMA concentration than multiple drug toxicity deaths ($U = 2189$, $p < 0.001$). There were no differences in median blood concentrations between intentional drug toxicity and accidental drug toxicity deaths. Overall, drug toxicity deaths had significantly higher blood MDMA concentrations than other cause deaths ($U = 10{,}611.5$, $p < 0.01$). Median MDMA concentrations over time varied widely and small numbers prevent confident interpretation of these trends (data not shown).

Table 4
Presence of other drugs in toxicology by cause of death, Australia.

Drug	Total *n* = 342* % (*n*)	Drug toxicity *n* = 215 % (*n*)	Other cause *n* = 127 % (*n*)
No other drug detected	15 (51)	21 (45)	5 (6)
Psychostimulants	54 (183)	55 (119)	50 (64)
Methamphetamine	44 (150)	44 (94)	44 (56)
Cocaine	15 (50)	19 (41)	7 (9)
PMA#	3 (9)	4 (9)	0 (0)
Other^	3 (9)	8 (18)	7 (9)
Alcohol	43 (148)	29 (62)	68 (86)
Opioids	30 (104)	46 (98)	5 (6)
Cannabis	25 (87)	18(39)	38 (48)
Benzodiazepines	23 (80)	33 (70)	8 (10)
Antidepressants	11 (39)	15 (32)	6 (7)
Ketamine	4 (13)	5 (10)	<5
GHB	3 (11)	5 (11)	0 (0)
Antipsychotics	3 (9)	4 (8)	<5

Paramethoxyamphetamine.
^ including methcathinone, 3,4-Methylenedioxy-N-ethylamphetamine - MDEA, methylenedioxypyrovalerone – MDPV, and 3′,4′-Methylenedioxy-α-pyrrolidinobutiophenone – MDPBP.
* 50 deaths did not have toxicology results available.

Other substances were commonly detected in addition to MDMA in post-mortem toxicology (Table 4). Psychostimulants (54%) (predominantly methamphetamine: 82% of psychostimulants; 44% of all toxicology cases) were the most common drug, followed by alcohol (43%), opioids (30%), cannabis (25%), benzodiazepines (23%) and antidepressants (11%). Other drugs such as ketamine, GHB, PMA, methcathinone, MDPV and MDEA were each detected in less than 10% of deaths.

Discussion

The current study provides novel data on long-term trends in MDMA deaths, their toxicology and the circumstances in which they occurred. Decedents were aged, on average, in their mid-twenties, were predominately male and likely to be employed. Females were on average three years younger than their male counterparts.

There appeared to be three distinct periods of MDMA deaths across

Table 3
Median blood concentrations of MDMA by cause of death and gender, Australia.

	All deaths *n* = 342* mg/L median (range)	Males *n* = 279 mg/L median (range)	Females *n* = 63 mg/L median (range)
Drug toxicity	0.59 (0.01–64.00)	0.50 (0.01–64.00)	0.85 (0.02–22.00)
Multiple drug toxicity	0.43 (0.01–22.00)	0.42 (0.01–9.40)	0.70 (0.02–22.00)
MDMA toxicity	1.20 (0.04–64.00)	1.30 (0.07–64.00)	1.10 (0.04–11.50)
Intentional toxicity	1.40 (0.01–64.00)	1.40 (0.01–64.00)	22 (22.00–22.00)
Accidental toxicity	0.60 (0.01–17.00)	0.50 (0.01–17.00)	0.90 (0.04–11.50)
Other cause	0.30 (0.01–8.40)	0.30 (0.01–8.40)	0.36 (0.17–5.10)
All Deaths	0.45 (0.01–64.00)	0.42 (0.01–64.00)	0.70 (0.02–22.00)

* 50 cases did not have toxicology results available.

the study: a marked increase between 2001 and 2007, a sharp decline between 2008 and 2010, and another marked increase between 2011 and 2016. During the latter period rates rose to exceed the peak of the first period. Notably, these patterns were observed for both toxicity and other cause deaths. These trends are consistent with changes in international MDMA market indicators, with the global shortage of the precursor safrole in 2008 having a major impact on MDMA manufacture and use globally, and the global increases in MDMA manufacture and use observed from 2011 onwards (European Monitoring Centre for Drugs & Drug Addiction, 2018; Mounteney et al., 2018; United Nations Office on Drugs & Crime, 2018). Global MDMA markets in the most recent period have also been characterised increasingly by higher purity MDMA being manufactured (Mounteney et al., 2018).

Despite much of the media attention on MDMA-related deaths focusing on deaths that occur at public events, more than half of all deaths (and three-quarters of drug toxicity deaths) in this study occurred in private locations, largely at home. Only 7% of drug toxicity deaths (and 4% of all deaths) occurred at music festivals or dance parties.

There were notable findings concerning MDMA blood concentrations. MDMA -related deaths attributed to drug toxicity had MDMA concentrations approximately twice that of MDMA-related deaths due to other causes. Moreover, deaths attributed solely to MDMA toxicity had concentrations three times that of those attributed to multiple drug toxicity. The higher concentration seen amongst female drug toxicity deaths (although not for other cause deaths) was marked, and puzzling, perhaps reflecting behavioural or, as has been suggested, physiological differences (Allott & Redman, 2007).

Concomitant drug use was prevalent among these deaths, with other psychostimulants most commonly detected in addition to MDMA. All psychostimulants place stress upon the cardiovascular system, and toxicity deaths are primarily due to these cardiovascular effects (Darke Lappin et al., 2019; Karch, 2015). The use of multiple psychostimulants is likely to increase the probability of psychostimulant toxicity. The high levels of alcohol detected among these MDMA-related deaths are also relevant. This finding is consistent with research among MDMA consumers showing that one-third of this group engages in high-risk drinking patterns in combination with their MDMA use (Kinner, George, Johnston, Dunn & Degenhardt, 2012). The use of alcohol with methamphetamine increases heart rate and blood pressure beyond that seen for methamphetamine use alone (Kirkpatrick, Gunderson, Levin, Foltin & Hart, 2012). The use of both these drugs in conjunction with MDMA is likely to increase the risk of a toxic reaction. Finally, there was frequent concomitant use of pharmaceutical drugs particularly benzodiazepines and antidepressants. Previous reports have noted the potentially fatal drug interactions between MDMA and a range of antidepressant drugs which may increase risk for a number of harms including serotonin toxicity (Pilgrim, Gerostamoulos & Drummer, 2011). Less is known about the interaction between MDMA and the use of central nervous system depressants such as benzodiazepines and opioids (Schifano, 2004). Of note, there were less than 10 deaths where known contaminants (e.g. paramethoxyamphetamine – PMA and dextromethorphan) were detected, and less than 10 deaths where other synthetic analogues (e.g. methylenedioxypyrovalerone – MDPV, methcathinone) were detected.

The findings of this study have clinical and public health implications. Importantly, the findings clearly show that fatal MDMA toxicity may occur in the absence of another substance. Harm reduction messages tend to focus on the risks associated with contaminants contained in MDMA (Kinner et al., 2012), however consumers need to be made aware of the dangers associated with MDMA toxicity alone. Given the age of decedents reported in this study, engagement of young consumers in the delivery of these messages is crucial. The preponderance of incidents occurring in private locations also suggests that dissemination of messages across a range of settings (e.g. secondary and tertiary education institutions, nightclubs and music festivals) is warranted. Research has shown that peer led interventions, delivering harm reduction messages about the risks of MDMA are effective in engaging young consumers attending nightclubs and festivals (Bleeker et al., 2009), and increased funding for peer education was amongst the key recommendations made by a recent government report into MDMA related harms (New South Wales Government, 2018). The role of other substances in increasing the likelihood of a toxic reaction also needs to be emphasised. Consistent with other research (Darke, Degenhardt & Mattick, 2007; Roxburgh et al., 2017), the majority of drug toxicity deaths in this study were due to MDMA in combination with other drug toxicity. This highlights the need for messages specifically targeting the dangers associated with concomitant MDMA and other substance use, and further research on these issues is required. Investigating the mechanism of death involved in MDMA fatalities is also an important focus for future research. Onsite medical support as well as spaces to rest have been successful harm reduction strategies employed at large scale events, and continued support for these services is important. Messages around the potential increased neurotoxicity of combining MDMA with other substances including alcohol, the potential for toxic reactions of overheating and overhydrating, and seeking medical assistance early, are critical to reduce MDMA-related harms in these environments. Finally, drug checking (both fixed-site and field-based) services have been in operation in Europe since the 1990s (Barratt, Kowalski, Maier & Ritter, 2018; Brunt, 2017), and more recently in the United Kingdom (UK), North America and Australasia (Barratt et al., 2018; Measham, 2019). Drug-checking services provide an opportunity to engage with young MDMA consumers, who may not be in contact with other services (Degenhardt et al., 2009; Measham, 2019), about the risks associated with MDMA use.

Strengths and limitations

A major strength of this study was the ability to differentiate deaths due to MDMA from amphetamine-related deaths, which is not possible using deaths data that are coded under the current ICD-10 coding system (World Health Organization, 2010). Another major strength was the use of investigative reports to report objective toxicology data on median MDMA blood concentrations and other drugs involved in these deaths. Finally, the availability of historical data from the NCIS over a period of 15 years provides a valuable opportunity to analyze longer-term trends in MDMA-related deaths at a national level.

As with all studies, however, limitations need to be considered. Firstly, the study may not have captured all the deaths that occurred within the study period, as not all deaths may have been reported to a coroner. Due to the complexities of coronial investigations, with some likely to be ongoing, some deaths may not yet be captured in the NCIS. Secondly, the study will not have captured deaths attributed to other drugs that were sold as MDMA to a decedent. Thirdly, it was not possible to account for the amount of MDMA consumed prior to death as in many cases this data was not reported. Finally, care must be taken in extrapolating these findings to other populations of MDMA users. The characteristics of these deaths were, however, comparable to those reported elsewhere.

Conclusions

MDMA-related deaths predominantly occurred among males aged in their mid-twenties, with females significantly younger than their male counterparts. Most incidents occurred in private locations. Three marked periods of increases and declines in death rates were observed, consistent with international MDMA supply trends. While most deaths were due to multiple drug toxicity, a notable proportion were attributed solely to MDMA toxicity. Also of note, almost one-third of deaths were due to accidents (predominantly motor vehicle accidents) that occurred during MDMA intoxication.

Engagement with young consumers about the risks associated with MDMA use alone, and in combination with other drugs, is critical, particularly in the context of rapidly changing drug markets globally, and the increasing purity of MDMA being manufactured (Mounteney et al., 2018). Implementation of harm reduction strategies across multiple settings is crucial.

Conflict of Interest Statement

Neither of the authors have any conflicts to declare.

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